

streptococcus is the most common cause of early-onset sepsis as well as early-onset bacterial meningitis. Infections with *Listeria* and *Candida* have longer latent periods between the time of microorganism inoculation and the appearance of clinical symptoms and present as late-onset sepsis.

Fetal Hydrops

Fetal hydrops refers to the accumulation of edema fluid in the fetus during intrauterine growth. Until recently, hemolytic anemia caused by Rh blood group incompatibility between mother and fetus (*immune hydrops*) was the most common cause, but with the successful prophylaxis of this disorder during pregnancy, causes of *nonimmune hydrops* have emerged as the principal culprits (Table 10-3). The intrauterine fluid accumulation can be quite variable, from progressive, generalized edema of the fetus (*hydrops fetalis*), a usually lethal condition, to more localized degrees of edema, such as isolated pleural and peritoneal effusions, or postnuchal fluid accumulation (*cystic hygroma*, see later) that are compatible with life.

Immune Hydrops

Immune hydrops is a hemolytic disease caused by blood group antigen incompatibility between mother and fetus. When the fetus inherits red cell antigenic determinants from the father that are foreign to the mother, a maternal

Table 10-3 Selected Causes of Nonimmune Fetal Hydrops

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| Cardiovascular |
| Malformations |
| Tachyarrhythmia |
| High-output failure |
| Chromosomal |
| Turner syndrome |
| Trisomy 21, trisomy 18 |
| Thoracic Causes |
| Cystic adenomatoid malformation |
| Diaphragmatic hernia |
| Fetal Anemia |
| Homozygous α -thalassemia |
| Parvovirus B19 |
| Immune hydrops (Rh and ABO) |
| Twin Gestation |
| Twin-to-twin transfusion |
| Infection (excluding parvovirus) |
| Cytomegalovirus |
| Syphilis |
| Toxoplasmosis |
| Genitourinary Tract Malformations |
| Tumors |
| Genetic/Metabolic Disorders |

The cause of fetal hydrops may be undetermined ("idiopathic") in up to 20% of cases. Data from Machin GA: Hydrops, cystic hygroma, hydrothorax, pericardial effusions, and fetal ascites. In Gilbert-Barness E, et al (eds): *Potter's Pathology of the Fetus, Infant, and Child*. St. Louis, Mosby, 2007, p 33.

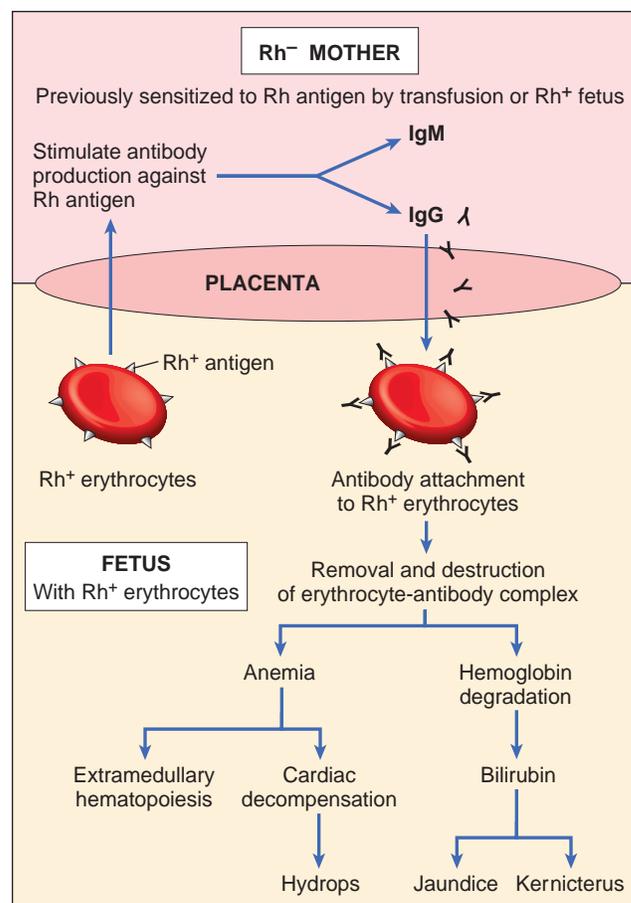


Figure 10-10 Pathogenesis of immune hydrops fetalis (see text).

immune reaction may occur. The major antigens known to induce clinically significant immunologic reactions are certain of the Rh antigens and the ABO blood groups. The reaction occurs in second and subsequent pregnancies in an Rh-negative mother with an Rh-positive father.

Etiology and Pathogenesis. The underlying basis of immune hydrops is the immunization of the mother by blood group antigens on fetal red cells and the free passage of antibodies from the mother through the placenta to the fetus (Fig. 10-10). Fetal red cells may reach the maternal circulation during the last trimester of pregnancy, when the cytotrophoblast is no longer present as a barrier, or during childbirth itself. The mother thus becomes sensitized to the foreign antigen. The initial exposure to Rh antigen evokes the formation of IgM antibodies, that unlike IgG antibodies, do not cross the placenta. Thus, Rh disease is uncommon with the first pregnancy. Exposure during a subsequent pregnancy generally leads to a brisk IgG antibody response and the risk of immune hydrops.

Of the numerous antigens included in the Rh system, only the D antigen is a major cause of Rh incompatibility. Several factors influence the immune response to Rh-positive fetal red cells that reach the maternal circulation.

- Concurrent ABO incompatibility protects the mother against Rh immunization, because the fetal red cells