Table 322-1) are nearly twice as likely to die as those with indirect causes of lung injury, while surgical and trauma patients with ARDS—especially those without direct lung injury—have a higher survival rate than other ARDS patients.

An early (within 24 h of presentation) elevation in pulmonary dead space (>0.60) and severe arterial hypoxemia (Pao_2/FIO_2, <100 mmHg) predict increased mortality risk from ARDS; however, there is surprisingly little additional value in predicting ARDS mortality from other measures of the severity of lung injury, including the level of PEEP (≥10 cm H_2O), respiratory system compliance (≤40 mL/cm H_2O), the extent of alveolar infiltrates on chest radiography, and the corrected expired volume per minute (≥10 L/min).

Functional Recovery in ARDS Survivors While it is common for patients with ARDS to experience prolonged respiratory failure and remain dependent on mechanical ventilation for survival, it is a testament to the resolving powers of the lung that the majority of patients recover nearly normal lung function. Patients usually recover maximal lung function within 6 months. One year after endotracheal extubation, more than one-third of ARDS survivors have normal spirometry values and diffusion capacity. Most of the remaining patients have only mild abnormalities in pulmonary function. Unlike mortality risk, recovery of lung function is strongly associated with the extent of lung injury in early ARDS. Low static respiratory compliance, high levels of required PEEP, longer durations of mechanical ventilation, and high lung injury scores are all associated with less recovery of pulmonary function. Of note, when physical function is assessed 5 years after ARDS, exercise limitation and decreased physical quality of life are often documented despite normal or nearly normal pulmonary function. When caring for ARDS survivors, it is important to be aware of the potential for a substantial burden of psychological problems in patients and family caregivers, including significant rates of depression and posttraumatic stress disorder.

WEBSITES

ARDS Support Center for patient-oriented education: *www.ards.org*
NHLBI ARDS Clinical Trials information: *www.ardsnet.org*
ARDS Foundation: *www.ardsusa.org*

Acknowledgment

The authors acknowledge the contribution to this chapter by the previous author, Dr. Steven D. Shapiro.

323 Mechanical Ventilatory Support

Bartolome R. Celli

MECHANICAL VENTILATORY SUPPORT

Mechanical ventilation is used to assist or replace spontaneous breathing. It is implemented with special devices that can support ventilatory function and improve oxygenation through the application of high-oxygen-content gas and positive pressure. The primary indication for initiation of mechanical ventilation is respiratory failure, of which there are two basic types: (1) *hypoxemic*, which is present when arterial O_2 saturation (Sao_2) <90% occurs despite an increased inspired O_2 fraction and usually results from ventilation-perfusion mismatch or shunt; and (2) *hypercarbic*, which is characterized by elevated arterial carbon dioxide partial pressure (PCO_2) values (usually >50 mmHg) resulting from conditions that decrease minute ventilation or increase physiologic dead space such that alveolar ventilation is inadequate to meet metabolic demands. When respiratory failure is chronic, neither of the two types is obligatorily treated with mechanical ventilation, but when it is acute, mechanical ventilation may be lifesaving.

INDICATIONS

The most common reasons for instituting mechanical ventilation are acute respiratory failure with hypoxemia (acute respiratory distress syndrome, heart failure with pulmonary edema, pneumonia, sepsis, complications of surgery and trauma), which accounts for ~65% of all ventilated cases, and hypercarbic ventilatory failure—e.g., due to coma (15%), exacerbations of chronic obstructive pulmonary disease (COPD; 13%), and neuromuscular diseases (5%). The primary objectives of mechanical ventilation are to decrease the work of breathing, thus avoiding respiratory muscle fatigue, and to reverse life-threatening hypoxemia and progressive respiratory acidosis.

In some cases, mechanical ventilation is used as an adjunct to other forms of therapy. For example, it is used to reduce cerebral blood flow in patients with increased intracranial pressure. Mechanical ventilation also is used frequently in conjunction with endotracheal intubation for airway protection to prevent aspiration of gastric contents in otherwise unstable patients during gastric lavage for suspected drug overdose or during gastrointestinal endoscopy. In critically ill patients, intubation and mechanical ventilation may be indicated before the performance of essential diagnostic or therapeutic studies if it appears that respiratory failure may occur during those maneuvers.

TYPES OF MECHANICAL VENTILATION

There are two basic methods of mechanical ventilation: noninvasive ventilation (NIV) and invasive (or conventional mechanical) ventilation (MV).

Noninvasive Ventilation NIV has gained acceptance because it is effective in certain conditions, such as acute or chronic respiratory failure, and is associated with fewer complications—namely, pneumonia and tracheolaryngeal trauma. NIV usually is provided with a tight-fitting face mask or nasal mask similar to the masks traditionally used for treatment of sleep apnea. NIV has proved highly effective in patients with respiratory failure arising from acute exacerbations of chronic obstructive pulmonary disease. It is most frequently implemented as bilevel positive airway pressure ventilation or pressure-support ventilation. Both modes, which apply a preset positive pressure during inspiration and a lower pressure during expiration at the mask, are well tolerated by a conscious patient and optimize patient-ventilator synchrony. The major limitation to the widespread application of NIV has been patient intolerance: the tight-fitting mask required for NIV can cause both physical and psychological discomfort. In addition, NIV has had limited success in patients with acute hypoxemic respiratory failure, for whom endotracheal intubation and conventional MV remain the ventilatory method of choice.

The most important group of patients who benefit from a trial of NIV are those with exacerbations of COPD and respiratory acidosis (pH <7.35). Experience from several randomized trials has shown that, in patients with ventilatory failure characterized by blood pH levels between 7.25 and 7.35, NIV is associated with low failure rates (15–20%) and good outcomes (as judged by intubation rate, length of stay in intensive care, and—in some series—mortality rates). In more severely ill patients with a blood pH <7.25, the rate of NIV failure is inversely related to the severity of respiratory acidosis, with higher failure rates as the pH decreases. In patients with milder acidosis (pH >7.35), NIV is not better than conventional treatment that includes controlled oxygen delivery and pharmacotherapy for exacerbations of COPD (systemic glucocorticoids, bronchodilators, and, if needed, antibiotics).

Despite its benign outcomes, NIV is not useful in the majority of cases of respiratory failure and is contraindicated in patients with the conditions listed in Table 323-1. NIV can delay lifesaving ventilatory support in those cases and, in fact, can actually result in aspiration or hypoventilation. Once NIV is initiated, patients should be monitored; a reduction in respiratory frequency and a decrease in the use of accessory muscles (scalene, sternomastoid, and intercostals) are good clinical indicators of adequate therapeutic benefit. Arterial blood gases should be determined at least within hours of the initiation of therapy to ensure that NIV is having the desired effect. Lack of benefit within