

Acute Decompensated Heart Failure and Pulmonary Edema

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart's job is to pump blood forward to every organ. The left side pumps oxygen-rich blood out to your brain, kidneys, and muscles. The right side pumps blood through your lungs to pick up oxygen. Your kidneys watch how much blood is flowing forward and decide whether to hold onto salt and water or let them go. When the heart pumps well, this whole system stays balanced. But when the heart suddenly weakens or stiffens, it cannot move blood forward anymore. Pressure builds up behind the failing pump. Blood backs up into the lungs, and fluid leaks into the tiny air sacs where you breathe. That fluid makes you feel like you are drowning. Your body tries to help by squeezing blood vessels tighter and speeding up your heart to keep your brain alive. But those fixes make the weak heart work even harder, so it falls further behind. Meanwhile, your kidneys still sense low forward flow, so they hold onto more salt and water, which makes the backup worse. The swelling in your legs, the struggle to breathe, and the exhaustion all come from this one problem: your heart cannot pump blood forward, so everything backs up.

What you might feel

- Severe trouble breathing (dyspnea) (shortness of breath) at rest
- Breathlessness when lying flat (Orthopnea) (shortness of breath when lying flat)
- Paroxysmal nocturnal trouble breathing (dyspnea) (sudden nighttime shortness of breath that wakes the patient)
- Pink frothy coughed-up mucus (sputum) (coughing up pink, foamy fluid)
- Crackles or crackles in the lungs (rales) (wet crackling sounds heard with a stethoscope in the lung bases)
- Elevated jugular venous pressure (swollen, bulging neck veins)
- Peripheral swelling (edema) (swelling in the ankles and legs that leaves a dent when you press it)
- A fast heart rate (Tachycardia) (heart rate faster than 100 beats per minute)

- Low oxygen in the blood (Hypoxemia) (low oxygen saturation, often below 90 percent on pulse oximetry)
- Heavy sweating (Diaphoresis) (sweating) and cool, clammy skin

Call the doctor right away if...

- Low blood pressure (Hypotension) (systolic blood pressure below 90 millimeters of mercury), suggesting cardiogenic shock where the heart cannot pump enough blood to keep organs alive
- Altered mental status (confusion, drowsiness, difficulty staying awake), meaning the brain is not getting enough oxygen
- Pink frothy coughed-up mucus (sputum), showing severe pulmonary edema (fluid flooding the lungs) with capillary damage
- Oxygen saturation below 90 percent despite supplemental oxygen, meaning respiratory failure is near
- Cool extremities and mottled skin (blotchy purple or pale patches), showing the body is shutting down blood flow to the arms and legs to protect vital organs

What the care team will do

- Place the patient in high Fowler position (sitting upright at 60 to 90 degrees) immediately.
- Apply high-flow oxygen by nonrebreather mask at 10 to 15 liters per minute, targeting oxygen saturation above 90 percent, and prepare for bilevel positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP) if oxygen saturation does not improve.
- Establish large-bore intravenous access and administer intravenous loop diuretic (furosemide 40 mg IV push or double the patient's home oral dose) within 30 minutes of arrival.
- Place the patient on continuous cardiac monitoring and obtain a 12-lead electrocardiogram within 10 minutes.
- Monitor blood pressure every 5 to 15 minutes and hold or call the provider before giving nitroglycerin or other vasodilators (medicines that open blood vessels) if systolic blood pressure drops below 90 millimeters of mercury.
- Measure and record strict intake and output every hour, including urine output, and notify the provider if urine output is less than 0.5 milliliters per kilogram per hour.
- Assess lung sounds every 1 to 2 hours, listening for crackles or crackles in the lungs (rales) that should decrease as diuresis (urination) removes fluid.

- Keep the patient calm, limit activity to bedrest with bedside commode privileges only, and cluster care to allow rest periods.

Medicines you may be given — and why

Intravenous loop diuretic (furosemide or bumetanide)

Furosemide blocks a tiny pump (called the Na-K-2Cl cotransporter) in a curved part of the kidney's filtering tube (the loop of Henle). Normally this pump pulls salt back into your blood. When the drug blocks it, salt stays in the urine. Water follows salt like a magnet, so the kidneys make much more urine. This pulls extra water out of your blood vessels. In heart failure your vessels are overfilled—like a sponge soaked with too much water. Less blood volume means less pressure pushing fluid into your lungs, so the tiny air sacs (alveoli) can dry out and you can breathe easier.

Intravenous nitroglycerin

Nitroglycerin releases a gas molecule called nitric oxide inside the smooth muscle cells that wrap around your blood vessels. Nitric oxide starts a chain reaction that lowers calcium inside those cells. Less calcium makes the muscle relax, so the vessels get wider. The drug widens veins more than arteries. Wider veins hold more blood, so less blood returns to the heart (this is called reducing preload). With less blood coming back, the heart's main pumping chamber (left ventricle) fills with less pressure. Lower pressure in the heart means lower pressure in the lung blood vessels, so fluid stops leaking into the air sacs and even moves back out. Slightly wider arteries also mean the heart does not have to push as hard (reducing afterload), which lowers the heart's oxygen needs.

Noninvasive positive-pressure ventilation (BiPAP or CPAP)

A machine pushes air through a tight mask on your face. With BiPAP it pushes harder when you breathe in (to help you inhale) and lighter when you breathe out (to keep airways open). The constant gentle push during exhale (called PEEP or positive end-expiratory pressure) acts like a splint, holding the tiny air sacs open so they cannot collapse. Open air sacs let oxygen into the blood. The positive pressure also presses on the big veins in your chest, so less blood flows back to the heart (reducing preload). When the heart gets less blood to pump, the pressure inside it and in the lungs drops, and fluid leaks back out of the air sacs into the blood vessels. The machine also helps your breathing muscles rest, so your whole body uses less oxygen.

Intravenous morphine (selective use)

Morphine binds to opioid receptors in the brain and blood vessels. In the brain it reduces the feeling of breathlessness and anxiety, which lowers the body's stress response (sympathetic

activation). This calms rapid breathing and heart rate. In blood vessels morphine causes mild widening (vasodilation), especially of veins, which pools blood in the body and reduces the volume returning to the heart (preload reduction). Older teaching said morphine was a core treatment for pulmonary edema (fluid flooding the lungs), but newer data show it does not improve outcomes and may increase the need for mechanical ventilation and ICU admission. Current guidelines recommend using it only for patients with severe anxiety or chest pain that other treatments have not relieved, and only in small, careful doses.

Inotropic support: dobutamine or milrinone (for cardiogenic shock)

Dobutamine and milrinone both make the heart muscle squeeze harder (increase contractility) so each beat pumps more blood forward. Dobutamine turns on beta-1 receptors on heart cells, which raises a molecule called cyclic AMP inside the cell. More cyclic AMP increases calcium available to the squeezing proteins (actin and myosin), so the muscle contracts more forcefully. Milrinone blocks an enzyme (phosphodiesterase-3) that breaks down cyclic AMP, so cyclic AMP stays high longer, also boosting calcium and contraction. Both drugs slightly widen blood vessels, lowering the resistance the heart pumps against. These drugs are used only when the heart is so weak that it cannot pump enough blood to keep organs alive (cardiogenic shock)—not for routine pulmonary edema (fluid flooding the lungs). They increase the heart's oxygen demand and can cause dangerous irregular rhythms, so they are given in the ICU with continuous monitoring.

Guideline-directed medical therapy for chronic heart failure with reduced ejection fraction (HFrEF): ACE inhibitor (or ARB), beta-blocker, mineralocorticoid receptor antagonist (MRA), and SGLT2 inhibitor; or ARNI instead of ACE-I/ARB

These four drug classes shut down the body's harmful long-term stress responses that make heart failure worse over time. An ACE inhibitor blocks the enzyme that makes angiotensin II (a chemical that squeezes blood vessels and tells the kidneys to hold salt). Less angiotensin II means vessels relax, blood pressure drops, and the heart does not have to work as hard. An ARNI blocks angiotensin receptors and also stops the breakdown of helpful peptides (BNP, ANP) that widen vessels and help kidneys dump salt. Beta-blockers block adrenaline receptors on the heart, slowing heart rate and protecting heart cells from constant stress-hormone damage. An MRA (like spironolactone or eplerenone) blocks aldosterone (a hormone that makes you hold salt and water and builds scar tissue in the heart). An SGLT2 inhibitor (like dapagliflozin or empagliflozin) blocks a sugar-and-salt pump in the kidney, making you pee out glucose and a little extra salt; somehow this also helps the heart use energy better and reduces inflammation. Together these drugs reduce hospital visits, slow or reverse heart enlargement, and help people live longer—but the benefit takes weeks to months, not hours.

Vasopressin receptor antagonist (tolvaptan): reserved for severe, refractory low sodium (hyponatremia)

Tolvaptan blocks vasopressin (also called antidiuretic hormone, or ADH) from binding to V2 receptors in the kidney collecting duct. Normally vasopressin tells the kidney to put water channels (aquaporin-2) into the duct walls so water can be pulled back into the blood, concentrating the urine. When tolvaptan blocks the signal, those channels stay inside the cell, so the kidney cannot reabsorb water. You make large amounts of dilute (watery) urine, which raises the sodium concentration in your blood. This is called an aquaretic (water removal without salt loss), different from a regular diuretic. Tolvaptan is used only in hospitalized heart-failure patients with severely low sodium (hyponatremia, typically below 125 mEq/L) that has not improved with fluid restriction and regular diuretics, because low sodium can cause confusion, seizures, and worse outcomes. The drug has serious liver-toxicity risk and is very expensive, so it is reserved for select cases.

Avoid routine intravenous crystalloid fluids

Acute decompensated heart failure means the body already has too much fluid in the blood vessels—the problem is not a lack of fluid but a backup of fluid because the heart cannot pump it forward. The lungs and body tissues are congested (overfilled). Giving extra IV fluids increases the volume in the vessels even more, which raises the pressure in the heart and lungs and pushes more fluid into the air sacs, making breathing worse. The goal is to pull fluid out (with diuretics) and relax the vessels (with vasodilators) to lower pressure, not add more volume. IV fluids should only be given in the rare situation when a patient is truly low on volume (hypovolemia)—for example, after too much diuretic, vomiting, or diarrhea, and confirmed by physical exam (flat neck veins, low blood pressure that rises quickly with a small fluid bolus). In most cases of acute heart failure, giving IV fluids causes harm.

Where this information comes from

- AHA/ACC 2022 Heart Failure Guideline
- ESC 2021 Acute and Chronic Heart Failure Guideline
- Cochrane Heart Failure Reviews
- HFSA 2023 Heart Failure Practice Guideline

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