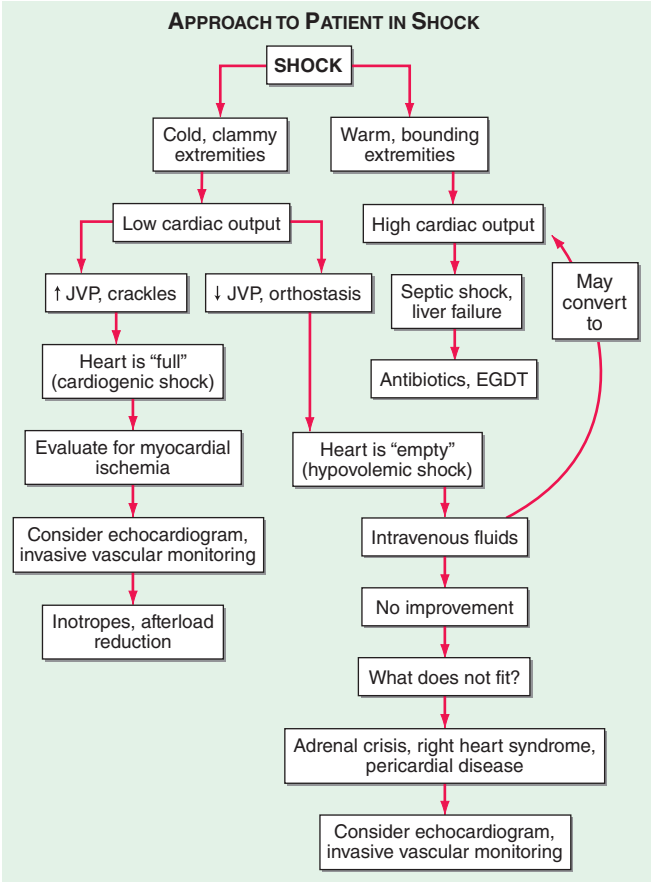

FIGURE 321-2 Approach to the patient in shock. EGDT, early goal-directed therapy; JVP, jugular venous pulse.

As such patients may be obtunded and unable to protect the airway, an early assessment of the airway is mandatory. Early intubation and mechanical ventilation often are required. Reasons for the institution of endotracheal intubation and mechanical ventilation include acute hypoxemic respiratory failure and ventilatory failure, which frequently accompany shock. Acute hypoxemic respiratory failure may occur in patients with cardiogenic shock and pulmonary edema (Chap. 326) as well as in those who are in septic shock with pneumonia or acute respiratory distress syndrome (ARDS) (Chaps. 322 and 325). Ventilatory failure often occurs as a consequence of an increased load on the respiratory system in the form of acute metabolic (often lactic) acidosis or decreased lung compliance due to pulmonary edema. Inadequate perfusion to respiratory muscles in the setting of shock may be another reason for early intubation and mechanical ventilation. Normally, the respiratory muscles receive a very small percentage of the cardiac output. However, in patients who are in shock with respiratory distress, the percentage of cardiac output dedicated to respiratory muscles may increase by tenfold or more. Lactic acid production from inefficient respiratory muscle activity presents an additional ventilatory load.

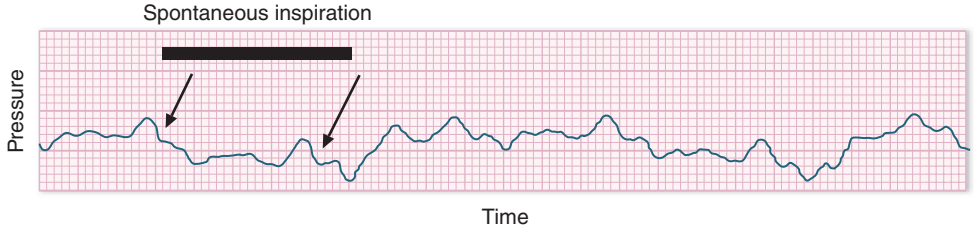

FIGURE 321-3 Right atrial pressure change during spontaneous respiration in a patient with shock whose cardiac output will increase in response to intravenous fluid administration. The right atrial pressure decreases from 7 mmHg to 4 mmHg. The horizontal bar marks the time of spontaneous inspiration.

Mechanical ventilation may relieve the work of breathing and allow redistribution of a limited cardiac output to other vital organs. Patients demonstrate respiratory distress by an inability to speak full sentences, accessory use of respiratory muscles, paradoxical abdominal muscle activity, extreme tachypnea (>40 breaths/min), and decreasing respiratory rate despite an increasing drive to breathe. When patients with shock are treated with mechanical ventilation, a major goal is for the ventilator to assume all or the majority of the work of breathing, facilitating a state of minimal respiratory muscle work. With the institution of mechanical ventilation for shock, further declines in MAP are frequently seen. The reasons include impeded venous return from positive-pressure ventilation, reduced endogenous catecholamine secretion once the stress associated with respiratory failure abates, and the actions of drugs used to facilitate endotracheal intubation (e.g., propofol, opiates). Accordingly, hypotension should be anticipated during endotracheal intubation. Because many of these patients may be fluid responsive, IV volume administration should be considered. Figure 321-2 summarizes the diagnosis and treatment of different types of shock **For further discussion of individual forms of shock, see Chaps. 324, 325, and 326.**

RESPIRATORY FAILURE

Respiratory failure is one of the most common reasons for ICU admission. In some ICUs, ≥75% of patients require mechanical ventilation during their stay. Respiratory failure can be categorized mechanistically on the basis of pathophysiologic derangements in respiratory function.

TYPE I: ACUTE HYPOXEMIC RESPIRATORY FAILURE

This type of respiratory failure occurs with alveolar flooding and subsequent intrapulmonary shunt physiology. Alveolar flooding may be a consequence of pulmonary edema, pneumonia, or alveolar hemorrhage. Pulmonary edema can be further categorized as occurring due to elevated pulmonary microvascular pressures, as seen in heart failure and intravascular volume overload or ARDS ("low-pressure pulmonary edema," Chap. 322). This syndrome is defined by acute onset (≤1 week) of bilateral opacities on chest imaging that are not fully explained by cardiac failure or fluid overload and of shunt physiology requiring positive end-expiratory pressure (PEEP). Type I respiratory failure occurs in clinical settings such as sepsis, gastric aspiration, pneumonia, near-drowning, multiple blood transfusions, and pancreatitis. The mortality rate among patients with ARDS was traditionally very high (50–70%), although changes in patient care have led to mortality rates closer to 30% (see below).

For many years, physicians have suspected that mechanical ventilation of patients with ARDS may propagate lung injury. Cyclical collapse and reopening of alveoli may be partly responsible for this adverse effect. As seen in Fig. 321-5, the pressure-volume relationship of the lung in ARDS is not linear. Alveoli may collapse at very low lung volumes. Animal studies have suggested that stretching and overdistention of injured alveoli during mechanical ventilation can further injure the lung. Concern over this alveolar overdistention, termed *ventilator-induced "volutrauma,"* led to a multicenter, randomized, prospective trial comparing traditional ventilator strategies for ARDS (large tidal volume: 12 mL/kg of ideal body weight) with a low tidal volume (6 mL/kg of ideal body weight). This study showed a dramatic reduction in mortality rate in the low-tidal-volume group from that in the high-tidal-volume group (31% versus 39.8%). In addition, a "fluid-conservative" management strategy (maintaining a low central venous pressure [CVP] or pulmonary capillary wedge pressure [PCWP]) is associated with fewer days of mechanical ventilation than a "fluid-liberal" strategy (maintaining a relatively high CVP or PCWP) in ARDS.