Newborn
Hyperbilirubinemia

A Self–Learning Module

2013
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Introduction

By completing this self-learning module, the learner will obtain the knowledge and skills to perform/provide a systematic approach to jaundice assessment, management and prevention of severe neonatal hyperbilirubinemia. Through proper screening, identification of risk factors for severe hyperbilirubinemia and appropriate use of phototherapy/treatment modalities, the health care team can make a difference and contribute to decreasing a newborn’s risk of neonatal encephalopathy.

Objectives

1. Summarize the mechanisms of bilirubin production and clearance (physiologic mechanism).
2. Identify newborns at risk for hyperbilirubinemia.
4. Describe a systematic process to screen for, assess and monitor neonatal hyperbilirubinemia.
5. Describe the recommended treatment modalities for hyperbilirubinemia.
6. Summarize the current consensus guidelines for screening, early intervention, treatment and follow-up of newborns ≥ 35 weeks gestation at risk for hyperbilirubinemia.
# 1. Types of Jaundice

<table>
<thead>
<tr>
<th>PHYSIOLOGIC</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal process occurs in the first few days of life after 24 hours of age</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Benign process</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Self-limiting</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Resolves by end of first week</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Requires no treatment</strong></td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>BREASTFEEDING</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Occurs in first few days of life</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Related to decreased breast milk intake and decreased frequency of feeding</strong></td>
<td></td>
</tr>
<tr>
<td><strong>May also be related to altered liver conjugation and bilirubin clearance due to inadequate caloric intake</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Prompt early initiation of breastfeeding and frequent, short unsupplemented feeding of colostrum and breast milk will prevent exaggeration of early physiologic jaundice</strong></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BREAST MILK</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Late onset jaundice beginning after 5th day of life, more uncommon</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Peaks during second or third week and continues for several weeks</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Caused by increased reabsorption of unconjugated bilirubin, perhaps due to unidentified factor in human milk</strong></td>
<td></td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>PATHOLOGIC</th>
<th>DESCRIPTION</th>
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</thead>
<tbody>
<tr>
<td><strong>Jaundice potentially arising from pathologic processes and usually appears within first 24 hours after birth</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Characterized by rapidly rising serum bilirubin and or elevated direct bilirubin concentration (&gt;34 micromols/L or &gt;20% of total serum bilirubin)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Combination of factors:</strong></td>
<td></td>
</tr>
<tr>
<td>a) <strong>increased production</strong> (polycythemia, sepsis, bruising, blood group incompatibilities)</td>
<td></td>
</tr>
<tr>
<td>b) <strong>decreased excretion</strong> (bowel obstruction, poor feeding, acidosis)</td>
<td></td>
</tr>
</tbody>
</table>
2. Bilirubin Metabolism

2.1 Bilirubin Production

Bilirubin is a product of the breakdown of the heme portion of hemoglobin that occurs when red blood cells are destroyed. Normally, bilirubin is excreted through the body after passing through the liver, spleen, kidneys and the gastrointestinal tract.

2.2 Types of Bilirubin

There are two types of bilirubin circulating in the bloodstream, *unconjugated* and *conjugated*.

*Unconjugated bilirubin* (or *indirect bilirubin*) can be found in circulating blood either bound to albumin or not. It is fat-soluble and therefore more potentially toxic since it can bind to the tissues. Most of the *unconjugated* bilirubin is bound to albumin and transported to the liver. There, it is converted to glucuronic acid aided by uridine diphosphate glucuronosyl transferase (UDGT) to produce conjugated bilirubin. Once it becomes conjugated, it is sent to the gut for excretion via the biliary system. The unbound, unconjugated bilirubin is most likely to cross the blood-brain barrier and settle in the tissues where it can cause temporary or permanent neurological damage. Once it settles in the brain, it is there forever. The unbound bilirubin is difficult to measure but it is thought that it is directly related to the amount of unconjugated bilirubin.

*Conjugated bilirubin* (or *direct bilirubin*) is water-soluble and therefore is a more stable and non-toxic form. This allows it to be easily excreted from the body in urine and stool. Elevated levels of conjugated bilirubin may indicate evidence of liver disease.

2.3 Conversion and Elimination of Bilirubin

```
Unconjugated (indirect) → Albumin → Unconjugated Bound → Liver → Conjugated (direct) → GI Tract → Excreted
```

Kernicterus
3. Factors Affecting Bilirubin Metabolism

3.1 Increased Production

Any disorder which causes increased numbers of red blood cells for example, polycythemia, will lead to increased amounts of bilirubin produced as these cells breakdown. If there is a decreased amount of albumin available, there will be decreased binding occurring and thus less processing from indirect to direct in the liver and more indirect bilirubin that could potentially cross the blood-brain barrier or settle in the tissues. Bruising will also increase the breakdown of RBCs and increase bilirubin levels.

3.2 Decreased Conjugation

Conditions such as acidosis and hypoxia can also affect the bilirubin/albumin ratio for binding. The presence of any type of liver disease or a metabolic or enzyme disorder will also affect the ability of the body to properly process bilirubin to the direct form to allow for excretion. Because bilirubin is changed in the gut to urobilinogen with the assistance of the normal intestinal flora, anything that affects normal gut function can affect the excretion of bilirubin from the body. We know that at birth, the infant’s gut is not fully developed so that any prematurity and/or any disorder of the bowel, as well as antibiotic therapy, can slow the excretion of bilirubin.

4. Maternal and Newborn Risk Factors for Development of Jaundice

<table>
<thead>
<tr>
<th>OVER PRODUCTION</th>
<th>UNDER SECRETION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MATERNAL</strong></td>
<td></td>
</tr>
<tr>
<td>– Feto-maternal factors such as ABO or Rh-incompatibility</td>
<td>– Maternal illness (such as diabetes mellitus, pre-eclampsia)</td>
</tr>
<tr>
<td>– Difficult delivery such as forceps or vacuum extraction</td>
<td>– Family history of jaundice, anemia, liver disease or splenectomy</td>
</tr>
<tr>
<td>– Use of oxytocin in labour (oxytocin binds to albumin)</td>
<td></td>
</tr>
<tr>
<td>– Mediterranean, Middle Eastern, East Asian, Aboriginal background</td>
<td></td>
</tr>
<tr>
<td><strong>INFANT</strong></td>
<td></td>
</tr>
<tr>
<td>– Polycythemia</td>
<td>– Prematurity</td>
</tr>
<tr>
<td>– Sepsis</td>
<td>– GI anomalies</td>
</tr>
<tr>
<td>– Excessive bruising</td>
<td>– Poor nutrition (dehydration, decreased fluid intake)</td>
</tr>
<tr>
<td>– Asphyxia, hypoxia, hypothermia, hypoglycaemia, acidosis</td>
<td></td>
</tr>
<tr>
<td>– Congenital enzyme, metabolic/endocrine disorders</td>
<td></td>
</tr>
</tbody>
</table>
Risk factors can also be illustrated by the following mnemonic:

J - Jaundice within 24 hours of birth
A – A sibling who had jaundice as a neonate, or an infant who has asphyxia, acidosis, or albumin < 3.0 g/dL
U – Unrecognized hemolysis (ABO, Rh or other blood incompatibility, red cell membrane defects)
N – Newborn born < 37 weeks gestation
D – Deficiency in glucose-6-phosphate dehydrogenase (G6PD)
I – Infection or infant of a diabetic mother
C – Cephalohematoma/bruising or central hematocrit > 65%
E – East Asian, Mediterranean, Middle Eastern, Aboriginal descent
D – Due to sub-optimal feeding (breast or formula) (CPS, 1999)

5. Bilirubin Encephalopathy (Bilirubin Toxicity)

Normally, hyperbilirubinemia resolves on its own as the infant processes the bilirubin and excretes it. However, in some infants, it can become harmful and will need further treatment. If left untreated and levels rise too high, some of the bilirubin may cross the blood brain barrier and settle into brain tissue where it can cause acute bilirubin encephalopathy. This encephalopathy can be detected by symptoms which, if not detected early and treated, will develop into kernicterus.

Kernicterus is a potentially fatal disease and results in permanent injury to specific parts of the brain. There are some clinical signs of acute bilirubin encephalopathy, which can occur in three phases:

Initial phase:
- lethargy, decrease in tone or activity

Intermediate phase:
- moderate stupor, irritability and variable activity
- increased tone, some retrocollis/opisthotonus
- minimal feeding, high-pitched cry

Advanced phase:
- deep stupor to coma, hypertonicity
- retrocollis/opisthotonus
- no feeding, shrill cry, seizures, death
6. Screening

The content of this section is based on: *Hyperbilirubinemia in Term and Late Preterm Infants - Screening, Testing and Treatment Guideline* and *Hyperbilirubinemia Toolkit* (2012) from the Champlain Maternal Newborn Regional Program (CMNRP) - http://www.cmnrp.ca/en/cmnrp/Hyperbilirubinemia_Toolkit_p3951.html

**NOTE**: This is a screening procedure for well infants who do not appear overtly jaundiced. Any infant appearing clinically jaundiced, particularly prior to 24 hours of age, should be assessed by a physician, nurse practitioner or midwife and have a serum bilirubin measured as appropriate.

6.1 Screening Steps

1. At the time of newborn screening (24-72h) draw a serum bilirubin (TSB) for analysis in the lab.
2. Note infant’s age in hours at the time of testing and gestational age at birth.
3. Plot bilirubin result on Hour-Specific Bilirubin Nomogram (Figure 1) to find Bilirubin Risk Zone (needed in step 5).
4. Determine presence or absence of the following risk factors for severe hyperbilirubinemia:
   - isoimmune (direct antibody test – DAT) or other hemolytic disease (e.g. G6PD deficiency, hereditary spherocytosis)
   - previous sibling with jaundice received phototherapy
   - cephalohematoma or significant bruising
   - East Asian, Black race
   - exclusive breastfeeding if not well established, as evidenced by good latch and milk transfer, or weight loss over 10%
5. Considering the infant’s gestational age at birth and presence or absence of risk factors above, follow algorithm path A or B or C (Figure 2) to determine whether to assess for phototherapy and timing of repeat testing or clinical follow up. Use Bilirubin Risk Zone determined in step 3 above as indicated in algorithm.
6. If evaluation for phototherapy is indicated, plot serum bilirubin on *Guidelines for Intensive Phototherapy* for Infants 35 or more weeks’ gestation (Figure 3).

- Use **total** bilirubin, do not subtract direct or conjugated bilirubin

- **Risk factors for bilirubin encephalopathy** to consider when determining which line to follow as cutoff for treatment (treatment line) include:
  - isoimmune hemolytic disease, G6PD deficiency
  - asphyxia
  - current and significant lethargy
  - unresolved temperature instability (requiring current, active treatment)
  - sepsis currently being treated
  - ongoing acidosis (not just low cord pH)
  - albumin < 30g/L (if measured)

**NOTE:**
- exclusive breast feeding DOES NOT affect treatment line
- other factors from step 4 not listed in bullets above DO NOT affect treatment line

7. If phototherapy is required:

- Begin with high intensity of at least 30 µw/cm²/nm
- Expose maximum skin surface; limiting interruptions to 20 minutes every 3 hours

8. If phototherapy is not required, schedule next bilirubin check as per algorithm (Figure 2).

9. If the infant is ready for discharge and the algorithm indicates a repeat test is required, discharge home with appropriate follow-up.

10. Advise parents when and where to obtain testing. Each hospital should have its own plan to ensure newborn follow-up.
Figure 1 - Hour-Specific Bilirubin Nomogram


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Figure 2 - Algorithm for determining whether to assess for phototherapy and timing of repeat testing & clinical follow up

A Gestational Age 35+0 to 37+6 weeks AND ANY hyperbilirubinemia risk factors from Step 4*

- Evaluate for phototherapy (Step 6)
- Serum Bilirubin in 4-8 hrs

B Gestational Age 35+0 to 37+6 weeks AND NO hyperbilirubinemia risk factors from Step 4* OR Gestation ≥38 weeks AND ANY hyperbilirubinemia risk factors from Step 4*

- Evaluate for phototherapy (Step 6)
- Serum Bilirubin in 4-24 hrs

C Gestational Age ≥38 weeks AND NO hyperbilirubinemia risk factors from Step 4*

- Follow-up within 2 days
- Consider Serum Bilirubin at follow-up

* Risk factors for severe hyperbilirubinemia: Isimmune or other hemolytic disease (eg G6PD deficiency, hereditary spherocytosis), previous sibling with jaundice requiring phototherapy, cephalohematoma or significant bruising. East Asian, Black race, exclusive breastfeeding if not well established or weight loss over 10%


Copyright CMNRP2012. Adapted with permission.
FIGURE 3 - Guidelines for Intensive Phototherapy for Infants at 35 or More Weeks Gestation


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7. Assessment and Monitoring of Jaundice

7.1 Physical Assessment

- **Visual assessment:** Jaundice moves from head to toe, then the eyes are affected last.
  
  **Serum bilirubin (approx.):**
  
  - $85 \text{ µmol/L}$ - When yellow tinge first becomes visible
  - $150 \text{ µmol/L}$ - Yellow tinge appears on trunk
  - $200 \text{ µmol/L}$ - Yellow tinge appears on legs
  - $250 \text{ µmol/L}$ - Eyes (sclera) are affected

- Although visual assessment alone cannot determine the degree of jaundice, a general assessment of spread of jaundice can be done under bright light:
  
  - Blanch skin to determine underlying colour
  - Press over bony prominence for best results (nose, forehead)
  - Check sclera
  - For dark skinned infants, the colour of the sclera, conjunctiva, and oral mucosa is most reliable indicator of level of jaundice
  - Petechiae may indicate underlying sepsis or haemolytic disease

- **Level of activity:**
  
  - Increasing levels of unconjugated bilirubin in the brain can lead to decreased levels of consciousness or awareness and thus infants may become more lethargic and less responsive

- **Level of hydration:**
  
  - Monitor intake and output
  - Adequate hydration is necessary to help maintain enough fluid to help with the absorption and excretion of conjugated bilirubin once it passes through the liver

- **Stools:**
  
  - Monitor frequency, type and colour of stools (transitional versus meconium)
  - Unconjugated bilirubin can accumulate in stool and thus has the potential to be reabsorbed
  - Conjugated bilirubin can also become deconjugated in the gut and become reabsorbed into the blood stream
7.2 Laboratory Assessment

- Obtain serum bilirubin levels as per algorithm (Figure 2).
- When bloodwork is being drawn, phototherapy lights should be turned off to prevent the sample from being affected by the lights. Some sites require the specimen to be protected from light during transfer to lab (e.g. wrap in tinfoil).
- The total bilirubin should be interpreted according to the infant’s age in hours to determine the severity of the situation.
- It is possible that bilirubin levels could rise again once phototherapy is discontinued although current methods of weaning phototherapy from high intensity (intensive) to low intensity (standard) phototherapy can prevent this. A follow up serum bilirubin should be taken 12-24 hours after therapy is discontinued.
- Other blood work that may be ordered:
  - Serum albumin - to help determine how much albumin is available for binding
  - CBC and differential – can help determine level of red blood cell destruction, haemolytic anemia, sepsis, or polycythemia
  - Direct Antiglobulin Test (DAT) - to look for presence of maternal antibodies in infant’s serum. (Note – Indirect Antibody Test {IAT} is done on maternal serum antenatally)
  - G6PD (glucose-6-phosphate dehydrogenase ) – helps maintain RBC wall integrity; a deficiency indicates enzyme deficiency and a possible metabolic reason for jaundice

8. Phototherapy

8.1 Phototherapy Principles

Effectiveness of phototherapy = 
Area of skin exposed + Radiant energy + Wavelength of light used
Phototherapy acts on unconjugated bilirubin to a depth of 2 to 3 mm. Through photoisomerization, fat soluble molecules are reconfigured to water soluble molecules and are excreted by the liver without actual conjugation.

**Structural photoisomerization**

- The most effective light sources for degrading bilirubin are those that emit blue-green light in a relatively narrow wavelength range (425 – 490 nanometers).
- When phototherapy is used, the fall in bilirubin level is proportionately greater in the skin than in the serum. The infant should have as much skin as possible exposed to the lights.
- It is possible to increase the efficacy of treatment by using multiple sources of phototherapy to optimize the amount of skin exposed.
- According to standards of care phototherapy is ordered as either intensive (high) or standard (low) intensity and expressed in nanometers of light.

Maisels & Mcdonagh, 2008

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8.2 Phototherapy Equipment

There are a variety of methods of delivering phototherapy. The method used depends on the equipment availability in each institution. The following are the current recommended methods:

- **Phototherapy lights**: Deliver light in the narrow spectrum most effective for reducing bilirubin. Their effectiveness depends on the distance from the baby as measured by a specific light meter.

- **Bili Bassinet**: Self-contained unit that combines a mattress area and three phototherapy light units to provide phototherapy and may be used as an adjunct therapy to phototherapy lights, but should not be used as the sole source of therapy.

- **Bili Blanket**: A small fiber optic pad placed under the infant. It can be used as an adjunct to overhead phototherapy lights but not as the only source of phototherapy. The advantage of a bili blanket is that it can remain in place for breastfeeding, providing continuity of therapy.

8.3 Potential Side Effects of Phototherapy

<table>
<thead>
<tr>
<th>SIDE EFFECT</th>
<th>SPECIFIC SIGNS / SYMPTOMS</th>
<th>RATIONALE/IMPLICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Altered Activity</td>
<td>• Lethargy or irritability</td>
<td>• May impact parent-infant interaction</td>
</tr>
<tr>
<td></td>
<td>• Decreased eagerness to feed</td>
<td>• May alter fluid and caloric intake</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Observe and support parental efforts and concerns.</em></td>
</tr>
<tr>
<td>Fluid Status</td>
<td>• Increased peripheral blood flow</td>
<td>• Increased fluid loss may alter uptake of I.M. medications</td>
</tr>
<tr>
<td></td>
<td>• Increased insensible water loss with open bed or warmer</td>
<td>• Due to increased evaporative water loss, metabolic rates and possible respiratory rate</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Monitor weight and fluid intake/output.</em></td>
</tr>
<tr>
<td>GI Function</td>
<td>• Increased number and frequency of watery, greenish-brown stools</td>
<td>• May be related to increased bile flow thus stimulating GI activity</td>
</tr>
<tr>
<td></td>
<td>• Decreased time for intestinal transit</td>
<td>• Increased stools water loss leading to risk of dehydration</td>
</tr>
<tr>
<td></td>
<td>• Decreased absorption, retention of nitrogen, water, electrolytes</td>
<td><em>Monitor output of stool.</em></td>
</tr>
<tr>
<td>Hematological Changes</td>
<td>• Increased rate of platelet turnover</td>
<td>• May be a problem in infants with low platelets and sepsis</td>
</tr>
<tr>
<td></td>
<td>• Damage to circulating red blood cells with decreased potassium and increased ATP (energy) activity</td>
<td>• May lead to hemolysis, increased energy needs <em>Observe for bruising and petechiae.</em></td>
</tr>
<tr>
<td>Ocular Effects</td>
<td>• Concerns about effects of lights vs. eye patches</td>
<td>• Lack of sensory input and stimulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Eye patches increase risk of eye infection, corneal abrasion, increased intracranial pressure (if applied too tight)</td>
</tr>
</tbody>
</table>
Newborn Hyperbilirubinemia: A Self-Learning Module

### 9. Nursing Care

Nursing care of the infant with hyperbilirubinemia is focused on assessment and management of the signs and symptoms of the disease. The main goals of treatment are to prevent the development of bilirubin-induced encephalopathy and reverse the haemolytic process. Therefore, most hospitalized infants will require the use of phototherapy initially to help reduce the amount of unconjugated (indirect) bilirubin, and there are certain aspects of care related to the use of the “Bili lights” to maintain a safe environment. The guidelines included here refer to newborns receiving phototherapy.

#### 9.1 Feeding /Nutrition

- Fluid intake is crucial to treatment success so this is an important factor to consider in caring for an infant with jaundice. Dehydration may be associated with increased serum bilirubin concentrations and intravenous (IV) therapy may be necessary to ensure adequate hydration.
- Breastfeeding should always be strongly encouraged and supported, even when the infant is receiving phototherapy (more frequent breastfeeding may be beneficial). Minimize the time that...
intensive phototherapy is interrupted to 20 minutes within a 3-hour period. Breastfeeding can continue with a Bili Blanket in place. If possible and as needed, provide a referral to a Lactation Consultant for a more specific assessment of breastfeeding.

- Accurate recording of intake and output, including stool pattern, is necessary. If the infant is receiving IV therapy, usual standards of care should be followed.

### 9.2 Skin Care and Thermoregulation

- Skin plays an important role in the regulation of body temperature and serves as a route of water excretion.
- Skin care includes observing colour, rashes, excoriation; cleaning the skin with warm water; cleaning the perineal area after stooling; changing position when providing care. Ensuring prompt cleansing of perianal area after stooling is important as photo-oxidation of bilirubin results in loose green caustic stools and can result in excoriation.
- Phototherapy is most effective when the exposed skin area is maximized, so infants in isolettes should be exposed as much as possible; diapers to catch urine and stool should be used.
- The infant’s temperature must be monitored at least every 4 hours to maintain a safe environment.

### 9.3 Eye Care

- The infant’s eyes must be protected from the phototherapy lights to prevent retinal damage. Check the manufacturer’s recommendations regarding eye protection.
- Place the pads correctly over the eyes:
  - Use the appropriate size of the eye pad or prefabricated eye protection.
  - Make sure the eyes are closed first to help prevent corneal abrasion, irritation and infection.
  - Check frequently to make sure the eye pads are not obstructing the nares.
- Eye protection should be removed every 2 to 4 hours and eyes cleansed with normal saline to reduce irritation and promote eye contact, socialization and attachment.
- Eye patches are not necessary if only the Bili Blanket is being used since the infant’s eyes are not being exposed to the blue lights directly as they would be with overhead lights.
10. References and Resources


The Champlain Maternal Newborn Regional Program (CMNRP) would like to thank the members of the Joint Orientation Sub-Committee for their work on the development of this Newborn Hyperbilirubinemia Self-Learning Module.

CMNRP also acknowledges the work of the following groups and health care professionals who have provided feedback and expertise:

- Members of the Interprofessional Education & Research Committee (IERC)
- Members of the Jaundice Working Group
- Pediatricians and Neonatologists
- CMNRP Perinatal Consultants and Neonatal Nurse Practitioners