

Heart Failure Exacerbation

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a two-sided pump. The left side pushes oxygen-rich blood out to your body. The right side pushes blood to your lungs to pick up oxygen. In heart failure, one or both sides cannot keep up with what your body needs. Fluid backs up wherever the pump is struggling—into the lungs (making you short of breath and cough), into your legs (making them swell), or into your belly. An flare-up (exacerbation) (flare-up) happens when that backup gets suddenly worse—often because you missed your water pill, ate a lot of salty food, caught an infection, developed an irregular heartbeat, or had a heart attack. Treatment focuses on getting the extra fluid out fast with diuretics (water pills), easing the workload on your heart with medicines like nitrates and ACE inhibitors, and figuring out what triggered the flare so we can stop it from happening again.

What you might feel

- Shortness of breath during activity, at rest, or when lying flat
- Needing to sleep propped up on two or more pillows (orthopnea)
- Waking up gasping for air one to two hours after falling asleep (paroxysmal nocturnal trouble breathing (dyspnea))
- Swelling in the ankles, legs, or lower back
- Feeling tired and weak all the time
- Gaining two to three pounds or more in one to two days
- Wet, crackling sounds (rales or crackles) when the doctor listens to your lungs
- Coughing up pink, frothy spit or spit with streaks of blood
- Bulging neck veins when sitting upright (jugular venous distension)
- Confusion, extreme sleepiness, cold clammy skin, and low blood pressure

Call the doctor right away if...

- Pink frothy coughed-up mucus (sputum), severe shortness of breath, and dropping oxygen levels—sign of flash pulmonary edema (fluid flooding the lungs) (the lungs are flooding fast). Sit the patient upright immediately, give high-flow oxygen or BiPAP (a mask that pushes air into the lungs), and start IV furosemide (a water pill) and IV nitroglycerin if blood pressure is high enough.
- Cold clammy skin, very narrow pulse pressure (the gap between top and bottom blood pressure numbers is tiny), and confusion—sign of cardiogenic shock (the heart is failing to pump enough blood to keep organs alive). The patient needs medicines to raise blood pressure (norepinephrine) and possibly to strengthen the heartbeat (dobutamine), plus urgent consultation with a heart specialist or ICU team.
- New changes on the ECG (heart tracing) or rising troponin (a blood marker that shows heart muscle damage)—sign that a heart attack is triggering the heart failure. The patient needs urgent cardiology consultation and possibly a heart catheterization (a camera threaded into the heart arteries) to open a blocked artery.
- Rapid atrial fibrillation (an irregular, quivering heartbeat) with a heart rate above 130 beats per minute—the chaotic fast rhythm is overwhelming the weak heart. Slow the rate urgently with diltiazem or amiodarone. If the heart is already weak (HFrEF, or heart failure with reduced ejection fraction), use beta-blockers very carefully because they can make pumping even weaker in the short term.
- Rising creatinine (a blood test showing the kidneys are getting worse) even though you are pulling fluid off with diuretics—sign of cardiorenal syndrome (the heart and kidneys are trapped in a vicious cycle). You must balance removing enough fluid to help the heart without starving the kidneys of blood flow. Call nephrology (kidney specialists) if the kidneys keep declining.

What the care team will do

- Daily weight is more important than I/O
- Titrate O₂ to keep SpO₂ ≥ 92% (88% if COPD)
- Monitor for electrolyte drops after diuresis
- Teach salt (2 g/day) and fluid restriction (1.5–2 L/day)
- Confirm GDMT restart before discharge (ACE/ARNI, BB, MRA, SGLT2i)

Medicines you may be given — and why

Furosemide (Lasix)

Tells the kidneys to dump salt and water into the urine fast. This shrinks the total volume of blood in the body, so less fluid backs up into the lungs and makes breathing easier.

Nitroglycerin (IV or under the tongue)

Relaxes the veins so less blood pours back to the heart each beat, which takes pressure off the lungs. At higher doses it also opens up arteries, making it easier for the heart to push blood forward. If a blocked artery is part of the problem, nitroglycerin opens the coronary arteries (the vessels that feed the heart muscle itself) and brings more oxygen to the muscle.

Spironolactone (Aldactone)

Blocks aldosterone, a hormone that tells the kidneys to hold onto salt and water and to dump potassium. By blocking it, spironolactone lets the kidneys release a little extra salt and water (which helps dry out fluid backup) while keeping potassium in the blood. Over weeks and months, it also stops aldosterone from scarring and stiffening the heart muscle.

Carvedilol, metoprolol succinate, or bisoprolol

Blocks adrenaline (epinephrine and norepinephrine) from landing on beta receptors in the heart. This slows the heart rate, lowers the force of each heartbeat, and over weeks to months lets the overstretched, overworked heart muscle rest, shrink back toward normal size, and pump more efficiently.

Lisinopril, enalapril, or other ACE inhibitor

Blocks the enzyme that makes angiotensin II, a powerful hormone that squeezes blood vessels tight and tells the kidneys to hold onto salt and water. By blocking it, ACE inhibitors relax arteries (making it easier for the heart to pump blood forward), lower blood pressure, reduce fluid buildup, and over time stop the heart from scarring and enlarging.

Sacubitril/valsartan (Entresto)

A two-in-one pill. One half (valsartan) blocks angiotensin II from squeezing blood vessels; the other half (sacubitril) stops the breakdown of natriuretic peptides (natural hormones the heart releases when it is stretched). Together they relax arteries, help the kidneys dump salt and water, and protect the heart from scarring—better than an ACE inhibitor alone.

SGLT2 inhibitor: dapagliflozin (Farxiga) or empagliflozin (Jardiance)

Blocks a transporter in the kidney that normally pulls sugar (glucose) back into the blood. When it is blocked, sugar spills into the urine and takes water and a little sodium with it. This

gentle, steady fluid loss takes pressure off the heart, improves how efficiently the heart burns fuel, and cuts the risk of heart failure hospitalization and death—even in people without diabetes.

Digoxin (Lanoxin)

Blocks a pump on heart muscle cells that normally pushes sodium out. When sodium builds up a little inside the cell, calcium builds up, too—and calcium is what tells the muscle to squeeze. The result is a stronger contraction. Digoxin also slows the AV node (the electrical gate between the atria and ventricles), which helps control a fast heart rate in atrial fibrillation (an irregular, quivering heartbeat).

Hydralazine and isosorbide dinitrate (H-ISDN)

Hydralazine relaxes arteries, making it easier for the heart to pump blood forward. Isosorbide dinitrate relaxes veins, so less blood floods back to the heart and the lungs stay drier. Together they lower both afterload and preload. This combination improves survival in Black patients with heart failure with reduced ejection fraction, and is used when a patient cannot take an ACE inhibitor, ARB, or ARNI.

Dobutamine

Mimics adrenaline and turns on beta-1 receptors in the heart muscle, telling it to squeeze harder and faster. This boosts cardiac output (the amount of blood pumped per minute) in severe heart failure or cardiogenic shock when the heart is too weak to keep organs alive. It is given only in the ICU through a central IV line because it is very strong and can cause dangerous rhythms.

Milrinone

Blocks an enzyme that breaks down cyclic AMP in heart muscle and blood vessel smooth muscle. With more cyclic AMP around, the heart squeezes harder (inotrope) and blood vessels relax (vasodilator). This boosts cardiac output while lowering the resistance the heart pumps against. Like dobutamine, it is given only in the ICU in severe heart failure or cardiogenic shock.

Where this information comes from

- AHA/ACC/HFSA 2022 Heart Failure Guideline
- ESC 2023 Acute and Chronic Heart Failure Guideline
- NEJM 2024
- UpToDate 2024

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