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Essentials in Critical Care Medicine

Narendran Selvakumar, Brian Casserly, and Sharon Rounds

INTRODUCTION

Critical care medicine has evolved dramatically with development of new technologies, and clinical trials have established standards for the management of the critically ill. Since the poliomyelitis epidemic in the 1950s, intensive care units have proved beneficial in treating acute, reversible disorders. However, because of the innovative nature of the technology used and the need for close monitoring and intensive management, the delivery of critical care medicine is expensive, accounting for up to 30% of total hospital costs.

Patients in intensive care units (ICUs) are a heterogeneous population treated for diverse conditions ranging from septic shock and respiratory failure to diabetic ketoacidosis and upper gastrointestinal bleeding. This chapter discusses a few of the most common conditions encountered in the ICU setting. Topics include acute respiratory failure and mechanical ventilation, acute lung injury, and shock.

ACUTE RESPIRATORY FAILURE

Acute respiratory failure results when the lung can no longer accomplish adequate gas exchange, a condition that is fatal if left untreated. Hypoxemic respiratory failure refers to respiratory failure associated with failure to oxygenate (type 1), whereas hypercarbic respiratory failure is the failure to ventilate (type 2). These disorders result from alterations in the arterial partial pressures of oxygen (Pao_2) and carbon dioxide ($Paco_2$), respectively.

Type 1 respiratory failure is hypoxia without hypercarbia that may result from interstitial lung diseases (e.g., pneumonia, emphysema), parenchymal diseases due to ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch or diseases of vasculature such as pulmonary embolism. Type 2 respiratory failure is the result of inadequate ventilation from various causes, including airway diseases, decreased respiratory drive, and disorders of the chest wall. Inadequate ventilation results in hypoxia and hypercarbia.

The values of Pao_2 and $Paco_2$ that define respiratory failure are somewhat arbitrary, but respiratory compromise is usually evident when the Pao_2 is less than 60 mm Hg or the $Paco_2$ is higher than 45 mm Hg. These values are not synonymous with the need for mechanical ventilation, and they do not preclude the need for mechanical ventilation.

The management of respiratory failure depends on the clinical presentation. Patients with respiratory failure who are awake, cooperative, and hemodynamically stable may tolerate aggressive respiratory therapy without intubation and mechanical ventilation, as long as gas exchange and overall status are continually monitored. Examples include patients with chronic obstructive pulmonary disease (COPD) who can tolerate $Paco_2$ levels as high as 85 mm Hg without severe respiratory acidosis. In contrast, patients in respiratory failure with evidence of severe respiratory distress (e.g., respiratory rate >30 breaths/min), mental deterioration (e.g., impaired judgment, confusion, hallucinations, somnolence), or hemodynamic instability (e.g., bradyarrhythmias, tachyarrhythmias, hypotension) usually require intubation and mechanical ventilation. In the latter circumstances, waiting for arterial blood gas determinations is unnecessary and may dangerously delay therapy. Although arterial blood gas evaluation is crucial when determining the need for mechanical ventilation in the patient with respiratory failure, the patient's clinical status ultimately dictates the course of action.

For a deeper discussion on this topic, please see Chapter 104, "Acute Respiratory Failure," in Goldman-Cecil Medicine, *25th Edition.*

MECHANICAL VENTILATION

Modern mechanical ventilation is positive-pressure ventilation. Air is forced into the central airways, increasing central airway pressure. Air follows the pressure gradient from the central airways to the alveoli, inflating the lungs. As the lungs inflate and the device stops forcing air into the central airways, the intra-alveolar pressure increases, and central airway pressure decreases. Exhalation occurs when the air follows the newly reversed pressure gradient from the alveoli to the central airways.

The principal benefits of mechanical ventilation during respiratory failure are improved gas exchange and decreased work of breathing. Mechanical ventilation improves gas exchange by improving $\dot{V}/\dot{Q}$ matching. Improved matching of the $\dot{V}/\dot{Q}$ ratio is primarily a consequence of decreased physiologic shunting. Altered lung mechanics (e.g., increased airway resistance, decreased compliance) and increased respiratory demand (e.g., metabolic acidosis) increase the work of breathing. The ventilatory muscles and diaphragm can tire while trying to maintain the elevated work of breathing, resulting in respiratory failure. Mechanical ventilation can alleviate some or all of the increased work of breathing, allowing recovery of fatigued ventilatory muscles. Deteriorating gas exchange, unresponsive to conservative measures, and respiratory distress are the most common