PULMONARY · CRITICAL CARE · SYSTEM FAILURE

Acute Respiratory Distress Syndrome

Sudden, life-threatening, non-cardiogenic respiratory failure marked by refractory hypoxemia and bilateral pulmonary infiltrates.

NGN NCLEX 2026 | High-Yield · Priority Topic | Mortality 30-45% | ICU · Mechanical Ventilation

SOURCE NOTE: This topic is not present in the uploaded reference notes. Content compiled from: *Saunders Comprehensive Review for the NCLEX-RN (2026)*, *Lippincott Manual of Nursing Practice*, the *Berlin Definition of ARDS (2012/2023 update)*, and the *ARDSnet ARMA Protocol* (NHLBI/NIH). Lung-protective ventilation evidence per *NEJM 2000* landmark trial.

1 Definition & Overview

ARDS is an **acute, diffuse, inflammatory lung injury** that develops within one week of a known clinical insult. It is the most severe form of acute respiratory failure.

- **Non-cardiogenic pulmonary edema** — fluid floods the alveoli but the heart is *not* the cause
- **Refractory hypoxemia** — low PaO_2 that does NOT improve with supplemental oxygen (the defining feature)
- **Bilateral "white-out"** on chest imaging — ground-glass / patchy infiltrates not explained by effusion, collapse, or nodules
- Mortality remains high (30–45%); survivors often have lasting pulmonary, cognitive, and functional impairment

> **Why it matters:** ARDS is a *syndrome*, not a disease — it is the lung's final common pathway for systemic injury. Recognizing early hypoxemia and the failure of O_2 to fix it is the nurse's first save.

2 Pathophysiology

Triggers (Direct vs Indirect lung injury):

Direct Injury

- Pneumonia (viral/bacterial)
- Aspiration of gastric contents
- Inhalation injury (smoke, toxic gas)
- Near-drowning
- Pulmonary contusion
- Fat or amniotic embolism

Indirect Injury

- **Sepsis** (most common cause)
- Severe trauma / shock
- Massive transfusion (TRALI)
- Pancreatitis
- Drug overdose
- DIC, burns

The cascade:

1. Insult triggers systemic inflammatory response → neutrophils & cytokines flood pulmonary capillaries
2. Alveolar-capillary membrane becomes **leaky** — protein-rich fluid pours into alveoli
3. Surfactant is inactivated → alveoli collapse (atelectasis)
4. **Hyaline membranes** form; lung becomes stiff (↓ compliance)
5. Severe **V/Q mismatch & intrapulmonary shunting** → blood passes unoxygenated alveoli
6. **Refractory hypoxemia** — supplemental O_2 no longer raises PaO_2

Three pathologic phases:

Exudative

Days 1–7

Edema, hyaline membranes, severe hypoxemia. Patient most unstable here.

Proliferative

Weeks 1–3

Type II pneumocytes regenerate; fibroblasts begin to lay collagen.

Fibrotic

After Week 3

Fibrosis stiffens lungs; some patients never fully recover compliance.

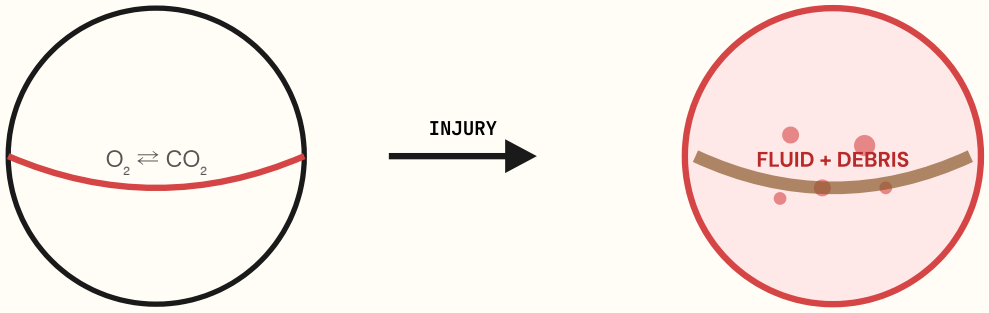

FIGURE 1 · ALVEOLAR DAMAGE IN ARDS

3 Signs & Symptoms

Early (within hours)

- **Restlessness, anxiety** — earliest hypoxia cue
- Dyspnea, tachypnea (RR > 20)
- Tachycardia
- Dry cough, fever
- Mild hypoxemia on room air
- ABG: **respiratory alkalosis** (blowing off CO_2)
- Crackles on auscultation

Late (12–48 hours)

- Severe dyspnea, accessory-muscle use
- Intercostal & substernal retractions
- Cyanosis, pallor, diaphoresis
- Coarse crackles, rhonchi throughout
- **Refractory hypoxemia** — PaO_2 won't rise with O_2
- ABG: **respiratory acidosis** (fatigue, CO_2 retention)
- ↓ LOC → coma → multi-organ failure

HALLMARK RED FLAG

Refractory hypoxemia — PaO_2 remains < 60 mmHg despite FiO_2 ≥ 60%. This is THE diagnostic clue. If the SpO_2 won't climb no matter how much O_2 you turn up — think ARDS.

EARLY WARNING

Restlessness in a patient with sepsis, trauma, pneumonia, or aspiration is hypoxia until proven otherwise. Do NOT sedate first — oxygenate first.

4 Diagnostic Criteria & Workup

Berlin Definition — All Four Required

- **Timing:** within 1 week of insult or new/worsening respiratory symptoms
- **Imaging:** bilateral opacities on CXR or CT — not explained by effusion, collapse, or nodules
- **Origin:** respiratory failure NOT fully explained by cardiac failure or fluid overload (use echo to rule out)
- **Oxygenation:** P/F ratio (PaO_2 / FiO_2) classifies severity on PEEP ≥ 5 cm H_2O

MILD	MODERATE	SEVERE
200–300	100–200	≤ 100

Other key diagnostics:

- **CXR:** bilateral "ground-glass" or "white-out" pattern (no cardiomegaly)
- **ABG:** early respiratory alkalosis → late respiratory acidosis with severe hypoxemia
- **BNP:** normal/low (rules out cardiogenic edema — BNP would be high in CHF)
- **Echocardiogram:** normal ejection fraction (confirms non-cardiogenic origin)
- **Sputum/blood cultures:** identify underlying infection

5 Priority Nursing Interventions

Use the **ABC framework** — airway/oxygenation is always first.

PRIORITY	INTERVENTION	RATIONALE
A — Airway	Prepare for intubation & mechanical ventilation	Refractory hypoxemia rarely responds to non-invasive O_2; early intubation prevents arrest
B — Breathing	Apply **lung-protective ventilation**: low tidal volume 4–6 mL/kg ideal body weight; plateau pressure ≤ 30 cm H_2O	Prevents volutrauma & barotrauma in already-damaged alveoli
B — Breathing	Titrate **PEEP** (5–20 cm H_2O) per protocol	Keeps alveoli open at end-expiration; improves oxygenation & reduces shunt
B — Position	**Prone positioning** 12–16 hr/day for moderate-severe ARDS (P/F < 150)	Redistributes perfusion to less-injured lung zones; ↓ mortality (PROSEVA trial)
C — Circulation	Conservative IV fluid management; monitor CVP, MAP, urine output	Excess fluid worsens pulmonary edema; balance against MAP for organ perfusion
Monitor	Continuous SpO_2, $ETCO_2$, ABG q4–6h, hemodynamics	Detect deterioration & guide ventilator titration
Prevent	VAP bundle: HOB 30°, oral chlorhexidine q4h, daily sedation interruption, DVT & stress-ulcer prophylaxis	Ventilator-associated pneumonia adds mortality on top of ARDS
Suction	Closed-system suction only when needed; pre-oxygenate with 100% O_2	Open suction loses PEEP and drops SpO_2 rapidly
Support	Enteral nutrition within 24–48 hr; sedation/analgesia per protocol	Maintains gut integrity, prevents muscle wasting, controls dyssynchrony

☆ NCJMM PEARL

If SpO_2 drops during prone positioning, **first action is to verify ETT placement and ensure all lines are patent**, then return to supine if instability continues. Do not delay calling the provider for sustained desaturation.

6 Pharmacology

DRUG / CLASS	MECHANISM	NURSING CONSIDERATIONS
Propofol (sedative)	GABA agonist; rapid sedation	Monitor BP (↓), triglycerides, lipase, amylase. Watch for **propofol infusion syndrome** (metabolic acidosis, rhabdo)
Midazolam (Versed) (benzodiazepine)	GABA-mediated sedation	Monitor LOC, respiratory depression. **Antidote: flumazenil**
Fentanyl (opioid analgesic)	μ-receptor agonist	Watch for respiratory depression, hypotension, ileus. **Antidote: naloxone**
Cisatracurium (Nimbex) (neuromuscular blocker)	Non-depolarizing paralytic at NMJ	**Patient must be sedated AND have analgesia first** — paralytics do not relieve pain or awareness. Monitor train-of-four. Use for severe ARDS only (P/F < 150)
Furosemide (Lasix) (loop diuretic)	Inhibits Na/K/2Cl in loop of Henle	Conservative fluid strategy. Monitor K^+ (hypokalemia), BUN, creatinine, daily weights
Norepinephrine (vasopressor)	α_1 agonist → vasoconstriction	Used if sepsis-induced hypotension. Central line preferred. Titrate to MAP ≥ 65 mmHg
Methylprednisolone (corticosteroid)	Anti-inflammatory	Selective use; monitor glucose, infection, GI bleeding. *Dexamethasone* if ARDS from COVID-19
Heparin SQ / Enoxaparin	Anticoagulant — DVT prophylaxis	Monitor aPTT (heparin) or anti-Xa. Bleeding precautions. **Antidote: protamine sulfate**
Pantoprazole / Famotidine	PPI / H_2 blocker	Stress-ulcer prophylaxis — critically ill ventilated patients are high-risk for GI bleed
Broad-spectrum antibiotics	Per culture / suspected source	Sepsis is the #1 trigger — give within **1 hour** of suspected sepsis (Surviving Sepsis bundle)

7 NCLEX Test Traps

~~WRONG: "Increase the FiO_2 to 100% — that will fix the hypoxemia"~~

RIGHT: Recognize refractory hypoxemia and anticipate intubation + PEEP

The defining feature of ARDS is that PaO_2 does **not** respond to supplemental oxygen. More O_2 alone is the wrong answer — PEEP and lung-protective ventilation are required.

~~WRONG: "Bilateral crackles + dyspnea = give furosemide, this is CHF"~~

RIGHT: Check BNP and echo first — ARDS is non-cardiogenic

In ARDS the BNP is normal/low and ejection fraction is preserved. Aggressive diuresis without confirming cardiogenic origin can drop MAP and worsen organ perfusion.

~~WRONG: "Give high tidal volumes (10-12 mL/kg) to push past the stiff lungs"~~

RIGHT: Low tidal volume 4-6 mL/kg of ideal body weight

High tidal volumes cause volutrauma in already-injured alveoli and increase mortality. Lung-protective ventilation is the standard of care.

~~WRONG: "The paralyzed patient on cisatracurium looks calm — no sedation needed"~~

RIGHT: Paralytics do NOT sedate or relieve pain — maintain analgesia + sedation continuously

A paralyzed but conscious patient experiences awareness and pain. This is a tested NCLEX trap. Always pair NMB with sedation and analgesia.

~~WRONG: "Restlessness in a post-op sepsis patient — give a benzo first"~~

RIGHT: Restlessness is hypoxia until proven otherwise — assess SpO_2, RR, breath sounds first

Early ARDS presents as restlessness and anxiety from hypoxemia. Sedating before oxygenating can mask deterioration and kill the patient.

~~WRONG: "Bolus 2 L NS — the patient is hypotensive"~~

RIGHT: Conservative fluid management; consider vasopressors after appropriate resuscitation

Excess fluid floods already-leaky alveoli. In ARDS, target euvolemia and use vasopressors (norepinephrine) for shock instead of aggressive crystalloid bolusing once initial resuscitation is complete.

8 Patient & Family Education

Most education occurs *during* the ICU stay (family) and *after* recovery (patient):

Family — ICU Phase

- Explain ventilator, sedation, restraints, alarms
- Encourage presence at bedside; talk to the patient
- ARDS recovery is **slow** — weeks to months
- Outcomes vary: some recover fully, others have lasting impairment
- Encourage rest, support resources, social work consult

Patient — Recovery Phase

- Pulmonary rehabilitation: graded exercise, breathing exercises
- Smoking cessation absolutely required
- Avoid respiratory irritants (smoke, dust, chemicals)
- Get annual flu vaccine + pneumococcal vaccine
- Recognize early infection: fever, cough, dyspnea → call provider
- Mental health: post-ICU PTSD, depression, "ICU brain" are common
- Nutrition, hydration, paced activity

☆ TEACH-BACK FOCUS

Patients and families often underestimate the **cognitive and emotional recovery** after ARDS. Validate that fatigue, memory lapses, and anxiety after discharge are real and expected, not failures.

9 Memory Boosters & Mnemonics

A · R · D · S

A **Assault** to the lung — sepsis, aspiration, trauma, transfusion

R **Refractory** hypoxemia — O_2 won't fix the PaO_2

D **Decreased** compliance — stiff, white-out lungs on CXR

S **Severe** — high mortality without lung-protective ventilation

P · A · L · E

P **PEEP** — positive end-expiratory pressure keeps alveoli open

A **ABGs** — alkalosis early, acidosis late

L **Low** tidal volume (4–6 mL/kg) — protect the lungs

E **Endotracheal** tube + prone position for severe cases

⚡ PATTERN RECOGNITION

Sepsis + new dyspnea + bilateral white-out CXR + SpO_2 won't climb = ARDS until proven otherwise.
This stem appears almost verbatim on NGN case studies.

10 NCJMM Clinical Judgment Scenario

> ***Stem:*** *A 58-year-old client was admitted 36 hours ago with septic shock from a perforated bowel. After surgery the client is in the ICU on 4 L NC at FiO_2 36%. The nurse notes the client is increasingly restless. RR 32, HR 118, BP 92/54, SpO_2 86% on 4 L, then 88% on a non-rebreather at 15 L. Bilateral crackles. Latest ABG: pH 7.48, $PaCO_2$ 28, PaO_2 56, HCO_3 22. CXR: new bilateral patchy infiltrates. BNP normal. Echo: EF 60%.*

STEP 1 · RECOGNIZE CUES

Restlessness, tachypnea (RR 32), tachycardia, hypotension, refractory hypoxemia (SpO_2 won't climb despite non-rebreather), bilateral crackles, bilateral infiltrates on CXR, respiratory alkalosis with severe hypoxemia (PaO_2 56 on high FiO_2), normal BNP, normal EF. Recent septic source.

STEP 2 · ANALYZE CUES

Pattern is non-cardiogenic pulmonary edema following a septic insult. P/F ratio ≈ 56/0.95 ≈ 59 → meets **severe ARDS**. Normal BNP and EF rule out CHF. Early respiratory alkalosis from compensatory hyperventilation.

STEP 3 · PRIORITIZE HYPOTHESES

#1 Acute Respiratory Distress Syndrome (severe). #2 ongoing septic shock. #3 (rule out) cardiogenic edema — already ruled out by BNP/echo.

STEP 4 · GENERATE SOLUTIONS

Notify provider/rapid response, prepare for intubation and mechanical ventilation with low tidal volume + PEEP, draw repeat ABG and cultures, anticipate prone positioning, continue vasopressor support (norepinephrine) for MAP ≥ 65, broad-spectrum antibiotics within 1 hour, conservative fluids, sedation/analgesia, VAP & DVT prophylaxis.

STEP 5 · TAKE ACTION

Call rapid response, set up suction and intubation equipment, place patient on 100% non-rebreather while preparing, position semi-Fowler, ensure two patent IVs, draw labs (ABG, lactate, cultures), administer ordered antibiotic STAT, document baseline neuro & breath sounds.

STEP 6 · EVALUATE OUTCOMES

After intubation and lung-protective ventilation with PEEP 12, target: SpO_2 88–95%, MAP ≥ 65, plateau pressure ≤ 30, urine output ≥ 0.5 mL/kg/hr, repeat ABG in 30 min showing improving oxygenation and stable pH. Reassess every 1–2 hours; escalate to prone positioning if P/F remains < 150.

Diane's NGN NCLEX 2026 Visual Study Library · ARDS · Compiled from Saunders 2026, Berlin Definition, ARDSnet Protocol, NHLBI/NIH guidelines