in PVR, fetal shunts (ductus arteriosus and foramen ovale) remain open allowing right-to-left shunting of blood through the persisting fetal shunts.

Inadequate pulmonary perfusion and ventilation produce hypoxemia and hypercapnia. Pulmonary arterioles, with their thick muscular layer, constrict in response to hypoxia. Thus, a decrease in oxygen tension causes vasoconstriction in the pulmonary arterioles that is further enhanced by a decrease in blood pH. This vasoconstriction contributes to a further increase in PVR.

Prolonged hypoxemia activates anaerobic glycolysis, which produces increased amounts of lactic acid. An increase in lactic acid causes metabolic acidosis; an inability of the atelectatic lungs to blow off excess carbon dioxide produces respiratory acidosis. Acidosis causes further vasoconstriction. With deficient pulmonary circulation and alveolar perfusion, partial pressure of oxygen in arterial blood continues to fall, pH falls, and the materials needed for surfactant production are not circulated to the alveoli.

Diagnostic Evaluation

The diagnosis of RDS is made on the basis of clinical signs (Box 8-4) and chest x-ray studies. Radiographic findings characteristic of RDS include (1) a diffuse granular pattern over both lung fields that closely resembles ground glass and represents alveolar atelectasis and (2) dark streaks, or bronchograms, within the ground glass areas that represent dilated, air-filled bronchioles. It is difficult to distinguish between RDS and pneumonia in infants with respiratory distress. The extent of respiratory compromise and acid–base status is determined by blood gas analysis. Criteria for visually evaluating the degree of respiratory distress are illustrated in Fig. 8-21. Pulse oximetry and carbon dioxide monitoring, as well as pulmonary function studies, assist in differentiating pulmonary and extrapulmonary illness and are used in the management of RDS.

Quality Patient Outcomes

Neonatal Respiratory Distress Syndrome