Unraveling the mystery of

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Acute respiratory distress syndrome (ARDS) is a life-threatening condition affecting about 190,000 patients a year in the United States, according to the National Heart Lung and Blood Institute. With prompt treatment, 7 out of 10 patients survive. We give you tried-and-true management strategies and nursing interventions and fill you in on what's new in treatment research.

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ARDS was first described in the medical literature in the 1960s as a mysterious condition associated with traumatic injuries and a mortality rate as high as 70%. However, case reports dating back as far as World War I suggested that injuries, including trauma and sepsis, adversely affected lung function. Despite a better understanding of the pathophysiology of ARDS and advances in mechanical ventilation and antibiotic therapy, it remains somewhat of a mystery. Although mortality rates have decreased to around 40%, ARDS is difficult to diagnose and can prove fatal within 48 hours of onset if not promptly diagnosed and treated. Some patients who survive ARDS recover completely, whereas others experience lasting damage to their lungs and other health problems. It's hopeful that the use of new treatment modalities, such as lung protective strategies and permissive hypercapnia, will help lower mortality rates.

In this article, we'll begin by reviewing how ARDS occurs, the pathophysiology behind it, and its diagnostic criteria; then we'll discuss current management strategies and nursing interventions and take a look at the possible future of treatment.

Lung injury gone haywire

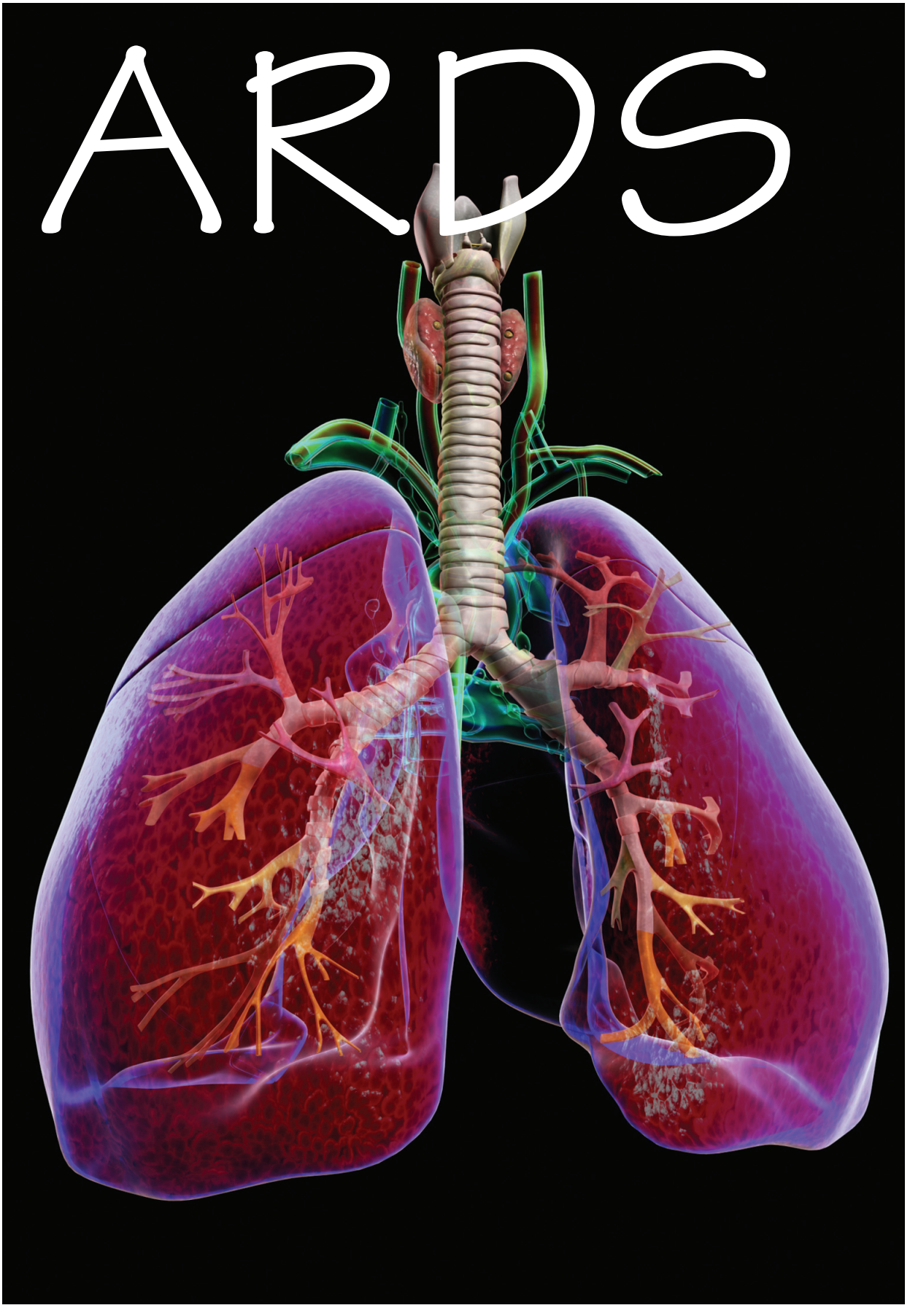
ARDS is a form of pulmonary edema characterized by severe hypoxemia that can rapidly lead to acute respiratory failure. It occurs as the result of a direct or indirect lung injury; it doesn't occur in isolation. Examples of a direct lung injury include:

- gastric aspiration
- bacterial, fungal, or viral pneumonia
- pulmonary contusion
- near drowning
- prolonged inhalation of high concentrations of oxygen, smoke, or toxic substances.

An indirect injury occurs outside the lungs. Examples include:

- sepsis
- shock (any cause)
- drug overdose
- fat embolism
- prolonged hypotension
- nonthoracic trauma
- cardiopulmonary bypass
- head injury

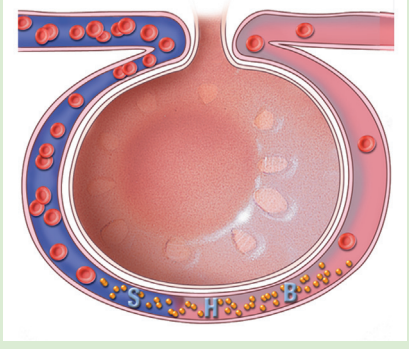
ARDS



A closer look at ARDS

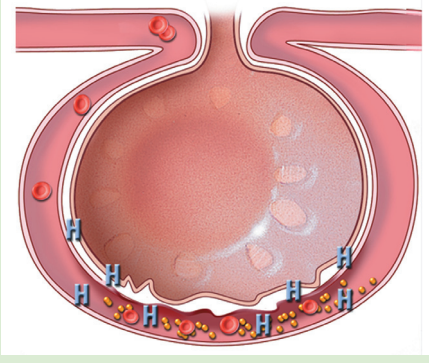
1 Phase

In *phase 1*, injury reduces normal blood flow to the lungs. Platelets aggregate and release histamine (H), serotonin (S), and bradykinin (B).



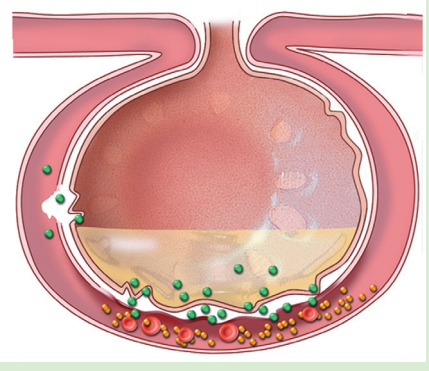
2 Phase

In *phase 2*, those substances—especially histamine—inflame and damage the alveolocapillary membrane, increasing capillary permeability. Fluids then shift into the interstitial space.



3 Phase

In *phase 3*, as capillary permeability increases, proteins and fluids leak out, increasing interstitial osmotic pressure and causing pulmonary edema.



- acute pancreatitis
- uremia
- hematologic disorders, such as disseminated intravascular coagulation, or multiple blood transfusions.

An acute response

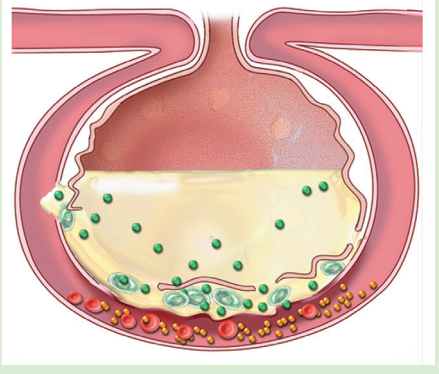
ARDS can be categorized into three main phases: the exudative phase, the proliferative phase, and the fibrotic phase (see *A closer look at ARDS* for a further breakdown of the underlying pathophysiologic phases). Let's take a closer look.

In the ***exudative phase***, an injury to the lungs, whether direct or indirect, leads to an acute inflammatory response, lasting up to 1 week. First, chemical mediators are called to the scene of the injury and released into the systemic circulation, including tumor necrosis factor alpha, numerous interleukins (IL-1; IL-4, 5, and 6; and IL-8), platelet-activating factor, nitric oxide (NO), prostacyclin, oxygen-free radicals, histamine, and bradykinin. These chemical mediators call on leukocytes to connect with cells involved in the injury and destroy the invading organisms. The most important of these leukocytes are neutrophils, which can adhere to the lining of blood vessels, squeeze between endothelial cells, and enter tissue. The neutrophils then phagocytize, or eat, the invading organisms. If further phagocytosis is needed, macrophages move in within 24 hours and take over the job of cleaning up dead tissue and any remaining organisms. However, the presence of macrophages leads to a chronic inflammatory process.

In the ***proliferative phase*** (sometimes called the fibroproliferative phase), the inflammatory process in the lungs occurs systemically throughout all tissues, leading to increased capillary permeability and movement of fluid out of the vascular space and into the tissue. In the lung bed, this leads to pulmonary and interstitial alveolar edema and inactivation of surfactant, which results in alveolar flooding and collapse (atelectasis) and reduced compliance, or stretch, of lung

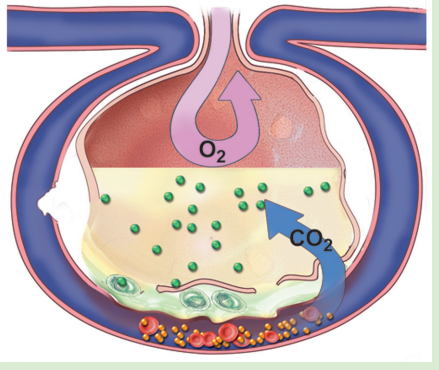
4 Phase

In *phase 4,* decreased blood flow and fluids in the alveoli damage surfactant and impair the cell's ability to produce more. As a result, alveoli collapse, impeding gas exchange and decreasing lung compliance.



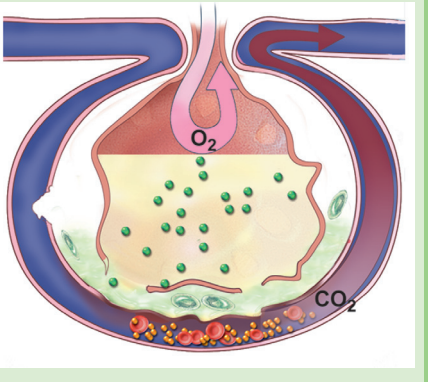
5 Phase

In *phase 5,* sufficient oxygen can't cross the alveolocapillary membrane, but carbon dioxide (CO_2) can and is lost with every exhalation. Oxygen (O_2) and CO_2 levels decrease in the blood.



6 Phase

In *phase 6,* pulmonary edema worsens, inflammation leads to fibrosis, and gas exchange is further impeded.



tissue. Decreased compliance, along with an increased work of breathing, leads to hypoxemia that's unresponsive to increasing levels of supplemental oxygen. The result is pulmonary edema, decreased ability to extract oxygen, and, eventually, cellular death.

In addition to leaky capillaries, the coagulation (clotting) and fibrinolytic (clot breakdown) systems are activated. Clots form in the small capillaries of the lungs. When blood is diverted around these clots, ventilation/perfusion, or V/Q, mismatch occurs because of impaired gas exchange due to decreased ventilation or perfusion at the alveolar capillary membrane, leading to worsening hypoxemia. In addition, small clots are formed all over the body, which leads to multiple organ dysfunction. As the processes of leaking capillaries and clot formation continue over time, the alveoli lose their elastic properties and become fibrotic. This phase can last up to 3 weeks.

In the ***fibrotic phase*** (sometimes called the resolution or recovery phase), the lungs begin to recover. Lung function may continue to improve over a period of 6 to 12 months. Typically, patients suffer long-term effects, such as permanent loss of lung tissue and diminished vital capacity, which lead to impaired pulmonary gas exchange and obstructive and restrictive pulmonary defects. Patients may have difficulty performing activities of daily living for the rest of their lives.

Rapid onset of signs and symptoms

The acute phase of ARDS is marked by rapid onset of severe dyspnea, usually occurring 12 to 48 hours after the initial injury (see *Is the stage set for ARDS?*). The patient will experience arterial hypoxemia that doesn't respond to supplemental oxygen and will have worsening bilateral infiltrates on chest X-ray. The acute lung injury will then progress to fibrosing alveolitis with persistent, severe hypoxemia. He'll also have increased alveolar dead space (ventilation to

the alveoli but poor perfusion) and decreased lung compliance.

Other signs of ARDS include:

- rapid, shallow breathing
- cyanosis
- intercostal retractions
- pulmonary crackles
- rhonchi
- altered mental status
- tachycardia.

With severe ARDS, signs include:

- hypotension
- decreased urine output
- respiratory alkalosis.

Diagnostic criteria

The criteria for establishing a diagnosis of ARDS include the following:

- the occurrence of an acute lung injury or a history of systemic or pulmonary risk factors
- acute onset of respiratory distress
- diffuse, bilateral infiltrates on chest X-ray (ARDS can occur in patients who have

Is the stage set for ARDS?

Timing after initial injury	Physiologic changes	Clinical and diagnostic indicators
Within 24 hours	• Neutrophils and protein-filled fluid leak into the alveoli. • Problems with surfactant production and function occur.	• Mild hypoxemia, dyspnea, and tachypnea, which may be masked by an underlying problem, such as pneumonia or exacerbation of chronic obstructive pulmonary disease • Possibly, evidence of alveolar edema on chest X-ray
Within 48 hours	• Surfactant decreases or becomes ineffective. • Inflammatory mediators and free radicals are released. The endothelium becomes inflamed and the interstitial spaces and capillaries become congested. • The patient develops widespread microatelectasis as his pulmonary microvasculature breaks down.	• Manifestations of acute respiratory failure, including agitation, tachypnea, tachycardia, hypertension or hypotension, and pallor • Patient generally needs endotracheal intubation and mechanical ventilation with PEEP • Increase in oxygen saturation level doesn't correspond to increased oxygen concentration delivered via the ventilator due to loss of functioning alveoli; this is a hallmark of ARDS • Patchy bilateral alveolar infiltrates on chest X-ray • Decreased partial pressure of arterial oxygen (Pao_2) and partial pressure of carbon dioxide in arterial blood ($Paco_2$) levels
2 to 10 days	• The alveoli become consolidated with cellular and fibrin deposits; hyaline membranes develop. • Surfactant production is greatly decreased.	• Manifestations of acute respiratory failure • Possibly, development of systemic inflammatory response syndrome (SIRS) and eventual multisystem organ dysfunction • Hemodynamic instability common, especially hypotension during position changes • Alveolar infiltrates and dependent atelectasis on chest X-ray • Eventually, partial pressure of carbon dioxide (Pco_2) levels rise
10+ days after injury	• Fibroproliferation results in fibrosing alveolitis. • Significant fibrosis may lead to chronic pulmonary fibrosis.	• Fever, continued SIRS • PEEP less effective at maintaining Pao_2 levels

undergone a pneumonectomy and have only one lung.)

- severe refractory hypoxemia, demonstrated by a partial pressure of arterial oxygen (Pa_{O_2})/fraction of inspired oxygen (Fi_{O_2}), or P/F, ratio of less than 200 mm Hg (To calculate the P/F ratio, divide the Pa_{O_2} by the Fi_{O_2}. A normal P/F ratio is greater than 300 mm Hg; with acute lung injury, the P/F ratio is less than 300 mm Hg.)
- no clinical evidence of left-sided heart failure (left atrial hypertension), demonstrated by a pulmonary capillary wedge pressure (PCWP) of less than 18 mm Hg. (In ARDS, a patient may exhibit pulmonary edema, but PCWP must be within normal limits [noncardiogenic pulmonary edema].)

True blue management strategies

Treatment for ARDS primarily involves supportive care in the ICU, including providing adequate oxygenation with avoidance of complications, drug therapy, nutritional support, prone positioning, and permissive hypercapnia.

Supportive therapy almost always includes endotracheal intubation and mechanical ventilation. In the past, patients with ARDS were mechanically ventilated using a high tidal volume, which often resulted in barotrauma (injury or damage to the lung tissue that can lead to entry of air into the pleural space [pneumothorax] or the tracking of air along the vascular bundle to the mediastinum [pneumomediastinum]). Practice changed after the National Heart Lung and Blood Institute ARDS Clinical Network, or ARDSNet, ALVEOLI Study, which showed improved survival rates in patients mechanically ventilated using low tidal volumes. From this study, lung protective strategies were derived.

The goal of lung protective strategies is to protect the lungs from overdistension (volutrauma) and end-expiratory collapse (atelectrauma) by using a low tidal volume, pressure-limited approach with low or moderately high positive end-expiratory pressure (PEEP) (see *Why high ventilator settings are out*). An initial tidal volume of 8 mL/kg is selected and then adjusted every 2 hours based on peak plateau pressure (the measure of pressure in the smaller airways and alveoli). Ideal peak plateau pressure is less than 30 cm H_2O. The goal is to titrate the tidal volume to 6 mL/kg. In addition to adjusting the tidal volume, Fi_{O_2} and PEEP are titrated to achieve an oxygen saturation level of 88% to 95% or a Pa_{O_2} value of 55 to 80 mm Hg at the lowest possible Fi_{O_2}, according to ARDSNet. PEEP should be kept between 5 and 20 cm H_2O.

Although a gold standard medication regimen for ARDS has yet to be developed, antibiotic therapy is often used in the treatment of sepsis-related ARDS or to treat confirmed or suspected underlying infection. A diuretic may be used to increase renal excretion of water, which decreases pulmonary interstitial and alveolar edema. A mechanically ventilated patient may need to be sedated. If sedation doesn't prevent the patient from "fighting" the ventilator, a neuromuscular blocking agent may be considered. The administration of fluids in patients with ARDS has been examined and still remains somewhat controversial. The debate centers around the need for fluid to achieve hemodynamic stability versus the fact that too much fluid may result in worsened pulmonary edema and pulmonary hypertension.

Nutritional support is critical for the patient with ARDS. Because metabolic demand is high, his caloric needs will be increased. Enteral nutrition is preferred; however, parenteral nutrition may also be considered. Adequate calories and protein should be provided,

memory jogger

Use the acronym **ARDS** to remember key treatments.

Antibiotics (if bacterial infection is present)
Respiratory support
Diuretics
Situate the patient in the prone position

Criteria for diagnosis of ARDS

cheat sheet

- Occurrence of an acute lung injury or a history of systemic or pulmonary risk factors
- Acute onset of respiratory distress
- Diffuse, bilateral infiltrates on chest X-ray
- Severe refractory hypoxemia (P/F ratio of less than 200 mm Hg)
- No clinical evidence of left-sided heart failure

including polyunsaturated fatty acids such as gamma linolenic acid, which can assist in decreasing platelet aggregation and the production of pro-inflammatory agents. Enteral nutrition formulas have been developed that provide a large amount of fat calories rather than carbohydrates because the breakdown of carbohydrates results in a surplus of carbon dioxide. Remember, the patient's injured lungs are already working overtime to get rid of carbon dioxide.

Prone positioning is a treatment modality that can be used for mechanically ventilated patients with ARDS who require high FiO_2 levels. Although it may potentially trigger complications, such as pressure ulcers, corneal abrasions, and brachial nerve injury, prone positioning can facilitate blood flow to areas of the lungs that are mildly injured and better oxygenated than those areas that are severely injured. It may also help diaphragm movement. It's recommended that prone positioning be considered early in the course of treatment. Additional research is needed to evaluate whether prone positioning may decrease ventilator days and mortality rates.

Permissive hypercapnia—the allowance of high levels of carbon dioxide in the bloodstream—is a newer management strategy for ARDS. It has been shown to reduce lung injury and is thought to provide a protective mechanism against injury from inflammation. Permissive hypercapnia is achieved by setting low tidal volumes on the ventilator. Research is ongoing to determine at what pH the best outcomes are achieved; currently, the patient's pH is allowed to reach levels as low as 7.2.

What's your role?

A patient with ARDS is critically ill and requires close monitoring in the ICU. Because your patient's condition could quickly become life-threatening, frequent assessment of his status, including arterial blood gas values and hemodynamic parameters, and evaluation of the effectiveness of treatment are necessary. But what else can you do?

Encourage frequent coughing if your patient can cough, which will help loosen excessive airway mucus and maintain open alveoli. If he can't cough, you can suction the airway if your assessment determines it's needed. If he's being mechanically ventilated, be sure to hyperventilate and hyperoxygenate him following your facility's protocol before suctioning the airway. Keep suctioning times as short as possible (less than 10 to 15 seconds), make as few passes with the suction catheter as possible (maximum of two suction passes), and don't interrupt PEEP.

Frequent turning and repositioning has been found to improve ventilation and perfusion in the lungs and enhance secretion drainage. If prone positioning is being used for your patient, closely monitor his response and for deterioration in oxygenation: Moving him from the supine position to the prone position can lead to changes in hemodynamic stability. Be alert to areas of pressure while your patient is in the prone position, including the knees, face, and abdomen. Apply eye lubrication as ordered to prevent corneal abrasions. Use foam support pillows to support your patient's head and face, chest, pelvis, genitals, and dorsa of the feet to reduce the chance of skin breakdown. And take care not to overextend his shoulders to reduce the chance of brachial plexus injury.

Why high ventilator settings are out

Historically, ventilator therapy for ARDS consisted of high tidal volumes (10 to 12 mL/kg) with high PEEP (20 to 25 cm H_2O) to improve oxygen delivery. But high airway pressures commonly caused patients to develop pneumothorax (barotrauma) and decreased venous return contributed to decreased cardiac output.

Research has shown that high tidal volumes and PEEP levels significantly overdistend the alveoli (volutrauma), which triggers release of inflammatory cytokines and decreases surfactant production. In ARDS, these effects exacerbate the underlying disease process.

The current ventilation strategy is to deliver a low tidal volume and low to moderately high PEEP to keep the alveoli open and diminish the negative effects of high-pressure settings.

Monitor your patient for signs and symptoms of cardiovascular compromise, particularly a decreased cardiac output, which may be caused by decreased venous return or because of positive pressure ventilation. Be alert for changes in BP; decreased pulse intensity, oxygen saturation, or urinary output; and mental status changes. Also monitor his lab values, especially the hemoglobin level because an adequate amount of hemoglobin is needed to carry oxygen to the tissues.

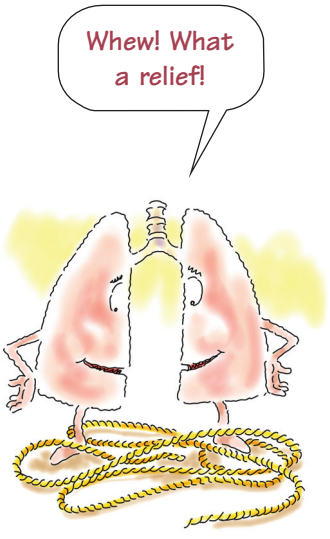
Your patient will be extremely anxious and agitated because of increasing hypoxemia and dyspnea. It's important to decrease his anxiety because anxiety increases heart rate and myocardial oxygen demand. Administer analgesia and sedation as indicated to optimize patient comfort and reduce anxiety.

He'll also be at risk for developing hospital-acquired conditions from invasive devices, such as ventilator-associated pneumonia, catheter-related bloodstream infection, and catheter-associated urinary tract infection. Follow the CDC's guidelines for proper hand hygiene to help prevent infection. To help prevent VAP, implement the Institute for Healthcare Improvement's ventilator bundle: Elevate the head of the bed from 30 to 45 degrees to prevent aspiration, administer peptic ulcer disease prophylaxis to decrease the risk of aspiration and to protect against a greater inflammatory response if aspiration does occur, use daily sedation vacations and assess the patient's readiness for extubation, and provide deep vein thrombosis prophylaxis to decrease the risk of venous thromboembolism.

Looking toward the future

Many new treatments are still being studied, including:

- ***continuous lateral rotational therapy***—involves placing the patient in a bed that turns or repositions him from one side to the other; it's recommended that rotational therapy be performed at least 18 hours/day for optimal effectiveness
- ***partial liquid ventilation***—involves gradually filling the lungs with a fluid called perfluorocarbon, which is believed to help carry oxygen to areas of the lungs that are filled with fluid and other substances and rid the lungs of harmful substances that are preventing the alveoli from opening; the patient must be sedated for this treatment and although it seems promising, improvement in mortality rates hasn't been seen so far
- ***corticosteroids***—the use of steroids to reduce inflammation in ARDS remains controversial; however, recent studies have shown some benefit to low-dose corticosteroid administration
- ***inhaled NO gas***—NO may be used as a rescue therapy for refractory hypoxemia in ARDS because it relaxes vascular smooth muscle, reducing pulmonary hypertension and improving oxygenation; the benefits generally don't last more than 24 hours
- ***surfactant replacement therapy***—this therapy has been used successfully in neonates with respiratory distress syndrome but the effectiveness in adults remains unclear; the use of aerosolized surfactant for adults continues to be studied.



A mystery still being solved

As we learn more about the complex processes associated with ARDS, we'll be able to develop new strategies to help decrease morbidity and mortality in this patient population. Meanwhile, the use of lung protective strategies is still the most effective way to manage patients with ARDS. Although the prognosis isn't always positive, your patient will have a better chance at survival with your help. ■

Learn more about it

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