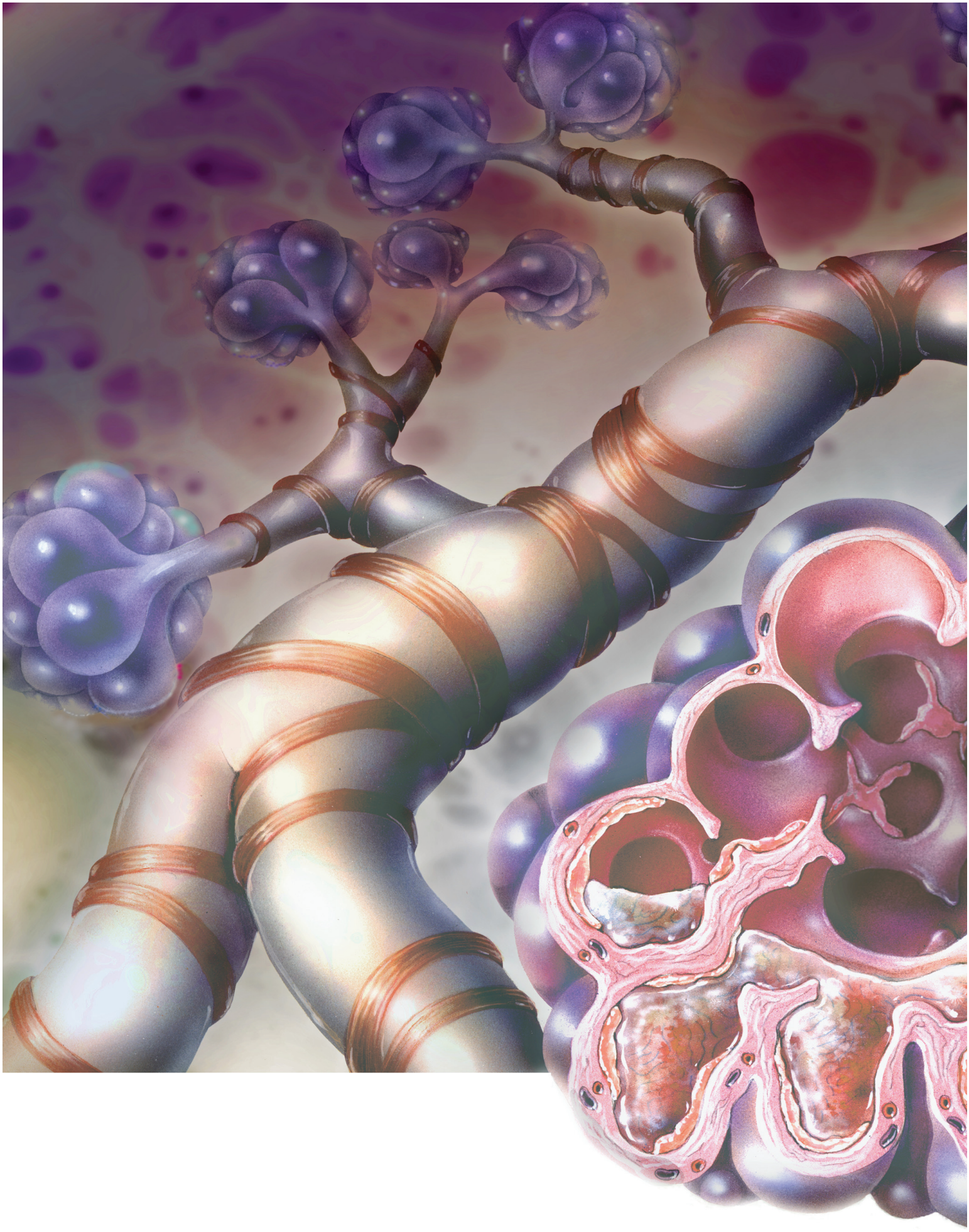

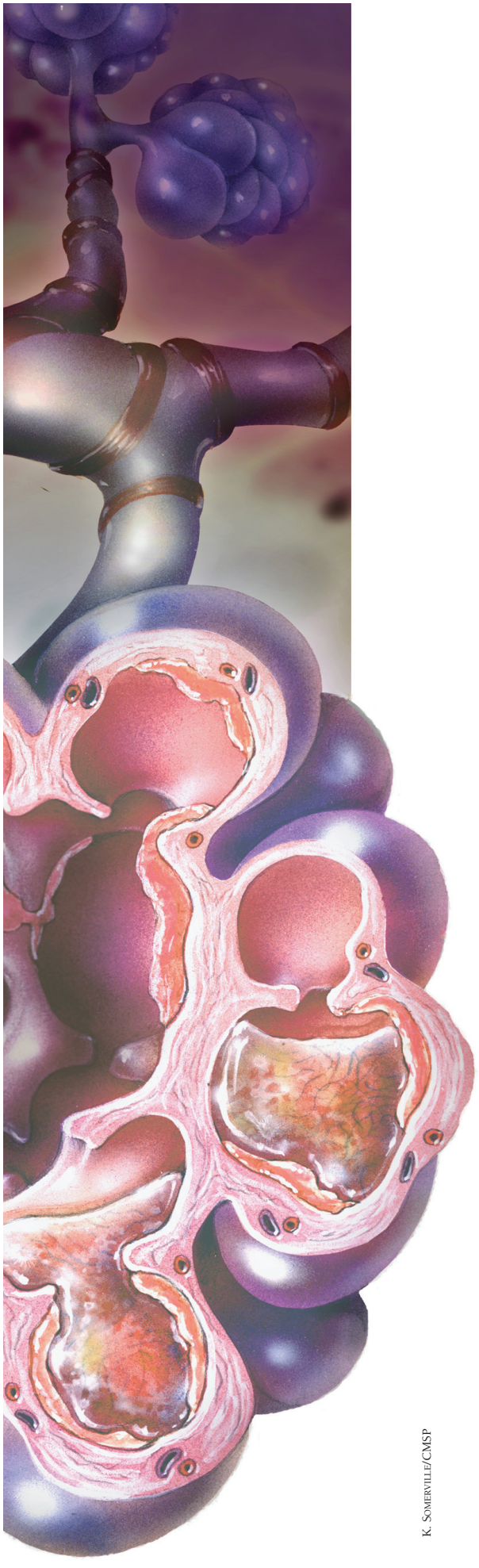

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Taking aim at ARDS

Initiate these best-practice interventions when your patient's critical illness or injury triggers this life-threatening pulmonary complication.

By John J. Gallagher, RN, CCNS, CCRN, RRT, MSN

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AMONG MANY COMPLICATIONS that threaten a critically ill patient is a potentially fatal syndrome of lung inflammation and injury: acute respiratory distress syndrome (ARDS). Whether caused by direct injury to the lungs or indirectly by severe illness, it's characterized by acute hypoxemia, tachypnea, decreased lung compliance, and alveolar collapse.

As advances in critical care interventions have improved, so have survival rates. Ironically, many patients who once would have died from a serious injury or illness now survive long enough to develop deadly complications such as ARDS.

Although our understanding of ARDS has improved, no definitive treatment currently exists, so nursing and medical care remains primarily supportive. This article reviews the pathophysiology, assessment findings, supportive treatment strategies, and nursing care for a patient with ARDS.

What causes ARDS?

Conditions that can activate the systemic inflammatory response syndrome (SIRS) have the potential to cause ARDS. These can be categorized based on whether they directly or indirectly injure the lung.

Direct mechanisms that lead to alveolar inflammation and injury include:

- aspiration of gastric contents
- pneumonia
- toxic inhalation, such as smoke inhalation
- pulmonary contusion or embolism
- oxygen toxicity
- near-drowning
- radiation
- reperfusion injury post–lung transplant.

Indirect mechanisms trigger ARDS from outside the lung through the release of tissue-damaging inflammatory cytokines that travel to the lungs. These causes include sepsis, trauma, massive transfusion, pancreatitis, drug overdose, burns,

disseminated intravascular coagulopathy, and shock.

ARDS is defined by the American-European Consensus Conference as the most severe form of *acute lung injury (ALI)*. Both conditions are defined by acute onset, the presence of bilateral infiltrates on chest X-ray, and absence of left atrial hypertension as evidenced by a pulmonary capillary wedge pressure (PCWP) of less than 18 mm Hg. In the definition for ALI, the partial pressure of oxygen/fraction of inspired oxygen ratio ($Pa_{O_2}/F_{I}O_2$ ratio) is less than or equal to 300 mm Hg. The distinguishing finding of ARDS is a $Pa_{O_2}/F_{I}O_2$ ratio less than or equal to 200 mm Hg.[1]

How ARDS develops

The progress of ARDS can be divided into three phases.

- *Acute exudative phase.* Lasting up to a week, this phase is associated with damage to the alveolar capillary endothelial cells and alveolar epithelial cells, as well as proteinaceous fluid flooding the alveoli. This alveolar edema compromises normal gas exchange and triggers diffuse alveolar collapse. To make matters worse, the fluid also inactivates surfactant, a protein that normally prevents the alveoli from collapsing. The result is worsening hypoxemia that doesn't respond to supplemental oxygen administration.[2]
- *Proliferative phase.* Lasting up to 3 weeks, this phase is marked by resolution of the acute phase and initial repair of the lung. A patient who reaches this phase may recover fully, or move on to the third phase.
- *Fibrotic phase.* In some patients, fibrotic tissue replaces the normal lung structure, generally causing progressive vascular occlusion and pulmonary hypertension. Many patients who progress to this stage require long-term support with mechanical ventilation and supplemental oxygen.[3,4]

Sorting out clinical clues

Key in detecting the onset of ARDS is recognizing those patients at risk for development of this condition. Patients who've experienced direct injury such as aspiration and those with systemic inflammatory conditions such as trauma or sepsis are at significant risk.

Your assessment findings in a patient with ARDS are similar to those found in anyone with respiratory failure: dyspnea, accessory muscle use (intercostal and suprasternal retractions), tachypnea, pallor, and diaphoresis. As you auscultate the patient's lungs, you may hear wheezes and crackles, with decreased breath sounds in all fields. These findings usually occur within hours or days of the initial injury and may even occur after the patient's condition had stabilized.

With worsening hypoxemia, the patient may develop restlessness, apprehension, decreased level of consciousness, motor dysfunction, and tachycardia. If hypoxemia isn't corrected, he may experience respiratory and metabolic acidosis, hypotension, oliguria, and eventually ventricular fibrillation or asystole.

Because ALI/ARDS can develop over hours to several days, patients who are initially stable may develop increasing respiratory distress. Assess your patient for tachypnea and complaints of shortness of breath. Auscultate the lungs to assess for presence of crackles, wheezes, and rhonchi. These findings are consistent with noncardiac pulmonary edema resulting from injury of the alveolar capillary membrane. You may also note increasing oxygen requirements and reduced oxygen saturation by pulse oximetry as lung edema worsens.

If your patient is on a ventilator, watch for increases in the ventilation pressure, also known as peak inspiratory pressure. This will rise as lungs become more congested and airway resistance increases. The patient is at risk for developing a pneumothorax as the lungs become less compliant. Suspect tension pneumothorax in any patient with decreased breath sounds, high-ventilation pressures, and hypotension. Immediate needle decompression of the affected lung followed by placement of a chest tube is the definitive treatment.

How do you know it's ARDS?

Several diagnostic studies may point to ARDS:

- *Chest X-ray* reveals a hallmark finding of bilateral fluffy infiltrates. Serial chest X-rays may be ordered to monitor disease progression and resolution.
- *Arterial blood gases* reveal hypoxemia and initially, respiratory alkalosis (increased arterial pH and decreased Pa_{CO_2}) as a result of tachypnea. As the patient becomes more fatigued, Pa_{CO_2} rises and respiratory acidosis results (low arterial pH with increased Pa_{CO_2} and decreased bicarbonate levels).

A pulmonary artery catheter may provide additional information to assist in diagnosing ARDS. A normal PCWP (less than 18 mm Hg) helps to distinguish ARDS from left atrial hypertension, a condition in which PCWP is elevated. Additionally, parameters such as continuous cardiac output measurement and mixed venous oxygen saturation (Sv_{O_2}) may be helpful in guiding interventions to improve oxygenation and tissue perfusion.

- *Pulse oximetry* may provide additional information as the patient's Sp_{O_2} level drops with progressive hypoxemia. This is a good tool for monitoring oxygenation noninvasively.
- *Lab work* (complete blood count; basic metabolic panel; coagulation studies; serum lactate level; blood, urine, and bronchial cultures; toxicology screen; and serum amylase level can help determine the triggering event, such as sepsis, pneumonia or other infection, shock, drug overdose, or pancreatitis. Lab work can also provide information to support a differential diagnosis.

Treatment options and patient support

Attempts to develop an ARDS-specific treatment have focused on interrupting the inflammatory cascade responsible for lung injury, but none of these treatments have been shown to improve patient outcome.[5] Current strategies are designed to correct the underlying cause of ARDS and support the patient by improving oxygen delivery to tissues and preventing multiorgan failure.

Mechanical ventilation is the mainstay of therapy for a patient with ARDS, but the lung/ventilator interaction is complex. Widespread lung edema and alveolar collapse can reduce normal total lung capacity to a fraction of normal, creating a "baby lung" condition. This makes mechanical ventilation especially challenging because using a standard tidal volume designed for normal lung capacity can overinflate and injure remaining areas of normal lung tissue.[3]

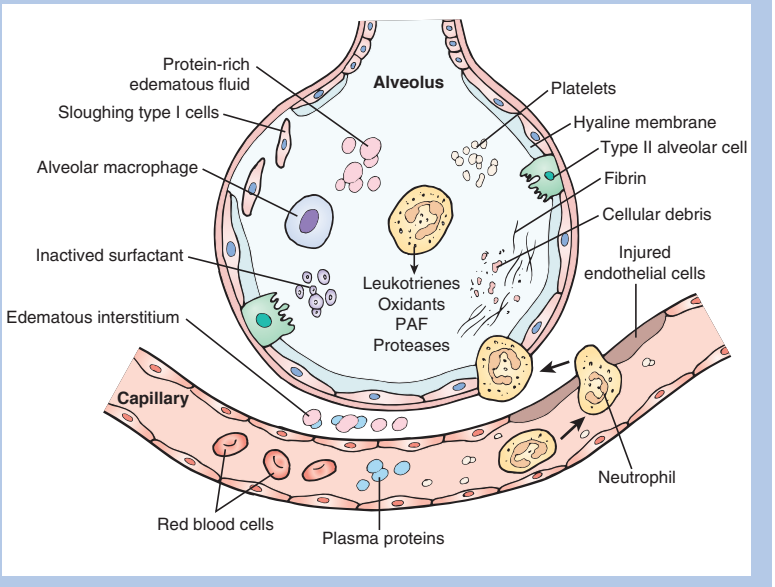
Lung-protective ventilation strategies are essential when you care for a patient with ARDS. To improve oxygenation and eventually restore normal ventilation, focus on two basic protective concepts:

- maintaining a safe level of pressure in the alveoli to prevent overstretch injury
- reexpanding collapsed alveoli and preventing them from collapsing again on exhalation.

Let's look at both these strategies more closely.

How ARDS damages the lungs

In ARDS, injury and increased permeability of the alveolar capillary membrane allow fluid, protein, cellular debris, platelets, and blood cells to move out of the vascular compartment and into the interstitium and alveoli. Activated neutrophils release various products, including oxidants, leukotrienes, proteases, and platelet-activating factor (PAF), that damage the alveoli, inactivate surfactant, and lead to formation of a hyaline membrane.



Source: Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States.* 2nd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2007: 513.

Maintaining safe alveolar pressures

Several ventilation strategies may be used to maintain safe alveolar pressures in a patient with ARDS. They're broadly classified as either volume-controlled modes or pressure-controlled modes. Each has advantages for different patient needs.

Volume-controlled ventilation modes deliver a set or "guaranteed" tidal volume with each ventilator breath. In ALI/ARDS, low tidal volume ventilation has been advocated to reduce or prevent ventilator-induced lung injury.

Low-tidal-volume ventilation involves using tidal volumes of 6 to 8 mL/kg rather than traditional tidal volumes of 10 to 12 mL/kg in order to prevent overstretching the lung. Reducing the tidal volume may cause the patient's $Paco_2$ to rise, but this is an acceptable effect known as permissive hypercapnia.[6]

Pressure-controlled ventilation modes deliver a flow of air into the lungs until a set pressure is achieved, but the tidal volume may vary depending on the compliance (stiffness) of the lungs and airway resistance. The advantage of pressure-controlled ventilation is that the peak pressure is achieved at the beginning of the breath and maintained for the entire inspiratory phase. This allows better expansion of collapsed alveoli while controlling the ventilation pressure.

Volume-assured pressure control modes combine the benefits of guaranteed tidal volumes while safely limiting the pressure required to deliver them. Using pressure-limited volume modes allows a set tidal volume on a breath-to-breath basis, but the ventilator delivers the breath within a set pressure limit by adjusting the speed (flow) with which the breath is delivered. The names of these modes vary according to the ventilator manufacturer. Examples of these modes include pressure-regulated volume control, volume support, volume control plus, pressure augmentation, and adaptive support ventilation.[7]

Pressure-controlled modes may be combined with *inverse-ratio ventilation* to further improve lung function. This involves changing the inspiratory/expiratory ratio of the patient breath from the normal 1:3 (shorter inspiratory phase) to a ratio that lengthens inspiration and shortens exhalation (2:1), so the patient doesn't fully exhale. This positive pressure is also known as *auto-positive end-expiratory pressure (PEEP)* or *intrinsic PEEP*. It's considered useful to prevent alveoli from collapsing while reexpanding collapsed alveoli over time.[7]

Reexpanding collapsed alveoli

Using PEEP helps expand and maintain collapsed alveoli by adding resistance on the exhalation phase of the ventilator breath. The positive resistance is designed to prevent alveoli from collapsing on exhalation. This improves oxygenation independent of increasing the oxygen concentration (F_IO_2) delivered to the patient. Generally, PEEP is set between 5 and

15 cm H_2O, but some patients require higher settings.

Because PEEP has the potential to cause pneumothorax and further lung damage at higher levels, closely monitor peak airway pressures. Watch for potential complications with thorough and frequent respiratory assessments. Be aware that the constant positive pressure in the thorax may reduce venous return to the right side of the heart, reducing cardiac output and causing hypotension. Hemodynamic values such as central venous pressure, pulmonary artery pressure, and PCWP may be falsely elevated from venous back pressure caused by PEEP. Monitor vital signs and hemodynamic values closely for changes related to PEEP settings.

Modes that allow spontaneous breathing

In the past, ventilated patients were often sedated and given paralytic agents to quell the intrinsic desire to breathe, because breathing in tandem with the ventilator can raise pressures and further injure the lungs. Today, thanks to improved ventilation modes, spontaneous breathing can now safely coexist with ventilator-delivered breaths. Advantages to spontaneous breathing include fewer adverse hemodynamic effects from positive pressure, less need for sedation and neuromuscular blockade, and ventilation of lower lung regions that can be achieved only when the diaphragm is engaged in spontaneous breathing.[8]

Similar to the pressure-controlled modes already discussed, these new ventilation modes provide two levels of pressure during the ventilator breath. The upper pressure limit (Pressure Hi) delivers the ventilator breath. At completion of the breath, exhalation occurs down to a set PEEP (Pressure Lo) to prevent alveolar collapse. Most importantly, these modes allow the patient to take spontaneous breaths along with superimposed machine breaths at both Pressure Hi and Pressure Lo. The names of ventilator modes in this category vary among manufacturers. Examples include bilevel ventilation and airway pressure release ventilation.

Other treatment strategies

High-frequency-oscillation ventilation uses a specialized ventilator to deliver very small tidal volumes at rates of more than 150 breaths/minute to maintain safe lung pressures while expanding collapsed alveoli. Though this method improves oxygenation, it calls for specialized equipment and training and hasn't been shown superior to other lung-protective ventilation strategies, so it's not widely used.

In some cases of severe ARDS, the inability to oxygenate the patient with mechanical ventilation may require the use of *extracorporeal membrane oxygenation* (ECMO), which takes over the lungs' gas exchange function through use of a cardiopulmonary bypass machine. Although a successful rescue therapy for some patients, ECMO hasn't gained widespread acceptance in adults because of its complexity and the need for trained staff and other resources, and it's not widely available. In addition, clinical research trials have failed to establish a clear benefit for most patients.[5]

Future developments in ventilation include modes such as *proportional-assist ventilation* designed to support the patient based on the changing work of breathing. Such a mode relies on the development of sensitive electronic patient/ventilator feedback loops that are still being perfected.

Medications play a role

Pharmacologic treatments to manage ARDS aim to reverse bronchoconstriction, vasoconstriction, abnormal coagulation, and the inflammatory processes that damage lung tissue. Let's consider the most commonly ordered medication types.

Pulmonary vasodilators such as prostacyclin and inhaled nitric oxide may be administered through the ventilator circuit to improve oxygenation and reduce pulmonary vascular resistance. Traveling with the ventilator breath to the most easily ventilated alveoli, these drugs cross the alveolar capillary membrane and dilate adjacent capillaries. This improves perfusion to the alveoli, allowing for better ventilation/perfusion matching and blood oxygenation. Also, vasodilation reduces pulmonary vascular resistance, improving blood flow through the lungs. Some studies have shown synergistic improvements in oxygenation when pulmonary vasodilators are combined with prone positioning.

Anti-inflammatory drugs, such as ibuprofen, have been proposed to limit inflammatory lung damage. However, use of these drugs has been primarily investigational.

Drotrecogin alfa (activated), a recombinant form of the natural anticoagulant-activated protein C, was approved by the FDA for use in severe sepsis after it was shown to improve survival. By reducing inflammatory cytokines and leukocyte adhesion and restoring normal balance in the coagulation cascade, it can limit inflammatory lung injury and the development of microclots in the pulmonary capillaries. Other drugs such as ketoconazole have been studied as platelet inhibitors for similar benefits.[9]

N-acetylcysteine and lisofylline, an investigational pentoxifylline derivative, have been suggested to reduce oxygen free radicals that produce lung damage in ARDS, though clinical research hasn't shown them to be effective.[5] More recent studies have been exploring the benefit of $beta_2$-adrenergic agents such as albuterol for their anti-inflammatory benefits and bronchodilator effects.[9]

Glucocorticoids such as methylprednisolone were among the earliest drugs used to treat inflammation in ARDS. To date, their main benefit has been to suppress fibrotic lung changes in the late stage. However, concerns over immunosuppression and potential infections have limited their use.

Surfactant therapy has been proposed to improve alveolar collapse and lung edema. Although successfully used in neonates with surfactant deficiency due to immature lungs, surfactant therapy hasn't proven as beneficial in adults with ARDS.[9]

Benefits of positioning therapy

Patient positioning can help improve lung function in several ways. For example, prone positioning or a specialty bed that provides continuous lateral rotation helps mobilize pulmonary secretions and may improve ventilation/perfusion matching.

Prone positioning provides the most aggressive approach: placing the patient face down improves ventilation in the posterior lung where edema settles when he's supine. Improved ventilation to the posterior regions of the lung allows for better ventilation/perfusion matching, which may improve oxygenation.

Although research has shown that these therapies improve oxygenation and may prevent complications such as atelectasis, improved survival hasn't been demonstrated.[5,9] If your patient is placed in a prone position to improve ventilation, assess for complications such as pressure ulcers, corneal abrasions, and brachial nerve injury. Be alert to areas of pressure while the 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 the head and face, chest, pelvis, genitals, and dorsum of the feet to reduce the chance of skin breakdown. Take care not to overextend the shoulders to reduce the chance of brachial plexus injury.

Administer analgesia and sedation as indicated to optimize patient comfort and reduce anxiety. Though not always required, administration of neuromuscular blocking agents may be necessary to facilitate ventilation while the patient is prone.

Setting care priorities

As you care for a patient with ARDS, keep in mind that the failure of his pulmonary system may herald the onset of multiple organ dysfunction. To prevent organ failure, focus your efforts on improving oxygen delivery to his tissues while limiting unnecessary oxygen consumption.

Begin optimizing oxygen delivery by protecting and maintaining his airway. Take measures to prevent disconnection of the ventilator/airway connection to prevent loss of PEEP. Because suctioning is stressful for the patient, suction only as needed and for as few passes as necessary to clear secretions. Hyperoxygenate him first, using a closed suction system. Avoid routine saline lavage during suctioning because it can worsen oxygenation and contribute to infection; research hasn't shown it to be effective for mobilizing and removing secretions.[10]

Maintain euvolemia to maintain adequate cardiac output for organ perfusion, taking care to avoid fluid volume overload, which would worsen pulmonary edema. Achieving this balance may require cardiac ultrasound or the insertion of invasive hemodynamic monitoring devices, such as arterial and pulmonary artery catheters, to closely track the patient's volume status. Measures to improve oxygen delivery may include transfusion of packed red blood cells and administration of inotropic agents such as dobutamine to improve cardiac muscle contractility and cardiac output.

The inflammatory response associated with ARDS can significantly increase tissue metabolism and oxygen demands.[11] To reduce unnecessary oxygen consumption for your patient, provide medication and care to eliminate controllable stressors such as pain, anxiety, and fever. Plan patient care to allow for uninterrupted rest and minimize stress. For example, limit or space routine care such as bathing and linen changes, dim the lights, and minimize noise.

On guard against complications

A patient with ARDS has a significant risk of complications related to both his underlying lung injury and treatments such as mechanical ventilation. By far, the most common risks are hospital-acquired infections such as ventilator-associated pneumonia (VAP), catheter-associated bloodstream infection, and urinary tract infections. All are related to invasive devices commonly used in critically ill patients.

Along with good hand hygiene, implement targeted evidence-based strategies to prevent infection. Because of pulmonary compromise, your patient is at particular risk for VAP. Diagnosis of new pneumonia is difficult because someone with ARDS typically has a fever and chest X-rays, white blood cell count, and sputum cultures are already abnormal. To help prevent VAP, implement the Institute for Healthcare Improvement's ventilator bundle, which is made up of four components:

- elevating the head of the bed from 30 to 45 degrees to prevent aspiration
- administering peptic ulcer disease prophylaxis to decrease the risk of aspiration and to protect against a greater inflammatory response if aspiration does occur
- using daily "sedation vacations" and assessing the patient's readiness for extubation. This has been shown to reduce the length of time the patient spends on the ventilator and the incidence of VAP.
- providing deep vein thrombosis (DVT) prophylaxis to decrease the risk of venous thromboembolism (VTE).[12]

Other interventions to prevent VAP include providing frequent oral care (to reduce pathologic bacteria and accumulation of oral secretions that may be aspirated), and in some cases, insertion of an endotracheal tube with a subglottic secretion aspiration port.

Besides infection, other complications associated with mechanical ventilation include pneumothorax, reduced cardiac output resulting from increased intrathoracic pressure, fluid retention, peptic ulcer, and malnutrition. Be alert for signs and symptoms that may indicate any of these complications. Monitor vital signs, respiratory status, and hemodynamics closely and report any changes. Be alert for worsening edema, changes in gastric pH, and fluid and electrolyte imbalances.

Hazards of immobility include VTE, pressure ulcers, and muscle deconditioning. Administer prophylaxis for DVT as ordered. Provide meticulous skin care and reposition the patient every two hours as tolerated. If possible,

perform passive range of motion exercises or help the patient perform active range of motion exercises.

Interruptions in the normal sleep/wake cycle associated with the ICU can trigger delirium, which is associated with increased complications and mortality. To catch the earliest signs of delirium, closely observe and document the patient's mental status. Reorient him as needed and provide emotional support.

Nutritional considerations

Any critically ill patient needs adequate nutrition to recover. When he's receiving mechanical ventilation, initiating nutritional support may be delayed and frequently interrupted for diagnostic procedures.

Unless clear contraindications exist, nutritional support via the enteral route should begin as quickly as possible. Advantages of using this route include broader selection of nutritional formulas, maintenance of healthy intestinal mucosa, and prevention of complications associated with parenteral nutrition. Consult with the nutritional support team for nutrition evaluation and recommendations to help optimize nutritional support for your patient.

Armed with a better understanding of ARDS and the necessary support strategies, you're prepared to provide the skilled care your patient needs to recover from this dangerous syndrome. ✧

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