erythroblastosis fetalis.

There is wide variability in the development of maternal sensitization to Rh-positive antigens. Sensitization may occur during the first pregnancy if the woman had previously received an Rh-positive blood transfusion. No sensitization may occur in situations in which a strong placental barrier prevents transfer of fetal blood into the maternal circulation. In approximately 10% to 15% of sensitized mothers, there is no hemolytic reaction in the newborn. In addition, some Rh-negative women, even though exposed to Rh-positive fetal blood, are immunologically unable to produce antibodies to the foreign antigen (Hensley, Coughlin, and Klein, 2009).

In the most severe form of erythroblastosis fetalis, **hydrops fetalis**, the progressive hemolysis causes fetal hypoxia; cardiac failure; generalized edema (anasarca); and fluid effusions into the pericardial, pleural, and peritoneal spaces (hydrops). The fetus may be delivered stillborn or in severe respiratory distress. Maternal Rh immunoglobulin (RhIg) administration, early intrauterine detection of fetal anemia by ultrasonography (serial Doppler assessment of the peak velocity in the fetal middle cerebral artery), and subsequent treatment by fetal blood transfusions or high-dose IVIG have dramatically improved the outcome of affected fetuses (Bagwell, 2014).

ABO Incompatibility

Hemolytic disease can also occur when the major blood group antigens of the fetus are different from those of the mother. The major blood groups are A, B, AB, and O. In the North American white population, 46% have type O blood, 42% have type A blood, 9% have type B blood, and 3% have type AB blood.

The presence or absence of antibodies and antigens determines whether agglutination will occur. Antibodies in the plasma of one blood group (except the AB group, which contains no antibodies) will produce agglutination (clumping) when mixed with antigens of a different blood group. Naturally occurring antibodies in the recipient's blood cause agglutination of a donor's RBCs. The agglutinated donor cells become trapped in peripheral blood vessels, where they hemolyze, releasing large amounts of bilirubin into the circulation.