respiratory passages, resulting in uniform expansion and maintenance of lung expansion at low intraalveolar pressure (Fig. 8-20). Deficient surfactant production causes unequal inflation of alveoli on inspiration and the collapse of alveoli on end expiration. Without surfactant, infants are unable to keep their lungs inflated and therefore exert a great deal of effort to reexpand the alveoli with each breath. With increasing exhaustion, infants are able to open fewer and fewer alveoli. This inability to maintain lung expansion produces widespread atelectasis.

**FIG 8-20** Preterm infant who is Rh positive born to an Rh-negative mother who received intrauterine transfusions and is receiving postnatal transfusions.

Following birth the oxygen concentration in the blood normally increases, the ductus arteriosus constricts and the pulmonary vessels dilate to decrease PVR. In the absence of alveolar stability (normal functional residual capacity) and with progressive atelectasis, PVR increases as resistance to blood flow into the lungs increases hypoperfusion to the lung tissue occurs. With the increase