

Chronic Hfref

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart's left side normally squeezes strong with every beat and pushes about 55-70% of its blood out to your body. When that squeeze gets weak (under 40%), we call it heart failure with reduced ejection fraction, or HFrEF. Many things can hurt the heart muscle this way: a heart attack that killed part of the muscle, years of high blood pressure that wore it out, a leaky or tight valve that overworked it, a virus that attacked it, alcohol or chemotherapy that poisoned it, or sometimes we never find the reason. Once the pump is weak, your body tries to help by releasing stress hormones like adrenaline and a chemical cascade called renin-angiotensin-aldosterone. At first these make vessels squeeze and the heart beat faster to keep blood moving, but over months and years they stretch and scar the heart, worsen the rhythm, and trap salt and water so you swell and get short of breath. The good news is we now have four medicine pillars that block this harmful stress response and let the heart rest and recover: an ARNI (sacubitril-valsartan), a beta-blocker (carvedilol, metoprolol succinate, or bisoprolol), an MRA (spironolactone or eplerenone), and an SGLT2 inhibitor (dapagliflozin or empagliflozin). Every person with HFrEF should be on all four unless there is a clear reason they cannot tolerate one, and each medicine should be pushed to the proven target dose. These medicines together cut the risk of going to the hospital, improve how you feel, and help you live years longer. If medicines are not enough, devices like an ICD (a defibrillator to stop deadly rhythms) or CRT (a special pacemaker to make both sides of the heart squeeze together) and sometimes a heart pump or transplant may be needed, but the foundation is always the four medicine pillars started early and pushed as high as you can take.

What you might feel

- Shortness of breath (dyspnea) during activity or at rest
- Feeling very tired (fatigue) and weak muscles
- Swelling in the ankles, legs, or belly (edema)
- Weight gain of more than 2 to 3 pounds in one day or 5 pounds in one week
- Hard to breathe when lying flat (orthopnea)
- Waking up at night gasping for air (paroxysmal nocturnal trouble breathing (dyspnea))

- Neck veins that bulge or stick out (jugular venous distension)
- Extra heart sound (S3 gallop)
- Crackling or bubbling sounds in the lungs (rales or crackles)
- Cough that may bring up white or pink frothy mucus

Call the doctor right away if...

- Sudden, severe shortness of breath or gasping for air (may signal acute pulmonary edema (fluid flooding the lungs) — fluid flooding the lungs)
- Chest pain or pressure (could mean a heart attack is causing or worsening the heart failure)
- Confusion, extreme fatigue, or fainting (may mean the brain is not getting enough oxygen because the heart is failing badly)
- Fast weight gain (more than 2–3 pounds overnight or 5 pounds in a week) or worsening leg swelling despite taking medicines
- Coughing up pink, frothy coughed-up mucus (sputum) (sign of severe fluid backup in the lungs)

What the care team will do

- Assess breathing rate, oxygen saturation with pulse oximeter, and lung sounds every 4 hours or more often if the patient is short of breath.
- Weigh the patient at the same time every morning before breakfast using the same scale, and report any gain of more than 2 pounds overnight or 5 pounds in one week.
- Monitor strict intake and output every shift, and watch for urine output less than 0.5 milliliters per kilogram per hour or 30 milliliters per hour.
- Check blood pressure and heart rate before giving each dose of beta-blocker, ARNI, or other blood-pressure medicine, and hold the dose if systolic pressure is below 90 or heart rate is below 50.
- Teach the patient to limit sodium (salt) to less than 2 grams per day and fluids to 1.5 to 2 liters per day if the doctor ordered fluid restriction.
- Inspect ankles, legs, sacrum (lower back when lying down), and belly every shift for pitting swelling (edema), and measure the swelling by pressing your finger into the skin for 5 seconds.
- Check potassium and creatinine (a kidney test) levels at least weekly or as ordered, especially when the patient is on spironolactone, eplerenone, or ARNI.
- Encourage the patient to sit up in a chair or elevate the head of the bed at least 30 to 45 degrees if they are short of breath when lying flat.

Medicines you may be given — and why

Sacubitril-valsartan (Entresto)

This drug does two jobs at once. First, it blocks a hormone called angiotensin II that squeezes blood vessels and makes the heart pump harder. Second, it stops the body from breaking down helpful chemicals called natriuretic peptides that relax blood vessels and help the kidneys get rid of extra salt and water. Together, these actions lower the workload on the weak heart and help it pump blood more easily.

Beta-blockers — carvedilol, metoprolol succinate (Toprol-XL), or bisoprolol

These drugs block adrenaline (the 'fight or flight' hormone) from reaching the heart. In heart failure, the body pours out too much adrenaline trying to keep blood pressure up, but this overdrive wears out the heart muscle even faster. Beta-blockers slow the heart rate, lower blood pressure, and give the tired heart time to rest and recover between beats.

Mineralocorticoid receptor antagonists (MRAs) — spironolactone or eplerenone

These drugs block aldosterone, a hormone that makes the kidneys hold onto salt and water and helps scar tissue build up in the heart. By blocking aldosterone, these medicines help the body release extra fluid, lower blood pressure, and slow down harmful remodeling (thickening and stiffening) of the heart muscle.

SGLT2 inhibitors — dapagliflozin (Farxiga) or empagliflozin (Jardiance)

These drugs were first made for diabetes, but they also help the failing heart. They block a protein in the kidneys that normally pulls sugar and sodium back into the blood. When this protein is blocked, the kidneys dump more sugar, sodium, and water into the urine. This lowers fluid overload, blood pressure, and stress on the heart. They may also protect the heart muscle and kidneys through other pathways scientists are still studying.

Loop diuretics — furosemide (Lasix), bumetanide, or torsemide

These are strong water pills that help the kidneys release large amounts of salt and water into the urine. They work in the loop of Henle, a key part of the kidney's filtering system, to stop sodium from being pulled back into the blood. When sodium leaves, water follows, reducing swelling in the legs, belly, and lungs and making it easier to breathe.

Hydralazine and isosorbide dinitrate (combination)

This combination relaxes blood vessels in two different ways. Hydralazine relaxes the arteries (the vessels that carry blood away from the heart), making it easier for the heart to push blood out. Isosorbide dinitrate relaxes the veins (the vessels that carry blood back to the heart),

reducing the amount of blood returning to the heart and easing the backup into the lungs. Together, they lower the heart's workload.

ACE inhibitors — enalapril, lisinopril, or ramipril

These drugs block an enzyme that makes angiotensin II, a hormone that squeezes blood vessels tight and raises blood pressure. By blocking this enzyme, ACE inhibitors relax blood vessels, lower blood pressure, and reduce the strain on the weak heart. They also slow down harmful remodeling (the heart getting stiffer and bigger in unhealthy ways).

Angiotensin receptor blockers (ARBs) — losartan, valsartan (alone), or candesartan

These drugs block the receptor where angiotensin II (a blood vessel-squeezing hormone) attaches. Even if your body makes angiotensin II, it can't do its job because the receptor is blocked. This relaxes blood vessels, lowers blood pressure, and reduces strain on the weak heart. ARBs work like ACE inhibitors but do NOT cause a cough.

Ivabradine (Corlanor)

This drug slows the heart rate without lowering blood pressure or weakening the heart's squeeze. It works on the heart's natural pacemaker cells by blocking a special channel (called the If channel) that controls how fast the pacemaker fires. A slower heart rate gives the heart more time to fill with blood and rest between beats, which can improve symptoms and reduce hospitalizations.

Digoxin (Lanoxin)

This old drug makes the heart squeeze a bit harder by increasing the amount of calcium available inside heart muscle cells. It also slows the heart rate by acting on the vagus nerve, which puts a brake on the heart's electrical system. Digoxin can help reduce hospitalizations and improve symptoms, but it does NOT help you live longer.

Vericiguat (Verquvo)

This newer drug boosts a natural signaling pathway inside cells that helps blood vessels relax and protects the heart muscle. It works by stimulating an enzyme called soluble guanylate cyclase (sGC), which makes more cyclic GMP (a messenger molecule). More cyclic GMP helps blood vessels stay open and may slow harmful changes in the heart muscle.

Where this information comes from

- AHA/ACC/HFSA 2022 Guideline for the Management of Heart Failure
- Cochrane Heart Failure Reviews

- ESC 2021 Heart Failure Guidelines (European Society of Cardiology)
- NEJM Heart Failure Clinical Reviews

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