The most common blood group incompatibility in the neonate is between a mother with O blood group and an infant with A or B blood group (see Table 8-3 for possible ABO incompatibilities). Naturally occurring anti-A or anti-B antibodies already present in the maternal circulation cross the placenta and attack the fetal RBCs, causing hemolysis. Usually, the hemolytic reaction is less severe than in Rh incompatibility; however, rare cases of hydrops have been reported (Black and Maheshwari, 2009). Unlike the Rh reaction, ABO incompatibility may occur in the first pregnancy. The risk of significant hemolysis in subsequent pregnancies is higher when the first pregnancy is complicated by significant hemolysis (Shamsi, Hossain, and Paidas, 2011).

TABLE 8-3

Potential Maternal–Fetal ABO Incompatibilities

Maternal Blood Group	Incompatible Fetal Blood Group
O	A or B
B	A or AB
A	B or AB

Clinical Manifestations

Jaundice may appear shortly after birth (during the first 24 hours) in newborns affected by HDN, and serum levels of unconjugated bilirubin rise rapidly. Anemia results from the hemolysis of large numbers of erythrocytes, and hyperbilirubinemia and jaundice result from the liver's inability to conjugate and excrete the excess bilirubin. Most newborns with HDN are not jaundiced at birth. However, hepatosplenomegaly and varying degrees of hydrops may be evident. If the infant is severely affected, signs of anemia (notably, marked pallor) and hypovolemic shock are apparent. Hypoglycemia may occur as a result of pancreatic cell hyperplasia.

Diagnostic Evaluation

Early identification and diagnosis of RhD sensitization are important in the management and prevention of fetal complications. A maternal antibody titer (**indirect Coombs test**) should be drawn at the first prenatal visit. Genetic testing allows early identification of paternal zygosity at the RhD gene locus, thus