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# TUTOR ICON

## ACUTE RESPIRATORY DISTRESS SYNDROME

#### **1. DEFINITION:**

ARDS is a clinical syndrome also called acute lung injury in which there is lung inflammation and severe hypoxemia and decreased compliance of the lungs, which leads to both oxygenation and ventilatory failure. Mortality is 34% to 58% and is improved with early intervention.

## 2. ETIOLOGY:

- Etiologies are numerous and can be pulmonary or nonpulmonary. These include (but are not limited to):
  - Infections, including aspiration, sepsis.
  - Shock (any cause), trauma, near drowning, direct or indirect lung injury, burns, pancreatitis.
  - Metabolic, hematologic, and immunologic disorders.
  - Inhaled agents—smoke, high concentration of oxygen, corrosive substances.
  - Major surgery including coronary artery bypass graft, fat or air embolism, lung or bone marrow transplantation.

### **3. PATHOPHYSIOLOGY:**

Pulmonary and/or nonpulmonary insult to the alveolar-capillary membrane causing fluid leakage into interstitial spaces.

Ventilation-perfusion (V/Q) mismatch caused by shunting of blood







# 4. CLINICAL MANIFESTATIONS:

- Acute onset of severe dyspnea, tachypnea, tachycardia, use of accessory muscles, cyanosis.
- Increasing requirements of oxygen therapy. Hypoxemia refractory to supplemental oxygen therapy.
- Scattered crackles and rhonchi heard on auscultation.

# **5. DIAGNOSTIC EVALUATION:**

- The hallmark sign for ARDS is a shunt; hypoxemia remains despite increasing oxygen therapy.
- Decreased lung compliance; increasing pressure required to ventilate patient on mechanical ventilation.
- Chest X-ray exhibits bilateral infiltrates.
- Pulmonary artery catheter readings show pulmonary artery wedge pressure >19 mm Hg, absence of left atrial hypertension, and no clinical signs of heart failure.

Tests used to diagnose ARDS include:

- Arterial blood gas
- Bronchoscopy
- CBC and blood chemistries
- Sputum cultures and analysis
- Tests for possible infections

Occasionally an echocardiogram or Swan-Ganz catheterization may need to be done to rule out congestive heart failure, which can look similar to ARDS on a chest x-ray.

### 6. MANAGEMENT:

- The underlying cause for ARDS should be determined so appropriate treatment can be initiated.
- Low  $V_T$  by mechanical ventilation (6 ml/kg of predicted body weight) reduces mortality compared to high volume ventilation. Monitor for respiratory acidosis. Protective ventilation (ie, maximum inspiratory pressure [MIP] of < 35 cm) should be instituted. PEEP should be used to improve PaO<sub>2</sub>. PEEP keeps the alveoli open, thereby improving gas exchange. Therefore, a lower oxygen concentration (FiO<sub>2</sub>) may be used to maintain satisfactory oxygenation.
- Fluid management must be maintained. The patient may be hypovolemic due to the movement of fluid into the interstitium of the lung. Pulmonary artery catheter monitoring and inotropic medication can be helpful.
- Medications are aimed at treating the underlying cause. Corticosteroids are used infrequently due to the controversy regarding benefits of usage.
- Adequate nutrition should be initiated early and maintained. Direct injury
- Infections, such as pneumonia, sepsis.
- Respiratory complications, such as pulmonary emboli, barotrauma, oxygen toxicity, subcutaneous emphysema, or pulmonary fibrosis.
- GI complications, such as stress ulcer, ileus.
- Cardiac complications, such as decreased cardiac output and dysrhythmias.
- Renal failure, disseminated intravascular coagulation.
- Multiorgan failure and sepsis, which may result in death.
- Cognitive impairment.

#### **Prone position**

Distribution of lung infiltrates in acute respiratory distress syndrome is nonuniform. Repositioning into the prone position (face down) might improve oxygenation by relieving atelectasis and improving perfusion. However, although the hypoxemia is overcome there seems to be no effect on overall survival.

#### Fluid management

Several studies have shown that pulmonary function and outcome are better in patients that lost weight or pulmonary wedge pressure was lowered by diuresis or fluid restriction.

### Corticosteroids

A Meduri et al. study has found significant improvement in ARDS using modest doses of corticosteroids. The initial regimen consists of methylprednisolone 2 mg/kg daily. After 3–5 days a response must be apparent. In 1–2 weeks the dose can be tapered to methylprednisolone 0.5-1.0 mg daily. Patients with ARDS do not benefit from high-dose corticosteroids. This was a study involving a small number of patients in one center. A recent NIH-sponsored multicenter ARDSnet LAZARUS study of corticosteroids for ARDS demonstrated that they are not efficacious in ARDS.

#### Nitric oxide

Inhaled nitric oxide (NO) potentially acts as selective pulmonary vasodilator. Rapid binding to hemoglobin prevents systemic effects. It should increase perfusion of better ventilated areas. There are no large studies demonstrating positive results. Therefore its use must be considered individually.

Almitrine bismesylate stimulates chemoreceptors in carotic and aortic bodies. It has been used to potentiate the effect of NO, presumably by potentiating hypoxia-induced pulmonary vasoconstriction. In case of ARDS it is not known whether this combination is useful.

#### Surfactant therapy

To date no prospective controlled clinical trial has shown a significant mortality benefit of exogenous surfactant in ARDS.

### 7. NURSING MANAGEMENT:

### **Nursing Interventions**

### Nursing Assessment

- Note changes suggesting increased work of breathing (dyspnea, tachypnea, diaphoresis, intercostal muscle retraction, fatigue) or pulmonary edema (fine, coarse crackles or rales, frothy pink sputum).
- Assess breath sounds.
  - Diminished or absent sounds suggest inability to ventilate the lungs sufficiently to prevent atelectasis.
  - Crackles may indicate ineffective airway clearance, fluid in the lungs.
  - Wheezing indicates narrowed airways and bronchospasm.
  - Rhonchi and crackles suggest ineffective secretion clearance.
- Assess level of consciousness (LOC) and ability to tolerate increased work of breathing.
  - Confusion, lethargy, rapid shallow breathing, abdominal paradox (inward movement of abdominal wall during inspiration), and intercostal retractions suggest inability to maintain adequate minute ventilation.
- Assess for signs of hypoxemia and hypercapnia.
- Analyze ABG and compare with previous values.
  - If the patient cannot maintain a minute ventilation sufficient to prevent CO<sub>2</sub> retention, pH will fall.
  - Mechanical ventilation or non-invasive ventilation may be needed if pH falls to 7.30 or below.
- Determine vital capacity (VC) and respiratory rate and compare with values indicating need for mechanical ventilation:
  - $\circ$  VC < 15 mL/kg.
  - Respiratory rate > 30 breaths/minute.
  - Negative inspiratory force < -15 to -25 cm  $H_2O$ .
  - Refractory hypoxia

• Determine hemodynamic status (blood pressure [BP], heart rate, pulmonary wedge pressure, cardiac output, SvO<sub>2</sub>) and compare with previous values. If patient is on mechanical ventilation with positive end-expiratory pressure (PEEP), venous return may be limited, resulting in decreased cardiac output.

### Nursing Diagnoses

- Impaired Gas Exchange related to inadequate respiratory center activity or chest wall movement, airway obstruction, and/or fluid in lungs
- Ineffective Airway Clearance related to increased or tenacious secretions

## Nursing Interventions

## **Improving Gas Exchange**

- Administer oxygen to maintain PaO<sub>2</sub> of 60 mm Hg or SaO<sub>2</sub> greater than 90% using devices that provide increased oxygen concentrations (aerosol mask, partial rebreathing mask, nonrebreathing mask).
- Administer antibiotics, cardiac medications, and diuretics as ordered for underlying disorder.
- Monitor fluid balance by intake and output measurement, daily weight, and direct measurement of pulmonary capillary wedge pressure to detect presence of hypovolemia or hypervolemia.
- Provide measures to prevent atelectasis and promote chest expansion and secretion clearance, as ordered (incentive spirometer, nebulization, head of bed elevated 30 degrees, turn frequently, out of bed when clinically stable).
- Monitor adequacy of alveolar ventilation by frequent measurement of SpO<sub>2</sub>, ABG levels, respiratory rate, and VC.
- Compare monitored values with criteria indicating need for mechanical ventilation (see section titled "Nursing Assessment"). Report and prepare to assist with noninvasive ventilation or intubation and initiation of mechanical ventilation, if indicated.

# **Maintaining Airway Clearance**

- Administer medications to increase alveolar ventilation—bronchodilators to reduce bronchospasm, corticosteroids to reduce airway inflammation.
- Teach slow, pursed-lip breathing to reduce airway obstruction and improve oxygen levels. Chest physiotherapy may be considered to remove mucus.
- Suction patient, as needed, to assist with removal of secretions.
- If the patient becomes increasingly lethargic, cannot cough or expectorate secretions, cannot cooperate with therapy, or if pH falls below 7.30, despite use of the above therapy, report and prepare to assist with intubation and initiation of mechanical ventilation.

# 8. PATIENT EDUCATION AND HEALTH MAINTENANCE:

- Instruct patient with preexisting pulmonary disease to seek early intervention for infections to prevent acute respiratory failure, pneumonia, and exacerbations.
- Teach patient about medication regimen.
  - Proper technique for inhaler use
  - Dosage and timing of medications
  - Monitoring for adverse effects of corticosteroids to report to health care provider: weight gain due to fluid retention, polyuria and polydipsia due to hyperglycemia, mood changes, insomnia, bruising, fragile skin; vision changes due to cataracts or glaucoma.

### COMMUNITY AND HOME CARE CONSIDERATIONS:

- Encourage patients at risk, especially the elderly and those with preexisting lung disease, to get pneumococcal pneumonia and yearly influenza immunizations.
  - Pneumococcal vaccine is 60% to 70% effective in preventing bacteremic pneumococcal infections in adults and children at least age 2.
  - If a person received their first pneumococcal vaccination before age 65, they should be revaccinated after age 65, if more than 5 years have elapsed since the previous dose.

- Vaccinate children under age 2 and those over age 2 with the following conditions, which increase risk for pneumococcal pneumonia or severe complications, as recommended by the Centers for Disease Control and Prevention (CDC):
  - Chronic cardiovascular disease (including heart failure).
  - Chronic pulmonary disease (eg, emphysema).
  - Diabetes.
  - Alcoholism.
  - Chronic liver disease (including cirrhosis).
  - Cerebrospinal fluid leaks.
  - Asplenia (including functional asplenia such as sickle cell disease).
  - Immunocompromised people (including human immunodeficiency virus [HIV]).
  - People living in environments at higher risk for pneumococcal disease (Alaskan natives, certain American Indian populations, and residents of nursing homes and long-term care facilities).
- Immunize annually for influenza in the following groups, according to the CDC:
  - People age 50 and older
  - Immunocompromised patients
  - Residents of nursing homes or chronic care facilities
  - People with cardiovascular disease
  - People with diabetes mellitus
  - Patients receiving long-term aspirin therapy
  - Pregnant women who will be in the second or third trimester of pregnancy during flu season
  - Health care workers
  - Household contacts of those at risk for influenza.
- Inactivated influenza vaccine should be given to people age 6 months to 5 years and in those ages 50 and older.
- Intranasal live attenuated vaccine is an alternative for people ages 5 to 49 without chronic conditions, HIV, or asthma.

### 9. COMPLICATIONS:

- Lung damage (such as pneumothorax) due to use of high settings on the breathing machine needed to treat the disease
- Multiple organ system failure
- Pulmonary fibrosis
- Ventilator-associated pneumonia

### 10. PROGNOSIS:

About a third of people with ARDS die from the disease. Survivors usually get back normal lung function, but many people have permanent, usually mild, lung damage.

Many people who survive ARDS have memory loss or other problems with thinking after they recover. This is due to brain damage that occurred when the lungs weren't working properly and the brain wasn't getting enough oxygen.

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