

Hfpef

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THE STORY IN ONE SENTENCE

The heart squeezes fine but cannot relax and fill with blood the way it should.

“Heart failure with preserved ejection fraction (HFpEF) means the heart pumps normally but its walls are stiff, so it cannot relax and fill between beats. SGLT2 inhibitors (like empagliflozin or dapagliflozin) and mineralocorticoid receptor antagonists (MRAs like spironolactone) help; other heart failure medicines are less proven here.”

Normal anatomy — the body's job

The left ventricle (the heart's main pumping chamber) squeezes to push blood out, then relaxes completely so it can fill back up with blood from the lungs. The walls are strong but flexible, like a healthy balloon. The arteries (the tubes carrying blood away from the heart) stay stretchy and match the ventricle's rhythm. This partnership—called arterial-ventricular coupling—keeps blood flowing smoothly at rest and when you move.

BODY'S JOB

The left ventricle's job is to squeeze and send oxygen-rich blood to the whole body, then relax and refill so it is ready for the next beat. Relaxing (called diastole) is just as important as squeezing (called systole). If the ventricle cannot relax, it cannot fill, and that starves the body of oxygen during activity.

1. Blood returns from the lungs, full of fresh oxygen, into the left atrium (the top left chamber of the heart).
2. The left atrium gently pushes that blood down through the mitral valve (a one-way door) into the left ventricle.
3. The left ventricle relaxes fully—its walls are soft and stretchy—so it can hold all the blood it needs without backup pressure.
4. Then the ventricle squeezes hard, sending blood out through the aortic valve into the aorta (the body's main artery) and onward to every organ and muscle.

5. The arteries stay flexible and expand slightly with each squeeze, then spring back, which keeps the flow steady and the ventricle's workload light.
6. This squeeze-relax-fill cycle repeats about 60 to 100 times a minute at rest, and speeds up smoothly when you exercise, because the heart can both speed up and fill faster.

What breaks

The left ventricle's walls become thick and stiff, so they cannot relax and fill with blood the way they should. At the same time, the arteries stiffen, and the heart loses its ability to speed up properly during exercise.

CLINICAL TERM

Left ventricular diastolic dysfunction (the ventricle cannot relax) due to concentric remodeling (the walls get thicker and stiffer) from chronic high blood pressure, type 2 diabetes, obesity, aging, and inflammation. Arterial stiffening (loss of vascular compliance) makes the ventricle work harder. Chronotropic incompetence (the heart cannot increase its rate enough with exercise) limits oxygen delivery.

How the body compensates

The body tries to fill the stiff ventricle by holding onto salt and water, and it revs up the nervous system to squeeze the heart harder and faster. These fixes keep blood pressure and oxygen delivery steady for a while, but they also raise filling pressures in the heart and lungs, which eventually causes symptoms and makes the stiffness worse.

1. {'what': 'The kidneys hold onto sodium and water (via the renin-angiotensin-aldosterone system, or RAAS) to increase blood volume and push more blood into the stiff ventricle.', 'why': 'More volume means higher filling pressure, which can force the stiff ventricle to fill—but it also backs fluid into the lungs and body tissues, causing shortness of breath and swelling.'}
2. {'what': 'The sympathetic nervous system (the body's fight-or-flight system) releases norepinephrine (a stress hormone) to make the heart beat faster and squeeze harder.', 'why': 'This boosts oxygen delivery temporarily, but chronic stress on the heart muscle worsens stiffness and can trigger irregular rhythms like atrial fibrillation (an irregular, quivering heartbeat).'}- 3. {'what': 'The left atrium (the chamber above the ventricle) squeezes harder to push blood into the stiff ventricle, a process called atrial kick.', 'why': 'This extra push helps fill the ventricle, but over time the atrium stretches and dilates from the high pressure, which sets the stage for atrial fibrillation (an irregular, quivering heartbeat).'}-

4. {'what': 'At rest, blood pressure and oxygen levels may look normal because these compensations are working.', 'why': 'This is the dangerous middle zone—the patient feels okay sitting still, so the team may miss how close the body is to tipping into failure during activity or stress.'}
5. {'what': 'The crash point to watch for is acute decompensation: sudden shortness of breath, fluid overload, atrial fibrillation (an irregular, quivering heartbeat) with rapid heart rate, or stroke from a clot forming in the stretched atrium.', 'why': 'Once the compensations max out, even a small trigger (a salty meal, a respiratory infection, skipping medicines) can flood the lungs and drop oxygen levels fast.'}
6. {'what': 'Trends matter more than snapshots. A patient who looks stable today but is gaining weight, needing more diuretics, or getting short of breath sooner each week is on a downhill slope.', 'why': 'Catching worsening trends early lets you adjust medicines and prevent a hospital admission; waiting for a crash closes the window to reverse course.'}

Domino effect

1. The left ventricle becomes thick and stiff from years of high blood pressure, diabetes, obesity, and aging. — High pressure forces the muscle to work harder and grow thicker, but instead of staying flexible, the walls stiffen and lose their ability to relax between beats.
2. The stiff ventricle cannot relax and fill with blood normally, so pressure builds up behind it in the left atrium and pulmonary veins (the blood vessels bringing blood from the lungs). — When the ventricle resists filling, blood has nowhere to go but backward, so it pools in the lungs and raises the pressure there.
3. Fluid leaks from the lung blood vessels into the air sacs (alveoli), causing shortness of breath, especially with exercise. — The backup pressure pushes fluid across the thin walls of the capillaries (tiny blood vessels in the lungs) into the spaces where oxygen exchange happens, so less oxygen reaches the blood and the person feels breathless.
4. The body holds onto salt and water to try to fill the stiff ventricle harder, but this extra fluid spills into the legs, ankles, and belly, causing swelling (edema). — The kidneys sense lower oxygen delivery and activate the renin-angiotensin-aldosterone system, which tells the body to keep sodium and water—but because the heart cannot pump it all forward, the fluid backs up into the tissues.
5. The left atrium stretches from chronic high pressure and begins to beat irregularly, leading to atrial fibrillation (an irregular, quivering heartbeat). — Stretched atrial muscle develops abnormal electrical circuits that fire chaotically, overriding the normal pacemaker and causing an irregular, often rapid heartbeat.

6. Atrial fibrillation (an irregular, quivering heartbeat) worsens symptoms because the ventricle loses the atrial kick (the extra push from the atrium's squeeze), so it fills even less and cardiac output drops. — In HFpEF, the stiff ventricle depends heavily on that final atrial squeeze to fill; without it, filling pressure must climb even higher or the person feels more tired and short of breath.
7. Blood pooling in the fibrillating atrium can form clots, which may break off and travel to the brain, causing a stroke. — When the atrium quivers instead of squeezing, blood swirls and stagnates, especially in a pouch called the left atrial appendage, and clots form in that slow-moving blood.
8. Every symptom—shortness of breath, fatigue, swelling, irregular heartbeat, risk of stroke—traces back to one upstream break: a stiff left ventricle that cannot relax and fill. — Fixing the stiffness (or at least slowing its progression) is the target of SGLT2 inhibitors, MRAs, blood pressure control, weight loss, and exercise—because all the dominoes fall from that single broken piece.

Symptoms · what the patient feels, and why

- Shortness of breath during activity or when lying flat: The left ventricle (the heart's main pumping chamber) becomes stiff and doesn't relax well between beats, so it can't fill up with enough blood. This causes blood to back up into the lungs (like water pooling behind a dam), making the lungs heavy and hard to expand.
- Swelling in the ankles, legs, or belly: Because the stiff heart can't handle all the blood coming back from the body, pressure builds in the veins. Fluid gets pushed out of the blood vessels and into the surrounding tissues, causing visible puffiness (edema).
- Feeling unusually tired or weak, especially with effort: The stiff heart can't pump enough oxygen-rich blood forward to the muscles and organs when you move or work. Your body feels like it's running on a low battery because the cells aren't getting the fuel they need.
- Fast or irregular heartbeat (palpitations): The heart tries to beat faster to make up for the fact that each beat doesn't move as much blood as it should. Over time, the strain on the atria (the heart's upper chambers) can also trigger atrial fibrillation (an irregular, quivering heartbeat) (a chaotic, quivering rhythm).
- Needing to urinate more often at night: During the day, fluid pools in your legs because of gravity. When you lie down at night, that fluid flows back into the bloodstream and the kidneys filter it out as urine. This is the body's way of unloading the extra fluid the heart can't handle.
- Sudden, severe shortness of breath with pink, frothy spit: This is a sign of acute pulmonary edema (fluid flooding the lungs)—fluid has suddenly flooded the air sacs in the lungs. The stiff heart can't keep up with incoming blood, pressure spikes in the lung vessels, and fluid leaks into the spaces where oxygen is supposed to pass into the blood.

- Chest pain or pressure, especially during exertion: The stiff, thickened heart muscle needs more oxygen than normal. If the coronary arteries (the vessels feeding the heart) can't deliver enough—often because of age, high blood pressure, or blockages—the heart complains with pain (angina).
- Confusion, trouble focusing, or feeling dizzy: When the heart can't pump enough blood forward, the brain gets less oxygen and nutrients. This can make thinking slow or fuzzy, especially in older adults, and can cause lightheadedness or fainting if blood pressure drops.

Focused exam

- Elevated jugular venous pressure (JVP) — the neck veins bulge when you sit the patient up 45 degrees: The stiff left ventricle cannot relax to accept blood, so pressure backs up through the left atrium, into the lungs, and all the way to the right heart. The neck veins swell because blood is stuck waiting to get into the heart. High JVP means the backup is severe.
- S4 gallop — a low, extra thump right before the main heartbeat, heard best at the apex with the bell of the stethoscope: The left atrium has to squeeze extra hard to shove blood into the stiff, thick left ventricle. That forceful squeeze makes the S4 sound. This is HFpEF's signature: the pump squeezes fine, but the muscle is too tight to relax and fill.
- Bilateral pitting swelling (edema) in the ankles and lower legs: High pressure in the veins makes fluid leak out of the blood vessels and into the tissue. Gravity pulls it down into the legs. The kidneys also hold onto salt and water to try to keep blood pressure up, which makes the swelling worse.
- Fine crackles at the lung bases, especially after lying flat or walking: The stiff ventricle creates high pressure that backs up into the pulmonary veins and lungs. Fluid seeps into the tiny air sacs (alveoli). You hear crackles low and in the back — that is where gravity and pressure push fluid first.
- Shortness of breath with mild exertion that seems worse than expected for the effort: When you exercise, your body asks the heart to pump more. In HFpEF, the stiff ventricle cannot fill faster or accept more blood, so pressure shoots up and backs into the lungs. The heart rate climbs, but the pump does not deliver. Shortness of breath comes fast.
- Atrial fibrillation (an irregular, quivering heartbeat) on EKG — irregularly irregular rhythm with no clear P waves: Years of high pressure stretch and tire the left atrium. It starts firing signals chaotically. When AF starts, you lose the atrial kick (that final squeeze before each beat), and HFpEF patients depend on that squeeze to fill the stiff ventricle. Blood also pools in the atrium, raising stroke risk.

Diagnostics · every test answers a question

- Transthoracic echocardiogram with Doppler → Is the left ventricle ejection fraction preserved ($\geq 50\%$), and is the ventricle stiff — does it fill poorly during diastole?
- B-type natriuretic peptide (BNP) or NT-proBNP blood test → Is the heart under stress and stretching from high pressure?
- Electrocardiogram (EKG) → Is the left ventricle thick (from years of high pressure), and is the heart rhythm regular or chaotic?
- Basic metabolic panel (BMP) — sodium, potassium, creatinine, blood urea nitrogen → Are the kidneys holding up, and is it safe to give diuretics or medicines that raise potassium?
- Chest X-ray → Is fluid backing up into the lungs, and are there other causes of shortness of breath like pneumonia or a big heart?
- H₂FPEF score or HFA-PEFF algorithm → Does this patient's clinical picture and echo findings fit HFpEF, and how certain are we?

Treatment logic

- Loop diuretic — furosemide (Lasix) or torsemide — to remove extra fluid → Pulls salt and water out of the blood through the kidneys, shrinking the fluid load the stiff heart has to deal with. Pressure drops in the veins and lungs, so shortness of breath and swelling improve within hours to days.
- SGLT2 inhibitor — empagliflozin (Jardiance) or dapagliflozin (Farxiga) → Blocks sugar reabsorption in the kidneys, so you pee out sugar, salt, and water. Lowers blood sugar, shrinks fluid load, helps you lose weight, and directly improves how the stiff left ventricle relaxes and fills. Large trials (EMPEROR-Preserved, DELIVER) show fewer hospitalizations and better survival in HFpEF.
- ACE inhibitor (lisinopril, enalapril) or ARB (losartan, valsartan) to control blood pressure → Blocks angiotensin II (a hormone that squeezes blood vessels and makes the heart work harder). Blood vessels relax, so the heart pumps against less resistance. Over months to years, this slows the buildup of stiffness and fibrosis in the ventricle wall.
- Mineralocorticoid receptor antagonist (MRA) — spironolactone or eplerenone — if potassium and kidney function are safe → Blocks aldosterone (a hormone that makes blood vessels and heart muscle stiff and tells the kidneys to hold onto salt and water). Reduces fibrosis in the heart and blood vessels, lowers fluid overload, and may improve long-term outcomes (TOPCAT trial showed benefit in some HFpEF patients).

- Beta-blocker (metoprolol, carvedilol) or non-dihydropyridine calcium channel blocker (diltiazem, verapamil) for rate control if atrial fibrillation (an irregular, quivering heartbeat) is present → Slows the heart rate so the stiff left ventricle has more time to fill between beats. In HFpEF with AFib, filling time is critical — if the heart races, the ventricle never fills and pressure backs up instantly. Target resting heart rate 60–80 bpm.
- Weight loss (target 5–10% body weight), aerobic exercise (30 minutes most days), and treatment of obstructive sleep pauses in breathing (apnea) with CPAP if present → Obesity, inactivity, and untreated sleep pauses in breathing (apnea) all drive high blood pressure, insulin resistance, inflammation, and oxidative stress — the upstream forces that make the ventricle stiff. Losing weight shrinks the heart's workload, improves filling, and reduces symptoms. Exercise training improves how the body uses oxygen and helps the heart fill better (SECRET-HF, REHAB-HF trials).
- Strict blood pressure control — target systolic < 130 mmHg per ACC/AHA guidelines — and diabetes control (HbA1c < 7%) → High blood pressure makes the left ventricle pump against resistance every beat, thickening and stiffening the muscle over years. Controlling BP stops the injury. High blood sugar stiffens the heart muscle directly (glycation of proteins, inflammation). Lowering HbA1c slows this process.

Medications

Furosemide (Lasix) or bumetanide (Bumex) · Loop diuretic

Tells the kidneys to dump extra salt and water into the urine, which shrinks the fluid load in the blood vessels and lungs. The heart doesn't have to push against as much fluid, so congestion (the soggy, heavy feeling in the lungs and legs) gets better.

1. The drug blocks a pump (the sodium-potassium-chloride cotransporter) on the wall of the loop of Henle.
2. Without that pump working, salt stays in the urine instead of going back into the blood.
3. Water follows salt, so the kidneys make a lot more urine.
4. The blood volume shrinks, so the pressure inside the blood vessels falls.
5. Less fluid leaks out of the lung capillaries and into the air sacs, which makes breathing easier.
6. Swelling in the legs and belly goes down because there is less fluid trying to seep out of the veins.

SAFETY WATCH

Can make potassium drop too low (hypokalemia), which can cause muscle cramps, weakness, or dangerous heart rhythms. · Can make you dizzy or lightheaded if it pulls off too much fluid and your blood pressure falls. · Can strain the kidneys if you get dehydrated; check kidney numbers (creatinine, BUN) regularly. · Can make gout flare up by raising uric acid. · Older adults may need a lower dose because their kidneys clear the drug more slowly.

Dapagliflozin (Farxiga) or empagliflozin (Jardiance) · SGLT2 inhibitor (sodium-glucose cotransporter-2 inhibitor)

Blocks a sugar pump in the kidney so that glucose spills into the urine and gets flushed out. That gentle fluid loss takes pressure off the heart, and the drug also seems to calm inflammation and protect the heart muscle in ways scientists are still mapping. It helps the heart pump better and keeps patients out of the hospital.

1. The drug blocks the SGLT2 pump on the wall of the proximal tubule.
2. Glucose that would normally go back into the blood stays in the urine instead.
3. The kidneys flush out the extra glucose, which also pulls some sodium and water along.
4. Blood volume drops gently, so the heart doesn't have to work against as much pressure.
5. The drug switches the heart muscle's fuel use from mostly glucose to more fat and ketones, which is easier on a stiff heart.
6. It lowers inflammation signals (like cytokines) and oxidative stress inside the heart muscle.
7. Over weeks to months, the left ventricle becomes a little less stiff and the patient breathes easier and has more energy.

SAFETY WATCH

Can cause yeast infections (vaginal or penile) because sugar in the urine feeds yeast; treat with antifungal cream. · Small risk of diabetic ketoacidosis (the acid crisis of missing insulin) (DKA) even if blood sugar is not sky-high—watch for nausea, vomiting, belly pain, or fruity breath and stop the drug if those appear. · Can drop blood pressure, so check for dizziness when standing up. · Rare risk of a serious infection in the genital area (Fournier gangrene); seek urgent care for pain, redness, swelling, or fever around the genitals or perineum. · May need to hold the drug before major surgery to lower DKA risk. · Check kidney function; do not start if eGFR is very low (thresholds vary by agent, typically do not start below 20–25 mL/min/1.73 m²).

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

Blocks aldosterone (a hormone that tells the kidneys to hold onto salt and water and dump potassium). By blocking aldosterone, the drug helps the kidneys get rid of extra fluid without wasting potassium, and it also slows down scarring (fibrosis) in the heart muscle. That makes the left ventricle a little less stiff over time.

1. The drug sits on the mineralocorticoid receptor and blocks aldosterone from docking there.
2. Without aldosterone's signal, the kidney stops pulling sodium back into the blood.
3. Sodium and water leave in the urine, gently lowering fluid overload.
4. Potassium stays in the blood instead of being dumped into the urine, so potassium levels stay normal or rise.
5. Over weeks to months, the drug also lowers the signals that tell heart cells to lay down collagen (scar tissue).
6. The heart muscle becomes a bit softer and more elastic, which helps it relax and fill with blood.

SAFETY WATCH

Can make potassium climb too high (hyperkalemia), especially if you also take an ACE inhibitor, ARB, or have kidney disease; check potassium within a week of starting and regularly after. · Can cause breast tenderness or enlargement (gynecomastia) in men because the drug also blocks some androgen receptors. · Can make periods irregular in women. · Avoid potassium supplements and salt substitutes (which are potassium chloride) while taking this drug. · Check kidney function before starting and regularly after; higher risk of high potassium (hyperkalemia) if eGFR is low.

Losartan (Cozaar), valsartan (Diovan), or sacubitril/valsartan (Entresto) · Angiotensin receptor blocker (ARB) or angiotensin receptor-neprilysin inhibitor (ARNI)

Blocks angiotensin II (a hormone that squeezes blood vessels tight and tells the heart to thicken and scar) from grabbing onto its receptor. This lets blood vessels relax, lowers blood pressure, and slows the remodeling (thickening and stiffening) of the heart muscle. Sacubitril/valsartan also blocks neprilysin (an enzyme that breaks down helpful peptides), so more of the body's natural relaxing and fluid-clearing signals stick around.

1. The drug blocks the AT1 receptor (the landing pad for angiotensin II).
2. Without angiotensin II's signal, blood vessels stay wider and blood pressure drops.

3. The heart muscle gets less of the 'grow and scar' message, so over months it becomes a bit less thick and stiff.
4. In the case of sacubitril/valsartan, sacubitril also stops neprilysin from breaking down natriuretic peptides (ANP, BNP) and bradykinin.
5. Those peptides tell the kidneys to dump salt and water and tell blood vessels to relax, so the combined effect is stronger than an ARB alone.

SAFETY WATCH

Can raise potassium, especially if combined with a MRA or ACE inhibitor; check potassium regularly. · Can cause a dry cough (less common than with ACE inhibitors, but still possible). · Rare risk of angioedema (sudden swelling of the lips, tongue, or throat); stop the drug and seek emergency care if this happens. · Do NOT use sacubitril/valsartan within 36 hours of taking an ACE inhibitor (raises angioedema risk). · Avoid in pregnancy (can harm the fetus). · Check kidney function before starting and a week or two after; a small rise in creatinine is expected, but a big jump means you may need to lower the dose or stop.

Metoprolol succinate (Toprol-XL) or carvedilol (Coreg) · Beta-blocker (beta-adrenergic receptor antagonist)

Blocks the receptors that adrenaline (epinephrine) and noradrenaline (norepinephrine) use to speed up the heart and make it squeeze harder. By turning down that overdrive signal, the heart beats slower and with less force, which gives it more time to relax and fill with blood. Over time, this also lowers blood pressure and can calm dangerous heart rhythms like atrial fibrillation (an irregular, quivering heartbeat).

1. The drug sits on beta-1 adrenergic receptors and blocks adrenaline and noradrenaline from docking.
2. The heart's pacemaker (SA node) slows down, so the heart rate drops.
3. Each heartbeat is less forceful, so the heart uses less oxygen.
4. The relaxation phase (diastole) lasts longer, which gives the stiff left ventricle more time to fill with blood.
5. Over weeks to months, the drug also lowers harmful remodeling signals, so the heart muscle becomes a bit less thick.
6. In patients with atrial fibrillation (an irregular, quivering heartbeat), the drug slows the signals traveling through the AV node, keeping the ventricles from racing.

SAFETY WATCH

Can make you feel tired, dizzy, or short of breath when you first start; the dose is usually started low and raised slowly. · Can drop blood pressure and heart rate too low; check both regularly. · Can hide the warning signs of low blood sugar (hypoglycemia) in people with diabetes, so check blood sugar more often. · Can make asthma or COPD worse by narrowing airways (beta-2 blockade); use with caution and pick a beta-1 selective agent like metoprolol if possible. · Do NOT stop suddenly—tapering off is important to avoid rebound high blood pressure or chest pain. · Avoid in people with severe a slow heart rate (bradycardia), heart block, or decompensated heart failure unless carefully supervised.

Red flags

- Sudden, severe shortness of breath with pink or bloody spit (sign of fluid filling the lungs)
- New or worsening chest pain, especially during activity (may signal a heart attack or dangerous lack of blood flow)
- Confusion, dizziness, or passing out (brain and body aren't getting enough oxygen)
- Rapid weight gain (more than 2–3 pounds in a day or 5 pounds in a week) or sudden increase in leg or belly swelling (sign that fluid is building up fast)
- Heart rate over 120 beats per minute at rest, or a new irregular rhythm (may be atrial fibrillation (an irregular, quivering heartbeat) or another dangerous arrhythmia)

Nursing priorities

1. Weigh the patient daily at the same time wearing similar clothing, and report weight gain of three pounds in one day or five pounds in one week to the provider immediately — Weight is the most reliable early sign of fluid overload because extra fluid adds body weight before swelling becomes visible; catching fluid buildup early prevents lung congestion and hospital readmission
2. Monitor intake and output closely, aiming for negative fluid balance when the patient has signs of fluid overload like swelling or shortness of breath — The kidneys hold onto salt and water in heart failure because low cardiac output (blood pumped per minute) makes the body think it is dehydrated; tracking how much fluid goes in versus comes out helps us adjust diuretic doses to remove extra fluid safely
3. Elevate the head of the bed at least thirty degrees and keep extra pillows at the bedside for patients with shortness of breath — Gravity pulls fluid down and out of the lungs when the patient sits upright, improving gas exchange (oxygen moving into blood); the stiff heart in HFpEF cannot handle the extra blood volume that flows back when lying flat

4. Check blood pressure and heart rate before giving ACE inhibitors, ARBs, beta-blockers, or other blood pressure medicines, and hold the dose if systolic blood pressure is below ninety or heart rate is below fifty — These medicines lower blood pressure and slow heart rate to reduce work on the heart, but giving them when pressure or rate is already low can cause dangerous drops in blood flow to the brain and kidneys
5. Monitor potassium and creatinine levels closely, especially when the patient takes spironolactone, ACE inhibitors, or ARBs together — These medicines block hormones that normally make the kidneys get rid of potassium, so potassium can build up to dangerous levels that cause deadly heart rhythm problems; they also can reduce kidney blood flow and raise creatinine (a kidney waste product)
6. Assess lung sounds every shift, listening for crackles or crackles in the lungs (rales) at the bases, and report new or worsening crackles immediately — Crackles are the sound of air moving through fluid in the alveoli (tiny air sacs in the lungs) and mean pulmonary edema (fluid flooding the lungs) (lung fluid buildup) from backward pressure when the left heart cannot pump forward; this can quickly become life-threatening
7. Check for pitting swelling (edema) in the ankles, legs, and sacrum (lower back area) every shift by pressing firmly for five seconds and watching how long the dent stays — Pitting swelling (edema) shows right heart failure causing venous congestion (backed-up blood in veins), and tracking whether it improves or worsens tells us if diuretic treatment is working; sacral swelling happens in patients who lie in bed most of the time because gravity pulls fluid there
8. Teach the patient to limit sodium intake to less than two grams per day and restrict fluids to two liters per day if provider orders fluid restriction — Sodium makes the body hold onto water through osmosis (water follows salt), and each gram of sodium holds about two hundred milliliters of extra fluid; limiting salt and fluid prevents overload and reduces how hard the stiff heart must work to pump

Sources

- AHA/ACC/HFSA 2022 Guideline for the Management of Heart Failure
- ESC 2021 Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure
- NEJM Clinical Practice Review on HFpEF
- Cochrane systematic reviews on heart failure

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