Case Based Learning Module SJH - Neonatal Jaundice

**Objectives:**

**Medical expert:**
1. To review pathophysiology of hyperbilirubinemia
2. To discuss differential diagnosis of jaundice
3. To become familiar with standardized bilirubin nomograms
4. To discuss investigations and evidence for management strategies for jaundice

**Communicator:**
1. To be able to explain to parents the etiology and treatment of jaundice in a newborn
2. To be able to present a pertinent history and relevant clinical findings along with a proposed management plan in a succinct and efficient manner

**Case:**
You are on call at St. Joseph's Hospital and you receive a call from the BANA clinic indicating they have a jaundiced baby boy they would like you to have a look at.

*What are your top 5 questions on history?*

**History:**
You meet with the parents to review the history. You learn that mom is a 33yo G3T2A1L1 healthy woman. She was HepB/VDRL/HIV negative and rubella immune. Her GBS swab at 35 weeks was negative. Her blood group is O positive. There were no significant infections during the pregnancy. She received regular pre-natal care at the maternity center.

Family has a 10 yrs old boy who did not have trouble with jaundice as an infant. Parents are Caucasian and non-consanguinous. There is no known history of liver disease or inborn errors of metabolism.

Mom was booked for C-section for transverse lie but went into spontaneous labour at 37 2/7. Baby was noted to be vertex at that point. The labour was augmented with oxytocin and baby Jayden delivered vaginally. There were no HR decelerations or maternal temperature noted. Membranes ruptured spontaneously 2 hours prior to delivery. Baby was vigorous with Apgars 9 (1) and 9 (5). Birth weight was 3306, length 49cm and head circumference 35.5cm. Baby was placed skin-to-skin immediately after delivery and attempted breast feeding in L&D. Cord gases' PHs were 7.19, 7.29.
Jayden was breast feeding in hospital and was discharged home at 36 hrs of age with discharge weight of 3125 (5% loss). Bilirubin at discharge was in High Intermediate Zone and follow up through Bana clinic was arranged. At home, Jayden continued to breast feed frequently and for prolonged periods >1 hr at breast. He is now 59 hours old and is having 2 stools and 4-5 wet diapers per day. He seems alert but then falls asleep at the breast.

**What are the top 4 things to look for on exam?**

**Examination**

Jayden's weight is down 11.6% from his birth weight. His sclera and skin appear jaundiced. He has moist membranes, normotensive fontanelle and normal skin turgour. There are no dysmorphic features noted. He awakens for examination and appears alert with normal tone. There is no hepatosplenomegaly.

**What are the top 5 investigations to order?**

- Bilirubin-261 at 59 hrs
- Lytes- Na 152
- Blood type-O positive
- Coomb’s- negative
- CBC-Hg 213, Ptl 176, WBC 9.8- no left shift

**What is your management plan?**

**Treatment plan:**

- Phototherapy- double- blanket and lights
- Breast feed with supplementation, for TFI=100 =33 cc q 3 hrs, to be offered by lactation aid, finger feed or bottle
- If too lethargic to feed, vomiting or not tolerating feeds - consider NG top ups
- If bilirubin or Na rising rapidly or clinical deterioration, consider need for re-admit to Neo for IV fluids
- Consider need for IVIG, exchange transfusion
- Recheck levels - how soon- 12 hrs?

**Approach to jaundice:**
Physiologic
Breast feeding jaundice
Breast milk jaundice

Resources:

CPS Guidelines:
http://www.cps.ca/documents/position/hyperbilirubinemia-newborn

AAP guidelines 2004:
http://pediatrics.aappublications.org/content/114/1/297.full.pdf

Lauer B, Hyperbilirubinemia In The Newborn, Pediatrics in Review 2011; Vol.32 No.8 p341

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Hyperbilirubinemia in the Newborn
Bryon J. Lauer and Nancy D. Spector
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Hyperbilirubinemia in the Newborn

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Author Disclosure
Drs Lauer and Spector have disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

Objectives After completing this article, readers should be able to:

1. List the risk factors for severe hyperbilirubinemia.
2. Distinguish between physiologic jaundice and pathologic jaundice of the newborn.
3. Recognize the clinical manifestations of acute bilirubin encephalopathy and the permanent clinical sequelae of kernicterus.
4. Describe the evaluation of hyperbilirubinemia from birth through 3 months of age.
5. Manage neonatal hyperbilirubinemia, including referral to the neonatal intensive care unit for exchange transfusion.

Introduction
For centuries, neonatal jaundice (icterus neonatorum) has been observed in newborns. As early as 1724, Juncker, in the Conspectus Medicinae Theoretico-Praticae, began distinguishing between “true jaundice” and “the icteric tinge which may be observed in infants, immediately after birth.” In 1875, Orth noticed during autopsies the presence of bilirubin in the basal ganglia of infants who had severe jaundice, which was labeled kernicterus by Schmorl in 1903. (1) In 1958, however, a nurse in the nursery of the General Hospital in Rothford, Essex, Great Britain, reported “an apparent fading away of the yellow pigmentation in the skin of the jaundiced babies when they had been a short time in sunlight.” (2)

Icterus neonatorum occurs in approximately two thirds of all newborns in the first postnatal week. Jaundice results from bilirubin deposition in the skin and mucous membranes. For most newborns, such deposition is of little consequence, but the potential remains for kernicterus from high bilirubin concentrations or lower bilirubin concentrations in preterm infants. (3) Although rare, kernicterus is a preventable cause of cerebral palsy.

Hyperbilirubinemia was treated aggressively in the 1950s to 1970s because of a high rate of Rh hemolytic disease and kernicterus. However, data from the 1980s and 1990s showed that pediatricians may have been too aggressive in their approach, almost making kernicterus a disease of the past. Pediatricians subsequently became less aggressive, discharging newborns earlier from nurseries before bilirubin concentrations peaked. These factors helped lead to an increase in kernicterus in the 1990s. (4) Because of these events, an American Academy of Pediatrics (AAP) Subcommittee on Hyperbilirubinemia established guidelines for the approach to neonatal jaundice. (5)

Bilirubin Metabolism
When red blood cells undergo hemolysis, hemoglobin is released. Within the reticuloendothelial system, heme oxygenase degrades heme into biliverdin and carbon monoxide. Biliverdin reductase reduces biliverdin to unconjugated (indirect) bilirubin. Unconjugated bilirubin binds to albumin and is transported to the liver. Unconjugated bilirubin can become unbound if albumin is saturated or if bilirubin is displaced from albumin by medications (eg, sulfisoxazole, streptomycin, chloramphenicol, ceftriaxone, ibuprofen). The unbound unconjugated bilirubin can cross the blood-brain barrier and is toxic to the central nervous system. (5)(6)

Once unconjugated bilirubin reaches the liver, it is conjugated by uridine diphosphate glucuronyltransferase (UGT1A1). Hepatic UGT1A1 increases dramatically in the first few weeks after birth. At 30 to 40 weeks’ gestation, UGT1A1 values are approximately 1%
Physiologic jaundice is an unconjugated hyperbilirubinemia that occurs after the first postnatal day and can last up to 1 week. Total serum bilirubin (TSB) concentrations peak in the first 3 to 5 postnatal days and decline to adult values over the next several weeks. The TSB concentrations vary greatly in infants, depending on race, type of feeding, and genetic factors. (8) Initially, the cord TSB concentration in term newborns is approximately 1.5 mg/dL (25.7 μmol/L). The TSB concentration peaks at approximately 5.5 mg/dL (94.1 μmol/L) by the third postnatal day in white and African American infants. The mean TSB concentration peaks are higher in Asian infants at approximately 10 mg/dL (171.0 μmol/L). (9) By 96 hours of age, 95% of infants have TSB concentrations of less than 17 mg/dL (290.8 μmol/L). Therefore, bilirubinemia above this value is no longer considered physiologic jaundice.

Physiologic jaundice occurs in infants for a number of reasons. They have a high rate of bilirubin production and an impaired ability to extract bilirubin from the body. Bilirubin production also is increased as a result of elevated hematocrit and red blood cell volume per body weight and a shorter life span of the red blood cells (70 to 90 days). (10) Finally, infants have immature hepatic glucuronosyl transferase, a key enzyme involved in the conjugation of bilirubin that facilitates excretion from the body. (5)(10)

PREMATURITY. Although preterm infants develop hyperbilirubinemia by the same mechanisms as term infants, it is more common and more severe in preterm infants and lasts longer. This outcome is related to the relative immaturity of the red blood cells, hepatic cells, and gastrointestinal tract. Sick preterm newborns are more likely to have a delay in initiating enteral nutrition, resulting in an increase in enterohepatic circulation. Despite the prevalence of hyperbilirubinemia in preterm newborns, kernicterus is extremely uncommon. However, kernicterus does occur at lower TSB concentrations, even without acute neurologic signs. (11) It is unclear, however, at what value of bilirubin central nervous system injury occurs. TSB values as low as 10 to 14 mg/dL (171.0 to 239.5 μmol/L) have resulted in milder forms of bilirubin-induced neurologic dysfunction (BIND) in preterm infants. (11)(12)

Pathologic

UNCONJUGATED HYPERBILIRUBINEMIA. Pathologic hyperbilirubinemia in a newborn can be separated into four categories: increased bilirubin production, defi-
iciency of hepatic uptake, impaired conjugation of bilirubin, and increased enterohepatic circulation (Table 1). (5) Increased production occurs in infants who have erythrocyte-enzyme deficiencies, blood group incompatibility, or structural defects in erythrocytes. ABO incompatibility may cause anemia in the first-born child, but Rh incompatibility rarely does. Pediatricians also should consider glucose-6-phosphate dehydrogenase (G6PD) deficiency, especially in African American infants. G6PD deficiency is a sex-linked disorder occurring in 11% to 13% of African American newborns in the United States and is a significant risk factor for kernicterus. (8)

Multiple conditions can cause hyperbilirubinemia through impaired bilirubin conjugation. Gilbert syndrome is an autosomal recessive condition in which UGT1A1 activity decreases mildly in hepatocytes, typically resulting in a benign unconjugated hyperbilirubinemia. The likelihood of severe hyperbilirubinemia is increased if the infant also has G6PD deficiency. In Crigler-Najjar syndrome type I, severe deficiency of UGT1A1 results in bilirubin encephalopathy in the first few days or month after birth. In Crigler-Najjar syndrome type II, the incidence of bilirubin encephalopathy is low. (5)

### CONJUGATED HYPERBILIRUBINEMIA

Conjugated hyperbilirubinemia is defined by a conjugated bilirubin concentration greater than 1 mg/dL (17.1 μmol/L) when the TSB concentration is 5 mg/dL (85.6 μmol/L) or less. If the TSB concentration is greater than 5 mg/dL (85.6 μmol/L), conjugated hyperbilirubinemia is defined when the value is 20% or greater of the TSB concentration. Elevated conjugated hyperbilirubinemia may be related to a urinary tract infection or sepsis. In an infant older than 3 weeks of age, total and conjugated bilirubin should be measured to rule out cholestasis and biliary atresia, which are associated with elevated conjugated bilirubin concentrations. The newborn screen also should be reviewed because thyroid abnormalities and galactosemia are additional causes of conjugated hyperbilirubinemia.

### Kernicterus

The term kernicterus was used originally for staining of the brainstem nuclei and cerebellum. Acute bilirubin encephalopathy describes the neurologic changes that occur in the first postnatal weeks from bilirubin toxicity. Kernicterus is the chronic or permanent neurologic sequela of bilirubin toxicity. (13) The level at which bilirubin toxicity occurs is not completely known, and multiple factors influence whether bilirubin toxicity does occur. Bilirubin can cross the blood-brain barrier and enter the brain tissue if it is unconjugated and unbound to albumin or if there is damage to the blood-brain barrier. Asphyxia, acidosis, hypoxia, hypoperfusion, hyperosmolality, and sepsis can damage the blood-brain barrier, allowing bilirubin bound to albumin to enter the brain tissue. Pedi-

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**Table. Risk Factors for Hyperbilirubinemia**

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<thead>
<tr>
<th>Increased Bilirubin Production</th>
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<tr>
<td>Hemolytic disease</td>
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<td>- Isoantibodies</td>
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<td>ABO</td>
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<td>Rh</td>
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<td>Minor antibodies</td>
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<td>- Enzyme defects</td>
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<td>Glucose-6-phosphate deficiency</td>
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<td>Pyruvate kinase deficiency</td>
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<tr>
<td>- Structural defects</td>
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<td>Spherocytosis</td>
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<td>Elliptocytosis</td>
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<tr>
<td>Birth trauma</td>
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<tr>
<td>- Cephalohematoma</td>
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<tr>
<td>- Excessive bruising</td>
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<td>Polycythemia</td>
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<tr>
<th>Impaired Bilirubin Conjugation</th>
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<tr>
<td>Gilbert syndrome</td>
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<td>Crigler-Najjar syndrome I and II</td>
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<td>Human milk jaundice</td>
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<tr>
<th>Decreased Bilirubin Excretion</th>
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<tr>
<td>Biliary obstruction</td>
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<td>- Biliary atresia</td>
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<td>- Choledochal cyst</td>
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<td>- Dubin-Johnson syndrome</td>
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<td>- Rotor syndrome</td>
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<th>Other/Combination</th>
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<td>Asian ethnicity</td>
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<tr>
<td>Prematurity</td>
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<td>Metabolic disorder</td>
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<td>- Hypothyroidism</td>
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<td>- Galactosemia</td>
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<td>Maternal diabetes mellitus</td>
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<td>Infection</td>
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<td>- Urinary tract infection</td>
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<td>- Sepsis</td>
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<td>Breastfeeding</td>
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<td>Drugs</td>
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<td>- Sulfinpyrazone</td>
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<td>- Streptomycin</td>
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<td>- Benzyl alcohol</td>
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Increased bilirubin concentration may occur if the mother’s blood type is O and the infant’s blood type is A or B. (13) Mother-infant ABO incompatibility occurs in approximately 15% of all pregnancies, but symptomatic hemolytic disease occurs in only 5% of these infants. Hyperbilirubinemia in infants who have symptomatic ABO hemolytic disease usually is detected in siblings, evaluation is appropriate for any infant who is receiving phototherapy or when the TSB crosses percentiles on the nomogram. A complete blood count with smear and direct bilirubin concentration should be checked in these instances. A reticulocyte count, G6PD measurement, and end-tidal carbon monoxide (ETCO) determination (if available) can be considered. (12) ETCO is a good indicator of ongoing bilirubin production. As noted previously, biliverdin and carbon monoxide are the byproducts of bilirubin breakdown. Measuring the ETCO allows identification of infants experiencing increased bilirubin production and possibly infants who have hemolytic disease. (5) The TSB concentration should be rechecked in 4 to 24 hours, depending on the infant’s age, TSB value, and risk factors. If the TSB is increasing despite phototherapy or if the infant is being considered for exchange transfusion, a reticulocyte count, bilirubin/albumin ratio, G6PD concentration, and ETCO should be checked. Urinalysis and urine
culture are appropriate if the infant has an elevated direct bilirubin value. If indicated by the history and physical examination, a sepsis evaluation should be completed. (13)

Although human milk jaundice is a common cause of prolonged jaundice in breastfed infants, more concerning conditions should be ruled out first. Total and direct bilirubin should be measured for the infant who develops jaundice or when jaundice persists after 3 weeks of age. In addition, the newborn screen should be reviewed specifically to rule out galactosemia and congenital hypothyroidism. An elevated direct bilirubin value should prompt an evaluation for cholestasis. (13)

Because TSB concentrations peak at 3 to 5 days of age, after many infants have left the nursery, it is important to perform a risk assessment on all infants before they leave the hospital, and appropriate follow-up evaluations should be stressed. Although some controversy surrounds screening and risk assessment, based on insufficient evidence, the AAP Subcommittee has recommended assessing TSB or TcB on all newborns before discharge. (16) The value should be plotted on the nomogram to assess the risk level. (13) Some authors suggest checking a TSB on all newborns when the newborn screen is obtained. Other authors argue that data are insufficient to justify screening all infants at discharge. (16)

Exclusive breastfeeding, phototherapy in a sibling, gestational age less than 37 weeks, jaundice in the first 24 hours, hemolytic disease, East Asian race, cephalohematomata or significant bruising, and a TSB or TcB in the high risk zone before discharge are the most common clinically relevant risk factors for severe hyperbilirubinemia. Each risk factor individually has little predictive value, but the greater the number of risk factors, the greater the likelihood of the baby developing severe hyperbilirubinemia. In general, a term infant who is fed predominately formula has a very low likelihood of developing severe hyperbilirubinemia. (13)

The timeframe for following up with a pediatrician once infants are discharged from the hospital depends on the baby’s age at the time of discharge. A newborn discharged at 48 to 72 hours of age should be evaluated for jaundice, weight gain or loss, stool patterns, voiding patterns, and adequacy of oral intake by 120 hours of age. The child should be evaluated at 96 hours of age if discharged between 24 to 48 hours and at 72 hours if discharged before 24 hours of age. Infants discharged before 48 hours of age may need a second visit to ensure evaluation during the time when the TSB peaks. Infants who have more risk factors may need more frequent follow-up evaluations. Also, if follow-up cannot be ensured, delaying discharge is appropriate until follow-up is determined or until the infant is older than 72 to 96 hours of age. One of the most important measures is educating all parents on the risks and assessment of hyperbilirubinemia as well as necessary follow-up evaluations. (13)

Treatment
Helping mothers breastfeed appropriately can decrease the likelihood of severe hyperbilirubinemia. Mothers should breastfeed at least 8 to 12 times in the first few days after birth to aid in bringing in the milk supply. Mother should be asked about any difficulties and lactation consultants involved when needed. The stool pat-
terns, voiding patterns, and weight of newborns are good indicators of whether the baby is receiving adequate milk. (6)(8)

Phototherapy

Since the discovery of the effects of sunlight on lowering bilirubin concentrations in 1958 (2), the need for exchange transfusions because of severe hyperbilirubinemia has decreased significantly. (17) Phototherapy works by converting bilirubin into a water-soluble compound called lumirubin, which is excreted in the urine or bile without requiring conjugation in the liver. The two biggest factors in the conversion of bilirubin to lumirubin are the spectrum of light and the total dose of light delivered. Bilirubin is a yellow pigment, so it most strongly absorbs blue light in the 460-nm wavelength. (5) Also, a phototherapeutic effect is seen only when the wavelength can penetrate tissue and absorb bilirubin. Lamps with output in the 460- to 490-nm range are the most effective in treating hyperbilirubinemia. Multiple types of phototherapy units are used today that contain daylight, cool white, blue, or “special blue” fluorescent tubes or tungsten-halogen lamps. Fiberoptic blankets are also available that provide light in the blue-green region. The special blue fluorescent lights are the most effective and should be used when intensive phototherapy is required. (5)(17) Ultraviolet light is not used for phototherapy. (17) Although sunlight has been shown to decrease bilirubin concentrations, it is not recommended because it is difficult to determine a timeframe that is safe to expose a naked infant to sunlight without getting sunburned. (13)

The total dose delivered, or spectral irradiance, is affected significantly by the distance the infant is from the light and the surface area to which he or she is exposed. Therefore, infants should be placed as close as possible to the light. Placing an infant in a bassinet rather than an incubator allows the light to be closer to the infant. When using fluorescent tubes, it is possible to bring the light source to within 10 cm of the infant without overheating him or her. Halogen lights can burn the infant, so the manufacturer’s instructions should be followed to determine the correct distance between the light source and the baby. (13)(17) Exposing the infant as much as possible while covering his or her eyes results in a faster decline in bilirubin concentrations. It is probably unnecessary to remove the diaper unless the bilirubin concentrations are approaching the level requiring an exchange transfusion. The bassinet should be lined with aluminum foil or white cloth when nearing the point of an exchange transfusion. In most instances, it is acceptable to interrupt phototherapy to feed the infant or for brief parental visits. Continuous phototherapy should be used if exchange transfusion is likely. (13)

While an infant is receiving phototherapy, his or her temperature and hydration status should be monitored. Because bilirubin is excreted in the urine and the stool, it is important to assure good urine output. If the infant is dehydrated, intravenous fluids should be started. Oral nutrition is sufficient for the infant who is not dehydrated. Supplementing breastfeeding with formula is an option to reduce enterohepatic circulation and decrease the TSB faster. (13)

Initiation of phototherapy should be based on the TSB concentration, age in hours, and risk factors, as recommended in guidelines from the AAP (Fig. 2). The TSB value should be used, and the direct bilirubin value should not be subtracted from the total when determining when to initiate therapy. There are no guidelines...
published for infants born earlier than 35 weeks’ gestation. When using intensive phototherapy, a decrease of 0.5 mg/dL (8.6/µmol/L) per hour can be expected in the first 4 to 8 hours. When the TSB does not decline or rises during phototherapy, ongoing hemolysis is likely. Discontinuation of phototherapy is not standardized. Therefore, clinical judgment is recommended. (5) Some authors suggest stopping once the bilirubin decreases 4 to 5 mg/dL (68.4 to 85.5 µmol/L). (5) Others state that the value should decrease to 13 to 14 mg/dL (222.4 to 239.5 µmol/L) if the child is readmitted for hyperbilirubinemia. A common misconception is that discontinuation of phototherapy results in a rebound hyperbilirubinemia. Rebound is a rare event in an infant who weighs more than 1,800 g and has no evidence of hemolysis. (5) Whether this observation holds true for smaller infants or those who have evidence of hemolysis is uncertain. A rebound bilirubin determination is not recommended, but if an infant is readmitted, a repeat TSB measurement or clinical follow-up in 24 hours is optional. (13)

Phototherapy is performed safely for millions of infants, but rare adverse effects do occur. The infant who has cholestatic jaundice with elevated conjugated hyperbilirubinemia has the potential for developing bronze infant syndrome. Affected infants develop a dark, grayish-brown color of the skin, serum, and urine. Generally, the syndrome is of little clinical significance. The only true contraindication to phototherapy is congenital porphyria or a family history of porphyria. Phototherapy in these patients could result in severe blistering and fotosensitivity. (17)

Exchange Transfusion

Exchange transfusion was the first successful treatment for severe hyperbilirubinemia. These procedures should be performed only in a neonatal intensive care unit by a trained physician. An exchange transfusion for an infant is a medical emergency, and the patient should be admitted directly to the neonatal intensive care unit, bypassing the emergency department. (9) Basically, the physician rapidly removes from the circulation bilirubin and any antibodies that may be contributing to ongoing hemolysis. The procedure involves taking small aliquots of the infant’s blood and replacing them with the same quantity of donor red cells via one to two central catheters until the infant’s blood volume has been replaced twice. (5) An infusion of albumin 1 to 4 hours before the procedure can increase the amount of bilirubin that is removed. Intravenous gamma globulin is recommended for infants who have isoimmune hemolytic disease if the TSB is rising despite phototherapy or the TSB is within 2 to 3 mg/dL (34.2 to 51.3 µmol/L) of the level for an exchange transfusion in hopes of avoiding an exchange transfusion. Another dose can be administered in 12 hours, if needed. (13)

Figure 3 shows guidelines for initiating an exchange transfusion. Exchange transfusion should be started immediately in a jaundiced infant demonstrating signs of acute bilirubin encephalopathy, even if the TSB value is falling. Risk factors for severe hyperbilirubinemia and the albumin/bilirubin ratio should be taken into account when considering when to start an exchange transfusion. (13)

Although exchange transfusions are successful in infants who have severe hyperbilirubinemia, there are many complications, including infection, portal venous thrombosis, and liver damage.
Summary

- Based on strong research evidence, breastfeeding, prematurity, significant jaundice in a previous sibling, and jaundice noted before discharge from the nursery are the most common risk factors associated with severe hyperbilirubinemia. (13)
- Based on research evaluating benefit versus harm, jaundice in the first 24 hours after birth is not physiologic jaundice and needs further evaluation.
- All newborns should undergo a risk assessment for hyperbilirubinemia before discharge from the newborn nursery and have appropriate follow-up evaluation after discharge.
- Visual assessment of jaundice does not assess the TSB reliably; clinicians should check either a TSB or TcB when in doubt.
- The infant’s age in hours is used when evaluating and managing bilirubin concentrations.

brosis, thrombocytopenia, necrotizing enterocolitis, electrolyte imbalances, graft versus host disease, and even death. The complication rate is reported to be approximately 12%. (5) Because of these risk factors, phototherapy should be maximized to reduce the need for an exchange transfusion. (13)

Conclusion

Kernicterus, although a rare event, is a preventable cause of cerebral palsy. Now that infants are being discharged at earlier ages, it is important to consider screening with a TcB or TSB before discharge because visual assessment is not always reliable. It is equally important to arrange for follow-up evaluation after discharge, ideally within 48 hours, for additional screening. Mothers should be educated about feeding to ensure that the infants are receiving adequate caloric intake and monitoring stool and urine output. Weight can be checked at the follow-up visit. When evaluating bilirubin concentrations, nomograms can be used to guide initiation of phototherapy and exchange transfusions. Guidelines and published nomograms can support clinical judgment and individualize the approach to the infant who has hyperbilirubinemia.

References


HealthyChildren.org Parent Resources From the AAP

The reader is likely to find material to share with parents that is relevant to this article by visiting this link: http://www.healthychildren.org/English/ages-stages/baby/Pages/Jaundice.aspx.
15. You are evaluating a 3-day-old term infant who has jaundice. His neonatal course was unremarkable and he is breastfed exclusively. He has approximately seven wet diapers and three stools per day. Findings on his physical examination are normal except for jaundice. His bilirubin measures 12 mg/dL (205.2 μmol/L) and is all unconjugated. Of the following, the most appropriate management is to

A. Admit the baby for phototherapy.
B. Continue breastfeeding.
C. Discontinue breastfeeding for 3 days, then resume.
D. Supplement the human milk with water.
E. Supplement the human milk with cow milk-based formula.

16. A 4-day-old girl is brought to your office for evaluation of jaundice. She was born at 39 weeks’ gestation and had no complications. Her and her mother’s blood types both are A+. She is breastfeeding well and has normal stools and urine output. She has significant jaundice on examination but is vigorous and well hydrated. At what total serum bilirubin value should phototherapy be initiated for this infant?

A. 10 mg/dL (171.0 μmol/L).
B. 12 mg/dL (205.2 μmol/L).
C. 15 mg/dL (256.5 μmol/L).
D. 17 mg/dL (290.8 μmol/L).
E. 20 mg/dL (342.1 μmol/L).

17. Which of the following is most likely to be present during the initial phase of acute bilirubin toxicity?

A. Cerebral palsy.
B. Chorea.
C. Opisthotonus.
D. Retrocollis.
E. Seizures.

18. Which of the following statements regarding the optimal use of phototherapy is true?

A. Infants receiving phototherapy should be placed in an incubator.
B. Infants should wear full clothing during phototherapy to prevent burns.
C. Intravenous fluids are required for all infants receiving phototherapy.
D. The light source should be 30 cm from the infant’s skin.
E. Stopping phototherapy to allow for breastfeeding is acceptable in most cases.
# Hyperbilirubinemia in the Newborn

Bryon J. Lauer and Nancy D. Spector  
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