

ARDS · Acute Respiratory Distress Syndrome

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, the tiny air sacs in your lungs (alveoli) are dry, stretchy, and coated with a slippery film called surfactant that keeps them open. Between each air sac and its blood vessel sits a wall so thin that oxygen slips across in a split second—but strong enough to keep blood fluid out. When something injures that wall—severe pneumonia, breathing in vomit, a crushing chest injury, a large transfusion, smoke inhalation, or overwhelming infection spreading through the body—the wall becomes leaky. Protein-rich fluid pours from the blood vessels into the air sacs, washing away the surfactant and filling the sacs with liquid. Some air sacs collapse, some fill like wet sponges, and only a small patch of lung (doctors call it the 'baby lung') stays open and working. Blood flowing past the flooded and collapsed sacs cannot pick up oxygen, so oxygen levels in the blood drop dangerously low—even when the person is breathing pure oxygen through a mask. The brain senses the drop and tells the body to breathe faster and harder, but the stiff, heavy lungs make every breath exhausting. Without a breathing machine to hold the air sacs open, suction out fluid, and deliver high oxygen, the breathing muscles tire out, carbon dioxide builds up, the right side of the heart strains against the swollen lung tissue, and other organs—kidneys, liver, brain—begin to fail from lack of oxygen. Every clue—the chest X-ray showing white clouds on both sides, the oxygen meter stuck at 85 percent despite a non-rebreather mask, the fast shallow breaths—points back to one broken piece: the damaged wall that turned dry air sacs into flooded ones.

What you might feel

- Severe shortness of breath (dyspnea) that starts suddenly
- Very fast breathing (tachypnea), often more than 30 breaths every minute
- Oxygen level stays dangerously low even when breathing 100% oxygen (refractory low oxygen in the blood (hypoxemia))
- Neck and chest muscles working visibly hard with every breath (use of accessory muscles)
- Fast heart rate (tachycardia)

- Blue or gray color of the lips, fingertips, or skin (cyanosis)
- Wet crackling or popping sounds heard with a stethoscope over both lungs (diffuse crackles)
- Confusion, restlessness, or sleepiness (altered mental status)
- Inability to speak a full sentence without gasping for air
- Sitting upright and leaning forward on the hands (tripod position)

Call the doctor right away if...

- Oxygen saturation stays below 88% even on a non-rebreather mask or high-flow nasal cannula (means the lungs are so damaged that oxygen cannot get into the blood)
- Breathing rate climbs above 35 breaths per minute, or suddenly slows down while the person becomes less alert (muscle exhaustion—respiratory arrest is imminent)
- New confusion, extreme drowsiness, or no response to voice or touch (the brain is starving for oxygen)
- Blood pressure drops below 90 systolic or signs of shock appear—cold, sweaty skin, weak pulse, no urine output (multiple organs are failing from low oxygen and widespread inflammation)
- Visible use of neck and chest muscles with every breath, tripod posture, or inability to speak more than a few words (severe respiratory distress—intubation needed urgently)

What the care team will do

- Monitor oxygen saturation continuously with pulse oximetry and keep SpO₂ above 88%; escalate immediately if saturation stays below 88% despite oxygen therapy
- Position the patient prone (lying on the stomach) for 12 to 16 hours daily if the physician orders it and the patient tolerates the position
- Limit IV fluid boluses and daily intake to the smallest amount needed to keep blood pressure safe; measure urine output, daily weight, and central venous pressure if available
- Check ventilator settings every hour: confirm tidal volume is 4 to 8 mL/kg ideal body weight (usually 6 mL/kg), plateau pressure stays below 30 cm H₂O, and driving pressure stays below 15 cm H₂O
- Suction the endotracheal tube only when you hear coarse crackles or see a drop in oxygen saturation; use inline suction and limit each pass to 10 seconds
- Raise the head of the bed to 30 to 45 degrees unless the patient is prone or with low blood pressure (hypotensive) (low blood pressure)
- Perform mouth care every 2 to 4 hours with chlorhexidine (an antiseptic rinse) and brush the teeth twice daily

- Assess sedation level hourly using a validated scale like RASS (Richmond Agitation-Sedation Scale) and coordinate daily sedation interruption with the physician if the patient is stable

Medicines you may be given — and why

Low tidal volume ventilation (6 mL/kg ideal body weight, plateau pressure < 30 cm H₂O, driving pressure < 15 cm H₂O)

Small breaths delivered by the ventilator protect the injured air sacs (alveoli) from being stretched too much. When alveoli overstretch, they tear and release chemicals that make the injury worse—like blowing up a balloon too far until it pops. We keep the pressure low (plateau < 30, driving < 15) so the lungs don't rupture and the remaining healthy parts stay open without damage. PEEP (positive end-expiratory pressure) is air pressure that holds the alveoli open between breaths—like keeping a wet tissue paper tube from collapsing—so oxygen has a surface to cross into the blood. After the patient is stable, we limit IV fluids to keep extra water from flooding the already-leaky lungs. Prone positioning (lying face-down for 16 hours) lets the back parts of the lungs open up because gravity now pulls fluid forward instead of squashing the back; this matches air and blood flow better. Muscle-paralyzing drugs for 48 hours stop the patient from fighting the ventilator, which lowers oxygen demand and lets the lungs rest. ECMO (a machine that takes blood out, adds oxygen, and puts it back) is used only when oxygen levels stay dangerously low despite everything else. Dexamethasone (a steroid) 6 mg daily calms lung inflammation in COVID-19 ARDS; evidence is weaker for non-COVID causes. Always treat the root cause: antibiotics for pneumonia, surgery to drain infections, diuretics (water pills) if heart failure caused the fluid backup.

Oxygen therapy (nasal cannula, high-flow nasal cannula, non-rebreather mask, mechanical ventilation)

ARDS makes it hard for oxygen to cross from the air sacs into the blood because fluid, protein, and dead cells clog the space. We deliver extra oxygen—higher than the 21% in room air—to push more oxygen molecules across whatever membrane surface is still working. Think of it like turning up the volume when a speaker is muffled. We start with the lowest method that works (nasal prongs at 1–6 liters per minute) and escalate to high-flow nasal cannula (warm, humidified oxygen up to 60 liters per minute, which also washes out dead-space CO₂ and provides a small amount of pressure to keep airways open), then to a non-rebreather mask (delivers close to 100% oxygen with a one-way valve so exhaled air doesn't mix back in), and finally to intubation (placing a breathing tube) and mechanical ventilation if the patient cannot maintain safe oxygen levels or is exhausting from the work of breathing.

Fluid management (restrictive strategy after resuscitation)

In the first hours of ARDS (especially if sepsis or shock triggered it), we give IV fluids (saline or balanced crystalloids like lactated Ringer's) rapidly to restore blood pressure and organ blood flow—this is resuscitation. Once the patient is stable (blood pressure adequate, urine output $\geq$ 0.5 mL/kg/hour, lactate clearing), we switch to a restrictive (conservative) fluid strategy: we stop or minimize ongoing IV fluids and may use diuretics (water pills like furosemide) to create a net-negative fluid balance (more out than in each day). Why? The alveolar-capillary membrane in ARDS is leaky—extra fluid in the bloodstream raises hydrostatic pressure (like turning up water pressure in a hose), pushing more fluid across the leak into the air sacs. Less fluid in the circulation means less pressure driving swelling (edema), so the lungs stay drier, gas exchange improves, and ventilator time shortens. Think of it like plugging a leaky bucket partway—you can't stop the leak, but you can avoid overfilling the bucket so it doesn't overflow.

Antibiotics (empiric broad-spectrum, then targeted)

Bacterial infection (pneumonia, bloodstream infection, abdominal abscess) is a common cause of ARDS. Bacteria release toxins (endotoxins from gram-negative bacteria like *E. coli*, exotoxins from gram-positive bacteria like *Staph aureus*) that activate the immune system, triggering a flood of cytokines (inflammatory chemicals) that damage the alveolar-capillary membrane and cause fluid to leak into the air sacs. Antibiotics kill bacteria by targeting their cell walls (beta-lactams like piperacillin-tazobactam), protein-making machinery (fluoroquinolones like levofloxacin), or DNA (metronidazole for anaerobes). Once bacteria die, toxin release stops, the immune storm calms, and the lungs can begin to heal. We start with broad-spectrum antibiotics (covering many types of bacteria) within 1 hour of recognizing sepsis, then narrow to targeted therapy once culture results identify the specific bug and its antibiotic sensitivities.

Dexamethasone (for COVID-19 ARDS)

COVID-19 can trigger an exaggerated immune response (cytokine storm) where the body releases floods of inflammatory chemicals (cytokines like IL-6, TNF-alpha) that injure the lungs, causing ARDS. Dexamethasone enters immune cells (T cells, macrophages, neutrophils) and binds to receptors inside the cell nucleus, turning OFF genes that make inflammatory proteins and turning ON genes that calm inflammation. This dampens the cytokine storm, reduces alveolar-capillary leak, and prevents progression to severe ARDS. The RECOVERY trial (published 2020) showed that dexamethasone 6 mg once daily for up to 10 days cuts death by one-third in hospitalized COVID-19 patients needing oxygen. It does NOT help patients who do NOT need oxygen (and may harm them), and evidence is weak for non-COVID ARDS.

Neuromuscular blocking agents (cisatracurium, for severe ARDS)

In severe ARDS, the patient may fight the ventilator (patient-ventilator dyssynchrony), which increases oxygen consumption by respiratory muscles, worsens lung injury from uncoordinated breaths, and makes it hard to deliver protective ventilation. Cisatracurium blocks acetylcholine receptors on the muscle side of the neuromuscular junction (the connection between nerve and muscle)—think of it like unplugging the wire between a switch and a light bulb. When the muscle cannot receive the nerve's signal, it relaxes completely. This eliminates all spontaneous breathing effort, allowing the ventilator to deliver precise low tidal volumes and controlled pressures without interference. The ACURASYS trial (2010) showed that 48 hours of cisatracurium infusion in early severe ARDS ($\text{PaO}_2/\text{FiO}_2 < 150$) improved survival and reduced barotrauma, possibly by reducing cytokine release from less vigorous breathing and better ventilator synchrony. Importantly, benefit is seen **ONLY** in the first 48 hours and **ONLY** in severe ARDS—longer use or use in less severe cases does not help and increases harm.

Where this information comes from

- ARDS Definition Task Force (Berlin Definition), JAMA 2012
- 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 2021
- National Heart, Lung, and Blood Institute ARDS Clinical Network (ARDSNet)

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