

ARDS • Acute Respiratory Distress Syndrome

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THE STORY IN ONE SENTENCE

Something—infection, breathing in stomach contents, major injury, bad blood transfusion, or other insult—damages the paper-thin wall between air sacs and blood vessels, so protein-rich fluid floods the air sacs and oxygen cannot cross into the blood.

“The wall between air sacs and blood vessels breaks and leaks fluid into the air sacs, drowning the lung from the inside.”

Normal anatomy — the body's job

Each alveolus (air sac) is a tiny, stretchy balloon with walls one cell thick, coated with surfactant—a slippery soap-like film that lowers surface tension so the sac stays open even at the end of a breath. The alveolar-capillary barrier (the wall between air and blood) is thinner than tissue paper, built from three layers: the alveolar cell, a shared basement membrane, and the capillary cell. This ultra-thin wall lets oxygen and carbon dioxide zip across in milliseconds but keeps protein and fluid locked in the blood.

BODY'S JOB

The lungs trade gases: oxygen from the air moves into the blood, and carbon dioxide from the blood moves out into the air. For this swap to work, alveoli must stay dry and open, and the barrier must stay thin and tight.

1. You breathe in: fresh air fills the alveoli with oxygen.
2. Oxygen crosses the paper-thin alveolar-capillary barrier in less than one second.
3. Red blood cells in the capillaries grab the oxygen and carry it to every organ.

4. At the same time, carbon dioxide (the waste gas from your cells) crosses the barrier in the opposite direction, from blood into the air sac.
5. You breathe out: the carbon dioxide leaves your body.
6. Surfactant keeps the alveoli from collapsing at the end of the breath, so they are ready to fill again.

What breaks

An injury or infection damages the thin wall between air sacs and blood vessels, turning it from a tight seal into a leaky sieve. Protein-rich fluid floods the air sacs, and the surfactant coating washes away or stops working.

CLINICAL TERM

An insult—direct (pneumonia, aspiration of gastric contents, inhalation injury, lung contusion) or indirect (sepsis, severe trauma, pancreatitis, massive transfusion)—triggers inflammation that injures both the capillary endothelium and the alveolar epithelium. The alveolar-capillary barrier becomes permeable (leaky), allowing protein-rich fluid to pour into the alveolar space (non-cardiogenic pulmonary edema (fluid flooding the lungs)). Surfactant is inactivated by the fluid and inflammatory proteins or washed out altogether. Alveoli collapse (atelectasis), especially dependent regions. Only a fraction of lung tissue—the 'baby lung'—remains aerated. Flooded and collapsed alveoli create intrapulmonary shunt: venous blood passes through the lung without being oxygenated, causing refractory low oxygen in the blood (hypoxemia) ($\text{PaO}_2/\text{FiO}_2$ ratio ≤ 300 mmHg by Berlin criteria, with severe ARDS defined as $\text{P}/\text{F} \leq 100$). The shunt does not improve with supplemental oxygen alone because the blood never contacts open alveoli.

How the body compensates

The body tries to keep oxygen delivery high enough by breathing faster and pumping harder, but when too much lung is out of commission, these efforts backfire—the work of breathing skyrockets, muscles tire, and the patient crashes. Vital signs may look acceptable until sudden decompensation.

1. {'label': 'Low oxygen in the blood (Hypoxemia) triggers fast breathing (tachypnea)', 'detail': 'Peripheral chemoreceptors in the carotid and aortic bodies sense falling arterial oxygen ($\text{PaO}_2 < 60$ mmHg) and signal the brainstem respiratory center to increase breathing rate (tachypnea, often 25–35 breaths per minute) and depth, raising minute ventilation (the total volume of air moved per minute).'} }

2. {'label': 'Heart rate climbs', 'detail': 'The sympathetic nervous system releases adrenaline (epinephrine), speeding the heart (tachycardia, often 100–130 bpm) to pump more blood per minute and deliver whatever oxygen is available to the brain, heart, and kidneys.'}
3. {'label': 'Work of breathing soars', 'detail': 'Flooded and collapsed alveoli are stiff (reduced compliance, often < 40 mL/cmH₂O), so each breath requires much higher pressure. The diaphragm and intercostal muscles (breathing muscles) work at near-maximal effort. Accessory muscles (neck and abdominal muscles) recruit, and you see retractions (skin sucking in between ribs and above the collarbone).')}
4. {'label': 'The dangerous middle', 'detail': 'The patient may have stable blood pressure and a reasonable oxygen saturation (SpO₂ 88–92 percent) on high-flow oxygen—but only because every compensatory mechanism is running at full throttle. Rising respiratory rate, increasing oxygen requirement (FiO₂ climbing from 0.5 to 0.8 to 1.0), and visible accessory muscle use are red flags that decompensation is imminent.'}
5. {'label': 'Refractory low oxygen in the blood (hypoxemia) persists', 'detail': 'Despite non-rebreather mask or high-flow nasal cannula delivering FiO₂ ≥ 0.6 , SpO₂ remains below 90 percent and PaO₂ stays below 60 mmHg because of the large intrapulmonary shunt. This defines the need for invasive mechanical ventilation with positive end-expiratory pressure (PEEP) to recruit collapsed alveoli.'}
6. {'label': 'Respiratory muscle fatigue and hypercapnic failure', 'detail': 'The diaphragm and intercostals cannot sustain the extreme work indefinitely. Fatigue sets in (you may see paradoxical abdominal breathing—belly sucking in during inspiration instead of expanding), tidal volume drops, and carbon dioxide (PaCO₂) begins to rise above 45 mmHg, causing respiratory acidosis (acid building up from trapped carbon dioxide) (pH < 7.35). Without mechanical ventilation, the patient will stop breathing.'}
7. {'label': 'Right heart strain (cor pulmonale)', 'detail': 'Alveolar low oxygen (hypoxia) and high airway pressures constrict pulmonary arteries (hypoxic pulmonary vasoconstriction), raising pulmonary vascular resistance. The right ventricle pumps against this increased afterload, dilates, and eventually fails, causing jugular venous distension, an enlarged liver (hepatomegaly) (enlarged liver), and peripheral swelling (edema).')}
8. {'label': 'Multi-organ failure', 'detail': 'Prolonged low oxygen in the blood (hypoxemia) (PaO₂ < 55 mmHg for hours to days) starves vital organs. Acute kidney injury develops (creatinine rises, urine output drops), hepatic transaminases climb, altered mental status appears (hypoxic encephalopathy), and lactic acidosis (acid from oxygen-starved tissues) worsens as cells switch to anaerobic metabolism.'}
9. {'label': 'Mortality in severe ARDS', 'detail': 'Even with lung-protective ventilation (tidal volume 6 mL/kg ideal body weight, plateau pressure ≤ 30 cmH₂O), prone positioning (flipping the patient face-down for 16 hours per day), neuromuscular blockade, and PEEP titration, mortality in severe ARDS (P/F ≤ 100) is 40–46 percent. Mild ARDS (P/F 200–300) carries 27–35 percent mortality, and moderate ARDS (P/F 100–200) 32–40 percent.'}

10. {'label': 'Watch the trend', 'detail': 'A patient who looks stable now but whose FiO_2 requirement is climbing ($0.4 \rightarrow 0.6 \rightarrow 0.8$ over hours), whose respiratory rate is increasing ($20 \rightarrow 28 \rightarrow 35$), or who is using accessory muscles is on the path to decompensation. Early intubation and lung-protective ventilation improve outcomes compared to waiting for full respiratory arrest.'}

Domino effect

1. An insult damages the alveolar-capillary barrier. — A direct hit (pneumonia, aspiration of gastric contents, lung contusion, inhalation of smoke or toxins) or an indirect wave of inflammation (sepsis from bloodstream infection, severe trauma, acute pancreatitis, massive transfusion) triggers release of pro-inflammatory cytokines ($\text{TNF-}\alpha$, IL-1, IL-6, IL-8) and recruits neutrophils (white blood cells) to the lung. These cells release reactive oxygen species and proteases (enzymes that break down proteins), which injure both the capillary endothelium (blood vessel lining) and the alveolar epithelium (air sac lining), disrupting tight junctions and creating gaps in the barrier.
2. Protein-rich fluid floods the alveoli. — The damaged barrier becomes permeable (leaky). Plasma proteins—albumin, fibrinogen, immunoglobulins—and fluid leak from the capillaries into the interstitium (the space around the alveoli) and then into the alveolar space itself. This non-cardiogenic pulmonary edema (fluid flooding the lungs) (fluid in the lungs not caused by heart failure) has high protein content (edema fluid-to-plasma protein ratio > 0.65), unlike the watery swelling (edema) of heart failure.
3. Surfactant is inactivated and alveoli collapse. — Surfactant—a mix of phospholipids (dipalmitoylphosphatidylcholine) and proteins (SP-A, SP-B, SP-C, SP-D) secreted by type II alveolar cells—normally lowers surface tension, keeping alveoli open at end-exhalation. Swelling (Edema) fluid dilutes surfactant, and plasma proteins (especially fibrinogen and albumin) bind to and inactivate surfactant phospholipids. Surface tension rises, and alveoli collapse (atelectasis) at low lung volumes, especially in dependent (gravity-dependent, back or bottom) regions. The functional residual capacity (the volume of air left in the lungs after a normal breath) drops from roughly 2.5–3.0 liters to less than 1.5 liters.

4. Intrapulmonary shunt develops and low oxygen in the blood (hypoxemia) worsens. — Blood flowing through capillaries next to flooded or collapsed alveoli cannot pick up oxygen because there is no air-blood interface for gas exchange. This shunted blood (venous blood with oxygen saturation around 75 percent) mixes with oxygenated blood from the remaining functional 'baby lung,' diluting arterial oxygen. A shunt fraction above 20 percent (normal < 5 percent) causes severe low oxygen in the blood (hypoxemia). Because the shunted blood never contacts ventilated alveoli, increasing inspired oxygen (FiO_2) has limited effect— PaO_2 remains low even at FiO_2 1.0 (100 percent oxygen). This refractory low oxygen in the blood ($\text{PaO}_2/\text{FiO}_2$ ratio ≤ 300 mmHg) is the hallmark of ARDS and meets the Berlin definition when bilateral infiltrates are present on chest imaging, PEEP ≥ 5 cmH₂O is applied, and the swelling (edema) is not fully explained by heart failure or fluid overload.
5. The patient develops fast breathing (tachypnea) and increased work of breathing. — Peripheral chemoreceptors in the carotid bodies detect falling PaO_2 (hypoxemia) and central chemoreceptors in the brainstem detect rising PaCO_2 (hypercapnia) and falling pH (acidosis). These sensors signal the respiratory center in the medulla to increase respiratory rate (often 25–40 breaths per minute) and tidal volume, raising minute ventilation. At the same time, lung compliance (stretchiness) falls from normal 50–100 mL/cmH₂O to 20–40 mL/cmH₂O because flooded and collapsed alveoli resist inflation. The pressure needed to move a given volume of air (airway resistance) also rises because small airways collapse. The diaphragm and intercostal muscles must generate much higher force, and accessory muscles (sternocleidomastoid, scalenes, abdominal muscles) recruit. You see suprasternal, supraclavicular, and intercostal retractions (skin sucking inward with each breath), nasal flaring, and tripod positioning (leaning forward, hands on knees).
6. Respiratory muscles fatigue and hypercapnic respiratory failure begins. — The diaphragm and intercostals are skeletal muscles with limited endurance at near-maximal workload. After hours to days of extreme effort, they fatigue—force generation drops, tidal volume falls (often below 5 mL/kg), and carbon dioxide (CO_2) production exceeds CO_2 elimination. PaCO_2 climbs above 45 mmHg, then 55, then 65 mmHg. Rising CO_2 lowers blood pH (respiratory too much acid in the blood (acidosis), pH < 7.35, then < 7.25, then < 7.20). Too much acid in the blood impairs cardiac contractility, causes arrhythmias (abnormal heart rhythms), and depresses mental status (confusion, somnolence, coma). Paradoxical abdominal breathing appears: the belly sucks inward during inspiration instead of expanding, a sign of diaphragm fatigue. Without mechanical ventilation to take over the work of breathing, the patient will develop respiratory arrest (cessation of breathing) and cardiac arrest.

7. Right heart strain (acute cor pulmonale) and pulmonary high blood pressure (hypertension) develop. — Alveolar low oxygen (hypoxia) (low oxygen in the air sacs) triggers oxygen-starved (hypoxic) pulmonary vasoconstriction: smooth muscle in the walls of small pulmonary arteries contracts to shunt blood away from poorly ventilated regions. When ARDS is widespread, vasoconstriction is diffuse, raising mean pulmonary artery pressure from normal 10–15 mmHg to 25–35 mmHg or higher. High airway pressures from mechanical ventilation (especially if tidal volume is too large or PEEP is excessive) compress alveolar capillaries, further increasing pulmonary vascular resistance (afterload on the right ventricle). The right ventricle, a thin-walled chamber designed to pump against low resistance, dilates and its wall thins (eccentric hypertrophy). Right ventricular stroke volume falls, causing jugular venous distension (JVD), an enlarged liver (hepatomegaly) (enlarged, tender liver), fluid buildup in the belly (ascites) (abdominal fluid), and peripheral swelling (edema) (swollen legs). The interventricular septum bows leftward (seen on echocardiography), impairing left ventricular filling and dropping cardiac output, which worsens low blood pressure (hypotension) and tissue blood flow (perfusion).
8. Multi-organ failure and death occur without effective treatment. — Prolonged low oxygen in the blood (hypoxemia) ($\text{PaO}_2 < 55\text{--}60$ mmHg, $\text{SpO}_2 < 88$ percent for hours to days) deprives vital organs of oxygen. The kidneys suffer acute tubular tissue death (necrosis); serum creatinine rises above 1.5–2.0 mg/dL, urine output drops below 0.5 mL/kg/hour (oliguria), and dialysis may be required. The liver shows blood-starved (ischemic) hepatitis; AST and ALT climb into the hundreds or thousands. The brain develops oxygen-starved (hypoxic) encephalopathy; altered mental status, agitation, or coma appear. The heart suffers demand starved blood flow (ischemia) (oxygen supply does not meet need), and troponin may rise; arrhythmias (atrial fibrillation, ventricular a fast heart rate (tachycardia)) become common. Lactic acid accumulates (lactate $> 2\text{--}4$ mmol/L) as cells switch from aerobic to anaerobic metabolism, causing metabolic acidosis (acid building up in the blood). Impaired blood clotting (Coagulopathy) (abnormal clotting) develops from liver dysfunction and consumptive impaired blood clotting. Despite lung-protective ventilation (tidal volume 6 mL/kg predicted body weight, plateau pressure ≤ 30 cmH₂O), prone positioning (16 hours per day face-down to recruit dorsal alveoli), conservative fluid management (CVP target 4–8 mmHg, avoiding fluid overload), and treatment of the underlying cause (antibiotics for pneumonia or sepsis, source control for intra-abdominal infection), mortality in severe ARDS ($\text{P/F} \leq 100$) remains 40–46 percent. Rescue therapies—inhaled pulmonary vasodilators (nitric oxide, epoprostenol), neuromuscular blockade (cisatracurium for 48 hours), recruitment maneuvers, and extracorporeal membrane oxygenation (ECMO)—are reserved for refractory cases and may reduce mortality by 10–15 percent in carefully selected patients.

Symptoms · what the patient feels, and why

- Severe shortness of breath (dyspnea) that starts suddenly: The tiny air sacs in the lungs (alveoli) fill with fluid and collapse, so oxygen cannot pass from the air into the blood. The brain senses low oxygen and triggers a desperate feeling of 'I need more air,' making the person breathe harder and harder.
- Very fast breathing (tachypnea), often more than 30 breaths every minute: The breathing control center in the brainstem detects dangerously low oxygen and rising carbon dioxide in the blood. It speeds up breathing to try to pull in more oxygen and blow off carbon dioxide.
- Oxygen level stays dangerously low even when breathing 100% oxygen (refractory low oxygen in the blood (hypoxemia)): Blood flows past flooded or collapsed air sacs without picking up oxygen—like water flowing past a blocked sponge. Giving more oxygen does not help because the air sacs are not open to let the oxygen in.
- Neck and chest muscles working visibly hard with every breath (use of accessory muscles): The lungs are so stiff and heavy with fluid that the normal breathing muscles (the diaphragm and ribs) cannot inflate them alone. The body recruits extra muscles in the neck and between the ribs to help pull air in.
- Fast heart rate (tachycardia): The heart pumps faster to circulate the small amount of oxygen-rich blood to the brain, heart, and kidneys. It is trying to make up for the fact that each liter of blood is carrying less oxygen than normal.
- Blue or gray color of the lips, fingertips, or skin (cyanosis): When the oxygen level in the blood drops very low, hemoglobin (the red protein in red blood cells that carries oxygen) turns dark purple. This dark blood shows through thin skin as a blue or gray color.
- Wet crackling or popping sounds heard with a stethoscope over both lungs (diffuse crackles): Fluid fills the air sacs and small airways. As air moves in and out, it pops through the fluid—like blowing bubbles in water—creating crackling sounds that travel to the chest wall.
- Confusion, restlessness, or sleepiness (altered mental status): The brain needs a constant supply of oxygen to function. When oxygen delivery drops, brain cells cannot work properly, leading to confusion, agitation, or a drowsy, unresponsive state.
- Inability to speak a full sentence without gasping for air: Speaking requires steady, controlled breathing. When the lungs are failing and every breath is a struggle, the person cannot hold enough air to finish a sentence—they must stop mid-phrase to breathe.
- Sitting upright and leaning forward on the hands (tripod position): Gravity helps drain fluid away from the back of the lungs when sitting up. Leaning forward and bracing the arms locks the shoulder muscles, giving the chest muscles a stable base to pull harder and move more air.

Focused exam

- Refractory low oxygen in the blood (hypoxemia) (blood oxygen stays dangerously low) even when breathing 100% oxygen through a mask or ventilator: This is the signature of ARDS. The tiny air sacs (alveoli) in the lungs are flooded with fluid, so oxygen cannot jump across the barrier into the bloodstream no matter how much oxygen you pump in. It is like trying to breathe through a wet sponge. This tells you the problem is damage to the thin membrane between air and blood, not a weak heart or blocked airway.
- White patches on both sides of the chest X-ray or CT scan that are NOT caused by heart failure, extra body fluid, or a collapsed lung lobe: When you see cloudy white areas scattered across both lungs, it means the injury spread everywhere through the bloodstream and damaged the air-blood barrier in thousands of air sacs at once. This pattern—plus the timing and a clear trigger like pneumonia or sepsis—separates ARDS from fluid backup caused by a failing heart. This finding is required by the Berlin Definition (the official diagnosis checklist for ARDS).
- Stiff, hard-to-inflate lungs with a plateau pressure above 30 cm H₂O or a driving pressure above 15 cm H₂O when the patient is on a breathing machine: Fluid and collapsed air sacs make the lungs rigid, like trying to blow up a thick rubber balloon. High pressures mean the barrier is badly injured and predict that the ventilator itself could cause more harm by overstretching fragile air sacs (this is called ventilator-induced lung injury). Driving pressure is the single best number for predicting who will survive.
- Very fast breathing with the patient using neck and shoulder muscles to help breathe, plus visible exhaustion: The body tries to make up for flooded air sacs by breathing faster and harder to pull in more oxygen and blow off carbon dioxide. When the patient starts to tire out and can no longer keep up this effort, breathing failure is just around the corner and a breathing tube with a ventilator will be needed urgently.
- Pink foamy fluid coughed up or suctioned from the breathing tube: This fluid is packed with protein and has leaked across the damaged barrier from blood vessels into the air sacs and is now spilling into the larger airways. It confirms that capillaries are leaking badly and that air sacs are severely flooded.
- Symptoms started within the past 7 days after a known injury or illness: pneumonia, throwing up and breathing it in (aspiration), bloodstream infection (sepsis), major trauma, pancreatitis, or receiving many units of blood quickly (massive transfusion): ARDS always has a trigger—something that injured the air-blood barrier either by directly hitting the lungs (like pneumonia or inhaling vomit) or by releasing inflammatory chemicals into the bloodstream that travel to the lungs (like sepsis or pancreatitis). Finding the trigger guides treatment of the root cause and warns you to watch for other organs failing at the same time.

Diagnostics · every test answers a question

- Chest X-ray (portable bedside film) or computed tomography (CT) scan of the chest → Are there white patches (opacities) on both sides of the lungs that match fluid-filled air sacs and cannot be fully explained by heart failure, fluid around the lungs (pleural effusions), or a collapsed lung lobe?
- Arterial blood gas (ABG) with calculation of the $\text{PaO}_2/\text{FiO}_2$ ratio (P/F ratio) → How bad is the oxygen problem, and is the air-blood barrier so damaged that oxygen cannot cross into the blood even with high oxygen delivery?
- Echocardiography (ultrasound of the heart) or blood test for brain natriuretic peptide (BNP) → Is the lung fluid caused by a weak or overloaded left side of the heart (cardiogenic pulmonary edema (fluid flooding the lungs)) or by damage to the air-blood barrier (ARDS)?
- Complete blood count (CBC) with differential, blood cultures, procalcitonin, and coughed-up mucus (sputum) or bronchoalveolar lavage (BAL) fluid cultures → What is causing ARDS, and is there an active infection that keeps injuring the lungs?
- Plateau pressure, driving pressure, and static compliance measured on the ventilator during a brief breath hold → Are the ventilator settings safe, or are high pressures overstretching already-damaged air sacs and causing more injury?
- Lactate level in arterial or venous blood → Are the tissues getting enough oxygen and blood flow, or is the body shifting to survival mode because of severe illness?
- Basic metabolic panel (BMP) or comprehensive metabolic panel (CMP) including creatinine and electrolytes → Are the kidneys working, and are electrolyte levels safe during fluid management and mechanical ventilation?
- D-dimer and, if elevated, CT pulmonary angiography (CTPA) to rule out pulmonary embolism (a clot lodging in a lung artery) (blood clot in lung arteries) → Could a blood clot blocking the lung arteries be mimicking or worsening ARDS?

Treatment logic

- Low tidal volume ventilation: deliver 4 to 8 mL per kilogram of ideal body weight (usually 6 mL/kg), keep plateau pressure below 30 cm H₂O, and keep driving pressure below 15 cm H₂O → This prevents the ventilator from overstretching fragile air sacs and causing more injury (ventilator-induced lung injury or VILI). Smaller, gentler breaths allow the damaged barrier to heal without adding mechanical trauma. The landmark ARDSNet trial showed this strategy cuts the risk of death by 9 percentage points compared to larger breaths.

- Positive end-expiratory pressure (PEEP): apply 8 to 15 cm H₂O (or higher if needed) to keep air sacs open at the end of each breath, adjusted based on oxygen levels, lung stiffness, and blood pressure tolerance → PEEP stops air sacs from collapsing completely when you breathe out (end-expiratory collapse or atelectasis). It reopens (recruits) collapsed air sacs and matches air flow with blood flow so oxygen can cross into the bloodstream more efficiently. Higher PEEP may let you lower the oxygen percentage (FiO₂) and reduce oxygen toxicity.
- Find and treat the root cause: give antibiotics for pneumonia (following local resistance patterns and IDSA/ATS pneumonia guidelines), drain infected fluid or remove infected tissue for sepsis (source control), prevent aspiration by elevating the head of the bed and using feeding tubes carefully, and stop any drug causing lung injury → Stopping the trigger halts ongoing damage to the air-blood barrier. Without eliminating the cause—whether it is bacteria, inhaled vomit, or inflammatory chemicals from pancreatitis—the barrier keeps leaking and ARDS gets worse no matter what the ventilator does.
- Prone positioning (flip the patient face-down) for 16 hours per day when ARDS is moderate to severe (P/F ratio below 150 mmHg) → Lying face-down takes weight off the back parts of the lungs (which collapse under the heart and belly when lying face-up) and reopens those air sacs. It balances air flow and blood flow better and improves oxygen levels. The PROSEVA trial showed that prone positioning cuts the risk of death in half in severe ARDS.
- Conservative fluid strategy once blood pressure and organ blood flow are stable: give only enough IV fluid to keep organs working, avoid overloading the patient, and use diuretics (water pills) cautiously if the patient is fluid-overloaded → Less fluid in the bloodstream lowers the pressure inside capillaries (hydrostatic pressure) that pushes fluid into air sacs. It helps the body reabsorb fluid from the lungs and shortens the time on the ventilator. The FACTT trial showed fewer ventilator days without causing more shock or kidney failure.
- Neuromuscular blockade with a continuous cisatracurium infusion for 48 hours in severe ARDS (P/F ratio below 150 mmHg) if the patient fights the ventilator despite deep sedation → Paralyzing the breathing muscles stops the patient from fighting the ventilator (patient-ventilator dyssynchrony), lowers oxygen use, decreases release of inflammatory chemicals (cytokines), and may improve oxygen levels. The ACURASYS trial suggested a survival benefit, but the later ROSE trial (using lighter sedation) showed no benefit. Use this selectively in severe cases.

- Extracorporeal membrane oxygenation (ECMO): remove blood from a large vein, run it through a machine that adds oxygen and removes carbon dioxide, then return it to the body—used for refractory low oxygen in the blood (hypoxemia) (P/F ratio below 80 mmHg on optimized settings) that does not respond to prone positioning and other rescue measures → ECMO bypasses the injured lungs completely, giving them a rest by taking over the job of adding oxygen to the blood. It allows ultra-gentle ventilator settings (very small breaths and low pressures) or even no ventilation at all, giving the barrier time to heal. It buys time for recovery.

Medications

Low tidal volume ventilation (6 mL/kg ideal body weight, plateau pressure < 30 cm H₂O, driving pressure < 15 cm H₂O) · Lung-protective mechanical ventilation strategy for ARDS (a severe lung injury where fluid leaks into the tiny air sacs, blocking oxygen from reaching the blood)

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.

1. Set the ventilator to deliver 6 mL per kilogram of ideal body weight each breath—this limits alveolar stretch and stops the release of cytokines (inflammation chemicals that make the injury spread like wildfire).
2. Keep plateau pressure (the pressure when the lung is fully inflated and holding still) below 30 cm H₂O to prevent barotrauma (lung rupture from too much pressure, like a tire blowout).

3. Keep driving pressure (plateau minus PEEP, the actual pressure change the lung feels with each breath) below 15 cm H₂O—studies show this number predicts survival better than tidal volume alone because it reflects how stiff or compliant the lung is.
4. Adjust PEEP using a standard table (matching oxygen need to PEEP level) or by measuring esophageal pressure (a balloon in the food tube estimates chest-wall squeeze) to balance opening collapsed alveoli against overstretching healthy ones.
5. Once blood pressure and organ function are stable, restrict IV fluids to net-even or slightly negative daily balance—this lowers hydrostatic pressure (the water-pushing force) across leaky capillaries so less fluid seeps into alveoli.
6. Start prone positioning if the PaO₂/FiO₂ ratio (a number that compares arterial oxygen to the oxygen percentage we deliver) is below 150 mmHg (severe low oxygen in the blood (hypoxemia)); keep the patient face-down for at least 16 consecutive hours each session to see benefit.
7. Consider a 48-hour continuous infusion of cisatracurium (a paralyzing drug) if PaO₂/FiO₂ stays below 150 despite optimized settings—paralysis stops the patient from breathing against the ventilator, cuts oxygen consumption by resting muscles, and may reduce lung injury from dyssynchrony (patient and machine fighting each other).
8. Evaluate for venovenous ECMO if PaO₂/FiO₂ remains below 80 mmHg on maximal support, or if pH drops below 7.25 from CO₂ buildup (hypercapnia) even when we accept higher CO₂ to protect the lungs—ECMO should only be done at specialized centers with experience.
9. Give dexamethasone 6 mg (IV or by mouth) once daily for up to 10 days in COVID-19 ARDS needing supplemental oxygen—this is recommended by NIH and WHO because it reduces death by calming the immune overreaction that damages lungs.
10. Identify and treat the trigger: start broad-spectrum antibiotics immediately for sepsis-related ARDS, perform emergency surgery to drain abscesses or remove infected tissue, use diuretics (furosemide) if heart failure caused fluid overload in the lungs.

SAFETY WATCH

Low blood pressure (Hypotension) (low blood pressure): high PEEP squeezes the heart and big veins, reducing the amount of blood returning to the heart—manage with IV fluids or vasopressors (drugs like norepinephrine that tighten blood vessels to raise pressure). · Barotrauma (pneumothorax, air escaping into the chest cavity): happens if plateau pressure goes above 30 cm H₂O—the lung pops like an overfilled water balloon, causing sudden oxygen drop and requiring a chest tube to drain the trapped air. · Hypercapnia (CO₂ builds up in the blood): we allow this down to a pH of 7.20–7.25 to keep tidal volumes low, but avoid it in patients with increased intracranial pressure (brain swelling) because high CO₂ dilates brain blood vessels and worsens swelling. · Prone positioning risks: pressure ulcers (skin breakdown on the face, chest, hips, and knees from lying face-down), accidental extubation (breathing tube pulled out during turning), brief oxygen drops during the turn—requires a trained team of at least four people and careful padding. · Neuromuscular blockade: prolonged muscle weakness (ICU-acquired weakness) if used longer than 48 hours or combined with steroids—always use train-of-four monitoring (a nerve stimulator checks how paralyzed the patient is by counting muscle twitches) to use the lowest effective dose. · ECMO complications: bleeding (blood thinners are needed to prevent clots in the tubing), limb starved blood flow (ischemia) (the leg where the catheter sits may not get enough blood), infection at catheter sites, red blood cell destruction (hemolysis) (red blood cells break apart in the machine), and stroke—only pursue at high-volume centers. · Corticosteroids (dexamethasone): high blood sugar (hyperglycemia) (blood sugar spikes, requiring insulin), secondary infections (bacteria like *Pseudomonas* or fungi like *Candida* take advantage of immune suppression), stomach ulcers and bleeding—use only in COVID-19 ARDS or in research protocols for other causes.

Oxygen therapy (nasal cannula, high-flow nasal cannula, non-rebreather mask, mechanical ventilation) · Supportive oxygen delivery to correct low oxygen in the blood (hypoxemia) (dangerously low oxygen in the blood)

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.

1. Start with low-flow nasal cannula (1–6 L/min) if the patient is alert, breathing comfortably, and SpO₂ can be maintained $\geq 90\%$ on room air with minimal support.
2. Escalate to high-flow nasal cannula (HFNC) up to 60 L/min at FiO₂ 40–100% if the patient needs more oxygen or is working hard to breathe—HFNC delivers heated, humidified gas that is more comfortable, clears CO₂ from the nasal cavity, and creates 3–5 cm H₂O of positive pressure to splint open airways.
3. Use a non-rebreather mask with reservoir bag at 10–15 L/min if SpO₂ remains low and HFNC is not available—this delivers near 100% oxygen by preventing exhaled air from mixing back into the next breath.
4. Intubate and start mechanical ventilation if SpO₂ stays below 88% despite maximal non-invasive oxygen, if the patient shows signs of exhaustion (rising CO₂, altered mental status, use of neck and belly muscles to breathe), or if airway protection is needed (patient cannot cough or swallow safely).
5. Target SpO₂ 88–95% (not higher) in ARDS—hyperoxia (too much oxygen) generates free radicals that damage cells and does not improve survival; the ARDS network and international guidelines recommend this range.
6. Monitor arterial blood gases every 4–6 hours in intubated patients to track PaO₂, PaCO₂, and pH—these numbers guide ventilator adjustments and tell you if the patient is improving or worsening.

SAFETY WATCH

Oxygen toxicity: prolonged exposure to FiO₂ > 60% (especially over 24–48 hours) damages alveolar cells by generating reactive oxygen species (free radicals)—use the lowest FiO₂ that keeps SpO₂ $\geq 88\%$. · CO₂ retention in chronic CO₂ retainers (patients with severe COPD): high oxygen can blunt their drive to breathe—target SpO₂ 88–92% in these patients, not higher. · Nasal dryness and a nosebleed (epistaxis) (nosebleed) with low-flow oxygen: humidify gas if flow is ≥ 4 L/min. · Aspiration risk with non-rebreather masks: if the patient vomits, the mask can trap vomit—remove immediately and consider intubation if mental status is impaired. · Delayed intubation: waiting too long to intubate a tiring patient can lead to cardiac arrest during the procedure—watch for rising heart rate, falling blood pressure, confusion, or inability to speak in full sentences.

Fluid management (restrictive strategy after resuscitation) · Intravenous fluid administration guided by hemodynamic monitoring to balance organ blood flow (perfusion) and pulmonary edema (fluid flooding the lungs)

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.

1. During initial resuscitation (first 6–12 hours), give IV crystalloid (normal saline or lactated Ringer's) in 500 mL boluses targeting mean arterial pressure $\geq$ 65 mmHg, urine output $\geq$ 0.5 mL/kg/hour, and lactate clearance—this restores blood flow (perfusion) to organs and prevents acute kidney injury and shock.
2. Once hemodynamically stable (stable blood pressure, adequate organ blood flow (perfusion), no ongoing losses), stop maintenance IV fluids or reduce them to the minimum needed for medication delivery.
3. Monitor daily fluid balance (total input minus total output over 24 hours); aim for net-even or net-negative balance (e.g., -500 to -1000 mL per day).
4. Use diuretics (furosemide 20–40 mg IV, titrate up if needed) to promote negative balance if the patient is volume-overloaded (edema, rising weight, worsening oxygenation)—ensure adequate intravascular volume first to avoid low blood pressure (hypotension) and kidney injury.
5. Measure central venous pressure (CVP) or use dynamic assessments (passive leg raise, stroke volume variation on the ventilator) to guide fluid administration in complex cases—goal is the lowest filling pressure that maintains organ blood flow (perfusion).
6. Track daily weight, lung compliance (how stiff the lungs are), and oxygenation ($\text{PaO}_2/\text{FiO}_2$ ratio)—improvement in these signals that the restrictive strategy is working.

SAFETY WATCH

Low blood pressure (Hypotension) and shock: aggressive diuresis without confirming adequate intravascular volume can drop blood pressure and reduce blood flow to kidneys, brain, and gut—always assess volume status before giving diuretics. · Acute kidney injury: restrictive fluids plus diuretics in a hypovolemic (low blood volume) patient can cause prerenal waste buildup in the blood (azotemia) (kidney function drops because not enough blood reaches the kidneys)—monitor creatinine and urine output closely. · Electrolyte imbalances: diuretics cause loss of potassium, magnesium, sodium, and chloride—check labs daily and replace as needed (oral or IV potassium chloride, magnesium sulfate). · Hypovolemia masking sepsis: if the patient becomes with low blood pressure (hypotensive) after fluid restriction, consider new infection, bleeding, or adrenal insufficiency—do not assume it is always from 'too dry'; reassess and give fluids if needed.

Antibiotics (empiric broad-spectrum, then targeted) · Antimicrobial agents to treat bacterial pneumonia or sepsis triggering ARDS

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.

1. Draw blood cultures (two sets from different sites) and respiratory cultures (sputum, endotracheal aspirate, or bronchoalveolar lavage) before giving the first antibiotic dose—but do not delay antibiotics to get cultures.
2. Start empiric broad-spectrum antibiotics within 1 hour of recognizing sepsis or septic shock (Surviving Sepsis Campaign guideline)—typical regimens for community-acquired pneumonia with ARDS: ceftriaxone 1–2 g IV daily plus azithromycin 500 mg IV daily (or doxycycline 100 mg IV twice daily); for hospital-acquired or ventilator-associated pneumonia: piperacillin-tazobactam 4.5 g IV every 6 hours (or meropenem 1 g IV every 8 hours) plus vancomycin 15–20 mg/kg IV every 8–12 hours (targeting MRSA).
3. Add anaerobic coverage (metronidazole 500 mg IV every 8 hours or already covered by piperacillin-tazobactam) if aspiration pneumonia is suspected (patient found down, vomiting, altered mental status).

4. Add antifungal therapy (e.g., fluconazole or an echinocandin like micafungin) if the patient is immunocompromised (chemotherapy, HIV, chronic steroids) or has risk factors for invasive candidiasis.
5. Reassess at 48–72 hours: review culture results and sensitivities, narrow antibiotics to the most specific effective agent (de-escalation), and stop antibiotics if cultures are negative and clinical suspicion for infection is low.
6. Typical duration: 7–10 days for uncomplicated pneumonia, longer (14–21 days) for complicated cases (empyema, abscess, bacteremia with *Staph aureus* or *Pseudomonas*).

SAFETY WATCH

Allergic reactions: penicillin and cephalosporin allergies are common—ask about rash, hives, or anaphylaxis; if true allergy, use aztreonam (gram-negative coverage) or a fluoroquinolone instead. · Clostridioides difficile infection: broad-spectrum antibiotics kill normal gut bacteria, allowing *C. diff* to overgrow and cause severe diarrhea, colitis, and toxic megacolon—use antibiotics only when needed, stop as soon as safe, and treat *C. diff* with oral vancomycin or fidaxomicin if it develops. · Acute kidney injury: vancomycin, aminoglycosides (gentamicin, tobramycin), and some beta-lactams are nephrotoxic—monitor creatinine daily, dose-adjust for kidney function, and check vancomycin trough levels to avoid toxicity. · QT prolongation: fluoroquinolones (levofloxacin, moxifloxacin) and azithromycin can lengthen the QT interval on ECG, risking torsades de pointes (a deadly arrhythmia)—avoid in patients with baseline long QT or on other QT-prolonging drugs. · Drug-drug interactions: many antibiotics interact with anticoagulants, antiepileptics, and immunosuppressants—check interactions and adjust doses accordingly. · Resistance emergence: overuse or underdosing breeds resistant bacteria (MRSA, ESBL-producing Enterobacteriaceae, carbapenem-resistant organisms)—always use the narrowest spectrum and shortest duration that is effective.

Dexamethasone (for COVID-19 ARDS) · Corticosteroid (a synthetic hormone that suppresses inflammation and immune overreaction)

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.

1. Confirm the patient has COVID-19 (positive PCR or antigen test) AND requires supplemental oxygen ($\text{SpO}_2 < 94\%$ on room air, or already on nasal cannula, high-flow, non-rebreather, or mechanical ventilation).
2. Give dexamethasone 6 mg once daily, either IV (preferred in ICU) or by mouth (if patient can swallow)—dose is the same regardless of weight.
3. Continue for up to 10 days or until hospital discharge, whichever comes first—stopping early may allow rebound inflammation.
4. Do NOT give dexamethasone to COVID-19 patients who do not need oxygen—the RECOVERY trial showed no benefit and possible harm in this group.
5. Monitor blood glucose closely (check fingerstick glucose every 6 hours) and start insulin if glucose rises above 180 mg/dL—steroids cause insulin resistance and high blood sugar (hyperglycemia).
6. Watch for secondary infections (bacterial pneumonia, fungal infections like invasive aspergillosis or candidiasis)—dexamethasone suppresses immune defenses, so maintain a high index of suspicion.

SAFETY WATCH

High blood sugar (Hyperglycemia) (high blood sugar): steroids make cells resistant to insulin, so glucose spikes—monitor fingerstick glucose every 6 hours and use sliding-scale or basal-bolus insulin to keep glucose 140–180 mg/dL. · Secondary infections: dexamethasone suppresses the immune system, raising risk of bacterial pneumonia (especially *Staph aureus*, *Pseudomonas*), invasive fungal infections (*Aspergillus*, *Candida*, *Mucorales*), and reactivation of latent TB or strongyloidiasis—watch for new fever, worsening infiltrates, or rising inflammatory markers and treat promptly. · Gastrointestinal bleeding: steroids increase stomach acid and reduce protective mucus—consider proton pump inhibitor (omeprazole 20–40 mg daily) in high-risk patients (history of ulcers, concurrent NSAIDs or anticoagulation). · Psychiatric effects: agitation, confusion, mania, or psychosis can occur, especially at higher doses or in patients with preexisting mental illness—reassure, consider short-term antipsychotic if severe, and taper steroids as soon as clinically appropriate. · Avascular tissue death (necrosis) (bone death, especially hip or shoulder): rare with short courses (10 days) but possible with prolonged or repeated steroid exposure—counsel patients to report new joint pain after discharge.

Neuromuscular blocking agents (cisatracurium, for severe ARDS) · Non-depolarizing neuromuscular blocker (paralyzes skeletal muscles by blocking the signal from nerve to muscle)

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.

1. Confirm severe ARDS: $\text{PaO}_2/\text{FiO}_2$ ratio < 150 mmHg (or < 120 in some protocols) within 48 hours of ARDS onset, already on lung-protective ventilation with optimized PEEP.
2. Ensure the patient is deeply sedated (Richmond Agitation-Sedation Scale -4 or -5 , meaning unresponsive to voice) with continuous infusions of sedatives (propofol or midazolam) and analgesics (fentanyl)—never paralyze an awake or inadequately sedated patient.
3. Start cisatracurium infusion at 15 mg/hour (or 0.15 mg/kg/hour) after a loading bolus of 0.2 mg/kg IV over 1–2 minutes.
4. Continue the infusion for 48 hours, then stop—do NOT extend beyond 48 hours unless absolutely necessary (refractory low oxygen in the blood (hypoxemia) despite all other interventions), as prolonged paralysis increases ICU-acquired weakness.
5. Monitor depth of paralysis using train-of-four (TOF) nerve stimulation every 4–6 hours: stimulate the ulnar nerve at the wrist and count thumb twitches—target 1–2 twitches out of 4, meaning partial but adequate blockade.
6. Ensure eye protection (artificial tears, taping eyelids closed) and frequent repositioning to prevent pressure ulcers, because the patient cannot blink or move.
7. Once the 48-hour infusion ends, allow paralysis to wear off (usually 30–60 minutes for cisatracurium, which is metabolized by plasma esterases and does not accumulate in kidney or liver failure), then resume spontaneous breathing trials or continue sedation as needed.

SAFETY WATCH

ICU-acquired weakness (critical illness myopathy or neuropathy): prolonged paralysis (especially beyond 48 hours) or combination with corticosteroids dramatically increases the risk of severe, persistent muscle weakness that can last months—some patients never fully recover strength; use the shortest duration possible. · Awareness under paralysis (accidental awareness): if sedation is insufficient, the patient is awake, aware, and unable to move or communicate—terrifying and traumatic; always confirm deep sedation (RASS –4 or –5, no response to voice or tactile stimulation) before and during paralysis, and use sedation scales and BIS (bispectral index) monitoring if available. · Prolonged paralysis after stopping: cisatracurium is usually cleared quickly, but in rare cases (renal failure with other non-depolarizing blockers, hypothermia, too much acid in the blood (acidosis)), paralysis can last hours—monitor TOF and support ventilation until recovery. · Pressure ulcers and corneal abrasions: the patient cannot shift weight or blink—reposition every 2 hours, use pressure-relief mattresses, apply artificial tears and tape eyelids shut. · Mask deterioration: because the patient cannot cough, trigger alarms, or show respiratory distress, close monitoring (continuous pulse oximetry, frequent arterial blood gases, ventilator alarms) is essential—any change in oxygenation or ventilator pressures must be investigated immediately (pneumothorax, mucus plug, ventilator malfunction). · Anaphylaxis: rare but possible with any neuromuscular blocker—presents as low blood pressure (hypotension), bronchospasm, rash; treat with epinephrine, fluids, and stop the drug.

Red flags

- 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)

Nursing priorities

1. Monitor oxygen saturation continuously with pulse oximetry and keep SpO₂ above 88%; escalate immediately if saturation stays below 88% despite oxygen therapy — The damaged alveolar-capillary membrane (the wall between air sacs and blood vessels) blocks oxygen transfer, so even small drops in saturation mean the brain and heart are not getting enough oxygen to survive.
2. 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 — Gravity redistributes blood flow to healthier parts of the lungs when prone, and the weight of the heart no longer compresses the back lung tissue, which improves oxygen levels by 60 to 80% in many ARDS patients.
3. 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 — Extra fluid leaks through the damaged alveolar-capillary barrier into the air sacs, worsening low oxygen in the blood (hypoxemia) (low blood oxygen) and making the lungs stiffer and harder to ventilate.
4. 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 — Large tidal volumes and high pressures stretch already-injured alveoli (air sacs), triggering more inflammation and causing ventilator-induced lung injury, which increases the risk of death by 20 to 30%.
5. 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 — Disconnecting the ventilator circuit or prolonged suctioning removes PEEP (positive end-expiratory pressure), so alveoli collapse instantly and oxygen levels can drop dangerously low within seconds.
6. 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) — An upright position uses gravity to keep the diaphragm (breathing muscle) lower in the chest, giving the lungs more space to expand and reducing the risk of ventilator-associated pneumonia by 40%.
7. Perform mouth care every 2 to 4 hours with chlorhexidine (an antiseptic rinse) and brush the teeth twice daily — Bacteria from the mouth migrate down the endotracheal tube into the injured lungs, and patients with ARDS already have damaged immune defenses in the alveoli, so infection can turn deadly within 24 to 48 hours.
8. 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 — Deep sedation paralyzes the diaphragm's natural movement, allows muscle wasting, and prolongs time on the ventilator; lighter sedation lets patients breathe with the ventilator and shortens ICU stay by 2 to 4 days.

Sources

- 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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