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ARDS: Acute Respiratory Distress Syndrome

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**ARDS Pathophysiology**

The pathophysiology of ARDS primarily involves an initial lung injury followed by mechanisms that increase fluid from the pulmonary microvasculature to the alveoli (Fujishima, 2014). Tissue injury disrupts this process and results in severe negative consequences, including:

- Decreased functional residual capacity and alveolar
discontinuity and air trapping (Hansen-Flaschen & Siegel, 2016).
- With proteins leaking out of the capillaries, the oncotic gradient no longer favors
  reabsorption of fluid into the intravascular space (Hansen-Flaschen & Siegel, 2016).
- The fluid of exudate into the airway perpetuates tissue injury leading to a further
  release of pro-inflammatory cytokines (Hansen-Flaschen & Siegel, 2016).

**The Innate Immune System and Inflammation**

The innate immune system plays a major role in the dysregulation of the inter- and intracellular molecules that increase vascular permeability and subsequent tissue damage, particularly the stimulation of alveolar macrophages and neutrophils. Once stimulated, macrophages recruit neutrophils and circulating macrophages to the site of injury where they perpetuate the inflammatory response by releasing the biologic mediators such as, proteases, reactive oxygen species, oxygen free radicals, and cytokines (Han & Mallampalli, 2015). These cytokotic mediators cause tissue necrosis as well as the inductors of cell death including the destruction of type II epithelial cells. This is of particular importance since type II epithelial cells are responsible for maintaining surfactant, a substance that lines the alveoli, forming support ventilation and prevents collapse (Han & Mallampalli, 2015). Some of the inflammatory cytokines that are shown to be elevated in ARDS patients includes IL-8, TNF-α, IL-6, and IL-10. Also of note is the research into pattern recognition receptors (PRRs) and their involvement in the development of ARDS. Particularly, the Toll-like receptor signaling pathway, which is believed to hyperactivate an extracellular matrix of glycosaminoglycan produced after tissue injury, initiates the inflammatory response in ARDS through TL2R2 and TL2R4 (Han & Mallampalli, 2015).

**Significance of Pathophysiology**

**Impaired Gas Exchange**

Damage to the endothelium allows a type of exudate into the lung parenchyma and eventually the alveolar airspace (Mannino, 2014). The increased lung weight causes dependent zones of the lungs to collapse, leaving only non-dependent areas to remain open for ventilation (Gattinoni & Quinzel, 2016). The resulting shunt from the increased gases areas and reduced lung size are the cause of the characteristic refractory hypoxemia seen in ARDS (Gattinoni & Quinzel, 2016).

**Decreased Lung Compliance**

Decreased lung compliance is a result of the activation of the coagulation cascade during the acute inflammatory process. It is triggered by the release of tissue factor from the damaged lungs (Mannino, 2014). During the process, fibrin deposits form in the lungs and can undergo remodeling to produce pulmonary thrombus (Mannino, 2014). The thrombus tissue, combined with collapsed alveoli, decrease the compliance of the lungs and result in progressive respiratory insufficiency.

**Increased Pulmonary Artery Pressure**

Increased pulmonary artery pressure is another significant consequence of the ARDS pathophysiology. It may be caused by hypoxic vasoconstriction, positive pressure ventilation, parenchymal distortion, airway collapse, and hypercarbia (Siegel, 2016). Although cor pulmonale is rare, right ventricular dysfunction associated with pulmonary hypertension increases the mortality of ARDS (Nagelhout & Platt, 2014).

**ARDS Nursing Implications**

**References**

In the 1960’s, military physicians discovered a distinct hypoxic condition involving both lungs simultaneously (Medvedkun & Gupta, 2015). In Vietnam, this condition was referred to by physicians as “shock lung.” Meanwhile, civilian physicians who encountered this condition termed it adult respiratory distress syndrome. The term was later modified to acute respiratory distress syndrome (ARDS) after determining that similar cases existed among all age groups (Medvedkun & Gupta, 2015). ARDS develops after insult to the lung tissue. There are many conditions that can precipitate such an injury. Some of the most common precipitating factors include:

- Sepsis
- Trauma
- Pancreatitis
- Pneumonia
- Hypothermia
- Hypoxia

Despite advances in treatment of ARDS, incidence and mortality remain high. In the United States, ARDS has an estimated incidence of 180,000 cases per year and a mortality rate of 26% to 38% (Medvedkun & Gupta, 2015). In recent studies, ARDS represented 10% of all patients with acute respiratory distress syndrome (Mallampalli & Han, 2014). Aspiration and hypoxemia are just two factors of ARDS. In addition to the symptoms related to respiratory distress and subsequent respiratory failure, ARDS patients with acute respiratory distress syndrome (ARDS) typically display symptoms of the precipitating disease process as well.

**Conclusion**

As of now, the only treatment for ARDS is supportive care aimed at improving gas exchange. The standard of care is lung protective ventilation strategies including low tidal volumes and limitation of positive end-expiratory pressure (PEEP). As well as the use of more aggressive strategies including neuromuscular blockers and prone positioning. Although these therapies are currently the only treatments shown to improve mortality, efforts are being made to identify interventions aimed at modulating the inflammatory response.

It is the responsibility of the nurse, and the rest of the treatment team, to be aware of the disease process, associated complications, and the most up-to-date therapy options. As a team, the various disciplines and medical specialties can utilize their expertise to optimize the patient’s ventilation status while treating the underlying cause. With a deeper understanding of the pathophysiology of ARDS, clinicians can better implement treatment plans to improve patient mortality.