

Pulmonary Edema

A plain-English guide for you and your family

WHAT IS HAPPENING

Your lungs are built like millions of tiny grape-sized air sacs called alveoli, each wrapped in super-thin blood vessels. Every breath you take pulls fresh air into those sacs, and oxygen slips across the paper-thin wall into your blood while carbon dioxide slips out. In pulmonary edema (fluid flooding the lungs), fluid leaks out of the blood vessels and floods the air sacs — like a basement flooding during a storm. When the sacs fill with water instead of air, oxygen cannot cross into the blood, so your body panics: you gasp for breath, your heart races, and you may cough up pink frothy spit because the fluid is churning in your lungs. The leak usually starts because your heart is too weak to pump blood forward (so blood backs up into the lungs), or because your kidneys cannot remove extra fluid (so your blood vessels overfill and leak), or because the lung's own blood vessels are injured by infection or toxins and become leaky like a torn screen. The faster the team spots the pattern — shortness of breath, crackling sounds in the lungs, low oxygen, and wet lungs on an X-ray or ultrasound — the faster they can pull the fluid out with diuretics (water pills), ease the heart's work with blood-pressure drugs, or support your breathing with oxygen or a mask that pushes air in. Catching it early stops the flood; waiting too long can drown the lungs and strain the heart past the point of easy rescue.

What you might feel

- Shortness of breath that gets worse when lying flat
- Pink, frothy coughed-up mucus (sputum) when coughing
- Fast, shallow breathing
- Crackling sounds (rales) heard through a stethoscope at the base of the lungs
- Anxiety or a feeling of panic
- Cold, clammy, pale or bluish skin
- Rapid heart rate
- Swelling in the legs or abdomen

Call the doctor right away if...

- Pink or blood-tinged frothy coughed-up mucus (sputum) — means severe leak and possible bleeding in the lungs
- Confusion, extreme drowsiness, or trouble staying awake — the brain is not getting enough oxygen
- Blue or gray lips, tongue, or fingernails (cyanosis) — very low oxygen in the blood
- Blood pressure dropping despite a fast heart rate — the heart is failing to pump enough blood forward

What the care team will do

- Sit the patient fully upright at 90 degrees or in high Fowler position immediately
- Apply oxygen by nasal cannula or nonrebreather mask to target SpO2 greater than 90 percent
- Establish IV access and prepare to give furosemide (a loop diuretic) as ordered, typically 40 to 80 mg IV push
- Obtain a 12-lead ECG and have it read within 10 minutes
- Monitor blood pressure every 5 to 15 minutes and watch for low blood pressure (hypotension) below 90 systolic
- Attach continuous pulse oximetry and cardiac telemetry (heart monitor)
- Measure strict intake and output starting immediately, and place a Foley catheter if the patient cannot use a urinal reliably
- Notify the provider immediately if respiratory rate stays above 30, SpO2 stays below 90 despite high-flow oxygen, or the patient shows altered mental status or extreme fatigue

Medicines you may be given — and why

Furosemide (Lasix)

Furosemide makes the kidneys dump extra salt and water into the urine, which shrinks the total volume of fluid in the blood vessels. When there's less fluid pushing through the lungs' tiny blood vessels, less water leaks into the air sacs, so breathing gets easier fast.

Nitroglycerin (IV or sublingual)

Nitroglycerin releases nitric oxide inside the smooth muscle cells that wrap around veins and arteries. Nitric oxide tells those muscles to relax, so the vessels widen. Wider veins mean less blood returns to the heart, and wider arteries mean the heart doesn't have to push as hard—both lighten the heart's workload and drop the pressure flooding the lungs.

Morphine sulfate (low-dose IV)

Morphine binds to opioid receptors in the brain and blood vessels. In the brain, it calms the panic and air-hunger feeling that makes people gasp and thrash, which lowers the body's stress hormone surge. In the veins, it causes mild widening (vasodilation), which—like nitroglycerin—reduces the volume of blood returning to the heart and eases pressure in the lungs.

Oxygen (supplemental)

Oxygen raises the percentage of oxygen molecules in each breath. Even though the air sacs are partly filled with water, flooding them with a higher concentration of oxygen pushes more oxygen molecules across the wet barrier and into the bloodstream, keeping vital organs fed until the fluid clears.

Noninvasive positive-pressure ventilation (CPAP or BiPAP)

A tight-fitting mask blows air into the lungs under steady or cycling pressure. That pressure acts like an invisible hand pushing the water that leaked into the air sacs back across the capillary wall and into the bloodstream, where the kidneys can then remove it. The pressure also props the alveoli open so they don't collapse under the weight of the fluid.

Dobutamine or Milrinone (in cardiogenic shock with low output)

When the heart is so weak it can't pump enough blood forward—causing organs to shut down—dobutamine and milrinone force the heart muscle to contract harder. Dobutamine mimics adrenaline at beta-1 receptors on heart cells; milrinone blocks an enzyme (phosphodiesterase-3) that normally breaks down the 'squeeze' signal inside the cell. Both boost the calcium surge that powers each heartbeat, so the heart ejects more blood with each pump, relieving backward congestion into the lungs.

Vasopressin or Norepinephrine (in distributive shock with low SVR)

When sepsis or another cause makes blood vessels leak and dilate, blood pressure crashes even though the total fluid volume is high. Vasopressors clamp down the vessel walls—norepinephrine through alpha-1 adrenergic receptors, vasopressin through V1 receptors—raising resistance and squeezing blood back toward the core. This restores pressure so the kidneys can filter and diuretics can pull out the extra water flooding the lungs.

Antibiotics (in infection-triggered ARDS or sepsis)

When bacteria invade the lungs or bloodstream, the immune system's inflammatory response can damage the alveolar-capillary barrier, causing non-cardiogenic pulmonary edema (fluid flooding the lungs) (ARDS). Antibiotics kill the bacteria by breaking their cell walls, stopping

protein synthesis, or blocking DNA replication. Once the infection is controlled, inflammation settles and the leaky barrier begins to heal, stopping further fluid leak.

Where this information comes from

- AHA/ACC Heart Failure Guideline 2022
- UpToDate: Pulmonary Edema
- NEJM review: Acute Decompensated Heart Failure
- Cochrane: Interventions for Cardiogenic Pulmonary Edema

Last reviewed: 2026-07-07