

HYPERBILIRUBINEMIA

Elevation of serum bilirubin levels is related to hemolysis of RBCs and subsequent re-absorption of unconjugated bilirubin from the small intestines. The condition may be benign or may place the neonate at risk for multiple complications/untoward effects.

NEONATAL ASSESSMENT DATA BASE

Activity/Rest

Lethargy, listlessness

Circulation

May be pale, indicating anemia
Residing at altitudes above 5000 ft
Cardiomegaly; increased bleeding tendencies (hydrops fetalis)

Elimination

Bowel sounds hypoactive.
Meconium passage may be delayed.
Stools may be loose/greenish brown during bilirubin excretion.
Urine dark, concentrated; brownish black (bronze baby syndrome).

Food/Fluid

History of delayed/poor oral feeding, poor sucking reflex.
More likely to be breastfed than bottle-fed.
Abdominal palpation may reveal enlarged spleen, liver.
Generalized edema, ascites (hydrops fetalis).

Neurosensory

Large cephalhematoma may be noted over one or both parietal bones related to birth trauma/vacuum extraction delivery.
Loss of Moro reflex may be noted.
Opisthotonos with rigid arching of back, bulging fontanel, shrill cry, seizure activity (crisis stage).

Respiration

History of asphyxia
Crackles, pink-tinged mucus (pleural edema, pulmonary hemorrhages)

Safety

History may be positive for infection/neonatal sepsis.
May have excessive ecchymosis, petechiae, intracranial bleeding.
May appear jaundiced initially on the face with progression to distal parts of the body; skin brownish black in color (bronze baby syndrome) as a side effect of phototherapy.

Sexuality

May be preterm, SGA infant, infant with IUGR, or LGA infant, such as IDM.
Birth trauma may have occurred associated with cold stress, asphyxia, hypoxia, acidosis, hypoglycemia, hypoproteinemia.
Occurs more often in male than female infants.

Teaching/Learning

May have congenital hypothyroidism, biliary atresia, cystic fibrosis (inspissated bile)
Family factors; e.g., ethnic descent (Asian, Greek, or Korean), history of hyperbilirubinemia in previous pregnancies/siblings, liver disease, cystic fibrosis, inborn errors of metabolism (galactosemia), blood dyscrasias

(spherocytosis, glucose-6-phosphate dehydrogenase [G-6-PD] deficiency)

Maternal factors, such as maternal diabetes; ingestion of medications (e.g., salicylates, oral sulfonamides late in pregnancy or nitrofurantoin [Furadantin]; Rh/ABO incompatibility; infectious illness (e.g., rubella, CMV, syphilis, toxoplasmosis)

Intrapartal contributing factors, such as preterm labor, delivery by vacuum extraction, oxytocin induction, delayed clamping of umbilical cord, or traumatic delivery

DIAGNOSTIC STUDIES

Coombs' Test on Newborn Cord Blood: Positive result of indirect Coombs' test indicates the presence of Rh-positive, anti-A, or anti-B antibodies in mother's blood. Positive result of direct Coombs' test indicates presence of sensitized (Rh-positive, anti-A, or anti-B) RBCs in neonate.

Infant and Maternal Blood Type: Identifies ABO incompatibilities.

Total Bilirubin: Direct (conjugated) levels are significant if they exceed 1.0–1.5 mg/dl, which may be associated with sepsis. Indirect (unconjugated) levels should not exceed an increase of 5 mg/dl in 24 hr, or should not be >20 mg/dl in a full-term infant or 15 mg/dl in a preterm infant (dependent on weight). Cord blood bilirubin 5 mg/dl indicates severe hemolysis.

Total Serum Protein: Levels <3.0 g/dl indicate reduced binding capacity, particularly in preterm infant.

CBC: Hb may be low (14 g/dl) because of hemolysis. Hct may be elevated (65%) with polycythemia, decreased (45%) with excess hemolysis and anemia. Cord-blood Hct of 40% indicates severe hemolysis.

Glucose: Dextrostix level may be 45% whole blood glucose 30 mg/dl, or serum glucose 40 mg/dl if newborn is hypoglycemic and begins to use fat stores and release fatty acids.

Carbon Dioxide (CO₂) Combining Power: Decreased level reflects hemolysis.

Transcutaneous Jaundice Meter: Identifies infants requiring serum bilirubin determination.

Reticulocyte Count: Elevation (reticulocytosis) indicates increased production of RBCs in response to hemolysis associated with RH disease.

Peripheral Blood Smear: May reveal abnormal or immature RBCs, erythroblasts in Rh disease, or spherocytes in ABO incompatibility.

Kleihauer-Betke Test: Evaluation of maternal blood smear for fetal erythrocytes.

Ultrasound: May be done prenatally in high-risk situations to diagnose hydrops fetalis before delivery, as noted by scalp edema, cardiomegaly, hepatomegaly, plural effusions, and ascites.

NURSING PRIORITIES

1. Prevent injury/progression of condition.
2. Provide support/appropriate information to family.

DISCHARGE CRITERIA:

1. Maintaining physiological homeostasis with bilirubin levels declining.
2. Complications prevented/resolving.
3. Parent(s) understand condition/prognosis and therapeutic regimen.
4. Plan in place to meet needs after discharge.

NURSING DIAGNOSIS:

Risk Factors May Include:

Possibly Evidenced By:

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

INJURY, risk for CNS involvement

Prematurity, hemolytic disease, asphyxia, acidosis, hypoproteinemia, and hypoglycemia

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

Display indirect bilirubin levels below 12 mg/dl in term infant at 3 days of age.

Show resolution of jaundice by end of the 1st wk of life.

Be free of CNS involvement.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Note infant/maternal blood group and blood type.

ABO incompatibilities affect 20% of all pregnancies and most commonly occur in mothers with type O blood, whose anti-A and anti-B antibodies pass into fetal circulation, causing RBC agglutination and hemolysis. Similarly, when an Rh-negative mother has previously been sensitized by Rh-positive antigens, maternal antibodies cross the placenta and attach to fetal RBCs, causing immediate or delayed hemolysis.

Review intrapartal record for specific risk factors, such as low birth weight (LBW) or IUGR, prematurity, abnormal metabolic processes, vascular injuries, abnormal circulation, sepsis, or polycythemia.

Certain clinical conditions may cause a reversal of the blood-brain barrier, allowing bound bilirubin to separate either at the level of the cell membrane or within the cell itself, increasing the risk of CNS involvement.

Note use of vacuum extractor for delivery. Assess infant for presence of cephalhematoma and excessive ecchymosis or petechiae.

Resorption of blood trapped in fetal scalp tissue and excessive hemolysis may increase the amount of bilirubin being released and cause jaundice.

Review infant's condition at birth, noting need for resuscitation or evidence of excessive ecchymosis or petechiae, cold stress, asphyxia, or acidosis.

Asphyxia and acidosis reduce affinity of bilirubin to albumin.

Keep infant warm and dry; monitor skin and core temperature frequently.

Cold stress potentiates release of fatty acids, which compete for binding sites on albumin, thereby increasing the level of freely circulating (unbound) bilirubin.

Initiate early oral feedings within 4–6 hr following birth, especially if infant is to be breastfed. Assess infant for signs of hypoglycemia. Obtain Dextrostix levels, as indicated.

Establishes proper intestinal flora necessary for reduction of bilirubin to urobilinogen; decreases enterohepatic circulation of bilirubin (bypassing liver with persistence of ductus venosus); and decreases reabsorption of bilirubin from bowel by promoting passage of meconium. Hypoglycemia necessitates use of fat stores for energy-releasing fatty acids, which compete with bilirubin for binding sites on albumin.

Evaluate maternal and prenatal nutritional levels; note possible neonatal hypoproteinemia, especially in preterm infant.

Hypoproteinemia in the newborn may result in jaundice. One gram of albumin carries 16 mg of unconjugated bilirubin. Lack of sufficient albumin increases the amount of unbound circulating (indirect) bilirubin, which may cross the blood-brain barrier.

Observe infant in natural light, noting sclera and oral mucosa, yellowing of skin immediately after blanching, and specific body parts involved. Assess oral mucosa, posterior portion of hard palate, and conjunctival sacs in dark-skinned newborns.

Note infant's age at onset of jaundice; differentiate type of jaundice (i.e., physiological, breast milk-induced, or pathological).

Apply transcutaneous jaundice meter.

Assess infant for progression of signs and behavioral changes: Stage I involves neurodepression (e.g., lethargy, hypotonia, or diminished/absent reflexes). Stage II involves neurohyperreflexia (e.g., twitching, convulsions, opisthotonos, or fever). Stage III is marked by absence of clinical manifestations. Stage IV involves sequelae such as cerebral palsy or mental retardation.

Evaluate infant for pallor, edema, or hepatosplenomegaly.

Collaborative

Monitor laboratory studies, as indicated:

Direct and indirect bilirubin;

Detects evidence/degree of jaundice. Clinical appearance of jaundice is evident at bilirubin levels $>7-8$ mg/dl in full-term infant. Estimated degree of jaundice is as follows, with jaundice progressing from head to toe: face, 4–8 mg/dl; trunk, 5–12 mg/dl; groin, 8–16 mg/dl; arms/legs, 11–18 mg/dl; and hands/feet, 15–20 mg/dl. Note: Yellow underlying pigment may be normal in dark-skinned infants.

Physiological jaundice usually appears between the 2nd and 3rd days of life, as excess RBCs needed to maintain adequate oxygenation for the fetus are no longer required in the newborn and are hemolyzed, thereby releasing bilirubin, the final breakdown product of heme. Breast milk jaundice usually appears between the 4th and 6th days of life, affecting only 1%–2% of breastfed infants. The breast milk of some women is thought to contain an enzyme (pregnanediol) that inhibits glucuronyl transferase (the liver enzyme that conjugates bilirubin), or to contain several times the normal breast milk concentration of certain free fatty acids, which are also thought to inhibit the conjugation of bilirubin. Pathological jaundice appears within the first 24 hr of life and is more likely to lead to the development of kernicterus/bilirubin encephalopathy.

Provides noninvasive screening of jaundice, quantifying skin color in relation to total serum bilirubin.

Excessive unconjugated bilirubin (associated with pathologic jaundice) has an affinity for extravascular tissue, including the basal ganglia of brain tissue. Behavior changes associated with kernicterus usually occur between the 3rd and 10th days of life and rarely occur prior to 36 hr of life.

These signs may be associated with hydrops fetalis, Rh incompatibility, and in utero hemolysis of fetal RBCs.

Bilirubin appears in two forms: direct bilirubin, which is conjugated by the liver enzyme glucuronyl transferase, and indirect bilirubin, which is unconjugated and appears in a free form in the blood or bound to albumin. The infant's potential for kernicterus is best predicted by elevated levels of indirect bilirubin. Elevated indirect bilirubin levels of 18–20 mg/dl in full-term infant, or 13–15 mg/dl in preterm or sick infant, are significant. Note: Stressed or preterm infant is susceptible to deposition of bile

Direct/indirect Coombs' test on cord blood;	pigments within brain tissue at far lower levels than nonstressed full-term infant.
CO ₂ combining power; Reticulocyte count and peripheral smear;	Positive results of indirect Coombs' test indicate presence of antibodies (Rh-positive or anti-A, anti-B) in mother's and newborn's blood; positive results of direct Coombs' test indicate presence of sensitized (Rh-positive, anti-A, or anti-B) RBCs in neonate. A decrease is consistent with hemolysis. Excessive hemolysis causes reticulocyte count to increase. Smear identifies abnormal or immature RBCs.
Hb/Hct;	Elevated Hb/Hct levels (Hb 22 g/dl; Hct 65%) indicate polycythemia, possibly caused by delayed cord clamping, maternal-fetal transfusion, twin-to-twin transfusion, maternal diabetes, or chronic intrauterine stress and hypoxia, as seen in LBW infant or infant with compromised placental circulation. Hemolysis of excess RBCs causes elevated levels of bilirubin with 1 g of Hb yielding 35 mg of bilirubin. Low Hb levels (14 mg/dl) may be associated with hydrops fetalis or with Rh incompatibility occurring in utero and causing hemolysis, edema, and pallor.
Total serum protein.	Low levels of serum protein (3.0 g/dl) indicate reduced binding capacity for bilirubin.
Calculate plasma bilirubin-albumin binding capacity.	Aids in determining risk of kernicterus and treatment needs. When total bilirubin value divided by total serum protein level is <3.7, the danger of kernicterus is very low. However, the risk of injury is dependent on degree of prematurity, presence of hypoxia or acidosis, and drug regimen (e.g., sulfonamides, chloramphenicol).
Initiate phototherapy per protocol, using fluorescent bulbs placed above the infant or bile blanket (except for newborn with Rh disease). (Refer to NDs: Injury, risk for side effects of phototherapy; Injury, risk for complications of exchange transfusions.)	Causes photo-oxidation of bilirubin in subcutaneous tissue, thereby increasing water solubility of bilirubin, which allows rapid excretion of bilirubin in stool and urine. Rate of hemolysis in Rh disease usually exceeds rate of bilirubin reduction related to phototherapy, so that an exchange transfusion is the only appropriate treatment.
Discontinue breastfeeding for 24–48 hr, as indicated. Assist mother as needed with pumping of breasts and reestablishment of breastfeeding.	Opinions vary as to whether interrupting breastfeeding is necessary when jaundice occurs. However, formula ingestion increases GI motility and excretion of stool and bile pigment, and serum bilirubin levels do begin to fall within 48 hr after discontinuation of breastfeeding.
Administer enzyme induction agent (phenobarbital, ethanol), as appropriate.	Stimulates hepatic enzymes to enhance clearance of bilirubin.

Assist with preparation and administration of exchange transfusion. Use same type of blood as infant's, but Rh-negative or type O-negative blood, if results of direct Coombs' test on cord serum are >3.5 mg/dl in the 1st wk of life, serum unconjugated bilirubin levels are 20 mg/dl in the first 48 hr of life, or Hb is 12 g/dl at birth in infants with hydrops fetalis. (Refer to ND: Injury, risk for complications of exchange transfusions.)

Exchange transfusions are necessary in cases of severe hemolytic anemia, which are usually associated with Rh incompatibility, to remove sensitized RBCs that would soon lyse; to remove serum bilirubin; to provide bilirubin-free albumin to increase binding sites for bilirubin; and to treat anemia by providing RBCs that are not susceptible to maternal antibodies.

NURSING DIAGNOSIS:

Risk Factors May Include:

Possibly Evidenced By:

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

INJURY, risk for side effects of phototherapy

Physical properties of therapeutic intervention and effects on body regulatory mechanisms

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

Maintain body temperature and fluid balance
WNL.

Be free of skin/tissue injury.

Demonstrate expected interaction patterns.

Display decreasing serum bilirubin levels.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Note presence/development of biliary or intestinal obstruction.

Measure quantity of photoenergy of fluorescent bulbs (white or blue light) using photometer.

Document type of fluorescent lamp, total number of hours since bulb replacement, and the measured distance between lamp surface and infant.

Phototherapy is contraindicated in these conditions because the photoisomers of bilirubin produced in the skin and subcutaneous tissues by exposure to light therapy cannot be readily excreted.

The intensity of light striking skin surface from blue spectrum (blue lights) determines how close to the light source the infant should be placed. Photometer should register between 8 and 9 $\mu\text{W}/\text{cm}^2/\text{nm}$ of light when placed flush with infant's abdomen. Blue and special blue lights are considered more effective than white light in promoting bilirubin breakdown, but they create difficulty in evaluating the newborn for cyanosis.

Light emission may decay over time. Infant should be placed approximately 18–20 in (45 cm) from light source for maximal benefit. Note: Use of fiberoptic blanket connected to an illuminator (light source) allows infant to be “wrapped” in therapeutic light without risk to corneas. In addition, infant can be held and fed without interrupting therapy.

Apply patches to closed eyes; inspect eyes every 2 hr when patches are removed for feedings. Monitor placement frequently.

Cover testes and penis of male infant.

Place Plexiglas shield between baby and light.

Monitor neonate's skin and core temperature every 2 hr or more frequently until stable (e.g., axillary temperature of 97.8°F (36.5°C), rectal temperature of 98.8°F (37.4°C). Regulate incubator/Isolette temperature, as appropriate.

Reposition infant every 2 hr.

Monitor fluid intake and output; weigh infant twice a day. Note signs of dehydration (e.g., reduced urine output, depressed fontanels, dry or warm skin with poor turgor, and sunken eyes). Increase oral fluid intake by at least 25%.

Note color and frequency of stools and urine.

Carefully wash perianal area after each passage of stool; inspect skin for possible irritation or breakdown.

Bring infant to parents for feedings. Encourage stroking, cuddling, eye contact, and talking to infant during feedings. Encourage parents to interact with infant in nursery between feedings.

Note behavioral changes or signs of deteriorating condition (e.g., lethargy, hypotonia, hypertonicity, or extrapyramidal signs).

Evaluate appearance of skin and urine, noting brownish black color.

Collaborative

Monitor laboratory studies, as indicated:

Bilirubin levels every 12 hr;

Prevents possible damage to the retina and conjunctiva from high-intensity light. Improper application or slipping of patches can cause irritation, corneal abrasions, and conjunctivitis, and compromise breathing by obstructing nasal passages.

Prevents possible testicular damage from heat.

Filters out ultraviolet radiation (wavelengths less than 380 nm) and protects infant if bulb breaks.

Fluctuations in body temperature can occur in response to light exposure, radiation, and convection.

Allows equal exposure of skin surfaces to fluorescent light, prevents excessive exposure of individual body parts, and limits pressure areas.

Increased water losses through stools and evaporation can cause dehydration. Note: Infant may sleep for longer periods in conjunction with phototherapy, increasing risk of dehydration if frequent feeding schedule is not maintained.

Frequent, greenish, loose stools and greenish urine indicate effectiveness of phototherapy with breakdown and excretion of bilirubin.

Early intervention helps prevent irritation and excoriation from frequent or loose stools.

Fosters attachment process, which may be delayed if separation required by phototherapy. Visual, tactile, and auditory stimulation helps infant overcome sensory deprivation. Intermittent phototherapy does not negatively affect photo-oxidation process. Note: Dependent on infant condition and policies/capabilities of hospital, phototherapy may be provided in conjunction with rooming-in.

Such changes may signify deposition of bile pigment in the basal ganglia and developing kernicterus.

An uncommon side effect of phototherapy involves exaggerated pigment changes (bronze baby syndrome), which may occur if conjugated bilirubin levels rise. The changes in skin color may last for 2–4 mo but are not associated with harmful sequelae.

Decreases in bilirubin levels indicate effectiveness of phototherapy; continued increases suggest continued hemolysis and may indicate the need for exchange transfusion. Note: Blood sample drawn for bilirubin

Hb levels;
 Platelets and WBCs.
 Administer fluids parenterally as indicated.

determination should be protected from light to prevent continued photo-oxidation.
 Continued hemolysis is manifested by progressive decreases in Hb level.
 Thrombocytopenia during phototherapy has been reported in some infants. Decrease in WBCs suggests a possible effect on peripheral lymphocytes.
 May be necessary to correct or prevent severe dehydration.

NURSING DIAGNOSIS:

INJURY, risk for complications of exchange transfusions

Risk Factors May Include:

Invasive procedure, abnormal blood profile, chemical imbalances

Possibly Evidenced By:

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

DESIRED OUTCOMES/EVALUATION CRITERIA—NEONATE WILL:

Complete exchange transfusion without complications.

Display decreasing serum bilirubin levels.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Note condition of infant's cord prior to transfusion, if umbilical vein is to be used. If cord is dry, administer saline soaks for 30–60 min prior to procedure.

Soaks may be necessary to soften cord and umbilical vein prior to transfusion for IV access and to ease passage of umbilical catheter.

Maintain NPO status for 4 hr prior to procedure, or aspirate gastric contents.

Reduces risk of possible regurgitation and aspiration during procedure.

Ensure availability of resuscitative equipment.

To provide immediate support if necessary.

Maintain infant's temperature prior to, during, and after procedure. Place infant under radiant warmer with servomechanism. Warm blood prior to infusion by placing in incubator, warm basin of water, or blood warmer.

Helps prevent hypothermia and vasospasm, reduces risk of ventricular fibrillation, and decreases blood viscosity.

Verify infant's and mother's blood type and Rh factor. Note blood type and Rh factor of blood to be exchanged. (Exchanged blood will be the same type as the baby's but will be Rh-negative or type O-negative blood that has been cross-matched with mother's blood beforehand.)

Exchange transfusions are most often associated with Rh incompatibility problems. Using Rh₀(D)-positive blood would only increase hemolysis and bilirubin levels, because antibodies in infant's circulation would destroy new RBCs.

Ensure freshness of blood (not more than 2 days old), with heparinized blood preferred.

Older blood is more likely to hemolyze, thereby increasing bilirubin levels. Heparinized blood is always fresh, but must be discarded if not used within 24 hr.

Monitor venous pressure, pulse, color, and respiratory rate/ease before, during, and after transfusion. Suction as needed.

Carefully document events during transfusion, recording amount of blood withdrawn and injected (usually 7–20 ml at a time).

Monitor for signs of electrolyte imbalance (e.g., jitteriness, seizure activity, and apnea; hyperreflexia, bradycardia, or diarrhea).

Assess infant for excessive bleeding from IV site following the transfusion.

Collaborative

Monitor laboratory studies, as indicated:

Hb/Hct levels prior to and following transfusion;

Serum bilirubin levels immediately following procedure, then every 4–8 hr;

Total serum protein;

Serum calcium and potassium;

Glucose;

Serum pH levels.

Administer albumin prior to transfusion if indicated.

Administer medications, as indicated:

5% calcium gluconate;

Establishes baseline values, identifies potentially unstable conditions (e.g., apnea or cardiac dysrhythmia/arrest), and maintains airway. Note: Bradycardia may occur if calcium is injected too rapidly.

Helps prevent errors in fluid replacement. Amount of blood exchanged is approximately 170 ml/kg of body weight. A double-volume exchange transfusion ensures that between 75% and 90% of circulating RBCs are replaced.

Hypocalcemia and hyperkalemia may develop during and following exchange transfusion.

Infusion of heparinized blood (or citrated blood without calcium replacement) alters coagulation for 4–6 hr following the exchange transfusion and may result in bleeding.

If Hct is 40% prior to transfusion, a partial exchange with packed RBCs may precede full exchange. Dropping levels following the transfusion suggest the need for a second transfusion. Bilirubin levels may decrease by half immediately following procedure but may rise shortly thereafter, necessitating a repeat transfusion.

Multiplying level by 3.7 determines the degree of elevation of bilirubin necessitating exchange transfusion.

Donor blood containing citrate as an anticoagulant binds calcium, thereby decreasing serum calcium levels. In addition, if blood is more than 2 days old, RBC destruction releases potassium, creating a risk of hyperkalemia and cardiac arrest.

Low glucose levels may be associated with continued anaerobic glycolysis within donor RBCs. Prompt treatment is necessary to prevent untoward effects/CNS damage.

Serum pH of donor blood is typically 6.8 or less. Acidosis may result when fresh blood is not used and infant's liver cannot metabolize citrate used as an anticoagulant, or when donor blood continues anaerobic glycolysis, with production of acid metabolites.

Although somewhat controversial, administration of albumin may increase the albumin available for binding of bilirubin, thereby reducing levels of freely circulating serum bilirubin. Synthetic albumin is not thought to increase available binding sites.

From 2–4 ml of calcium gluconate may be administered after every 100 ml of blood infusion to

Sodium bicarbonate;
Protamine sulfate.

correct hypocalcemia and minimize possible cardiac irritability. Note: Some controversy exists as to the purpose and effectiveness of this practice.
Corrects acidosis.
Counteracts anticoagulant effects of heparinized blood.

NURSING DIAGNOSIS:**KNOWLEDGE deficit [Learning Need], regarding condition, prognosis, and treatment needs****May Be Related To:**

Lack of exposure, misinterpretation, unfamiliarity with information resources

Possibly Evidenced By:

Request for information, statement of problem/misconceptions, inaccurate follow-through of instructions

DESIRED OUTCOMES/EVALUATION CRITERIA—PARENT(S) WILL:

Verbalize understanding of cause, treatment, and possible outcomes of hyperbilirubinemia.

Demonstrate appropriate care of infant.

Identify signs/symptoms requiring prompt notification of healthcare provider.

ACTIONS/INTERVENTIONS**RATIONALE**

Independent

Provide information about types of jaundice and pathophysiological factors and future implications of hyperbilirubinemia. Encourage questions; reinforce or clarify information, as needed.

Promotes understanding, corrects misconceptions, and can reduce fear and feelings of guilt. Neonatal jaundice may be physiological, breast milk-induced, or pathological, and protocol of care depends on its cause and contributing factors.

Demonstrate means of assessing infant for increasing bilirubin levels (e.g., observing blanching of skin over bony prominence or behavior changes), especially if infant is to be discharged early. Provide parents with 24-hr emergency telephone number and name of contact person, stressing importance of reporting increased jaundice.

Enables parents to recognize signs of increasing bilirubin levels and to seek timely medical evaluation/intervention.

Discuss home management of mild or moderate physiological jaundice, including increased feedings, diffused exposure to sunlight (checking infant frequently), and follow-up serum testing program.

Parents' understanding helps foster their cooperation once infant is discharged. Information helps parents to carry out home management safely and appropriately and to recognize the importance of all aspects of management program. Note: Exposure to direct sunlight is contraindicated as infant's tender skin is highly susceptible to thermal injury.

Provide information about maintaining milk supply through use of breast pump and about reinstating breastfeeding when jaundice necessitates interruption of breastfeeding.

Review rationale for specific hospital procedures/therapeutic interventions (e.g., phototherapy, exchange transfusions) and changes in bilirubin levels, especially in the event that neonate must remain in hospital for treatment while mother is discharged.

Discuss need for Rh immune globulin (RhIg) within 72 hr following delivery for an Rh-negative mother who has an Rh-positive infant and who has not been previously sensitized.

Assess family situation and support systems. Provide parents with appropriate written explanation of home phototherapy, listing technique and potential problems, and safety precautions. Discuss appropriate monitoring of home therapy, e.g., periodic recording of infant's weight, feedings, intake/output, stools, temperature, and proper reporting of infant status.

Provide appropriate referral for home phototherapy program, if necessary.

Make appropriate arrangements for follow-up testing of serum bilirubin at same laboratory facility.

Discuss possible long-term effects of hyperbilirubinemia and the need for continued assessment and early intervention.

Helps mother maintain adequate milk supply to meet infant's needs when breastfeeding is resumed.

Assists parents in understanding importance of therapy. Keeps parents informed about infant's status. Promotes informed decision making. Note: Some hospitals have overnight rooms that allow mother/father to remain with infant.

In Rh₀(D)-negative client with no Rh antibodies, who has given birth to an Rh₀(Du)-positive infant. Rh-Ig may reduce incidence of maternal isoimmunization in nonsensitized mother and may help to prevent erythroblastosis fetalis in subsequent pregnancies.

Home phototherapy is recommended only for full-term infants after the first 48 hr of life, whose serum bilirubin levels are between 14 and 18 mg/dl with no increase in direct reacting bilirubin concentration.

Lack of available support systems and education may necessitate use of visiting nurse to monitor home phototherapy program.

Treatment is discontinued once serum bilirubin concentrations fall below 14 mg/dl, but serum levels must be rechecked in 12–24 hr to detect possible rebound hyperbilirubinemia.

Neurological damage associated with kernicterus includes death, cerebral palsy, mental retardation, sensory difficulties, delayed speech, poor muscle coordination, learning difficulties, and enamel hypoplasia or yellowish green staining of teeth.

(Refer to CP: The Parents of a Child with Special Needs; ND: Knowledge deficit [Learning Need].)