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Discussion of some of the questions follows:
(1 – 3) Persistent pulmonary hypertension occurs in term infants who were asphyxiated
at birth. The heart and lungs are normal on chest roentgenogram. Metabolic acidosis
follows the hypoxemia. Normal temperature will reduce oxygen needs. Hyperviscosity
is common and reduction will improve perfusion. Tolazoline is used to dilate the
pulmonary vasculature. The patient is not in cardiac failure, and ventilation without
oxygen is of little value. Reducing ductal shunting is counterproductive
(4,5) In severe RDS, there is hyperventilation, minimal response to ventilation, and
hypercarbia. There often is ductal shunting as well as impaired diffusion, alveolar
ventilation, and perfusion differences. Due to the acidosis, the dissociation curve
will be shifted to the right (lower saturation).
(6) Pneumonia, hypoglycemia, and CNS hemorrhage may all produce cyanosis. PDA
with persistence and subsequent cardiac failure is more common in low-birth-weight
infants. Transient tachypnea in term infants occurs by 3 to 4 days of life, and is due
to slow reabsorption of lung fluid.
(7) You would anticipate a response to breathing 100% oxygen in conditions due to
alveolar hyperventilation (cardiac failure, CNS hemorrhage). Paco2 would not
be normal in pneumonia. Methemoglobinemia would respond to oxygen administration
but a cardiac shunt would not.
(8) In iron deficiency anemia the red blood cells are microcytic and hypochromic, the
bone marrow shows erythroid hyperplasia. The serum iron is reduced while Coombs
reaction is negative and hemoglobin electrophoresis is normal. The findings in TEC are
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