

Acute Hypoxemic Respiratory Failure

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, tiny air sacs in your lungs—called alveoli—pull oxygen from the air you breathe and hand it off to blood flowing through the lungs. The blood then carries that oxygen to every cell in your body. When this job fails, something is blocking that oxygen handoff. Pneumonia (lung infection), ARDS (widespread lung inflammation from sepsis, trauma, or aspiration), fluid in the lungs from a weak heart, a blood clot in the lung artery, a collapsed lung, bleeding inside the lungs, or scarred lung tissue—each of these fills, collapses, or blocks parts of the lung. When oxygen cannot cross, your oxygen level—called SpO₂, the percent of your red blood cells carrying oxygen—drops. Your body fights back: you breathe faster and harder, your heart races, you feel anxious, and then you get tired because breathing becomes exhausting work. None of this is random—every sign is your body buying time while it tries to fix the oxygen problem. The real danger is not seeing the crash coming: if the team misses how fast things are sliding, the window to turn it around closes.

What you might feel

- Trouble breathing (Dyspnea) (shortness of breath)
- Fast breathing (Tachypnea) (breathing faster than 20 times per minute)
- Low oxygen in the blood (Hypoxemia) (oxygen saturation below 90% or blood oxygen pressure below 60 on room air)
- Use of accessory muscles (neck, shoulder, and belly muscles visibly working hard with every breath)
- A fast heart rate (Tachycardia) (heart beating faster than 100 times per minute)
- Bluish skin (Cyanosis) (blue color on lips, tongue, or fingernails)
- Altered mental status (confusion, restlessness, or being very sleepy)
- Heavy sweating (Diaphoresis) (heavy sweating)
- Inability to speak in full sentences
- Tripod positioning (sitting upright, leaning forward with hands on knees)

Call the doctor right away if...

- Oxygen saturation stays below 90% even when the patient is on high-flow oxygen or a non-rebreather mask at 15 liters per minute (this means blood is flowing past air sacs that are not working at all, or a thick barrier is blocking oxygen—supplemental oxygen cannot fix these problems)
- Breathing rate above 30 or below 8 breaths per minute (over 30 means the breathing muscles are about to give out from exhaustion; under 8 means the brain's breathing center is shutting down from too much carbon dioxide or sedation)
- Confusion, extreme sleepiness, or unable to cough or swallow safely (this signals the brain is not getting enough oxygen or carbon dioxide is building up dangerously high, causing the blood to turn acidic)
- Blood pressure drops below 90 systolic, heart rate climbs above 130, or signs of shock such as cold clammy skin and weak pulses (the heart and blood vessels are starting to fail because of severe oxygen lack or overwhelming infection)
- PaO₂ to FiO₂ ratio below 300 (this is the cutoff for acute lung injury by the Berlin definition) or below 200 (this defines moderate to severe ARDS and requires lung-protective ventilator settings to prevent further damage)

What the care team will do

- Place the patient on continuous pulse oximetry and capnography (a monitor that measures exhaled carbon dioxide) with alarm limits set at oxygen saturation below 90 percent and respiratory rate above 30 or below 8 breaths per minute, and respond to alarms within 30 seconds.
- Start supplemental oxygen immediately using the lowest flow that keeps oxygen saturation between 92 and 96 percent (or 88 to 92 percent if the patient has COPD), titrating up from nasal cannula to non-rebreather mask or high-flow nasal cannula as needed, and notify the provider if saturation stays below 90 percent despite a non-rebreather at 15 liters per minute.
- Position the patient upright at 45 to 90 degrees (high Fowler position) or in prone position (lying on stomach) if awake and able to tolerate it for at least 1 hour at a time, monitoring oxygen saturation during position changes.
- Assess respiratory rate, work of breathing (use of neck and shoulder muscles, nasal flaring, belly breathing), and mental status every 15 minutes until stable, then every 1 hour, and immediately notify the provider if respiratory rate climbs above 30, accessory muscle use increases, or the patient becomes confused or drowsy.

- Establish large-bore IV access (18-gauge or larger) and administer fluids cautiously at keep-vein-open rate (10 to 30 milliliters per hour) unless the provider orders a bolus for sepsis or shock, checking lung sounds before and after any fluid bolus.
- Keep the patient NPO (nothing by mouth) except small sips for pills until respiratory status stabilizes and the provider clears them to eat, and elevate the head of the bed at least 30 degrees during any oral intake.
- Draw arterial blood gas (ABG) from the radial artery or use venous blood gas (VBG) if arterial access is not available, and review results with the provider within 15 minutes, looking for pH below 7.35, PaCO₂ (blood carbon dioxide pressure) above 45, and PaO₂ (blood oxygen pressure) below 60 on room air or PaO₂-to-FiO₂ ratio below 300.
- Prepare intubation equipment at the bedside (endotracheal tube, laryngoscope, bag-valve-mask, suction, sedation and paralytic medications) and ensure the code cart and difficult airway supplies are within 30 seconds reach, reviewing the intubation plan with the respiratory therapist and provider.

Medicines you may be given — and why

Supplemental oxygen (nasal cannula, simple face mask, high-flow nasal cannula)

Oxygen therapy raises the amount of oxygen in each breath you take. Normally, room air is about 21% oxygen. When your lungs are hurt or sick, they cannot move enough oxygen from the air into your blood. By giving you richer oxygen—anywhere from 24% up to 100%—we boost the amount of oxygen that gets into your bloodstream, even if the lungs are not working perfectly. Think of it like turning up the brightness on a dim light: the bulb (your lungs) is the same, but more light (oxygen) gets through.

Noninvasive positive-pressure ventilation (CPAP or BiPAP)

CPAP and BiPAP are breathing machines that push air into your lungs through a tight-fitting mask over your nose or mouth—no tube goes down your throat. CPAP delivers one steady pressure all the time, like holding a balloon inflated; this keeps collapsed air sacs open. BiPAP gives you a higher push when you breathe in and a gentler pressure when you breathe out, making it easier to exhale. Both reduce the work your chest muscles must do and open up lung units that have collapsed, improving oxygen transfer.

Mechanical ventilation (intubation and ventilator)

When lungs are too sick or you are too weak or unconscious to breathe safely, a doctor slides a plastic tube through your vocal cords into your windpipe (trachea). This is called intubation. A ventilator machine then pushes oxygen-rich air into your lungs under controlled pressure and volume. The machine takes over the work of breathing so your body can rest and heal. We use

lung-protective settings—small breath sizes (tidal volumes of 4 to 8 mL per kilogram of your ideal weight) and end-pressure (PEEP, positive end-expiratory pressure) that keeps air sacs from collapsing—to avoid further injuring fragile lungs.

Prone positioning (flipping the patient face-down)

In severe lung injury (like ARDS, acute respiratory distress syndrome), the back parts of the lungs collapse under the weight of fluid and swelling because you lie on your back. When we flip you onto your stomach (prone positioning), gravity pulls that weight forward and opens up the back lung zones—the ones that were squashed. Blood flow also shifts to better-aired parts of the lung, improving the match between air and blood (ventilation-blood flow (perfusion) or V/Q matching). Think of it like flipping a wet sponge—the bottom side gets a chance to drain and re-expand.

Furosemide (Lasix)

Furosemide tells your kidneys to dump salt and water into your urine. It blocks a pump (the sodium-potassium-chloride cotransporter) in the thick ascending limb of the loop of Henle, a deep part of the kidney's filtering tubes. When sodium cannot be pulled back into the blood, water follows it out into the urine by osmosis (water goes where salt is). This shrinks the total volume of fluid in your body, lowering the pressure in your blood vessels and reducing fluid backup into the lungs. Think of it like opening a drain in a flooded basement—the water level drops.

Heparin (unfractionated) or enoxaparin (Lovenox, low-molecular-weight heparin)

Heparin and enoxaparin slow down your body's clotting system. They boost the power of antithrombin (a natural protein in your blood that blocks clotting enzymes). Heparin binds to antithrombin, making it work hundreds of times faster to block thrombin (the enzyme that glues clotting proteins together to form a clot) and Factor Xa (an enzyme earlier in the clotting chain). This does not dissolve clots that are already there, but it stops them from growing bigger and prevents new clots from forming. Your body's own cleanup crew (plasmin in the fibrinolytic system) then slowly dissolves the existing clot over days to weeks. Think of it like putting out a fire—you stop it from spreading, and the embers burn out on their own.

Antibiotics (e.g., ceftriaxone, azithromycin, cefepime, piperacillin-tazobactam, vancomycin, linezolid)

Antibiotics kill or stop the growth of bacteria that are causing infection in your lungs (pneumonia). Different antibiotics attack bacteria in different ways. Beta-lactams (like ceftriaxone, cefepime, and piperacillin-tazobactam) punch holes in the bacterial cell wall so the cell bursts. Macrolides (like azithromycin) block the bacterial ribosome (the factory that makes

proteins the bacteria need to live). Glycopeptides (like vancomycin) and oxazolidinones (like linezolid) also jam protein-making or cell-wall building in bacteria that are resistant to other drugs. The choice of antibiotic depends on where you caught the infection (community, hospital, or ventilator-associated pneumonia), local resistance patterns, and your allergies.

Albuterol (ProAir, Ventolin, Proventil)

Albuterol relaxes the smooth muscle wrapped around your breathing tubes (bronchi and bronchioles). It binds to beta-2 adrenergic receptors on those muscle cells, starting a chemical chain reaction inside the cell that raises cyclic AMP (cAMP, a messenger molecule inside cells that tells the muscle to relax). Rising cAMP tells the muscle to relax and the airway to widen. This makes it easier for air to flow in and out, and mucus can be coughed up more easily. Think of it like loosening a belt around your chest—you can breathe deeper and easier. Albuterol is most helpful when airways are tight (bronchoconstriction), as in asthma, COPD exacerbations, or acute bronchitis overlying respiratory failure.

Corticosteroids (e.g., methylprednisolone, prednisone, dexamethasone, hydrocortisone)

Steroids turn down the volume on your immune system's inflammatory response. They enter cells and bind to glucocorticoid receptors in the cytoplasm (the jelly-like inside of the cell). The drug-receptor complex then moves into the nucleus (the cell's control center) and changes which genes are turned on or off. This reduces production of inflammatory chemicals (cytokines like interleukin-6 and tumor tissue death (necrosis) factor-alpha, and prostaglandins, which are messengers that trigger swelling and pain) and stabilizes blood vessel walls, so less fluid and fewer white blood cells leak into tissues. In ARDS, severe asthma or COPD exacerbations, COVID-19 pneumonia, or certain other pneumonias (like eosinophilic or Pneumocystis), steroids calm the storm of inflammation that is damaging the lungs.

Neuromuscular blocking agents (e.g., cisatracurium, rocuronium, vecuronium)

Neuromuscular blockers temporarily paralyze all your voluntary muscles—the ones you use to move, breathe, and even blink—by blocking the signal between nerves and muscles. Normally, a nerve releases acetylcholine (a chemical messenger) at the junction with a muscle; acetylcholine binds to nicotinic receptors (a type of docking port on the muscle surface) and tells it to contract. These drugs sit on those same receptors and block acetylcholine, so the muscle gets no signal and stays relaxed (paralyzed). In severe ARDS, paralyzing you for 24 to 48 hours can reduce fighting against the ventilator (dyssynchrony), lower the pressure needed to inflate stiff lungs, and improve oxygen. This is used only in the sickest patients when the ventilator alone is not enough.

Where this information comes from

- American Thoracic Society / European Society of Intensive Care Medicine / Society of Critical Care Medicine Clinical Practice Guideline: Mechanical Ventilation in ARDS (2017)
- Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock (2021)
- American Thoracic Society (ATS)
- Society of Critical Care Medicine (SCCM)

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