nomogram within 3 days of discharge if discharged at less than 24 hours of age. Newborns discharged at 24 to 48 hours should receive follow-up evaluation within 4 days (96 hours), and those discharged between 48 and 72 hours should receive follow-up within 5 days (American Academy of Pediatrics, Subcommittee on Hyperbilirubinemia, 2004; Blackburn, 2011). The serum bilirubin may be obtained at the time of the metabolic screening, thus precluding the need for additional blood sampling. The newest guidelines for monitoring and treating neonatal hyperbilirubinemia are published extensively elsewhere, and readers are referred to "Management of Hyperbilirubinemia in the Newborn Infant 35 or More Weeks of Gestation (Clinical Practice Guideline)" (American Academy of Pediatrics, Subcommittee on Hyperbilirubinemia, 2004) for an in-depth overview of management guidelines.

Complications

Unconjugated bilirubin is highly toxic to neurons; therefore, an infant with severe jaundice is at risk of developing **bilirubin encephalopathy**, a syndrome of severe brain damage resulting from the deposition of unconjugated bilirubin in brain cells. **Kernicterus** describes the yellow staining of the brain cells that may result in bilirubin encephalopathy. The damage occurs when the serum concentration reaches toxic levels, regardless of cause. There is evidence that a fraction of unconjugated bilirubin crosses the blood–brain barrier in neonates with physiologic hyperbilirubinemia. When certain pathologic conditions exist in addition to elevated bilirubin levels, there is an increase in the permeability of the blood–brain barrier to unconjugated bilirubin and thus potential irreversible damage. The exact level of serum bilirubin required to cause damage is not yet known.

Multiple factors contribute to bilirubin neurotoxicity; therefore, *serum bilirubin levels alone do not predict the risk of brain injury.* Factors that are known to enhance the development of bilirubin encephalopathy include metabolic acidosis, lowered serum albumin levels, intracranial infections (such as meningitis), and abrupt fluctuations in BP. In addition, any condition that increases the metabolic demands for oxygen or glucose (e.g., fetal distress, hypoxia, hypothermia, hypoglycemia) also increases the risk of brain damage at lower serum levels of bilirubin.