Chapter 67

Acute Respiratory Failure and Acute Respiratory Distress Syndrome

# KEY POINTS

## Acute Respiratory Failure

- *Acute respiratory failure* (ARF) results from a failure of oxygenation and/or ventilation or both, which occurs when the transfer of $O_2$ and $CO_2$ between the atmosphere and the blood, is inadequate.
- Respiratory failure is not a disease but a symptom of underlying pathology affecting lung function. The major threat of ARF is the inability of the lungs to meet the $O_2$ demands of the tissues.
- ARF is classified as hypoxemic or hypercapnic.
  - *Hypoxemic respiratory failure* is often defined as a $PaO_2$ less than or equal to 60 mm Hg when the patient is receiving an inspired $O_2$ concentration greater than or equal to 60%.
    - Common causes include pneumonia, pulmonary edema, pulmonary emboli, heart failure, shock, and alveolar injury related to inhalation of toxic gases.
    - Four main physiologic mechanisms may cause hypoxemia and hypoxemic respiratory failure: V/Q mismatch, shunt, diffusion limitation, and alveolar hypoventilation.

• In *hypercapnic respiratory failure*, the lungs are often normal, and the main problem is ventilatory failure (insufficient $CO_2$ removal).

- It is defined as a $PaCO_2$ greater than 50 mm Hg in combination with acidemia (arterial pH less than 7.35).
- Disorders that compromise $CO_2$ removal include drug overdoses, central nervous system (CNS) depressants, neuromuscular diseases, acute asthma, exacerbation of chronic obstructive pulmonary disease (COPD), and spinal cord injury.

• *Hypoxemia* occurs when the amount of $O_2$ in arterial blood is less than normal. *Hypoxia* occurs when the $PaO_2$ falls enough to cause signs and symptoms of inadequate oxygenation.

- Hypoxemia can lead to hypoxia if not corrected.
- Cyanosis is an unreliable indicator of hypoxemia and the severity of ARF.

Clinical Manifestations

- Signs of respiratory failure are related to the extent of change in $PaO_2$ and/or $PaCO_2$, the rapidity of change (acute versus chronic), and the ability of the body to compensate
- One of the first signs of acute hypoxemic failure is a change in the patient's mental status. Other early signs include tachycardia, tachypnea, slight diaphoresis, and mild hypertension.
- A morning headache and slow respiratory rate suggest hypercapnia.

• Other signs and symptoms that provide information about the patient's respiratory status and work of breathing include:

- Position that the patient assumes
- Patient's ability to speak

- Use of pursed-lip breathing
- Presence of muscle retractions of the intercostal spaces or the supraclavicular area
- Use of the accessory muscles
- Work of breathing (WOB)

• Auscultate breath sounds. Crackles may indicate pulmonary edema and COPD. Absent or decreased breath sounds may occur with atelectasis or pleural effusion. Bronchial breath sounds over the lung periphery result from lung consolidation due to pneumonia.

Diagnostic Studies

• Arterial blood gases (ABGs) are done to identify oxygenation ($PaO_2$) and ventilation ($PaCO_2$) status, and give information about acid-base balance.

• A chest x-ray can help identify possible causes of respiratory failure.

• Other diagnostic studies include a complete blood cell count, serum electrolytes, sputum and blood cultures, urinalysis, and electrocardiogram.

## Nursing and Interprofessional Management: Acute Respiratory Failure

- The overall goals for the patient with ARF include (1) maintain a patent airway, (2) absence of dyspnea or recovery to baseline breathing patterns, (3) effectively cough and able to clear secretions, (4) normal ABG values or values within the patient's baseline, and (5) breath sounds within the patient's baseline.

• Perform a thorough physical assessment and history to identify the patient at risk for ARF, then initiate appropriate interventions.

• The major goals of care for ARF include maintaining adequate oxygenation and ventilation and correcting acid-base imbalance.

• Choose an $O_2$ delivery device with the goal of correcting hypoxemia.

• The general standard for $O_2$ administration is to give the patient the lowest concentration that results in a $PaO_2$ of greater than or equal to 60 mm Hg.

- Retained pulmonary secretions may cause or worsen ARF. They can be mobilized through proper patient positioning, effective coughing, chest physiotherapy, suctioning of the oral airway, humidification, adequate hydration and early mobilization.
- If intensive measures fail to improve ventilation and oxygenation, non-invasive positive pressure ventilation (NIPPV) is provided through a nasal or face mask.

• Goals of drug therapy include to (1) reduce airway inflammation and bronchospasm, (2) relieve pulmonary congestion; (3) treat infection; and (4) reduce anxiety, pain, and restlessness.

- Corticosteroids are used in alone, or, in conjunction with bronchodilators when inflammation and bronchospasm are present.
- Short-acting bronchodilators can reverse bronchospasms.
- Diuretics, nitroglycerin, and/or opioids (e.g., morphine) may decrease pulmonary congestion caused by heart failure.
- IV antibiotics are given if infection is present.
- Give sedation and analgesia at the lowest possible dose for non-intubated patients, to decrease anxiety, restlessness, and pain.

## Gerontologic Considerations: Acute Respiratory Failure

• Multiple factors contribute to an increased risk of ARF in older adults. These include the reduction in ventilatory capacity, decreased respiratory muscle strength, and delayed responses in respiratory rate and depth to falls in $PaO_2$ and rises in $PaCO_2$.

## Acute Respiratory Distress Syndrome

• *Acute respiratory distress syndrome* (ARDS) is a sudden, progressive form of ARF in which the alveolar-capillary membrane becomes damaged and more permeable to intravascular fluid, causing the alveoli to fill with fluid.

• The progression of ARDS varies. Factors that affect the course of ARDS include the nature of the initial injury and the extent and severity of co-existing cardiac and/or respiratory diseases.

• A direct or indirect lung injury can cause ARDS.

• The most common cause of ARDS is sepsis. It may develop because of multiple organ dysfunction syndrome (MODS).

• The pathophysiologic changes of ARDS are divided into 3 phases: injury or exudative, reparative or proliferative, and the fibrotic or chronic phases.

### Clinical Manifestations and Diagnostic Studies

- ARDS diagnostic criteria include: development within 1 week of a known clinical insult or new or worsening respiratory symptoms, chest x-ray with new bilateral opacities, and a low $PaO_2/FIO_2$ (P/F) ratio:
  - Mild ARDS: P/F ratio $\leq$ 300 with PEEP or CPAP $\geq$ 5 cm $H_2O$
  - Moderate ARDS: P/F ratio $\leq$ 200 with PEEP or CPAP $\geq$ 5 cm $H_2O$

- Severe ARDS: P/F ratio < 100 with PEEP or CPAP ≥ 5 cm $H_2O$

• During the injury (exudative) phase, the patient may only have dyspnea, tachypnea, cough, and restlessness. Lung auscultation may be normal or reveal fine, scattered crackles. ABGs in early ARDS usually show mild hypoxemia and respiratory alkalosis caused by hyperventilation.

• In early ARDS, the chest x-ray may be normal or show minimal scattered interstitial infiltrates. As ARDS progresses, diffuse and extensive bilateral opacities (termed "whiteout") are common.

• As ARDS progresses, changes in mental status, tachycardia, hypotension, and severe changes in oxygen, ventilation, and acid-base balance occur. Refractory hypoxemia and profound respiratory distress require intubation and mechanical ventilation.

• Complications include changes in lung function, ventilator associated pneumonia, stress ulcers, VTE, acute kidney injury, and mental health issues.

## Nursing and Interprofessional Management: Acute Respiratory Distress Syndrome

• Collaborative care for the patient with ARF applies to the patient with ARDS.

• Overall goals for a patient with ARDS include $PaO_2$ greater than 60mmHg, a patent airway, clear lungs on auscultation, and absence of any complications.

• Care of the patient with ARDS is multifaceted and requires collaboration of the entire interprofessional team in the ICU.

- General care includes monitoring hemodynamic stability and proper positioning of the patient to promote oxygenation and ventilation. In severe cases, prone positioning may be used.
- Oxygen is administered with the goal of correcting hypoxemia.
- Many patients need mechanical ventilation with a pressure-control type of ventilation.

- Low tidal volume ventilation at 4-8ml/kg helps prevent over-distention of stiff, difficult to inflate lungs, decreasing the risk of barotrauma and volutrauma
- Because of low tidal volume ventilation, $PaCO_2$ will climb.
- Patients asynchronous with mechanical ventilation may, after addressing treatable causes of restlessness, be given analgesia, sedation, and neuromuscular blocking agents (NMBA)
- During positive pressure ventilation, it is common to apply positive end-expiratory pressure (PEEP) to maintain $PaO_2$ at greater than or equal to 60 mm Hg.

• Complications of PEEP include decreases in preload, cardiac output, and impeded venous drainage from the vessels in the head and neck.

- Placing the patient in a prone position helps "recruit" dorsal (posterior) alveoli for oxygenation and ventilation.
- Extracorporeal membrane oxygenation (ECMO) is an advanced therapy that involves cannulation of a major blood vessel, removal of the patient's blood through a catheter, adding $O_2$ and removing carbon dioxide, and re-infusing the blood back to the patient.

• Supportive therapy includes maintaining cardiac output with IV fluids, drugs, or both, keeping the patient normovolemic or on the "dry" side, initiating enteral or parenteral nutrition, and preventing ventilator-associated pneumonia (VAP).

**Answer Keys to Questions**

- Rationales for Bridge to NCLEX Examination Questions

1. **Correct answers**: a, b, d

**Rationale:** Clinical manifestations that occur with hypoxemic respiratory failure include cyanosis, tachypnea, and paradoxical chest or abdominal wall movement with the respiratory cycle. Manifestations of hypercapnic respiratory failure include morning headache, pursed-lip breathing, and decreased respiratory rate with shallow breathing.

2. **Correct answer**: d

**Rationale:** The selected $O_2$ delivery system must be able to deliver oxygen in enough concentration to maintain partial pressure of $O_2$ in arterial blood ($PaO_2$) at 55 to 60 mm Hg or higher and arterial $O_2$ saturation ($SaO_2$) at 90% or higher (at the lowest $O_2$ concentration possible). This might range from a simple face mask to intubation and mechanical ventilation. Several methods are available to provide $O_2$ to patients in ARF. The device selected depends upon the patient's overall physiological condition, degree of respiratory failure, ability to maintain a patent airway, the amount of $FIO_2$ that the device can delivered, and most importantly, the patient's ability to sustain spontaneous ventilation.

3. **Correct answer**: a

**Rationale:** The initial presentation of acute respiratory distress syndrome (ARDS) is often subtle. At the time of the initial injury and for up to 48 hours, the patient may not have respiratory symptoms, or the patient may have only dyspnea, tachypnea, cough, and restlessness.

4. **Correct answer:** b, c, d, e

**Rationale:** Management strategies for patients with ARDS include administration of analgesia and sedation for comfort, ETT tolerance, and to help prevent ventilator dyssynchrony. Management of fluid balance includes maintaining normovolemia or keeping the patient on the

"dry" side. Avoid aggressive IV fluid resuscitation as the lungs are already fluid overloaded. Measures to help reduce ventilator associated pneumonia include head of bed elevated 30 to 45 degrees and, strict infection control measures. Surfactant replacement therapy in adults is experimental at this time.

5. **Correct answer**: c

**Rationale:** Low-tidal volume ventilation helps avoid the risk of volutrauma. The delivery of large tidal volumes of air into stiff, non-compliant lungs is associated with volutrauma and barotrauma. Volutrauma causes damage or tears in the alveoli and movement of fluids and protein into the alveolar spaces. Suctioning the patient is done as needed and has no effect on volutrauma. Increasing PEEP and inspiratory flow increase the risk of barotrauma.

• Answer Guidelines for Case Study in the text.

*1. What is the cause of ARDS for J.N.? Is this a direct or indirect cause?*

The cause for J.N.'s ARDS diagnosis is his perforated colon. This is an indirect cause. The perforated colon would have leaked abdominal contents into the abdominal cavity, causing inflammation and infection. Septic mediators would have entered the bloodstream, travelled to the lungs, where they started the process of lung injury.

*2. How does the pathophysiology of ARDS predispose him to refractory hypoxemia?*

Two basic pathophysiologic processes occur in ARDS contribute to refractory hypoxemia. First, damage to the alveolar-capillary membrane results in increased capillary permeability. This leads to interstitial edema and eventually alveolar edema. It creates an intrapulmonary shunt and severe V/Q mismatch because the alveoli fill with fluid and the blood passing through them

cannot be oxygenated. Secondly, alveolar type I, and, type II cells which produce surfactant, are damaged. Inactivation of surfactant causes the alveoli to become unstable and collapse, resulting in widespread atelectasis. This creates a severe V/Q mismatch. It contributes to hypoxemia because airflow to the alveoli is limited. Hyaline membrane formation on the inner aspect of each alveolus contributes to the development of fibrosis and atelectasis, leading to a decrease in gas-exchange capability and lung compliance. Overall, the severe V/Q mismatch and shunting of pulmonary capillary blood result in hypoxemia unresponsive to increasing concentrations of $O_2$. $O_2$ cannot be delivered to the alveoli nor diffuse across the alveolar-capillary membrane.

*3. What manifestations does J.N. have that support a diagnosis of ARDS?*

Manifestations of ARDS shown by J.N. include refractory hypoxemia (despite increasing levels of $FIO_2$) respiratory acidosis on ABGs, sinus tachycardia, decreased $O_2$ saturation and crackles through-out all lung fields.

*4. Calculate the PaO2/FIO2ratio. What does this value tell you about the seriousness of his condition?*

The PaO2/FiO2 ratio is 59. This shows severe ARDS.

*5. What other complications is J.N at risk for developing from ARDS?*

Multiple complications can result from ARDS including sepsis, pulmonary emboli, stress ulcers, acute kidney injury, dysrhythmias, decreased cardiac output, anemia, thrombocytopenia, disseminated intravascular coagulation (DIC), and multiple organ dysfunction syndrome (see Table 67.7). Complications also occur from treatment of ARDS. They include ventilator-associated pneumonia, pulmonary barotrauma, and laryngeal and tracheal injury from endotracheal intubation. If J.N. survives, there may be mental health concerns.

6. ***Evidence-Based Practice:*** *You are orienting a new nurse who asks you why you allowed J.N.'s family to stay at the bedside during your physical assessment and morning rounds with the ICU team. How would you respond?*

You explain that the American Association of Critical-Care Nurses (AACN) has endorsed the presence of family members during resuscitation and other procedures (e.g., physical assessment). Evidence favors engaging families as important members of the healthcare team. Family presence during procedures and team rounds is desired by families and beneficial to patients, families, and staff. Source: AACN: Family presence during resuscitation and invasive procedures. Retrieved from *www.aacn.org/wd/practice/content/family-presence-practice-alert.pcms?menu=practice*.

7. ***Priority Decision:*** *What priority interventions should be implemented to improve J.N.'s respiratory status and hypoxemia?*

Mechanical ventilation and PEEP are used to keep the lungs partially expanded to prevent the alveoli from totally collapsing. Even with mechanical ventilation and PEEP, it may be necessary to increase the $FIO_2$ >60% to keep the $PaO_2$ ≥60 mm Hg. In this case, prone positioning may promote oxygenation by increasing perfusion to less ventilated alveoli that are "recruited" as a result of changing from supine to prone position.

8. ***Patient-Centered Care:*** *What information would you give to J.N's family about his health status?*

At all times, the patient's family, significant others, and/or caregiver(s) should be informed of all interventions and the patient's response to the interventions. Encourage them to ask questions. Consider having a family conference to bring the caregivers and interprofessional team together to ensure accurate information is communicated about the patient's treatments and prognosis. Finally, arrange for a visit by a clergy member, if appropriate.

9. ***Patient-Centered Care:*** *Given the guidelines in the patient's advance directive, what legal/ethical issues could you encounter?*

The patient has an advance directive that states he does not want to be kept alive by artificial means. Ethically, the interprofessional team must determine the chance of J.N. surviving and fully recovering from this event (i.e., being able to breathe on his own). Should J.N. develop further organ failure, his risk for dying increases. The patient's family may need to determine whether removing the patient from the ventilator is an option that would be honoring his wishes.