pathophysiologic change. Increased secretions seen in asthma often lead to atelectasis and hypoxia, whereas restrictions of expiratory airflow may lead to hypercarbia. Progression to respiratory failure results from peripheral airway obstruction, extensive atelectasis, and resultant hypoxemia and retention of CO_2.

TREATMENT

Initial treatment of patients in respiratory distress includes addressing the ABCs (see Chapter 38). **Bag/mask ventilation** must be initiated for patients with apnea. In other patients, oxygen therapy is administered using appropriate methods (e.g., simple mask). Administration of oxygen by nasal cannula allows the patient to entrain room air and oxygen, making it an insufficient delivery method for most children in respiratory failure. Delivery methods, including intubation and mechanical ventilation, should be escalated if there is inability to increase oxygen saturation appropriately.

Patients presenting with hypercarbic respiratory failure are often hypoxic as well. When oxygenation is established, measures should be taken to address the underlying cause of hypercarbia (reversal of drug action, control of fever, or seizures). Patients who are hypercarbic without signs of respiratory fatigue or somnolence may not require intubation based on the P_{CO_2} alone; however, patients with marked increase in the work of breathing or inadequate respiratory effort may require assistance with ventilation.

After identification of the etiology of respiratory failure, specific interventions and treatments are tailored to the needs of the patient. External support of oxygenation and ventilation may be provided by **noninvasive ventilation** methods (heated humidified high-flow nasal cannula, continuous positive airway pressure, biphasic positive airway pressure, or negative pressure ventilation) or through invasive methods (traditional **mechanical ventilation,** high-frequency oscillatory ventilation, or extracorporeal membrane oxygenation). Elimination of CO_2 is achieved through manipulation of minute ventilation (tidal volume and respiratory rate). Oxygenation is improved by altering variables that affect oxygen delivery (fraction of inspired oxygen) or mean airway pressure (PEEP, peak inspiratory pressure, inspiratory time, gas flow).

COMPLICATIONS

The major complication of hypoxic respiratory failure is the development of organ dysfunction. **Multiple organ dysfunction** includes the development of two or more of the following: respiratory failure, cardiac failure, renal insufficiency/failure, gastrointestinal or hepatic insufficiency, disseminated intravascular coagulation, and hypoxic-ischemic brain injury. Mortality rates increase with increasing numbers of involved organs (see Table 38-3).

Complications associated with mechanical ventilation include pressure-related and volume-related lung injury. Both overdistention and insufficient lung distention (loss of functional residual capacity) are associated with lung injury. Pneumomediastinum and pneumothorax are potential complications of the disease process and overdistention. Inflammatory mediators may play a role in the development of chronic fibrotic lung diseases in ventilated patients.

PROGNOSIS

Prognosis varies with the etiology of respiratory failure. Fewer than 1% of previously healthy children with bronchiolitis die. Asthma mortality rates, although still low, have increased. Despite advances in support and understanding of the pathophysiology of ARDS, the mortality rate remains approximately 30%.

PREVENTION

Prevention strategies are explicit to the etiology of respiratory failure. Some infectious causes can be prevented through active immunization against organisms causing primary respiratory disease (pertussis, pneumococcus, *Haemophilus influenzae* type b) and sepsis (pneumococcus, *H. influenzae* type b). Passive immunization with respiratory syncytial virus immunoglobulins prevents severe illness in highly susceptible patients (prematurity, bronchopulmonary dysplasia). Primary prevention of traumatic injuries may decrease the incidence of ARDS. Compliance with appropriate therapies for asthma may decrease the number of episodes of respiratory failure (see Chapter 78).

Chapter 40

SHOCK

ETIOLOGY AND EPIDEMIOLOGY

Shock is the inability to provide sufficient perfusion of oxygenated blood and substrate to tissues to meet metabolic demands. **Oxygen delivery** is directly related to the arterial oxygen content (oxygen saturation and hemoglobin concentration) and to cardiac output (stroke volume and heart rate). Changes in metabolic needs are met primarily by adjustments in cardiac output. Stroke volume is related to myocardial end-diastolic fiber length (preload), myocardial contractility (inotropy), and resistance of blood ejection from the ventricle (afterload) (see Chapter 145). In a young infant whose myocardium possesses relatively less contractile tissue, increased demand for cardiac output is met primarily by a neurally mediated increase in heart rate. In older children and adolescents, cardiac output is most efficiently augmented by increasing stroke volume through neurohormonally mediated changes in vascular tone, resulting in increased venous return to the heart (increased preload), decreased arterial resistance (decreased afterload), and increased myocardial contractility.

HYPOVOLEMIC SHOCK

Acute hypovolemia is the most common cause of shock in children. It results from loss of fluid from the intravascular space secondary to inadequate intake or excessive losses (vomiting and diarrhea, blood loss, capillary leak syndromes, or pathologic renal fluid losses) (Table 40-1). Reduced blood volume decreases preload, stroke volume, and cardiac output. Hypovolemic shock results in increased sympathoadrenal activity, producing an increased heart rate and enhanced myocardial