from hemorrhage at childbirth is correlated with malaria-induced anemia.

■ MALARIA IN CHILDREN

Most of the >400,000 deaths from falciparum malaria each year are in young African children. Convulsions, coma, hypoglycemia, metabolic acidosis, and severe anemia are relatively common among children with severe malaria, whereas deep jaundice, oliguric acute kidney injury, and acute pulmonary edema are unusual. Severely anemic children may present with labored deep breathing, which in the past has been attributed incorrectly to “anemic congestive cardiac failure” but in fact is usually caused by metabolic acidosis, sometimes compounded by hypovolemia. In general, children tolerate antimalarial drugs well and respond rapidly to treatment.

■ TRANSFUSION MALARIA

Malaria can be transmitted by blood transfusion, needlestick injury, or organ transplantation. The incubation period in these settings is often short because there is no preerythrocytic stage of development, and thus there are no relapses of *P. vivax* and *P. ovale* infections. The clinical features and management of these cases are the same as for naturally acquired infections although primaquine is not needed for vivax or ovale malaria as there are no liver stages.

CHRONIC COMPLICATIONS OF MALARIA

■ HYPERREACTIVE MALARIAL SPLENOMEGALY

Chronic or repeated malarial infections produce hypergammaglobulinemia; normochromic, normocytic anemia; and, in certain situations, splenomegaly. Some residents of malaria-endemic areas in tropical countries exhibit an abnormal immunologic response to repeated infections that is characterized by massive splenomegaly, hepatomegaly, marked elevations in serum IgM and malarial antibody titers, hepatic sinusoidal lymphocytosis, and (in Africa) peripheral B-cell lymphocytosis. This syndrome has been