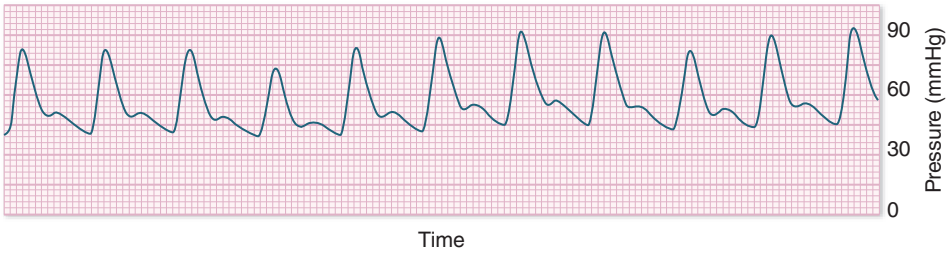

FIGURE 321-4 Pulse pressure change during mechanical ventilation in a patient with shock whose cardiac output will increase in response to intravenous fluid administration. The pulse pressure (systolic minus diastolic blood pressure) changes during mechanical ventilation in a patient with septic shock.

TYPE III RESPIRATORY FAILURE

This form of respiratory failure results from lung atelectasis. Because atelectasis occurs so commonly in the perioperative period, this form is also called *perioperative respiratory failure*. After general anesthesia, decreases in functional residual capacity lead to collapse of dependent lung units. Such atelectasis can be treated by frequent changes in position, chest physiotherapy, upright positioning, and control of incisional and/or abdominal pain. Noninvasive positive-pressure ventilation may also be used to reverse regional atelectasis.

TYPE II RESPIRATORY FAILURE

This type of respiratory failure is a consequence of alveolar hypoventilation and results from the inability to eliminate carbon dioxide effectively. Mechanisms are categorized by impaired central nervous system (CNS) drive to breathe, impaired strength with failure of neuromuscular function in the respiratory system, and increased load(s) on the respiratory system. Reasons for diminished CNS drive to breathe include drug overdose, brainstem injury, sleep-disordered breathing, and severe hypothyroidism. Reduced strength can be due to impaired neuromuscular transmission (e.g., myasthenia gravis, Guillain-Barré syndrome, amyotrophic lateral sclerosis) or respiratory muscle weakness (e.g., myopathy, electrolyte derangements, fatigue).

The overall load on the respiratory system can be subclassified into resistive loads (e.g., bronchospasm), loads due to reduced lung compliance (e.g., alveolar edema, atelectasis, intrinsic positive end-expiratory pressure [auto-PEEP]—see below), loads due to reduced chest wall compliance (e.g., pneumothorax, pleural effusion, abdominal distention), and loads due to increased minute ventilation requirements (e.g., pulmonary embolus with increased dead-space fraction, sepsis).

The mainstays of therapy for type II respiratory failure are directed at reversing the underlying cause(s) of ventilatory failure. Noninvasive positive-pressure ventilation with a tight-fitting facial or nasal mask, with avoidance of endotracheal intubation, often stabilizes these patients. This approach has been shown to be beneficial in treating patients with exacerbations of chronic obstructive pulmonary disease; it has been tested less extensively in other kinds of respiratory failure but may be attempted nonetheless in the absence of contraindications (hemodynamic instability, inability to protect the airway, respiratory arrest).

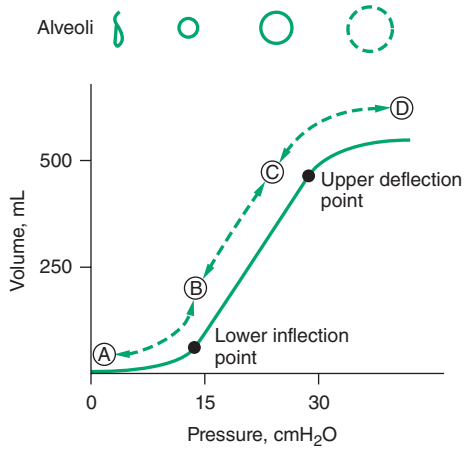

FIGURE 321-5 Pressure-volume relationship in the lungs of a patient with acute respiratory distress syndrome (ARDS). At the lower inflection point, collapsed alveoli begin to open and lung compliance changes. At the upper deflection point, alveoli become overdistended. The shape and size of alveoli are illustrated at the top of the figure.

TYPE IV RESPIRATORY FAILURE

This form results from hypoperfusion of respiratory muscles in patients in shock. Normally, respiratory muscles consume <5% of total cardiac output and oxygen delivery. Patients in shock often experience respiratory distress due to pulmonary edema (e.g., in cardiogenic shock), lactic acidosis, and anemia. In this setting, up to 40% of cardiac output may be distributed to the respiratory muscles. Intubation and mechanical ventilation can allow redistribution of the cardiac output away from the respiratory muscles and back to vital organs while the shock is treated.

CARE OF THE MECHANICALLY VENTILATED PATIENT

(See also Chap. 323) Whereas a thorough understanding of the pathophysiology of respiratory failure is essential for optimal patient care, recognition of a patient's readiness to be liberated from mechanical ventilation is likewise important. Several studies have shown that daily spontaneous breathing trials can identify patients who are ready for extubation. Accordingly, all intubated, mechanically ventilated patients should undergo daily screening of respiratory function. If oxygenation is stable (i.e., Pa_{O_2}/FI_{O_2} [partial pressure of oxygen/fraction of inspired oxygen] >200 and PEEP ≤5 cmH_2O), cough and airway reflexes are intact, and no vasopressor agents or sedatives are being administered, the patient has passed the screening test and should undergo a spontaneous breathing trial. This trial consists of a period of breathing through the endotracheal tube without ventilator support (both continuous positive airway pressure [CPAP] of 5 cmH_2O and an open T-piece breathing system can be used) for 30–120 min. The spontaneous breathing trial is declared a failure and stopped if *any* of the following occur: (1) respiratory rate >35/min for >5 min, (2) O_2 saturation <90%, (3) heart rate >140/min or a 20% increase or decrease from baseline, (4) systolic blood pressure <90 mmHg or >180 mmHg, or (5) increased anxiety or diaphoresis. If, at the end of the spontaneous breathing trial, none of the above events has occurred and the ratio of the respiratory rate and tidal volume in liters (f/V_T) is <105, the patient can be extubated. Such protocol-driven approaches to patient care can have an important impact on the duration of mechanical ventilation and ICU stay. In spite of such a careful approach to liberation from mechanical ventilation, up to 10% of patients develop respiratory distress after extubation and may require resumption of mechanical ventilation. Many of these patients will require reintubation. The use of noninvasive ventilation in patients in whom extubation fails may be associated with worse outcomes than are obtained with immediate reintubation.

Mechanically ventilated patients frequently require sedatives and analgesics. Opiates are the mainstay of therapy for pain control in mechanically ventilated patients. After adequate pain control has been ensured, additional indications for sedation include anxiolysis; treatment of subjective dyspnea; psychosis; facilitation of nursing care; reduction of autonomic hyperactivity, which may precipitate myocardial ischemia; and reduction of total O_2 consumption (V_{O_2}).

Neuromuscular blocking agents are occasionally needed to facilitate mechanical ventilation in patients with profound ventilator dyssynchrony despite optimal sedation, particularly in the setting of severe