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Acute Respiratory Distress Syndrome

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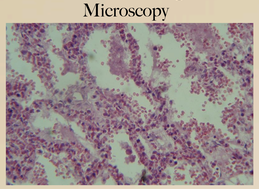
Pathophysiological Process

Underlying Pathophysiology

- Acute alveolar injury results in release of inflammatory cytokines (tumor necrosis factor, IL-1, IL-6, IL-8) that enlist neutrophils to become active in the lungs and inflict damage to the capillary endothelium and alveolar epithelium through discharge of toxins (Butt et al., 2016).
- The damaged capillary membrane leaks protein from the vascular space and disrupts the oncotic gradient that normally regulates absorption of fluid, allowing fluid to leak into the interstitial space (Butt et al., 2016).
- The interstitial lymphatic system, which normally returns fluid to the vascular system, is unable to displace the large volume of fluid and becomes inefficient (Butt et al., 2016).
- The airway spaces become flooded with excess fluid made up of proteins and waste products that are results of cell injury (Albert, 2012).
- Surface tension is lost due to decreased amount of surfactant that leads to collapse of the alveoli (Albert, 2012).

Diffuse Alveolar Damage Under Microscopy



Signs and Symptoms

- Typically occur within 6 to 72 hours of initial lung injury, most often caused by sepsis, pneumonia, aspiration, trauma and blood administration (Laffey, Pham, & Bellani, 2017).
- Often present with shortness of breath, respiratory distress, diffuse crackles, tachypnea, tachycardia and hypoxemia (Gong et al., 2016).
- Arterial blood gas measurement features hypoxemia and respiratory acidosis, even on high levels of delivered fraction of inspired oxygen (fi02) (Modrykamien & Gupta, 2015).
- Chest radiograph may show alveolar infiltrates or edema, decreased aeration and diffuse airspace opacities (Laffey et al., 2017).
- In mechanical ventilation, plateau pressures exceed 30 cm H20 (Gong et al., 2016).

Significance of Pathophysiology

- Impaired gas exchange results in refractory hypoxemia due to ventilation-perfusion mismatch and physiologic shunting (Albert, 2012).
- Increased physiologic dead space results in diminished ventilation, causing hypercarbia (Kallet, Zhuo, Liu, Caffee, & Matthay, 2014).
- Inflammatory changes lead to decreased lung compliance, causing high airway pressures and difficulty in ventilation (Gong, Thompson, Taylor, & Gong, 2016).
- Pulmonary hypertension may occur as a result of vasoconstriction secondary to hypoxia and physical compression due to high levels of positive airway pressure in mechanically ventilated patients (Viellard-Baron et al., 2016).
- In the final fibro-proliferative phases, scar tissue will form, leading to permanent pulmonary dysfunction (Albert, 2012).

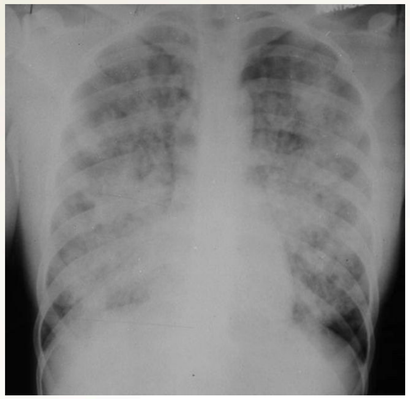
Table 1. ARDS Berlin definition.

The Berlin definition of acute respiratory distress syndrome	
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]	Bilateral opacities — not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload. Need objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]	
Mild	200 mmHg < PaO_2/FIO_2 ≤300 mmHg with PEEP or CPAP ≥5 cmH_2O[c]
Moderate	100 mmHg < PaO_2/FIO_2 ≤200 mmHg with PEEP ≥5 cmH_2O
Severe	PaO_2/FIO_2 ≤100 mmHg with PEEP ≥5 cmH_2O

Abbreviations: CPAP, continuous positive airway pressure; F_IO_2, fraction of inspired oxygen; PaO_2, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure; [a]Chest radiograph or computed tomography scan; [b]If altitude is higher than 1,000 m, the correction factor should be calculated as follows: [PaO_2/FIO_2_(barometric pressure/760)]; [c]This may be delivered noninvasively in the mild acute respiratory distress syndrome group.

Introduction

- Acute respiratory distress syndrome (ARDS) is a complex disease state with varied causality, multiple pathophysiologic mechanisms and numerous treatment modalities.
- Patients who develop ARDS are often extraordinarily ill and require prolonged stays in the intensive care unit with extensive medical intervention. The development of identification and treatment of ARDS has a storied history and continues to evolve in clinical practice with little progress toward improved patient outcomes.
- "Mortality from ARDS has been estimated at 26-58%" in the United States (U.S.) despite decades of study and advances in therapy (Modrykamien & Gupta, 2015, p. 163).
- Extensive research has been conducted regarding ventilation modalities and medical management with minimal improvement in rates of morbidity and mortality (Butt, Kurdowska, & Allen, 2016).
- As the overall health of the general population continues to decline as evidenced by rising obesity rates, an generally aging population and increased incidence of multiple medical comorbidities, treatment of the patient in ARDS becomes more challenging (Hibbert, Rice, & Malhotra, 2012).
- An advanced practitioner armed with a thorough understanding of the history, pathophysiology and evidenced-based treatment modalities is best equipped to care for patients with ARDS.



Why ARDS?

- ARDS is a syndrome that affects patients of many different populations and the tenets of disease management are concepts that are integral in the care provided as a Certified Registered Nurse Anesthetist (CRNA).
- ARDS affects approximately 86 of 100,000 patients and there are roughly 190,000 cases in the United States every year (Gong, Thompson, Taylor, & Gong, 2016).
- The pathophysiology of ARDS is complex and requires an in-depth understanding of several concepts including pulmonary, inflammatory, acid-base imbalances and distribution of body fluids.
- There has been a significant amount of research regarding the management of ARDS and in-depth knowledge of evidence-based practice guidelines is essential to the care of a CRNA

Implications for Nursing Care

Diagnostic Criteria

- Symptoms occur within seven days of initial lung injury (Laffey, Pham, & Bellani, 2017).
- Impaired oxygenation as evidenced by Pa02/Fi02 ratio of <200 mm Hg for moderate ARDS and <100 mm Hg for severe ARDS (Laffey et al., 2017).
- Chest radiograph reveals presence of non-cardiogenic pulmonary edema as bilateral opacities (Laffey et al., 2017).

Adjunct Therapies and Supportive Care

- Minimize oxygen consumption by treating fever, pain and anxiety (Laffey et al., 2017).
- Sedation and neuromuscular blockade are often required to suppressive the patient's intrinsic respiratory pattern and optimize mechanical ventilation (Gong, Thompson, Taylor, & Gong, 2016).
- Judicious fluid administration decreases further fluid leakage through capillary beds (Viellard-Baron et al., 2016).
- Hemodynamic monitoring allows for tightly controlled treatment of hemodynamic instability and intravascular volume status (Viellard-Baron et al., 2016).
- Prone positioning, extracorporeal membrane oxygenation (ECMO) and high frequency oscillation ventilation (HFOV) may be considered in severe cases of refractory hypoxemia (Albert, 2012).

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Ventilator Management

- Low tidal volume ventilation (LTVV) aims to prevent alveolar overdistention and barotrauma (Mosier et al., 2015).
- LTVV is achieved through calculating and delivering tidal volumes of 6 ml/kg using predicted body weight (Davies et al., 2015).
- Increased respiratory rate (maximum 35 breaths per minute) compensates to achieve adequate minute ventilation (Mosier et al., 2015).
- Goal plateau pressure is less than 30 cm H20 (Davies et al., 2015).
- Fi02 and positive end expiratory pressure (PEEP) are increased in tandem (Mosier et al., 2015).
- Goal arterial oxygen tension (PaO_2) is 55-80 mmHg and ideal oxygen saturation is between 88 and 95% (Mosier et al., 2015).

Conclusion

- ARDS is a complex disease state with varied pathophysiologic factors and multiple treatment modalities.
- Early recognition and aggressive management contribute to improved patient outcomes due to the syndrome's high incidence and impact on critically ill patients.
- Lung protective ventilation is imperative and must be managed closely.
- Understanding of the diagnosis and treatment is an important quality for the advanced practice nurse caring for critically ill patients.

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