Acute respiratory distress syndrome

Usually, ARDS starts with acute lung injury (ALI), which kills about 75,000 Americans a year and whose risk increases with age.
ARDS itself develops from an injury to the alveoli, the site of gas exchange.
ALI and ARDS have these characteristics in common:
• Acute onset
• Bilateral infiltrates on chest X-ray
• Pulmonary artery wedge pressure (PAWP) of less than 18 mm Hg.
What distinguishes ALI from ARDS is the P/F ratio, the comparison of arterial partial pressure of oxygen (PaO2) with inspired fractional concentration of oxygen (FiO2). Simply put, the P/F ratio is a comparison of the amount of oxygen given to a patient with the amount of oxygen actually entering the patient's bloodstream.
The higher the P/F ratio, the better the gas exchange. The normal measurement is around 500 mm Hg. A P/F ratio below 300 mm Hg regardless of the positive end-expiratory pressure (PEEP) measurement indicates ALI. A P/F ratio below 200 mm Hg regardless of the PEEP measurement indicates ARDS.
If a patient's oxygen requirements continue increasing while oxygen saturation levels (based on finger probe readings and arterial blood gas [ABG] measurements) remain low, ALI is progressing to ARDS.
This condition is called *refractory hypoxemia*. A patient with ARDS has decreased functional residual lung capacity, which may lead to organ failure and death. Typically, ARDS requires admission to an intensive care unit (ICU) and mechanical ventilation.

What's the damage?
ARDS is marked by inflammation, increased permeability of alveoli membranes, and cytokine activation. Damage to the endothelium, or lining of the capillary, interferes with alveolar gas exchange. The damage causes macrophages to release cytokines, inflicting more damage on the alveolar endothelium. Protein levels build, pulling fluid into the alveolar spaces and causing noncardiac pulmonary edema and a reduced surfactant level.
Normally, surfactant decreases surface tension and allows the alveoli to open easily. In ARDS, the edema and reduced surfactant level compromise gas exchange, causing decreased oxygen and increased carbon dioxide in the blood. The result is hypoxemia, pulmonary hypertension, and decreased pulmonary compliance. In later stages of ARDS, progressive alveolitis and fibrosis— stiff lungs—further decrease pulmonary function.

What causes ARDS?
Acute respiratory distress syndrome (ARDS) starts with a lung injury, either direct or indirect.
Direct injuries
With direct injuries, damage results from such events as:
• Near-drowning
• Aspiration
• Chemical inhalation

• Aspiration pneumonia.

Indirect injuries
With indirect injuries, damage results from an inflammatory response to an acute condition, such as:
• Sepsis
• disseminated intravascular coagulation
• Shock
• Pancreatitis
• Multiple trauma.
Cigarette smoking and alcohol abuse can also cause indirect injuries.

Signs and symptoms
In the first 24 to 48 hours, signs and symptoms include dyspnea, tachypnea, dry cough, fatigue, and tachycardia. Even with supplemental oxygen, a patient's skin may look cyanotic and mottled. Auscultation reveals adventitious breath sounds (crackles, rhonchi, and wheezes) or increasingly diminished breath sounds. As oxygenation and perfusion diminish, the patient may become agitated, anxious, confused, and restless.
A chest X-ray shows diffuse infiltrates, and ABG results indicate respiratory alkalosis with very low PaO2 levels. In the later stages, hypercapnia may develop. Further metabolic imbalances can lead to mixed acidosis, signalling a low ventilation- to-perfusion (V /Q) ratio and a deteriorating P/F ratio. To rule out a cardiogenic cause of pulmonary oedema, a physician may order PAWP measurements.

5 P's of ARDS therapy
Managing patients with ARDS requires maintaining the airway, providing adequate oxygenation, and supporting hemodynamic function.
The five P's of supportive therapy include ;

1. Perfusion,
2. Positioning,
3. Protective lung ventilation,
4. Protocol weaning, and
5. Preventing complications.

Perfusion
The goal of care for ARDS patients is to maximize perfusion in the pulmonary capillary system by increasing oxygen transport between the alveoli and pulmonary capillaries.
To achieve the goal, you need to increase fluid volume without overloading the patient. Give either crystalloids or colloids to replace the fluids that have leaked from the capillaries into the alveolar spaces.
Blood transfusions can improve oxygen delivery, but remember they can also cause an increased inflammatory response and increase the risk of infection and death. Evaluate the patient's volume status by measuring blood pressure, respiratory variations of pulmonary and systemic arterial pulse pressure, central venous pressure, and urine output. Confirm intravascular status with pulmonary artery catheter data, cardiac output, cardiac index, pulmonary vascular resistance, and venous oxygen saturation (SvO2).

Certain drugs can also help increase perfusion. Inotropics such as dobutamine (Dobutrex) can increase cardiac output to boost oxygenation. Milrinone lactate (Primacor), another inotropic, improves perfusion by causing vasodilation in the pulmonary bed.
Vasopressors, such as norepinephrine and dopamine, promote systemic vasoconstriction, thus increasing blood pressure and perfusion. When administering these drugs, monitor vital signs, skin color and temperature, and the patient's tolerance to therapy.

Positioning
Patient positioning also affects perfusion. If a patient is standing, blood flow moves to the base of the lung and away from the apex. If a patient is supine, the posterior area of the lung will be more perfused than the anterior area. Because the better aerated surfaces of the lungs are the nondependent areas, the result is a ventilation/perfusion mismatch.
Immobility, a major cause of pulmonary complications, greatly influences perfusion distribution. Three positioning therapies can decrease these complications and improve perfusion in ARDS patients:
• Kinetic Therapy (bilateral turning of a patient 40 degrees or more per side)
• Continuous lateral rotational therapy (bilateral turning of a patient no more than 40 degrees per side)
• Prone positioning.

These therapies improve oxygenation by mobilizing secretions, resolving atelectasis, improving V/Q ratio, recruiting functional but collapsed or consolidated alveolar units, and decreasing interstitial fluid accumulation. Rotational therapy reduces nosocomial pneumonia, skin breakdown, ICU length of stay, and the number of ventilator days.
Kinetic Therapy effectively prevents and treats severe respiratory complications of prolonged immobilization.
When started early, it prevents and treats pneumonia and ARDS, saving hospital resources and lives. Prone positioning improves the V /Q ratio. Aeration improves because the heart no longer compresses the posterior areas of the left lung as it does in the supine position. With the patient in the prone position, most lung tissue, which is in the posterior areas, moves toward the anterior, clearing the airways of debris, decreasing atelectasis, reducing lung inflammation, and producing more efficient oxygenation and perfusion.

In prone position
Turn the patient from the supine **(A)** to the prone **(B)** position with pillows or cushions supporting the chest and pelvis.When the patient is in the prone position, arrange the arms in the swimmer's pose **(C)** and place the headrest appropriately **(D)**. Be sure to align all tubes and drains at the head or foot of the bed to prevent dislodgement.
Disadvantages of prone positioning
Prone positioning does have its share of disadvantages, including possible tube dislodgement, patient desaturation, skin breakdown, and facial edema.

With diligent nursing care and awareness, however, you can prevent or treat most of these complications.

Protective Lung Ventilation

During the early stages of ARDS, use mechanical ventilation to open collapsed alveoli. The primary goal of ventilation is to support organ function by providing adequate ventilation and oxygenation while decreasing the patient's work of breathing. But mechanical ventilation itself can damage the alveoli, making protective lung ventilation necessary.

Current recommendations for protective lung ventilation include:

• limiting plateau pressures to less than 30 cm H_2O
• maintaining PEEP
• reducing FiO_2 to 50% to 60%, if doing so doesn't compromise PaO_2
• providing low VTs (6 ml/kg of ideal body weight).

Protocol weaning

Weaning protocols can reduce the time and cost of care while improving outcomes for ARDS patients. The rule of thumb is: The patient either needs full ventilatory support or should be weaning. ***Evidence-based guidelines suggest the following:***

• Using spontaneous breathing trials instead of synchronous intermittent mechanical ventilation
• Designing and implementing protocols for all appropriate healthcare professionals, not just physicians
• Tailoring protocols not as rigid rules but as guidelines to patient care
• Using protocols to enhance clinical judgment, not replace it
• Using sedation goals to reduce the duration of mechanical ventilation and ICU length of stay.

Preventing complications

The most common complications are VILI, deep vein thrombosis (DVT), pressure ulcers, decreased nutritional status, and ventilatorassociated pneumonia (VAP).

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