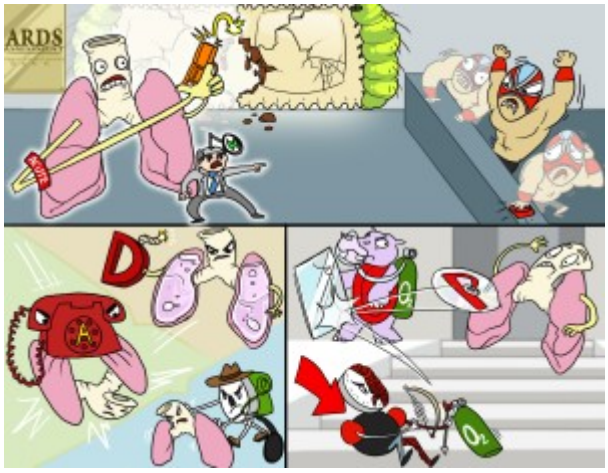
Acute Respiratory Distress Syndrome (ARDS) Assessment

ARDS is a sudden and progressive failure of the respiratory system in which the alveolar-capillary membrane becomes damaged. Damage to this membrane makes it more permeable to fluid, which can lead to difficulty breathing, atelectasis, and hypoxemia that is unresponsive to oxygen therapy. Patients who develop ARDS are typically afflicted by another illness or injury such as COPD, pneumonia, tuberculosis, aspiration, sepsis, shock, or fluid overload. Patients with this condition may also develop pulmonary hypertension, which is a late indicator of decreased lung compliance.



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Cause

Damaged Alveolar-Capillary Membrane
Damaged Ravioli-Caterpillar Membrane
Damage to the alveolar–capillary membrane in acute respiratory distress syndrome (ARDS) leads to the release of inflammatory cytokines such as TNF-?, IL-1, and IL-6. These mediators increase capillary permeability, causing protein-rich fluid to leak into the alveoli and interstitial spaces. This noncardiogenic pulmonary edema results in diffuse alveolar damage, hyaline membrane formation, and severe impairment of gas exchange.

Assessment

Restlessness
Restlessness-wrestler
Early in ARDS, patients may appear restless, anxious, and diaphoretic due to hypoxemia. The sympathetic nervous system responds by increasing heart rate and respiratory rate in an attempt to compensate for low oxygen levels.

Dyspnea
Disc-P-lungs
Damage to the alveoli and the resulting decrease in lung compliance cause dyspnea and tachypnea. The patient often develops respiratory alkalosis initially due to hyperventilation, followed later by respiratory acidosis as fatigue and worsening hypoxemia occur.

Refractory Hypoxemia
Reflecting Hippo-blood-O2
A hallmark of ARDS is hypoxemia that does not improve with supplemental oxygen. This is due to intrapulmonary shunting and ventilation–perfusion (V/Q) mismatch, as alveoli are filled with fluid or collapsed, preventing oxygen from entering the bloodstream despite adequate perfusion.

Decreased PaO2
Down-arrow Partial Pressure-gauge Artery-archer with O2-tank
Despite high concentrations of inspired oxygen, arterial oxygen tension (PaO?) remains low because of impaired diffusion and alveolar flooding. The PaO?/FiO? ratio is used diagnostically, with a ratio less than 300 indicating ARDS severity.

Diffuse Pulmonary Infiltrates
D-fuse Lungs Filled-with-liquid
Chest imaging reveals bilateral diffuse alveolar infiltrates, often described as “whiteout” or “white lung” on chest X-ray. This radiographic pattern reflects widespread alveolar consolidation and edema.

Atelectasis
A-telephone-collapsing-lungs
Injury to type II pneumocytes reduces surfactant production, promoting alveolar collapse. Together with fluid accumulation, this atelectasis further worsens hypoxemia and decreases lung compliance.

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Pulmonary Hypertension

Lungs Hiker-BP

Chronic inflammation and hypoxic vasoconstriction increase pulmonary vascular resistance, leading to pulmonary hypertension and potential right heart strain as a late complication of ARDS.

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