Chapter 39

RESPIRATORY FAILURE

ETIOLOGY

Acute respiratory failure occurs when the pulmonary system is unable to maintain adequate gas exchange to meet metabolic demands. The resulting failure can be classified as hypercarbic ($Paco_2$ >50 mm Hg in previously healthy children), hypoxemic (Pao_2 <60 mm Hg in previously healthy children without an intracardiac shunt), or both. **Hypoxemic respiratory failure** is frequently caused by ventilation-perfusion mismatch (perfusion of lung that is not adequately ventilated) and shunting (deoxygenated blood bypasses ventilated alveoli). **Hypercarbic respiratory failure** results from inadequate alveolar ventilation secondary to decreased minute ventilation (tidal volume × respiratory rate) or an increase in dead space ventilation (ventilation of areas receiving no perfusion).

Respiratory failure may occur with **acute lung injury (ALI)** or **acute respiratory distress syndrome (ARDS)**. The definitions of these are in the process of revision; however, currently ALI is defined as having the following four clinical features: acute onset, bilateral pulmonary edema, no clinical evidence of elevated left atrial pressure, and a ratio of Pao_2 to $Fio_2 \leq 300$ mm Hg regardless of the level of positive end-expiratory pressure (PEEP). ARDS is a subset of ALI with more severe hypoxemia (Pao_2/Fio_2 of ≤ 200 mm Hg). These syndromes can be triggered by a variety of insults, including sepsis, pneumonia, shock, burns, or traumatic injury, all resulting in inflammation and increased vascular permeability leading to pulmonary edema. Numerous mediators of inflammation (tumor necrosis factor, interferon-γ, nuclear factor κB, and adhesion molecules) may be involved in the development of ARDS. Surfactant action also may be affected.

EPIDEMIOLOGY

Respiratory failure is frequently caused by bronchiolitis (often caused by respiratory syncytial virus), asthma, pneumonia, upper airway obstruction, and sepsis/ARDS. Respiratory failure requiring mechanical ventilation develops in 7% to 21% of patients hospitalized for respiratory syncytial virus.

Asthma is increasing in prevalence and is the most common reason for unplanned hospital admissions in children 3 to 12 years of age in the United States. Environmental factors (exposure to cigarette smoke) and prior disease characteristics (severity of asthma, exercise intolerance, delayed start of therapy, and previous intensive care unit admissions) affect hospitalization and near-fatal episodes. The mortality rate of asthma for children younger than 19 years of age has increased by nearly 80% since 1980. Deaths are more common in African-American children.

Chronic respiratory failure (with acute exacerbations) is often due to chronic lung disease (bronchopulmonary dysplasia, cystic fibrosis), neurologic or neuromuscular abnormalities, and congenital anomalies.

CLINICAL MANIFESTATIONS

Early signs of hypoxic respiratory failure include **tachypnea and tachycardia** in attempt to improve minute ventilation and cardiac output and to maintain delivery of oxygenated blood to the tissues. Further progression of disease may result in dyspnea, nasal flaring, grunting, use of accessory muscles of respiration, and diaphoresis. Late signs of inadequate oxygen delivery include **cyanosis** and **altered mental status** (initially confusion and agitation). Signs and symptoms of hypercarbic respiratory failure include attempts to increase minute ventilation (tachypnea and increased depth of breathing) and altered mental status (somnolence).

LABORATORY AND IMAGING STUDIES

A chest radiograph may show evidence of the etiology of respiratory failure. The detection of atelectasis, hyperinflation, infiltrates, or pneumothoraces assists with ongoing management. Diffuse infiltrates or pulmonary edema may suggest ARDS. The chest radiograph may be normal when upper airway obstruction or impaired respiratory controls are the etiology. In patients presenting with stridor or other evidence of upper airway obstruction, a lateral neck film or computed tomography (CT) may delineate anatomic defects. Direct visualization through flexible bronchoscopy allows identification of dynamic abnormalities of the anatomic airway. Helical CT helps diagnose a pulmonary embolus.

Pulse oximetry allows noninvasive, continuous assessment of oxygenation but is unable to provide information about ventilation abnormalities. Determination of CO_2 levels requires a blood gas measurement (arterial, venous, or capillary). An **arterial blood gas** allows measurement of CO_2 levels and analysis of the severity of oxygenation defect through calculation of an alveolar-arterial oxygen difference. A normal Pco_2 in a patient who is hyperventilating should heighten concern about the risk of further deterioration.

DIFFERENTIAL DIAGNOSIS

Hypoxic respiratory failure resulting from impairment of alveolar-capillary function is seen in ARDS; cardiogenic pulmonary edema; interstitial lung disease; aspiration pneumonia; bronchiolitis; bacterial, fungal, or viral pneumonia; and sepsis. It also can be due to intracardiac or intrapulmonary shunting seen with atelectasis and embolism.

Hypercarbic respiratory failure can occur when the respiratory center fails as a result of drug use (opioids, barbiturates, anesthetic agents), neurologic or neuromuscular junction abnormalities (cervical spine trauma, demyelinating diseases, anterior horn cell disease, botulism), chest wall injuries, or diseases that cause increased resistance to airflow (croup, vocal cord paralysis, post-extubation edema). Maintenance of ventilation requires adequate function of the chest wall and diaphragm. Disorders of the neuromuscular pathways, such as muscular dystrophy, myasthenia gravis, and botulism, result in inadequate chest wall movement, development of atelectasis, and respiratory failure. Scoliosis rarely results in significant chest deformity that leads to restrictive pulmonary function. Similar impairments of air exchange may result from distention of the abdomen (postoperatively or due to ascites, obstruction, or a mass) and thoracic trauma (flail chest).

Mixed forms of respiratory failure are common and occur when disease processes result in more than one