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# Acute Respiratory Distress Syndrome

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## Introduction

Acute Respiratory Distress Syndrome is an inflammatory response resulting from injury to the alveolar-capillary membrane. This injury is caused by a systemic inflammatory response that involves either direct trauma to the lung cells, such as a pneumonia, or indirect, such as sepsis. The inflammatory response that is triggered results in leaky alveolar-capillary beds and infiltration of the lungs (Villar, 2011). This is very common with approximately 150,000 cases annually in the United States and a very high mortality rate of 60,000 deaths per year (Pipeling & Fan, 2010). Despite the high mortality rate, 15-35%, there is no set of guidelines for treatment of this condition, and methods of mechanical ventilation are only supportive (Zaglam, Jouvet, Flechelles, Emeriaud & Cheriet, 2014). The most severe form of ARDS is refractory hypoxemia, a life threatening condition, in which there is not an adequate amount of oxygen delivered to the tissues (Villar & Kacmarek, 2013). With more than 60 causes of ARDS, it is essential for nursing staff working directly with these patients to be aware of signs and early detection allowing more rapid initiation of treatment modalities in the hope of decreasing patient mortality (Taylor, 2005).

## Pathophysiology

### Signs and Symptoms

Presenting symptoms of ARDS usually occur within 24-48 hours after the initial injury. In the presence of sepsis, the onset of symptoms can be even more rapid, within 24 hours. Patients generally present with dyspnea, tachypnea, and hypoxemia (York & Kane, 2012). They may also have adventitious breath sounds including crackles, rhonchi, and wheezes (Taylor 2005).

Diagnosis is made through exclusion of other possible causes and through the Berlin criteria, see Table 1 (Luks, 2013). Determining the ratio of the arterial partial pressure of oxygen to the fractional concentration of inspired oxygen (PaO2/FiO2) is referred to as the P/F ratio and is used as a marker for the severity of hypoxemia. Arterial blood gas initially may show respiratory alkalosis due to tachypnea and a decreased PaCO2. As the patient fatigues and gas exchange worsens the patient will shift into a reparatory acidosis and eventually a metabolic acidosis from tissue hypoxia and anaerobic metabolism (York & Kane, 2012).

### Underlying Pathophysiology

Beginning with either a direct lung cell injury or an indirect lung cell injury, such as widespread infection, the pathophysiology and precipitating symptoms of ARDS are resultant from an inflammatory response. Diffuse injury to the alveolar-capillary membrane causes local inflammation, and in ARDS an exaggerated inflammatory response follows. Circulating neutrophils are activated and release tissue damaging products, furthering the inflammatory cascade. Damaged capillary walls causes increased pulmonary microvascular permeability, allowing protein-rich fluid to flood the alveoli and interstitium of the distal airspaces (Marino, 2014, p. 447). As a result, type 2 pneumocytes are damaged, reducing surfactant production and impairing fluid removal. Consequently, atelectasis, or collapsing of the alveoli, follows. With less alveoli to participate in gas exchange, there is a decrease in lung volume and dead space ventilation causes intrapulmonary shunts to be created (Taylor, 2005). Lung compliance is also diminished as a result of decreased surfactant. The need for elevated pressures to deliver tidal volumes because of the decreased compliance can further lung injury and continue the cycle. All these factors cause severe hypoxemia in the patient because of impaired gas exchange (Luks, 2013).

***Table 1. Berlin Criteria for Diagnosis of ARDS***

| | | | |
|---|---|---|---|
| **Timing** | Within one week of known clinical insult or new or worsening respiratory symptoms | | |
| **Chest Imaging** | 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** | **Mild** | **Moderate** | **Severe** |
| | PaO2/FiO2: 200-300 mm Hg<br>with PEEP or CPAP ≥ 5 cm H2O | PaO2/FiO2: 100-200 mm Hg<br>with PEEP or CPAP ≥ 5 cm H2O | PaO2/FiO2: < 100 mmHg<br>with PEEP or CPAP ≥ 5 cm H2O |

(Ferguson et al., 2012)

### Common Causes

**Direct Lung Injury**
- Pneumonia
- Aspiration
- Pulmonary Contusion
- Near-Drowning
- Inhalation injury
- Fat emboli

**Indirect Lung Injury**
- Sepsis
- Multiple trauma
- Acute Pancreatitis
- Drug Overdose
- Cardiopulmonary Bypass
- Blood Transfusion

(Villar, 2011)

### Significance of Pathophysiology

The pathophysiology is what has driven treatment modalities for ARDS. Understanding that ARDS stems from an inflammatory process from lung injury is essential to utilizing effective therapy. There is a fine balance between improving patient oxygenation and causing further lung damage, which exacerbates the inflammatory process and worsens ARDS (Luks, 2013). By understanding the pathophysiology, nursing and healthcare staff can be more competent and efficient in the care of ARDS patients.

## Implications for Nursing Care

Since there is no known treatment that can stop the underlying inflammatory process that causes ARDS, nurses should understand that therapy is supportive, focusing on maintaining adequate gas exchange. The current standard of care is to use lung protective ventilation. This can be defined as using a tidal volume of 6 ml/kg of the patient's predicted body weight. Protective ventilation is essential in this patient population to prevent high distending pressures that are a result of low compliance. Decreased compliance causes the alveoli and lung tissue to be very fragile and high distending pressures cause further lung injury or volutrauma and can worsen the underlying ARDS (Luks, 2013). The goal of using a decreased tidal volume is to maintain a plateau pressure, or the peak pressure in the alveoli at the end of inspiration, that is less than or equal to 30 cm H2O. Additionally, the use of positive end-expiratory pressure (PEEP) prevents atelectrauma, which is caused by repetitive opening and closing of the alveoli (Marino, 2014, p. 455). PEEP also recruits collapsed alveoli to take part in gas exchange, decreasing the physiologic shunting (Taylor, 2005).

As the advocate for the patient, nursing staff must pay close attention for the need for sedation. Not only does patient-ventilator dyssynchrony increase further lung damage and complicates the patients oxygenation issues, but use of mechanical ventilation can be uncomfortable for the patient causing anxiety. The healthcare team should ensure patient comfort and thereby improve ventilator synchrony and optimize oxygenation (York & Kane, 2012).

Although some patients may require further rescue measures (see refractory hypoxemia) the main cause of death from ARDS is a result of complications developed in the ICU. It is vital for nursing staff to continue prophylactic measures against further complications. Using prophylactic measures against catheter-related blood stream infections, catheter-associated urinary tract infections, venous thromboembolism, ventilator-associated pneumonia, and gastrointestinal bleeding are essential to decreasing mortality for the ARDS patient (Luks, 2013).

Regular Chest Radiograph

ARDS Chest Radiograph

(Zaglam et al., 2014)

## Conclusion

ARDS continues to be a prominent and deadly complication in the intensive care unit. Although a great deal of research is being done, treatment protocols are still only supportive of patient gas exchange. Many rescue therapies exist for refractory hypoxemia, although most improve patient oxygenation, they do not necessarily improve mortality rates, leaving the use of these interventions to the discretion of the physician (Collins & Blank, 2011). Nursing staff and healthcare providers should possess a knowledge of the pathophysiologic process behind ARDS, to improve recognition and allow for earlier intervention (Taylor, 2005). Understanding the mechanisms of treatment and how they are related to the physiologic process of ARDS is essential for new innovations and improved patient outcomes.

## Refractory Hypoxemia

The most severe form of ARDS is known as refractory hypoxemia. There is no standard definition for refractory hypoxemia, but this group of patients can be described as having significant hypoxemia that is refractory to standard treatment despite increased oxygen delivery and high levels of PEEP (Villar & Kacmarek, 2013). While refractory hypoxemia is an infrequent cause of death in ARDS patients (<15%), for this group of patients the use of protective ventilation may not be enough and may require one or a combination of rescue methods (Villar & Kacmarek, 2013).

### Rescue Methods

These strategies are used for patients with refractory hypoxemia. While they may improve oxygenation, none of these modalities have been shown to decrease patient mortality. For the most effective use of these strategies, they should be implemented within 96 hours of ARDS onset, requiring rapid recognition of the disease process and failure of conventional methods (Collins & Blank, 2011).

### Recruitment Maneuvers

The goal of lung recruitment maneuvers is to recruit atelectatic alveoli and improve gas exchange but avoid atelectrauma by repetitive opening and closing alveoli. Maneuvers apply a higher than normal pressure either intermittently for a few minutes or sustained for up to about 40 seconds (Villar & Kacmarek, 2013). While this may temporarily increase oxygenation and lung mechanics there has been no correlation for improved patient outcome (Collins & Blank, 2011).

### Neuromuscular Blockade

Neuromuscular blockade is often used in the hypoxemic patient. Two benefits result from paralytic agents, the patient has improved ventilator synchrony and there is an elimination of muscle activity and therefor a decrease in oxygen consumption, which is beneficial for the patient with a very limited oxygen supply. Research has shown that continuous NMBA infusions have improved outcomes, which allows this pharmacologic intervention to be contemplated earlier in patients with ARDS (Luks, 2013).

### High-Frequency Oscillation Ventilation (HFOV)

This method of ventilation utilized a very low tidal volume with a very high respiratory rate. This prevents over-distention and allows a higher end-expiratory lung volume. Similar to other rescue methods, HFOV may improve oxygenation in patients, but there is no evidence of decreased mortality rates (Collins & Blank, 2011).

### Inhaled Vasodilators

Inhaled pulmonary vasodilators such as nitric oxide and prostacyclin cause local vasodilation of healthy lung tissue allowing for increased perfusion and therefor improving ventilation-perfusion matching and arterial oxygenation. Again, this treatment modality does improve oxygenation in the ARDS patient, but has shown no benefit in mortality rates (Luks, 2013).

### Prone Positioning

Due to the dependent nature of atelectasis in lung tissue, prone positioning is used to counteract this issue and improve patient oxygenation. Additional benefits of prone positioning include secretion clearance, decreased compression of the lungs by the heart, and increased end-expiratory volume (Luks, 2013). However, to experience optimal benefits the patient may be required to be in the prone position for more than 20 hours. Adverse effects from this prolonged position include aspiration, tube or line displacement, and pressure ulcers (York & Kane, 2012). While prone positioning has been shown to increase oxygenation, there is no data to support a reduction in patient mortality (Villar & Kacmarek, 2013).

### Extracorporeal Membrane Oxygenation (ECMO)

ECMO is a method of respiratory support that uses an external circuit to provide gas exchange. This treatment creates time for recovery from the underlying disease. The use of ECMO for ARDS is still controversial since there is no evidence in improved survival when compared to conventional methods of mechanical ventilation (Rozé, Repusseau, & Ouattara, 2014).

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