ARDS Pathophysiology

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Acute Respiratory Distress Syndrome

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Introduction
Acute respiratory distress syndrome (ARDS) is a hypoxic respiratory disorder that causes quick decline in intensive care unit (ICU) patients with a mortality of 34-55% (Sagul & Fargo, 2012). The hallmark of ARDS is that it causes damage to the alveoli of the lungs from products produced by the systemic inflammatory response and spread through the blood flow to the lung, or is caused by trauma precisely to lung tissue resulting in lung collapse and pulmonary edema (Koulouras et al., 2016). It is important to understand the mechanism of change because of its high mortality rate and how quickly it occurs in critical patients. The ARDS syndrome as we know it today was originally seen during the Vietnam War and has undergone multiple research studies to identify best treatment and diagnostic options since then (Koulouras et al., 2016).

Working in a medical ICU, I see a lot of ventilated patients who are being treated for ARDS or who develop ARDS. Flu season is when we see most ARDS cases and those patients either don’t recover or have a very poor prognosis. I picked ARDS for this assignment because it doesn’t discriminate; any person who has an inflammatory or infectious process systemically or in the lung is at risk for ARDS no matter their age or gender.

Underlying Pathophysiology
In a healthy lung, the membrane surrounding the alveoli allows for oxygen to pass into capillaries of the lung and be circulated to the rest of the body in the blood stream (Sagul & Fargo, 2012). When alveoli are damaged by trauma or infection they attract the constituents of the body’s inflammatory response which cause capillary permeability permitting pulmonary edema and the breakdown of the alveolar membrane causing impaired gas exchange (Sagul & Fargo, 2012). The excess fluid and mediators compress the alveoli further decreasing gas exchange and causing lung collapse and systemic hypoxia (Sagul & Fargo, 2012). As ARDS progresses, fibrosis of the lung occurs as a result of the initial inflammation leading to long term problems with lung expansion and oxygenation in the patient (Sagul & Fargo, 2012).

Significance of Pathophysiology
The pathophysiology leads to an obvious worsening clinical picture of the patient. The patient may now meet criteria that makes them require mechanical ventilation and if they are already being ventilated, the clinician may need to adjust ventilator settings to prevent further damage to lung tissue. Since the pathophysiology leads to similar manifestations of congestive heart failure, the absence of heart failure will lead the clinician to want to rule out ARDS or begin treating the patient for ARDS. If ARDS is determined, then cardiac testing such as an echocardiogram may be ordered to check for heart dysfunction as a result of ARDS. The pathophysiology may also indicate that now there are systemic inflammatory processes going on if sepsis was not the underlying cause of ARDS. The disease process of ARDS happens abruptly in the patient, so the significance to the clinician would mean implementing ARDS treatments early and quickly obtaining the appropriate diagnostic data to best treat the patient.

Signs and Symptoms
ARDS is caused by injury to the lung tissue from the systemic immune response or from trauma. Sepsis and pneumonia are common causes of ARDS.

❖ Low oxygen blood level that doesn’t improve with oxygen administration
❖ Fever
❖ Cough with increased sputum production
❖ Shortness of breath
❖ Increased respiration rate
❖ Changes in patient’s mentation
❖ Increased work of breathing
❖ Pleuritic chest pain

Nursing Implications
❖ Diagnostic criteria based on The American European Consensus Conference definition of ARDS include: symptoms witnessed within 7 days of the initial injury or a decline in the patient; chest x-ray showing infiltrates in both lungs; lung failure without underlying heart dysfunction; and a P/F ratio of less than or equal to 300mmHg with PEEP greater than or equal to 5 (Drahank & Custer, 2015).
❖ Low tidal volume (VT) and high positive end expiratory pressure (PEEP) ventilator settings for the patient with ARDS; ideally a VT of 8 ml/kg PBW, but more research needs conducted (Chang et al., 2013).
❖ Paralytics have been proven to be successful in decreasing the time that the patient spends on the ventilator, increases the patient’s tolerance of the ventilator, reduces heart rate variability, and improves oxygenation (Ettinger, 2012).

Conclusion
Although there are many underlying causes of, ARDS, the advancements made in treatment of this process have led to a significant decrease in mortality. More research needs done regarding artifacts, low VT, high PEEP, blood temperature and its impact, and ECMO as effective modes of treatment. There is sufficient evidence for close monitoring of fluid status and being more cautious with fluid administration relating to positive outcomes (Modykamien & Gupta, 2015). Despite successful interventions, the biggest predictor in patient mortality comes down to comorbidities of the patient which can lead to death within 1 year ARDS diagnosis and treatment (Chang et al., 2014). Nursing remains one of the biggest roles in diagnosing and managing ARDS making the point for further education and research regarding ARDS (Koulouras et al., 2016).

References