position places pressure on bony prominences in the sacrum and hips, pressure areas must be relieved at intervals. Most of the care of PE occurs in the ICU, which is anxiety provoking for the child and family. (For other nursing care activities, see the Acute Respiratory Distress Syndrome section.)

Acute Respiratory Distress Syndrome

ARDS is a potentially life-threatening inflammatory lung condition that may occur in both children and adults. The syndrome may be caused by direct injury to the lungs or by systemic insults that lead indirectly to lung injury with subsequent hypoxemia and respiratory failure due to non-cardiogenic PE. Sepsis, trauma, viral pneumonia, aspiration, fat emboli, drug overdose, reperfusion injury after lung transplantation, smoke inhalation, and near-drowning, among others, have been associated with ARDS. Mechanical ventilation is often required.

The diagnostic criteria established by the American European Consensus Conference (Bernard, Artigas, Brigham, et al, 1994) have been superseded by the Berlin definition of ARDS (ARDS Definition Task Force, Ranieri, Rubenfeld, et al, 2012). According to the Berlin definition, ARDS occurs within 1 week of a known clinical insult or new or worsening respiratory symptoms, is characterized by bilateral opacities on chest imaging not fully explained by effusions, lobar/lung collapse or nodules, and manifests as respiratory failure not fully explained by cardiac failure or fluid overload (ARDS Definition Task Force, Ranieri, Rubenfeld, et al, 2012). Hypoxemia is expressed in terms of the ratio of partial pressure of oxygen (PaO_2) to the fraction of inspired oxygen (FiO_2) or P/F ratio. In the setting of a PEEP or CPAP ≥5 cm H_2O, mild, moderate and severe ARDS are defined by P/F ratios between 200 and 300, between 100 and 200, and ≤100 mm Hg, respectively.

Pathologically, the hallmark of ARDS is increased permeability of the alveolar-capillary membrane. During the acute phase of ARDS, inflammatory mediators cause damage to the alveolocapillary membrane, with an increasing pulmonary capillary permeability with resulting interstitial edema. Later stages are characterized by pneumocyte and fibrin infiltration of the alveoli, with the start of