2. Physical characteristics of the postterm newborn may include: *(Select all that apply.)*
   1. Long fingernails
   2. Loose skin
   3. Cracked, dry skin
   4. Abundant lanugo

3. Retinopathy of prematurity (ROP) is primarily caused by:
   1. Oxygen toxicity
   2. Hypoglycemia
   3. Heat loss
   4. Hyperbilirubinemia

4. The nurse is caring for a newborn diagnosed with thickening of the alveolar sacs with the occurrence of atelectasis and scarring. The newborn's history and physical indicate this diagnosis is the result of the child receiving supplemental oxygen for a prolonged time. The nurse is aware that this complication of prolonged oxygen therapy is:
   1. Necrotizing enterocolitis (NEC)
   2. Patent ductus arteriosus (PDA)
   3. Bronchopulmonary dysplasia (BPD)
   4. Retinopathy of Prematurity (ROP)

5. Which statement made by a student nurse regarding the preterm newborn demonstrates the need for further education?
   1. "Preterm newborns may become irritable when hyperstimulated by the environment."
   2. "Preterm newborns are unable to perceive pain."
   3. "The preterm newborn may be positioned on the back with the head of the mattress slightly elevated."
   4. "The preterm newborn has high caloric needs."

6. Most prematurely born infants catchup to the appropriate developmental level by:
   1. 6 months of age
   2. 12 months of age
   3. 18 months of age
   4. 24 months of age

7. Management of necrotizing enterocolitis (NEC) includes which intervention(s)? *(Select all that apply.)*
   1. Nasogastric suctioning
   2. Frequent measurement of abdominal girth
   3. Decreased number of oral feedings
   4. Administration of intravenous fluids
   5. Administration of corticosteroids

Critical Thinking Questions

1. A woman has just given birth to a preterm newborn, 32 weeks' gestation, weight 1.4 kg (3 lbs). She is anxious and is expressing how she had looked forward to a "normal baby girl." What approach will you take with the woman as you assist her in providing newborn care?

2. A woman gave birth to a 4.4 kg (9 lbs, 11 oz) boy. She had a difficult labor but did deliver vaginally. What patient teaching should you give to the woman?

chapter

16 The Newborn at Risk: Acquired and Congenital Conditions

evolve

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Objectives

1. Define key terms listed.
2. Discuss the prenatal diagnosis of Down syndrome.
3. Recognize three genetic inborn errors of metabolism.
4. Compare the metabolic disorders hypoglycemia, maple syrup urine disease, hypothyroidism, and phenylketonuria; their effect on the newborn; and the nursing implications.
5. Describe common congenital anomalies.
6. Interpret signs associated with elevated bilirubin in the newborn.
7. Explain the nursing interventions used in phototherapy.
8. Articulate the principles of newborn resuscitation.
9. Outline the common respiratory problems in the newborn.
10. Characterize the effect of maternal diabetes on the newborn.
11. Outline six problems of infants born to mothers with diabetes mellitus.
12. Explain factors responsible for newborn sepsis, and state the nurse's role in reducing the risks.
13. Discuss the nursing assessment that would lead the nurse to suspect newborn sepsis.
14. Identify the defects involved in the tetralogy of Fallot and common manifestations.
15. Compare the alteration of blood flow of cyanotic and noncyanotic congenital heart defects.
16. Explain the pathophysiology of noncyanotic congenital heart defects.
17. Describe care of the newborn who has neonatal abstinence syndrome.

Key Terms

galactosemia (gă-lăk-tō-SĒ-mē-ă, p. 323)
hemolysis (hē-MŎL-ī-sĭs, p. 329)
hyperglycemia (hī-pĕr-glī-SĒ-mē-ă, p. 336)
hypoglycemia (hī-pō-glī-SĒ-mē-ă, p. 332)
infants of diabetic mothers (IDMs) (p. 336)
kernicterus (kĕr-NĬK-tĕr-ŭs, p. 329)
macrosomia (măk-rō-SŌ-mē-ă, p. 336)
meconium aspiration syndrome (MAS) (mĕ-KŌ-nē-ŭm, p. 334)
neonatal sepsis (nē-ō-NĀ-tăl, p. 337)
phenylketonuria (PKU) (fēn-ŭl-kē-tō-NŪ-rē-ă, p. 322)
phototherapy (p. 330)

Genetics is the scientific study of the transmission of characteristics from parent to child (see Chapters 1 and 3). Genetic defects are common causes of acute and chronic conditions that manifest during fetal life; immediately after birth; or during childhood, adolescence, or adulthood. An understanding of human genetics, including genetic challenges, disorders, and technology, is an integral part of maternity nursing and medical obstetric practice.

Birth defects, abnormalities that are apparent at birth, occur in 3% to 4% of all live births. The rate is even higher if the defects that become evident later in life are counted. An abnormality of structure, function, or metabolism may result in a physical or mental disability, may shorten life, or may be fatal. Box 16-1 shows the system of classification of birth defects. Because these disorders include so many conditions, it is necessary to limit the number discussed in the chapter and to place others in relevant areas of the text (see the index for specific conditions). Defects that are manifested later in life are discussed in pediatric textbooks.

Defects present at birth often involve the skeletal system; limbs may be missing, malformed, or duplicated. Some abnormalities (e.g., congenital hip dysplasia) are more subtle, and the nurse must be alert to detect them. *Inborn errors of metabolism* include a number of inherited diseases that affect body chemistry. There may be an absence or a deficiency of a substance necessary for cell metabolism. The deficient substance is usually an enzyme. Almost any organ of the body may be damaged. Examples of inborn errors of metabolism include cystic fibrosis and phenylketonuria (PKU). In *disorders of the blood,* there is a reduced or missing blood component or an inability of a component to function adequately. Sickle cell disease, thalassemia, and hemophilia fall into this category. *Chromosomal abnormalities* number in the thousands. Most involve some type of mental retardation, and others are incompatible with life. The newborn with Turner's syndrome or Klinefelter's syndrome may have impaired physical growth and sexual development. *Perinatal injuries* have many causes and are seen

Box 16-1 Classification and Examples of Birth Defects

MALFORMATIONS PRESENT AT BIRTH

Structural defects, including:

- Hydrocephalus*
- Spina bifida*
- Congenital heart malformations*
- Cleft lip and palate*
- Clubfoot*
- Developmental hip dysplasia*
- Tracheoesophageal fistula*
- Hypospadias

METABOLIC DEFECTS (BODY CHEMISTRY)

- Cystic fibrosis
- Phenylketonuria (PKU)*
- Galactosemia*
- Maple syrup urine disease*
- Hypothyroidism*
- Tay-Sachs disease
- Family hypercholesterolemia (high cholesterol that often causes early heart attack)

BLOOD DISORDERS

- Sickle cell disease
- Hemophilia
- Thalassemia
- Defects of white blood cells and immune defense

CHROMOSOMAL ABNORMALITIES

Many abnormalities, most involving some combination of mental retardation and physical malformations that range from mild to fatal, including:

- Down syndrome*
- Klinefelter's syndrome
- Turner's syndrome
- Trisomies 13, 18, and 21

PERINATAL INJURY

- Infections*
- Drugs*
- Maternal disorders
- Abnormalities unique to pregnancy (hyperbilirubinemia,* difficult labor or delivery, premature birth)
- Meconium aspiration syndrome*

*Topics discussed in this chapter. More detailed discussions of other conditions can be found in other chapters within this text.

in various forms, the most common of which is premature birth.

As the March of Dimes Foundation (2007) points out, "Few birth defects can be attributed to a single cause. The majority are thought to result from an interplay between environment and heredity, depending on inherited susceptibility, stage of pregnancy, and degree of environmental hazard." Newborns with birth defects may need to remain in the neonatal unit for an extended time for intensive care and treatment. Screening tests are performed prenatally to detect many genetic defects and, in some cases, enable appropriate treatment before or immediately after birth (see Chapter 5).

CHROMOSOMAL DISORDERS

The human body is made up of 23 paired chromosomes, with one pair from the mother and one pair from the father. These chromosomes contain deoxyribonucleic acid (DNA) and other complex proteins. An abnormal chromosome number or arrangement can cause a congenital defect. Sometimes an extra chromosome is present or a chromosome is broken or missing. The resulting imbalance can provide the newborn with too little or too much genetic material. As the embryo grows, the scrambled genetic information may translate into various types of congenital defects.

DOWN SYNDROME

Trisomy 21, also known as **Down syndrome,** is one of the most common chromosomal syndromes, occurring in 1 in 600 to 800 live births (Gabbe, Simpson, & Niebyl, 2007). It affects all races and economic levels equally. Down syndrome often results from having an extra chromosome (usually chromosome 21) and is called trisomy 21 because it was first identified with this chromosome. Occasionally, the extra chromosome 21 is attached to another chromosome in the egg or sperm, which can create a translocation (an alteration in location). The translocation in either parent greatly increases the chances of having another child with Down syndrome. Mothers older than 35 years are at the greatest risk of having a Down syndrome newborn.

The American Academy of Pediatrics recommends screening of all pregnant women for Down syndrome in the first trimester of pregnancy. First trimester screening includes ultrasound assessment of the thickness of the fetal nuchal fold (called *nuchal translucency*). An ultrasound demonstrating absence of the nasal bone (Cicero, Avgidou, Rembouskos, et al., 2006) and a second trimester "quad test" involving blood testing of alpha-fetoprotein (AFP), human chorionic gonadotropin (hCG), unconjugated estriol (UE), and inhibin A (a placental hormone) are additional screening tests for Down syndrome. A low AFP or a high hCG and inhibin A, with a low UE, may indicate a high risk for Down syndrome for the developing fetus. A test of pregnancy-associated plasma protein A (PAPP-A) may also indicate a risk for Down syndrome. Positive tests in the first or second trimester may indicate a need for amniocentesis to confirm the diagnosis.

Characteristics of the Down syndrome newborn are most noted in the craniofacial features (Figure 16-1). The eyes have an upward slant because of an epicanthal fold, speckles known as Brushfield's spots are seen in the iris, the nose is small with a wide nasal bridge, and the ears are low set. The tongue appears large and protrudes from the newborn's mouth, fingers are short and broad, often there is an unusually wide space between the first two toes, and a single

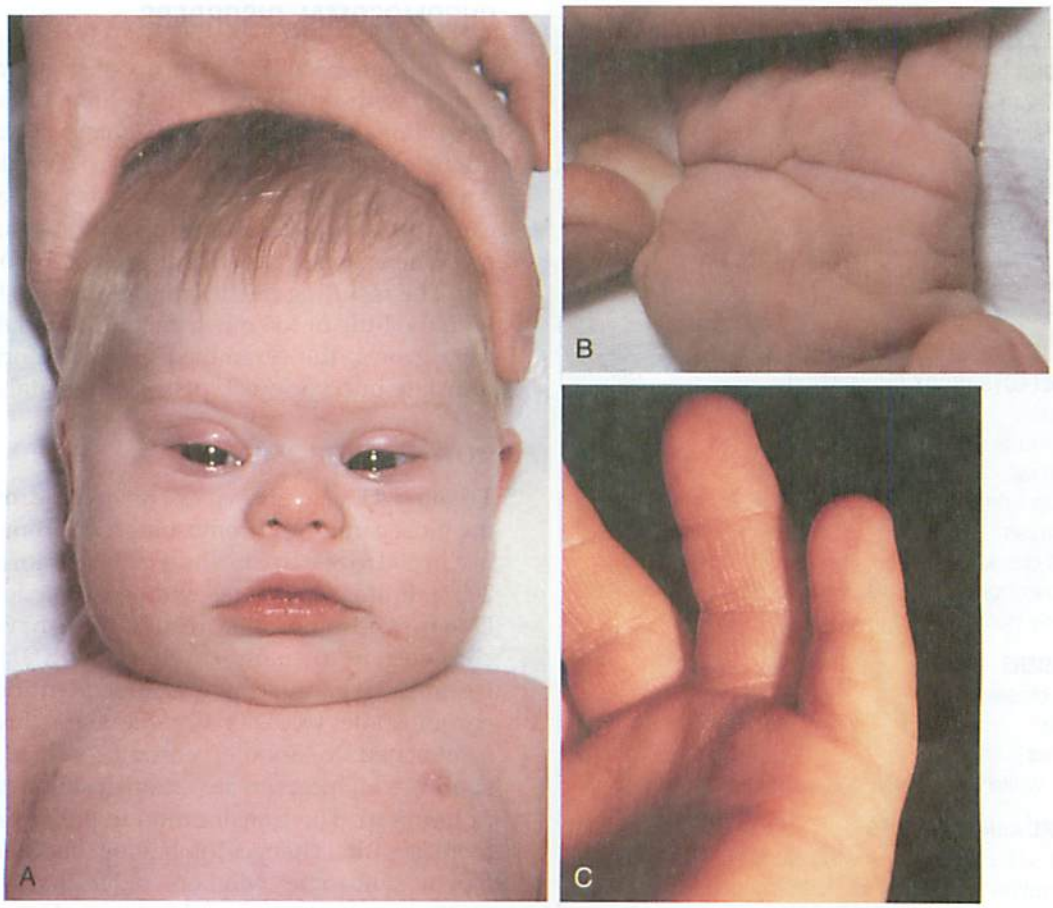


FIGURE 16-1 **Down syndrome. A,** The typical facial appearance of a newborn with Down syndrome shows the upward slant of the canthal folds of the eyes, protruding tongue, and short, thick neck. **B,** The straight simian crease in the palm of the hand is a typical finding in children with Down syndrome. **C,** The short fifth finger is a typical finding in children with Down syndrome. The tip of the fifth finger does not extend to the distal joint of the adjoining finger.

palmar crease (simian crease) may be present. Relatively common internal anomalies in trisomy 21 newborns include heart defects and duodenal atresia. Mental retardation is exhibited, with the mean IQ of approximately 50 (range: 25 to 70). Females are fertile; however, it is rare for males to be fertile.

At birth, these newborns are usually hypotonic (limp and flaccid) and may have feeding difficulties. They also have increased susceptibility to respiratory tract infections. Parents need guidance in feeding and preventing infections and encouragement to stimulate their newborns developmentally. Children with Down syndrome are usually very affectionate. Parents should be encouraged to join a support group for parents of children with Down syndrome. Developmental disabilities, including mental retardation, are common, with hearing and speech difficulties that complicate efforts at education. Cardiac, orthopedic, and thyroid dysfunction are also common. Alzheimer's disease often develops early in the third decade of life, and an altered immune response increases susceptibility to respiratory and dental infection.

INBORN ERRORS OF METABOLISM

Inborn errors of metabolism do not always manifest symptoms at birth. Screening tests for specific errors of metabolism are required by law in most states. It is important for early identification so that treatment can be started as soon as possible to minimize effects on the newborn.

PHENYLKETONURIA

Phenylketonuria (PKU) is an autosomal recessive inherited inborn error of phenylalanine metabolism that occurs in 1 in 15,000 live births (Trahms, 2008). It is caused by the faulty metabolism of phenylalanine, an amino acid that is essential to life and found in all protein foods. The hepatic enzyme phenylalanine hydrolase, which is required to convert phenylalanine into tyrosine, is missing. As a result, when the newborn ingests protein (found in milk and all protein foods), phenylketones accumulate in the blood and can rise as high as 20 times the normal level. Its byproduct, phenylpyruvic acid, appears in the urine within the first weeks of life. These phenylketones accumulate

in the brain and cause irreversible brain damage, resulting in severe mental retardation. Early detection and treatment are essential because by the time the urine test is positive, brain damage has already occurred. The newborn appears normal at birth but begins to show delayed development at approximately 4 to 6 months of age. The newborn may show evidence of failure to thrive or have eczema or other skin conditions. Characteristically, these newborns have an unusual musty odor to their body and in their urine.

A diagnosis is made by the **Guthrie test.** Blood is obtained from a simple heel prick, and a few drops of capillary blood are placed on a filter paper and mailed to the laboratory for screening. It is recommended that the blood be obtained after 48 to 72 hours of life, preferably after ingestion of proteins, to reduce the possibility of false results. All states in the United States require that the test be performed in all newborns before they leave the nursery, but, because of early discharge, the test is often repeated after discharge. The newborn can be tested at home by a public health nurse or at the physician's office or clinic. Confirmation of the diagnosis requires quantitative elevations of phenylalanine compound in both blood and urine. The nurse must stress to the mother the importance of the return visit of the newborn to the physician or clinic for the repeat testing.

Since the development of newborn screening for PKU, several women who had been diagnosed with PKU in the newborn period and then were treated with a phenylalanine-restricted diet have grown up not suffering the damages of untreated PKU. However, newborns born to women with PKU who do not follow the restricted diet during pregnancy have teratogenic effects if high concentrations of phenylalanine are circulating in the mother's blood. Congenital heart defects, microcephaly, and mental retardation are the most commonly seen effects. Therefore, for these women, following dietary restrictions from conception (especially during embryonic development) is critical.

The newborn diagnosed with PKU is fed a special formula (Lofenalac) that has had the phenylalanine reduced or removed. Phenyl-Free is given to children, and Phenex-2 is given to adolescents. The goals of the diet are to provide enough essential proteins to support growth and development while maintaining phenylalanine blood levels between 2 and 10 mg/dL. A phenylalanine level less than 2 mg/dL may result in growth retardation, and a level greater than 10 mg/dL can result in significant brain damage. Levels are monitored throughout childhood.

A dietitian should be consulted for parental guidance and support in maintaining the dietary regimen. This attention is especially needed for the school-age child and the adolescent. The intake of most meat, dairy products, and diet drinks needs to be restricted, and protein intake is restricted to that required for basic growth. The milk substitute can be flavored with a fruit powder or a chocolate substitute, which can increase the child's compliance. Aspartame, a sugar substitute, must be avoided. Genetic counseling is important for future family planning.

GALACTOSEMIA

Galactosemia is an inborn error of metabolism that occurs in 1 in 53,000 live births (Tarini & Freed, 2007). The newborn has a deficiency of the enzyme necessary to convert galactose to glucose, resulting in an increased amount of galactose in the blood (galactosemia), liver, brain, kidney, and urine (galactosuria). An early diagnosis is important so that a milk substitute can be prescribed because galactose is present in milk. Galactosemia can be detected by measuring blood levels of galactose, and screening of all newborns is performed in most states across the United States. Failure to thrive, cataracts, jaundice, cirrhosis of the liver, sepsis, and mental retardation are manifestations of untreated cases. Therapy consists of eliminating galactose from the diet and providing lactose-free formula, such as Nutramigen. Because breast milk contains lactose, breastfeeding must be discontinued. Medications that contain lactose fillers as inactive ingredients also must be avoided. Parents and children often experience frustration and anxiety and must be educated and supported in following this dietary program.

HYPOTHYROIDISM

Congenital hypothyroidism is the result of an inborn error of metabolism caused by a maternal iodine deficiency or the use of antithyroid drugs by the mother. It occurs 1 in 4000 live births (Palma-Sisto, 2004). Thyroxine (T_4) is measured from a drop of blood obtained from heel stick at 2 to 5 days of age. If not treated with thyroid replacement, the infant may develop hypothermia, poor feeding, lethargy, jaundice, and cretinism. The infant has a large protruding tongue, thick lips, and a generally dull appearance.

An important note is that one blood sample can be used to test all three metabolic disorders: PKU, galactosemia, and hypothyroidism. The screening test for hypothyroidism is mandated in all states across the United States and is often performed before discharge from the nursery. Early diagnosis and treatment are essential to maintain normal physical and mental growth and development.

MAPLE SYRUP URINE DISEASE

Maple syrup urine disease is a disorder of amino acid metabolism in which the amino acids leucine, isoleucine, and valine cannot be metabolized because of missing enzymes. Elevated levels of leucine can cause cerebral edema and central nervous system (CNS) symptoms such as seizures. Body fluids have a sweet odor, similar to maple syrup. Some states require

routine screening of all newborns for this condition (see Chapter 9). Nursing responsibilities include educating the family concerning strict dietary and exercise limitations throughout the infant's lifetime.

COMMON CONGENITAL ANOMALIES

Although congenital anomalies are generally treated in the pediatric setting, they are usually identified soon after birth. The family of the newborn with the disorder may have been aware of the condition through prenatal testing, or they may be surprised by the birth of a newborn who is not completely normal. The family will need a great deal of support and sensitivity from the health care providers. Some congenital anomalies are noted in Table 16-1. The nursing student is urged to read a pediatric textbook for more detailed information concerning the management of congenital anomalies in the newborn.

COMMON ACQUIRED DISORDERS

HYPERBILIRUBINEMIA (PHYSIOLOGIC JAUNDICE)

Hyperbilirubinemia is defined as an abnormally high level of bilirubin in the blood. This condition occurs when the normal pathways of bilirubin metabolism and excretion in the newborn are altered because of excess products (from normal hemolysis), liver immaturity, delayed feeding (which prevents development of intestinal flora), trauma, or cold stress. An increase in bilirubin also can occur as a result of cephalhematoma, extensive bruising, infections, and acidosis that causes a decrease in liver function. Maternal use of sulfa or salicylates can interfere with conjugation of bilirubin. The result is clinical jaundice (icterus neonatorum), which is seen in approximately 60% of term newborns; it is even more common in preterm newborns, typically occurring after the third day of life. Total serum bilirubin levels greater than 12 mg/dL in the term newborn usually indicate hyperbilirubinemia.

The newborn is born with an excessive amount of red blood cells (RBCs) and, at birth, begins to destroy the RBCs he or she no longer needs. The infant does not need the excess RBCs because he or she is now in an atmosphere of higher oxygen concentration than was available in utero. Bilirubin inside the RBC is released into the bloodstream when the RBC is destroyed. The bilirubin combines with albumin in the blood and is transported to the liver. Under the influence of the enzyme glucuronyl transferase, the bilirubin is conjugated into a water-soluble form and excreted subsequently into the small intestine; most of it is excreted from the body in the feces. Some bilirubin is converted to unconjugated bilirubin, is reabsorbed, and is recirculated back into the blood. The amount of bilirubin in the blood is described in milligrams of bilirubin per deciliter (mg/dL). When the bilirubin accumulates in the blood, it contributes to a condition called *icterus neonatorum*, or physiologic jaundice. The skin and whites of the eyes assume a yellow-orange cast. The higher the bilirubin level the deeper the jaundice. An increase of more than 5 mg/dL in 24 hours or a bilirubin blood level of 12.9 mg/dL or more requires investigation and intervention.

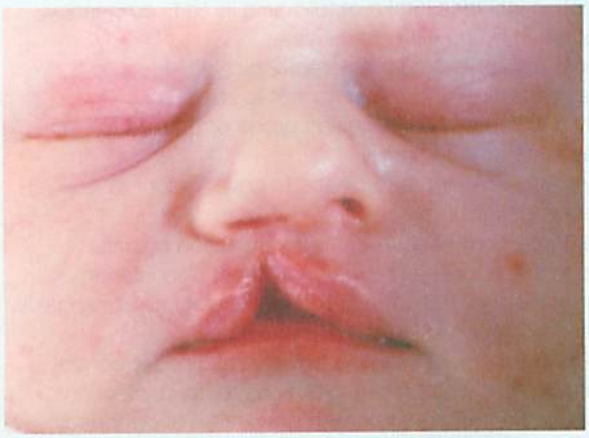
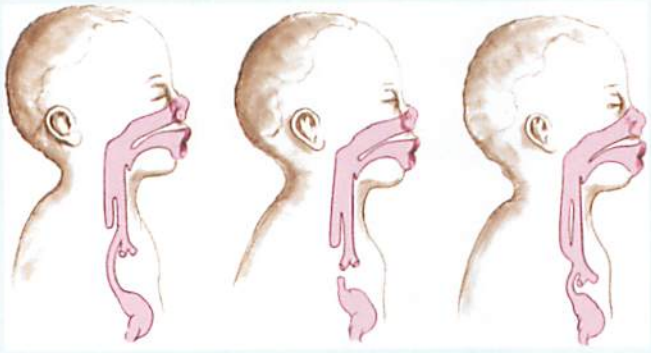
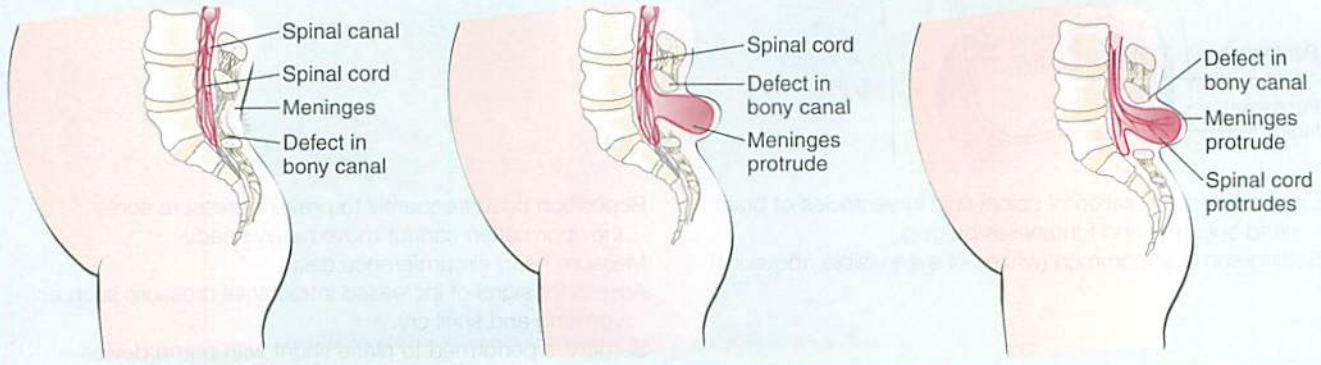
The bilirubin level in newborns typically peaks between 3 and 5 days of age. Therefore, the early discharge of newborns requires follow-up observation within a few days. Conjugation of the bilirubin can be inhibited by a lack of bacteria in the intestines or a low level of glucuronyl transferase enzyme. The immature liver may also be slow to take up the bilirubin that flows to it. For these reasons, the liver may not be able to "clear" the bilirubin from the blood and excrete it from the body at a rapid rate. High levels of bilirubin in the blood can stain the basal nuclei of the brain, causing long-term neurologic problems. This condition is called **kernicterus.**

Because bilirubin conjugated by the liver is excreted by the body via the intestinal tract, the stimulation of meconium stool passage is an important part of the care plan. The initiation of early feedings enhances the passage of meconium and therefore plays an essential role in the management of hyperbilirubinemia. Colostrum, in breast milk, has a natural laxative effect, and therefore breastfeeding at least 10 times a day is recommended for the neonate. Glucose water supplements should be avoided because little bilirubin is excreted by the kidneys and decreased caloric intake is associated with decreased passage of stool, allowing for the reabsorption of bilirubin before it can be excreted. Although there is a factor in human milk that may increase reabsorption of bilirubin from the intestines, this rarely causes a significant rise in serum bilirubin. Some physicians may prefer to feed the infant bottled formula for a 12- to 24-hour period to avoid this small increase. The mother should be encouraged to pump milk from the breast during this short period to enable easy return to breastfeeding.

Assessment and Management of Physiologic Jaundice

All newborns with visible jaundice and all infants under 35 weeks' gestation should have serum bilirubin levels drawn (Rennie, 2010). The visual blanch test is used to help distinguish jaundice from normal skin color. Pressure is applied with a finger over a bony area on the newborn, such as the nose, forehead, or sternum, for several seconds to empty the capillaries in that area. A yellow tinge in the blanched area indicates jaundice. When pressure is released, the capillaries refill. The conjunctivae of the eyes and the buccal mucosa can also be visually assessed to detect jaundice. Assessing for jaundice should be done under natural light

Table 16-1 Common Congenital Anomalies

ANOMALY	TREATMENT AND NURSING CARE
Gastrointestinal	
Cleft Lip and Palate  Failure of fusion of upper lip (may be unilateral or bilateral) Failure of fusion of hard and soft palate Defect caused by both genetic and environmental factors	Degree of cleft determines care. Encourage parents to verbalize concerns. Determine most effective nipple (soft preemie, lamb's, cleft nipple, rubber-tipped Asepto). Feed in upright position to decrease risk of aspiration. Feed slowly, burp frequently (tendency to swallow air). Cleanse mouth with water after feedings. Support parents. Refer parents to support group. Management is through lip surgery (approximately 10 weeks) and palate repair in stages (approximately 1 year). Long-term follow-up, including speech therapy, is necessary.
Esophageal Atresia (Failure of Esophagus to Connect with Stomach)  Associated with maternal hydramnios Excessive mucous secretions (drooling) Periodic cyanotic episodes and choking Abdominal distention after birth Immediate regurgitation of feeding with risk of aspiration	Withhold feeding until esophageal patency is determined. Elevate head of crib to prevent reflux of gastric juices. Explain surgical repair to parents. First feeding of all newborns should be supervised by a nurse to observe for this anomaly.
Neurologic	
Spina Bifida **Occulta** **Meningocele** **Meningomyelocele**  	

Continued

Table 16-1 Common Congenital Anomalies—cont'd

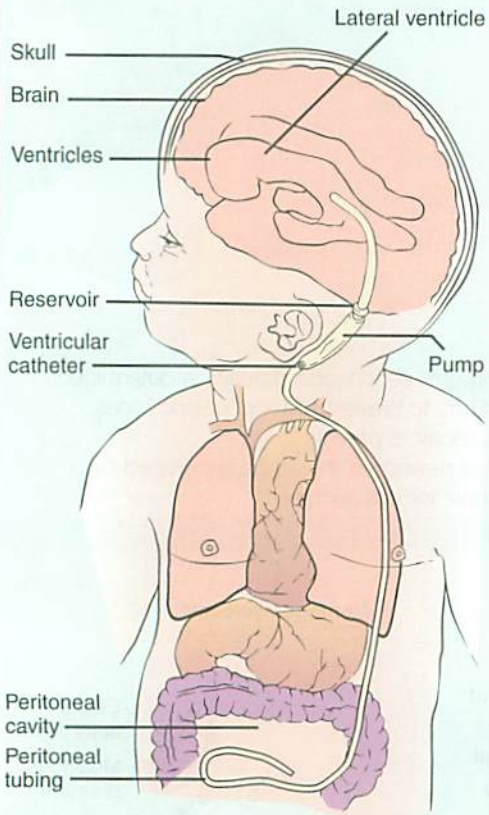
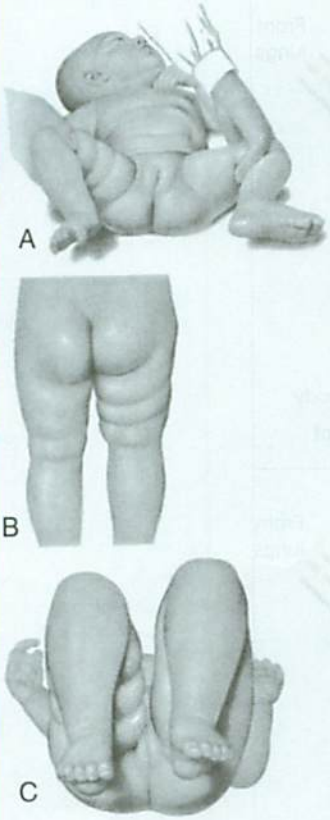
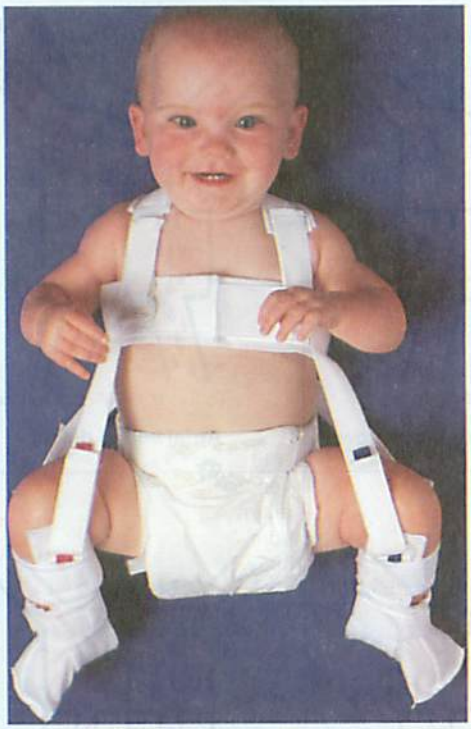
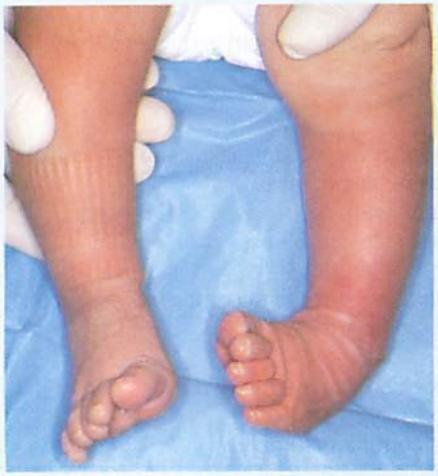
ANOMALY	TREATMENT AND NURSING CARE
Neurologic	
Spina Bifida—cont'd	
Occulta: Failure of vertebral arch to close (may have a dimple or tuft of hair over lumbosacral region) *Meningocele:* Protrusion of meninges, covered by skin or thin membrane *Meningomyelocele:* Protrusion of both meninges and spinal cord; degree of paralysis depends on location of defect	Prevention includes intake of folic acid in early pregnancy. Protect membrane with sterile cover. Observe sac for leakage of cerebrospinal fluid. Assess sensation and movement of legs. Gently handle newborn; position prone or on side to prevent trauma to sac. Apply sterile dressing and plastic to cover defect to prevent drying. Prevent infection; keep free of contamination by urine and feces; dribbling of urine may affect skin integrity. Measure head circumference to identify early hydrocephalus. Assess for increased intracranial pressure. Long-term treatment includes bowel and bladder control management with prevention of urinary tract infections, management of paralysis with prevention of orthopedic and skin complications, and prevention of obesity. Watch for latex allergy; 73% of children with spina bifida are sensitive to latex and must be cared for in a latex-free environment.
Hydrocephalus  	
Excessive accumulation of spinal fluid in ventricles of brain; head enlarged and fontanelles bulging Setting-sun sign common (whites of eyes visible above iris)	Reposition head frequently to prevent pressure sores (newborn often cannot move heavy head). Measure head circumference daily. Assess for signs of increased intracranial pressure such as vomiting and shrill cry. Surgery is performed to place shunt with pump device (directed from ventricle to peritoneal cavity [ventriculoperitoneal shunt]).

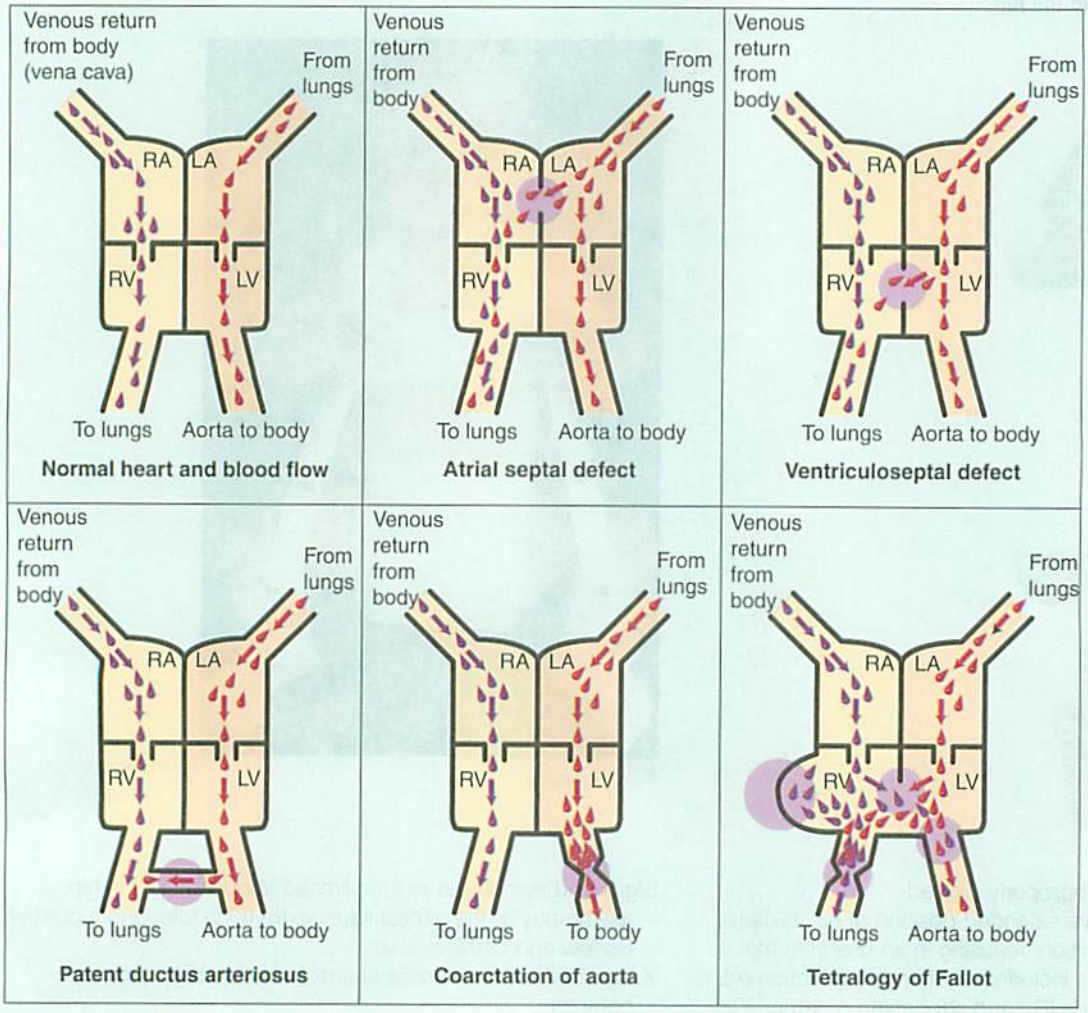
Table 16-1 Common Congenital Anomalies—cont'd

ANOMALY	TREATMENT AND NURSING CARE
Musculoskeletal	
Developmental Dysplasia of the Hip	
 A, B, C	 Pavlik harness
Femoral head and hip improperly aligned May be genetic or involve extended position of hip in utero, such as a breech position, resulting in an unstable hip Early signs of dislocation include limitation of abduction *(A)*, asymmetry of skin folds *(B)*, and shortening of femur *(C)*	Signs and symptoms include limited abduction of the hip, asymmetry of the gluteal folds, extra thigh fold, and a positive Barlow and Ortolani test. X-ray studies are not reliable until bone formation is more complete. Treatment may be use of Pavlik harness to maintain hip flexion. Traction and spica cast may be required.
Clubfoot	
	
Talipes equinovarus: an abnormal twisting of the foot out of normal alignment	Early treatment is essential before ossification of bone is complete. Exercise and casting are the treatments of choice soon after birth.

Continued

Table 16-1 Common Congenital Anomalies—cont'd

ANOMALY	TREATMENT AND NURSING CARE
Cardiac (Heart Defects)*	



Patent Ductus Arteriosus

Noncyanotic heart defect

Failure of the ductus arteriosus, connecting the pulmonary artery and the aorta, to close after birth

Cyanosis does not occur because blood recirculates to the lung and is fully oxygenated when it flows to general circulation

Heart defects may or may not be identified immediately.

Newborn with heart defect may exhibit murmurs, abnormal heart rate or rhythm, breathlessness, and fatigue while feeding.

Surgery may be postponed until newborn is physiologically stable.

Tetralogy of Fallot

Cyanotic heart defect

Involves four characteristic defects: a ventricular septal defect, aorta positioned over the ventricular septal defect, stenosis of the pulmonary valve, and hypertrophy of the left ventricle

Cyanosis results from venous blood from the right ventricle flowing through the septal defect and directly into the overriding aorta; blood flow to the lungs is decreased because of the narrowed pulmonary valve; cyanosis occurs because unoxygenated blood reaches the general circulation

Monitor closely; observe for respiratory difficulties, cyanosis, tachycardia, tachypnea, diaphoresis.

Conserve newborn's energy to reduce workload on heart.

Gavage feedings or oral feedings with special nipple may be given.

Elevate newborn's head and shoulders to improve respirations and reduce cardiac workload.

Prevent infection.

Place in knee-chest position for respiratory distress during "tet" attack.

Management includes corrective surgery.

LA, left aorta; *LV*, left ventricle; *RA*, right aorta; *RV*, right ventricle.

*For other common congenital heart defects, students should consult their pediatric textbooks.

because reflections of wall color can influence the appearance of skin color (Skill 16-1). The cephalocaudal (head-to-toe) pattern of circulation results in a head-to-toe progression of jaundice in the newborn so that jaundice affecting the head and upper body may be a reflection of a lower serum bilirubin level than in infants who evidence jaundice of the chest and lower body. Serum bilirubin levels are taken whenever the jaundice levels appear before 24 hours of age. A bilirubin threshold table is available as a guideline in the management of infants with hyperbilirubinemia (Rennie, 2010).

Noninvasive Methods of Bilirubin Measurement

The trauma, cost, and inconvenience of obtaining a blood sample for serum bilirubin levels can be saved by measuring the transcutaneous bilirubin (TcB). Hand-held electronic devices such as the BiliCheck (Respironics) measure TcB levels. TcB monitoring is a screening tool. Those infants with TcB bilirubin levels above 14.6 mg/dL should have serum bilirubin levels drawn and referred for further follow-up.

HYPERBILIRUBINEMIA (PATHOLOGIC JAUNDICE)

A major cause of hyperbilirubinemia is hemolytic disease, in which there is an excessive breakdown of RBCs of the newborn as a result of maternal antibodies passing through the placenta to the fetus in the uterus. Isoimmune hemolytic disease, also known as **erythroblastosis fetalis,** occurs when an Rh-negative mother is pregnant with an Rh-positive fetus and transplacental passage of maternal antibodies occurs. When maternal antibodies enter the fetal circulation, they destroy the fetal RBCs. The fetal system responds by increasing the RBC production with a marked increase in immature RBCs (erythroblasts). A high level of maternal antibodies may have developed during a previous pregnancy, abortion, amniocentesis, or abruptio placentae. A previous blood transfusion with Rh-positive blood would also cause the development of maternal antibodies. At birth, the newborn with hemolytic disease caused by Rh incompatibility has a positive direct Coombs' test, which reveals the presence of antibody-coated (sensitized) Rh-positive RBCs in the newborn. The indirect Coombs' test measures the amount of Rh-positive antibodies in the mother's blood. If the mother prophylactically receives $Rh_o(D)$ immune globulin (RhoGAM), maternal development of antibodies to Rh-positive blood greatly decreases (Figure 16-2). Pathologic jaundice, such as that caused by Rh incompatibility, is evident *before* the third day of life because the hemolysis begins before birth.

? Did You Know?

Pathologic Jaundice

In pathologic jaundice of erythroblastosis (Rh hemolytic disease), jaundice is evident ***before*** the third day of life. Jaundice that is first evident ***after*** the third day of life is usually caused by physiologic icterus neonatorum.

ABO incompatibility may also cause pathologic jaundice. Mothers with type O blood are most likely to be involved; however, the **hemolysis** (destruction of RBCs) is much less than with Rh incompatibility. Pathologic jaundice also can result from infection, hypothyroidism, and biliary atresia.

Management of the Jaundiced Newborn

The goals of management are prompt identification of newborns who are at risk for jaundice (based on the woman's Rh history and antibody levels) and prompt treatment to prevent the development of **kernicterus**, a staining of the basal nuclei of the brain that results in toxicity to the CNS. Newborn hyperbilirubinemia is considered pathologic if clinical jaundice is evident within the first 24 hours of life, the clinical jaundice persists for more than 14 days, serum bilirubin level

Skill 16-1 Detecting Jaundice in the Newborn

PURPOSE

To determine whether a blood test for bilirubin level is indicated.

Steps

1. Place the newborn in a well-lighted area.
2. With one finger, press the area of the skin at the bridge of the nose near the forehead to cause blanching of the skin.
3. Observe the skin and subcutaneous tissue for yellowish color as the skin is blanched.
4. Blanch areas of the body at the sternum and below. As the degree of jaundice increases, the yellow-orange color will be observable in the torso and lower extremities as well as the face.
5. Document findings.
6. Report abnormal findings.

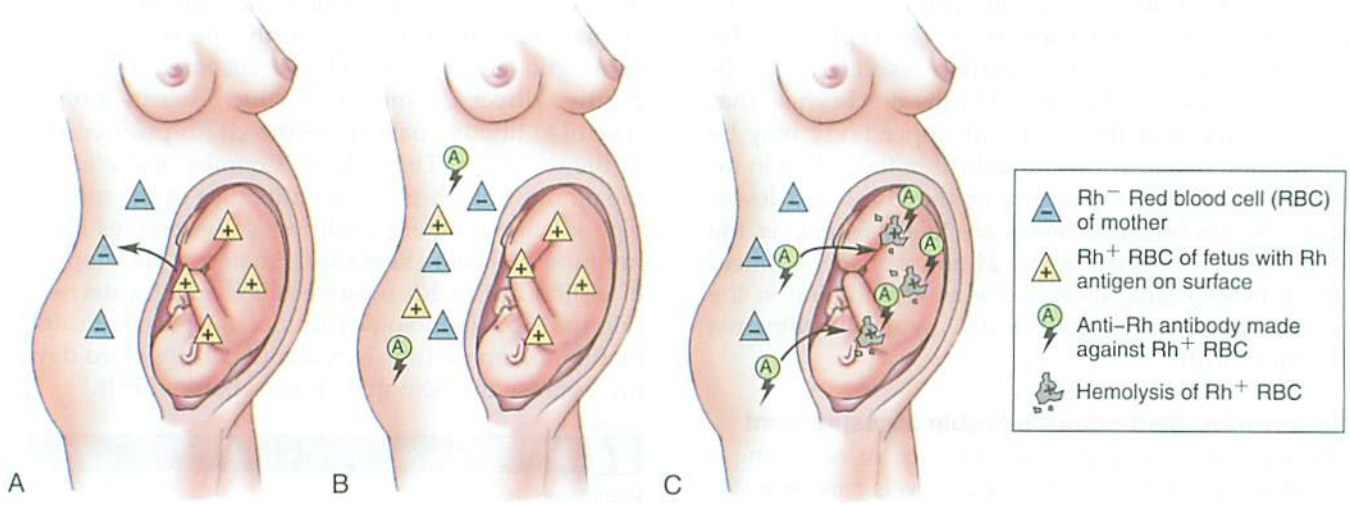


FIGURE 16-2 Maternal sensitization producing erythroblastosis in newborn. A, During the first pregnancy the mother is sensitized to the Rh-positive (*Rh*⁺) antigen from the fetus. **B,** The mother produces Rh antibodies to the Rh antigen to which she was exposed. **C,** During a second pregnancy, these Rh-positive antibodies cross the placenta to the fetus and destroy the fetal Rh-positive blood cells. *Rh*⁻, Rh negative.

rises more than 5 mg/dL/day, or the total bilirubin level is greater than 12 mg/dL.

Clinical signs associated with the development of kernicterus include temperature instability, poor feeding, decreased muscle tone, poor Moro's reflex, lethargy, high-pitched cry, rigidity, irritability, opisthotonos position (arched back), seizures, upward gaze, dark urine, and light stools. In preterm newborns, apnea and seizures may also occur.

Phototherapy. Management includes **phototherapy** as the primary treatment of choice for an infant with serum bilirubin levels above 12 mg/dL (Figure 16-3). Exposing the newborn to high-intensity light in the blue light spectrum decreases bilirubin levels by converting unconjugated bilirubin into isomers, called *photobilirubin*, which is transported to the liver, where it combines with bile and is excreted in the feces (without conjugation by the liver). Some light-oxidized bilirubin is also excreted in the urine. It is important for frequent feedings to continue to facilitate the excretion process. Phototherapy can be provided by a bank of green or fluorescent blue lights placed above an incubator, a fiberoptic blanket attached to a halogen light source wrapped around the body of the infant, or a fiberoptic mattress attached to a halogen light source placed under the infant (Figure 16-4). The newborn receiving standard phototherapy will have his or her eyes covered by a shield or mask to prevent potential retinal damage caused by the lights (Figure 16-5). The eye patches are removed, and the newborn is removed from the incubator during feeding and short parental visits. It is also recommended that the newborn's genital area be shielded to protect the testicles and ovaries. Surgical masks can be used like a "bikini" diaper to

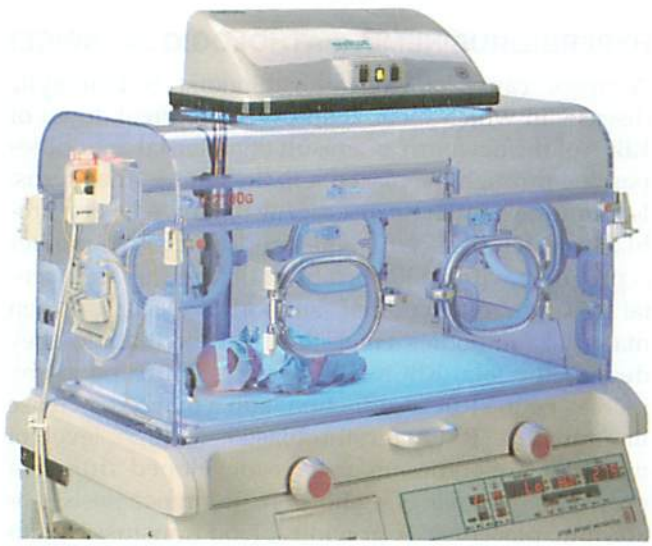


FIGURE 16-3 Phototherapy. The neoBLUE provides a high-intensity, narrow band of blue light that helps break down excess bilirubin. A flip of a switch allows change from conventional to intense phototherapy treatment. Note: In the clinical setting, the infant would be diapered only, not fully clothed.

provide protection. Repeat bilirubin measurements should be taken q6-12 hours and then again 12 hours after phototherapy is stopped (Rennie, 2010).

Frequent stools occur with rapid decrease in bilirubin and can lead to perineal excoriation; thus, careful and frequent cleansing of the newborn's skin is essential. Ointment should not be used because it could cause a burn to the skin during the phototherapy. The newborn should be assessed for dehydration and the intake and output accurately monitored. The newborn's temperature is monitored frequently, and the newborn's position is changed a minimum of every 2 hours.

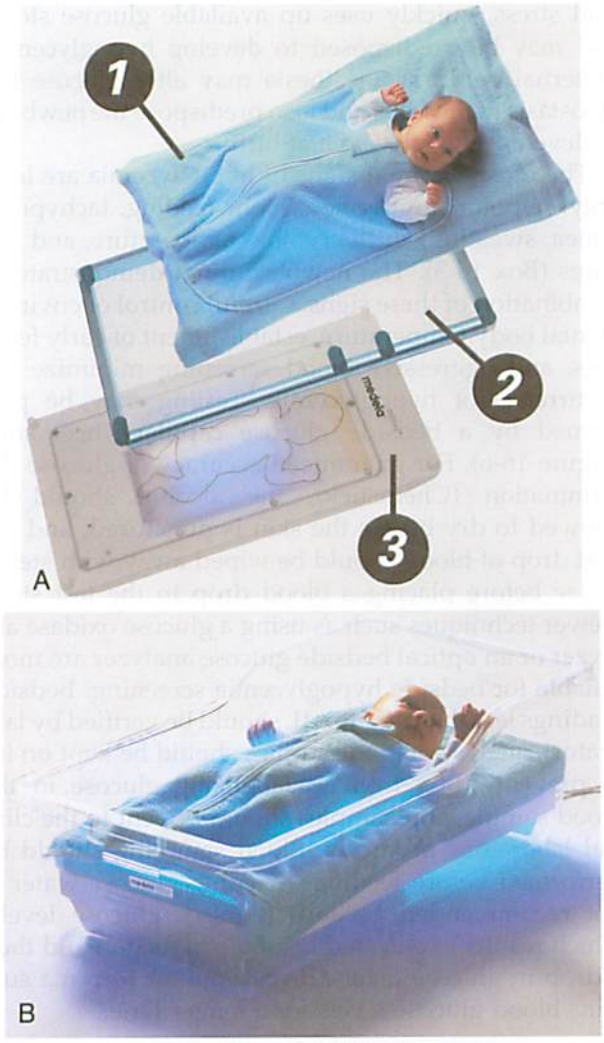


FIGURE 16-4 **Phototherapy BiliBed. A,** Newborn lies on a transparent film over a blue light source. **B,** Romper blanket in which the newborn is wrapped has a light, porous fabric on the anterior and posterior surfaces. Newborn's eyes do not need covering, and newborn can room with the mother during therapy.

Advantages of using a fiberoptic blanket or mattress method of phototherapy are that the eyes do not have to be covered, the newborn is accessible for care and interaction, and fluid and weight loss are usually not problems.

If the newborn is to receive phototherapy using home equipment, parents are taught how to assess the baby and use the equipment (see Chapter 18). Breastfeeding may continue, and the infant should not be given additional fluids routinely (Rennie, 2010).

Exchange Transfusion. Treatment by blood exchange transfusions may be indicated if the bilirubin level is greater than 25 mg/dL within 6 hours of phototherapy (Rennie, 2010).

An exchange transfusion is accomplished by alternately removing a small amount (5 to 10 mL) of the newborn's blood from the umbilical vessels and replacing it with 5 to 10 mL of donor blood. A maximum of 500 mL of donor blood is transfused, and the technique results in an approximately 75% exchange of the newborn's total blood volume.

The newborn's blood is Rh positive. Because antibodies are present in the newborn's blood against the Rh-positive factor, transfusing Rh-positive donor blood to the newborn will cause the antibodies to destroy the new blood cells and result in an increased bilirubin level. For this reason, Rh-negative blood is used for the exchange transfusion. The Rh-positive antibodies will not destroy Rh-negative blood cells, and the bilirubin levels will not increase. The life cycle of all RBCs is approximately 180 days. Therefore, after 180 days, the Rh-positive antibodies are no longer active in the newborn's blood and the Rh-negative blood cells are no longer present. The newborn will produce his or her own RBCs, reverting to the Rh-positive blood type the newborn was born with.

During the procedure, the newborn's vital signs are monitored, infection control measures are maintained, and the newborn is closely observed for transfusion reactions (hypocalcemia), such as jitteriness, convulsions, and edema or signs of fluid volume overload. Research is ongoing to develop a safe drug that will prevent the development of elevated blood bilirubin levels.

Administration of RhoGAM. Prevention of erythroblastosis by administering RhoGAM to the mother is routine. An intramuscular injection is given to the Rh-negative mother within 72 hours of delivery of an Rh-positive newborn, provided she has not been previously sensitized. Also, RhoGAM is usually given to the Rh-negative pregnant woman at 28 weeks' gestation. Rh-negative women need to have RhoGAM when they have had an abortion, after an amniocentesis, or when they have bleeding during pregnancy. Fetal blood may leak into the mother's circulation at these times and

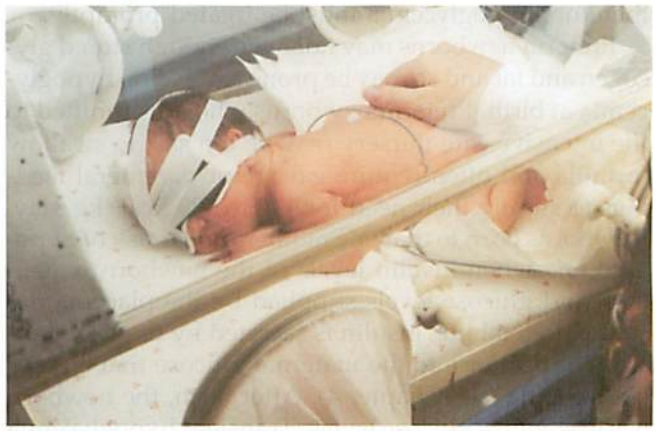


FIGURE 16-5 This newborn is in an incubator receiving phototherapy; note the eyes covered for protection. The mother provides gentle touch and massage. The newborn's position is changed frequently to expose all areas to the light.

Box 16-2 Rh Sensitization

- The potential for sensitization occurs when an Rh-negative woman and an Rh-positive man conceive a fetus that is Rh positive.
- If the woman becomes sensitized, her body will produce antibodies to her fetus's Rh-positive blood.
- Tests used to detect antibody formation or sensitization are (1) an indirect Coombs' test on the mother's blood to measure the amount of Rh-positive antibodies and (2) a direct Coombs' test on the baby's blood to detect antibody-coated Rh-positive red blood cells.
- Rh_o(D) immune globulin (RhoGAM) is given at 28 weeks' gestation (prenatally) to reduce the potential fetal Rh-positive cell antibody development in the Rh-negative mother's bloodstream.
- RhoGAM should be administered after each amniocentesis, abortion, or ectopic pregnancy and within 72 hours or less after birth of an Rh-positive newborn.
- Phototherapy is the treatment of choice for newborns who develop hyperbilirubinemia.

stimulate antibody production (Box 16-2). Rophylac, approved by the FDA in 2004, is used in many facilities in place of RhoGAM. Rophylac is derived from human plasma and has never contained thimerosal (mercury). It can be administered IM or IV but cannot be administered at the same time as administration of other vaccines.

HYPOGLYCEMIA

Blood glucose levels decrease during the first 2 hours of life to approximately 50 mg/dL and then start to rise and stabilize. **Hypoglycemia** (*hypo* refers to below, and *glycemia* refers to sugar in the blood) is usually based on two consecutively low values on blood samples taken 30 minutes apart. Plasma glucose levels of less than 40 mg/dL indicate hypoglycemia and are considered abnormal. Blood glucose less than 25 mg/dL is usually treated with intravenous glucose solutions. The brain requires a constant supply of glucose, and therefore hypoglycemia must be treated promptly.

Preterm newborns may not have enough stored glycogen and fat and so may be prone to develop hypoglycemia at birth. Preterm newborns who are admitted to the intensive care nursery may be too sick to swallow formula and often require gavage or parenteral feedings to supply their need for 110 to 130 kcal/kg/day. Newborns born to diabetic mothers are at risk of hypoglycemia when insulin levels of the newborn remain high and glucose levels supplied by the placenta decrease. In fetal life, insulin is secreted by the fetal pancreas in response to the maternal glucose that crosses the placenta (see Chapter 4). After birth, the newborn has to stabilize glucose levels. Transient hypoglycemia most often occurs during the first 24 hours after birth, but it may be delayed for up to 72 hours. Any newborn who experiences stress at birth, such as asphyxia or cold stress, quickly uses up available glucose stores and may be predisposed to develop hypoglycemia. Maternal epidural anesthesia may alter glucose homeostasis in the fetus and also predispose the newborn to develop hypoglycemia at birth.

The most common signs of hypoglycemia are lethargy, hypotonia, jitteriness, poor feeding, tachypnea, apnea, sweating, shrill cry, low temperature, and seizures (Box 16-3). The newborn often demonstrates a combination of these signs. Careful control of environmental body temperature, establishment of early feedings, and aggressive blood screening minimize the occurrence of hypoglycemia. Testing may be performed by a bedside glucose capillary heel stick (Figure 16-6). For guaranteed accuracy of glucose determination (Chemstick), the alcohol should be allowed to dry before the skin is punctured, and the first drop of blood should be wiped away with sterile gauze before placing a blood drop in the test strip. Newer techniques such as using a glucose oxidase analyzer or an optical bedside glucose analyzer are more reliable for bedside hypoglycemia screening. Bedside readings less than 40 mg/dL should be verified by laboratory analysis. Blood samples should be kept on ice to prevent RBCs from metabolizing glucose in the blood sample, and samples should be sent to the clinical laboratory promptly. Blood sampling should be performed before feedings. Feeding glucose water is not recommended because it raises glucose levels, which results in elevated insulin production and then a drop in glucose levels. Breast milk or formula sustains blood glucose levels for a longer time.

RESPIRATORY DISORDERS

The respiratory system plays a critical role in successful adaptation to extrauterine life. At birth, interruption of the fetoplacental circulation requires the newborn to achieve effective gas exchange immediately. Maturation of the respiratory system in utero is essential for extrauterine life. Pulmonary surfactant is an antiatelectasis factor located in the alveolar lining layer, which provides a low surface tension to the

Box 16-3 Signs of Hypoglycemia

- Poor feeding
- Jitteriness or tremors
- Tachypnea
- Cyanosis
- Lethargy
- Hypotonia (possible weak swallowing reflex)
- Irritability
- Temperature instability
- Apnea
- Seizures and coma

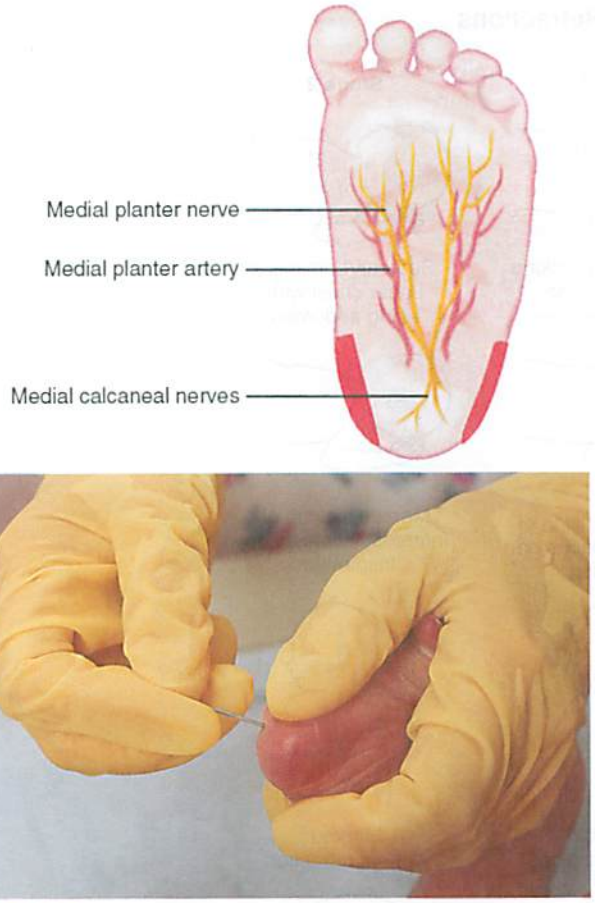


FIGURE 16-6 The shaded areas at the side of the heel are used for heel sticks in newborns to avoid the nerves, blood vessels, and bony areas. Warming the heel before puncture will promote better blood flow.

tissues that prevents collapse of the alveoli during expiration.

Common respiratory disorders that manifest during the beginning of extrauterine life include respiratory distress syndrome, meconium aspiration syndrome (MAS), transient tachypnea of the newborn, persistent pulmonary hypertension of the newborn, and sepsis.

RESPIRATORY DISTRESS SYNDROME

Respiratory distress syndrome (RDS), also known as hyaline membrane disease, is a major cause of newborn morbidity and death. Impaired or delayed surfactant appears to play a key role in this condition, and it often occurs in preterm infants. Hypoxemia occurs and causes metabolic acidosis, and both contribute to pulmonary vasoconstriction. The newborn then has decreased ability to exchange oxygen and carbon dioxide necessary for perfusion of oxygenated blood to vital organs and for removal of metabolic waste products.

Newborns with RDS are typically seen initially with a combination of tachypnea, nasal flaring, subcostal and intercostal retractions, cyanosis, and expiratory grunting. Retractions occur as the result of the soft rib cage being drawn in on inspiration. The expiratory grunt results from partial closure of the glottis during an expiration, which is a means of trapping alveolar air. Clinical signs of the disorder often occur within 1 hour of birth. Signs of respiratory distress in the newborn are illustrated in Figure 16-7. Principles of newborn resuscitation are reviewed in Table 16-2.

Prevention and Treatment

Essential preventive measures for RDS include avoiding preterm birth (from either elective cesarean birth or premature labor). If preterm delivery is necessary, the administration of corticosteroids to the mother before birth stimulates fetal lung production of surfactant. After birth, the severity of RDS may be lessened by administration of surfactants by trachea using strict sterile technique. Nursing responsibilities include:

- Weighing the newborn to ensure the proper dosage is administered.
- Deep suctioning before the procedure to ensure the medication will not be mixed with thick mucus. Suctioning is not performed for 2 hours after the procedure to avoid removing the instilled liquid.
- Administering appropriate sedatives as prescribed.
- Assisting with chest x-ray examinations as needed.
- Positioning the newborn during procedure. Positioning required is often head down, turned in specific directions, and held in place for 30 seconds after each dose is administered. A respiratory therapist should be available.
- Monitoring the newborn's vital signs and status. Bradycardia, decreased oxygen saturation, and poor general color may indicate a pause in the procedure is necessary.
- Monitoring oxygen saturation, vital signs, signs of respiratory distress, and signs of cerebral hemorrhage after the procedure.

Assisted ventilation is given with high-speed jet ventilation or a high-frequency oscillation ventilator (HFOV), which delivers small volumes of gas at high frequencies and limits development of high airway pressure. An intermittent positive pressure ventilation (IPPV), a continuous positive airway pressure (CPAP), high-frequency extracorporeal membrane oxygenation (ECMO), or nitric oxide inhalation is also used. Careful assessment and parenteral nutritional support are essential. Documenting an accurate intake and output is essential.

Safety Alert

Signs of Respiratory Distress in Neonates

- Pale, mottled skin color
- Tachypnea and periods of apnea
- Retractions on inspiration (substernal or intercostal)
- Flared nares on inspiration
- Expiratory grunt
- Decreased response to stimuli

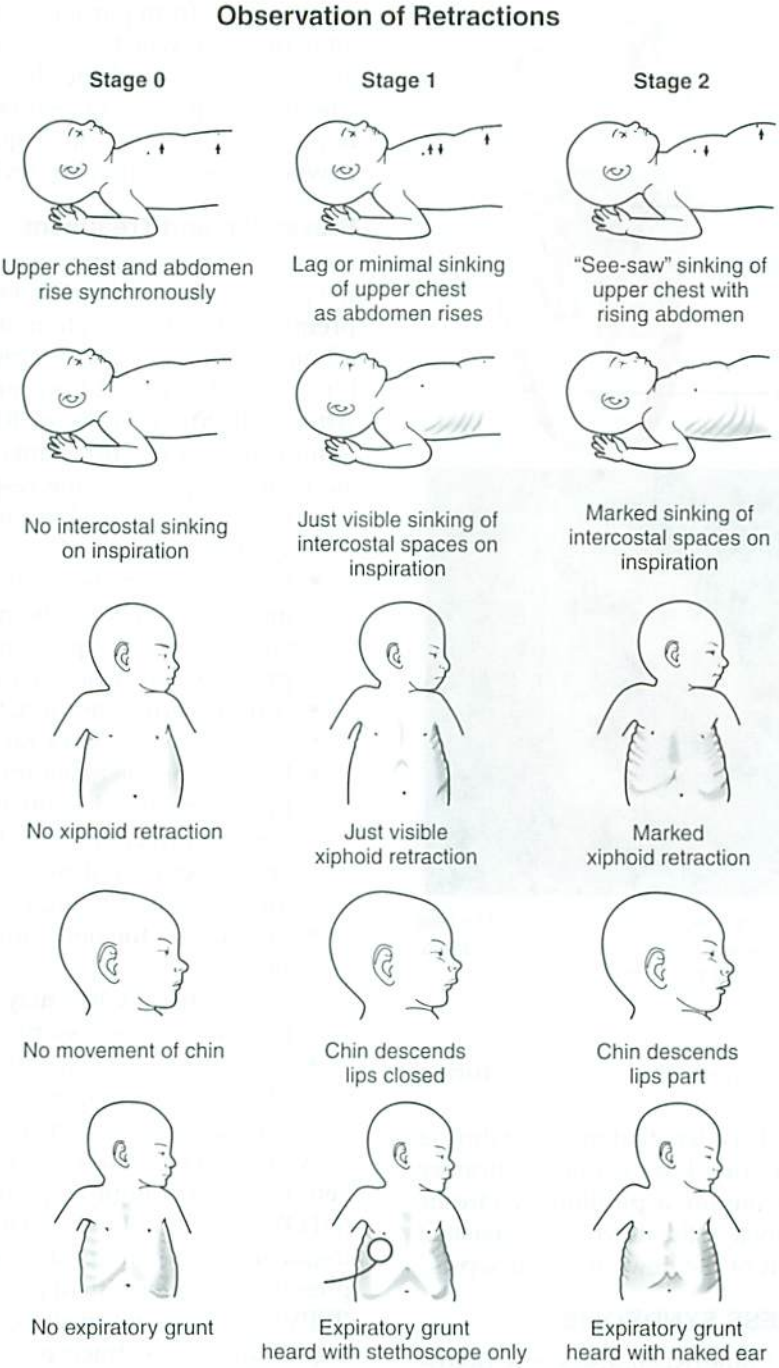


FIGURE 16-7 Assessment of respiratory distress. The Silverman-Andersen index is used to score the newborn's degree of respiratory difficulty. The score for individual criteria matches the grade, with a total maximum score of 10, indicating severe distress.

MECONIUM ASPIRATION SYNDROME

The fetal physical response to asphyxia in utero is increased intestinal peristalsis, relaxation of the anal sphincter, and the passage of meconium into the amniotic fluid. When the fetus experiences hypoxia, gasping movements can draw meconium into the fetal airways. Interestingly, the passage and subsequent aspiration of meconium are rarely seen before 34 weeks' gestation. **Meconium aspiration syndrome (MAS)** primarily affects postterm newborns and those who have had a prolonged labor and intrauterine asphyxia. The presence of thick (particulate or pea soup–like) amniotic fluid increases the risk for MAS. The key to management of MAS is its prevention and, using the skilled team approach, immediate routine upper airway suctioning at birth as soon as the head is delivered to the perineum. After birth, tracheal suctioning should be performed when the Apgar score is low and must be completed before using positive pressure ventilation.

When meconium is aspirated, obstruction of the newborn's airways can occur. Obstruction of the large or upper airways results in an acute hypoxic emergency.

Table 16-2 Steps in Newborn Resuscitation

INTERVENTION	RATIONALE AND NURSING RESPONSIBILITY
Place infant in head-down position.	Avoids aspiration of oropharyngeal secretions.
Suction.	Suctioning nose and mouth establishes a patent airway.
Rub infant's back.	Provides noninvasive respiratory stimulation.
Provide positive pressure puffs of inflation at 40-60 breaths/minute with infant's head in neutral (sniff) position.	Sniff position opens the airway (avoid hyperextension of the neck).
Intubate.	Have resuscitation equipment and medications on hand. In delivery room, may be given by umbilical vein. • Epinephrine • Sodium bicarbonate • Intravenous dextrose 10% • Naloxone (Narcan) • Normal saline (intravenous) • Lactated Ringer's solution (intravenous) • Dopamine

Presence of meconium in the lungs produces a ball-valve action, in which air is allowed in but cannot escape on expiration. This problem results in overdistention of alveoli, which leads to alveolar rupture, pulmonary air leaks, chemical inflammation (pneumonitis), and atelectasis (incomplete expansion of lungs). With air leaks, pneumothorax often occurs. These newborns can have extreme acidosis from cardiac shunting and decreased perfusion, and extreme hypoxia may result, even with 100% oxygen concentration and ventilatory assistance. Clinical signs of MAS include the skin, nails, and umbilical cord stained a yellowish green; tachypnea; retractions; generalized cyanosis; and metabolic acidosis.

Prophylactic surfactant therapy may be indicated, and chest physiotherapy is usually prescribed. Fortunately, the use of amnioinfusion (the infusion of sterile saline into the uterine cavity to dilute the meconium-stained fluid) during labor has reduced the severity of MAS (see Chapter 7). The nurse should be alert to and report abnormal fetal heart rates and meconium in the vaginal discharge of women in labor, and they should prepare for the prevention and management of meconium aspiration.

PERSISTENT PULMONARY HYPERTENSION OF THE NEWBORN

Persistent pulmonary hypertension of the newborn (PPHN) refers to the combination of pulmonary hypertension and persistence of right-to-left shunting. Blood bypasses the lungs by flowing through the foramen ovale or ductus arteriosus. PPHN may be present either as a single entity or as the main component of MAS, pneumonia, sepsis, or diaphragmatic hernia. PPHN is also known as **persistent fetal circulation.** Actually, any process that interferes with the transition from fetal to extrauterine circulation may precipitate PPHN. Maternal use of aspirin, nonsteroidal antiinflammatory drugs, or general hypoxia is implicated as a contributory factor.

When there is a sustained elevation of pulmonary vascular resistance after birth, the transition to extrauterine circulation is hindered. When right-to-left shunting occurs, hypoxemia results and progresses to hypoxia and metabolic acidosis, which cause a worsening of pulmonary vasoconstriction. ECMO may be used. Incubators, ECMO, parenteral nutrition, and minimum external stimulation have achieved positive results in the treatment of PPHN.

TRANSIENT TACHYPNEA OF THE NEWBORN

Transient tachypnea of the newborn (TTN) is seen more often in newborns delivered by cesarean birth, probably because of insufficient thoracic squeeze resulting in retained fetal lung fluid, and in large infants of diabetic mothers (IDMs). Shortly after birth, a transient elevation in respiratory rate occurs in an effort to get rid of amniotic fluid in the newborn's lungs. Newborns breathing room air will show expiratory grunting, nasal flaring, and mild cyanosis. Respirations may reach 100 to 140 breaths/minute. It is a self-limiting disorder resulting from a slight lack of surfactant or a delayed reabsorption of fetal lung fluid. The newborn is given oxygen, respiratory support, parenteral nutrition, and intravenous fluids because the infant is placed on NPO (nothing by mouth) status when respiratory rates are increased. Improvement is often seen within 48 hours (Figure 16-8).

INFANTS OF DIABETIC MOTHERS

Maternal diabetes is a problem for the mother and the newborn (see Chapter 13). The effect of diabetes on pregnancy depends on the type of diabetes and how well it is controlled. Newborns of mothers with long-term diabetes may have a deficiency of nutrients as a result of decreased blood flow reaching the fetus. Hypertension, which occurs more often in diabetic

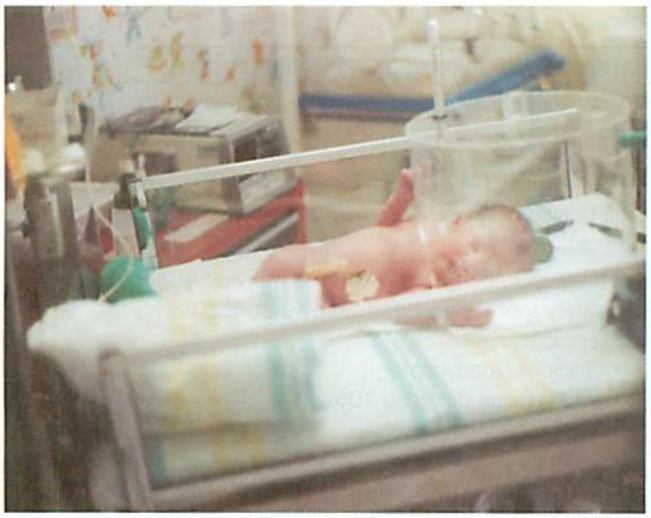


FIGURE 16-8 Oxygen is administered by an oxygen hood. The newborn is accessible for treatment without interrupting oxygen supply.

mothers compared with other women, can further compromise the uteroplacental blood flow and can cause fetal growth restriction and even death.

A high maternal glucose level can result in a large fetus (macrosomia) for the gestational age and an increased risk for problems at birth. The most common congenital defects associated with uncontrolled **hyperglycemia** throughout pregnancy include congenital heart defects, tracheoesophageal fistulas, and CNS anomalies. Close observation in the first few days of life is essential. **Infants of diabetic mothers (IDMs)** have an increased risk of RDS compared with healthy newborns because high levels of insulin appear to interfere with the production of surfactant in the lungs. Another complication for the IDMs is an increased risk for hypoglycemia after birth. After birth, the maternal glucose is no longer available; however, the newborn's pancreas continues to produce an increased amount of insulin (overproduction of insulin), and hypoglycemia results. For this reason, early and frequent heel sticks are performed to assess the newborn's blood glucose level. A Chemstick determination of less than 40 mg/dL indicates hypoglycemia in a term newborn. *Levels less than 30 mg/dL indicate hypoglycemia in the preterm newborn.* Early feedings or intravenous therapy is administered to maintain normal glucose levels. If hypoglycemia is left untreated, seizures, brain damage, and death can occur. See Box 16-3 on p. 332 for signs of hypoglycemia.

Polycythemia (hematocrit level greater than 65%) may be a problem with IDMs because these newborns often produce more erythrocytes than normal because of poor oxygenation during fetal life. Polycythemia often results in *hyperbilirubinemia* as the excessive RBCs break down after birth. *Hypocalcemia* may be a problem in response to a long and difficult birth process, and the large-for-gestational-age (LGA) newborn may exhibit tremors. Polycythemia from intrauterine hypoxia, combined with an immature liver, can predispose the LGA newborn to hyperbilirubinemia. IDM babies may have immature lungs and insufficient surfactant at birth and so are susceptible to *respiratory distress.* Administration of surfactant by endotracheal tube is often necessary. Birth defects may occur if the mother had uncontrolled diabetes mellitus during pregnancy.

IDMs can vary in their appearance. The newborn may be small for gestational age if he or she experiences growth restriction in utero because of poor diabetic control and maternal vascular involvement. **Macrosomia** (large newborn) is the result of maternal hyperglycemia in which elevated maternal levels of amino acids and fatty acids, along with hyperglycemia, cross the placenta. The resulting accelerated protein synthesis and fat stores produce the typical macrosomia newborn, with a round, puffy face and characteristic cushingoid appearance from increased subcutaneous fat. The macrosomic infant differs from the typical LGA infant who is symmetric in appearance and does not have excess fatty deposits and (hypertrophy) enlarged organs (Figure 16-9).

The risk of birth trauma resulting from macrosomia, including cephalhematoma, paralysis of the facial nerve (seventh cranial nerve), fracture of the clavicle, brachial plexus paralysis, and Erb-Duchenne (upper right arm) paralysis, is increased.

Nursing Management

The onset of hypoglycemia in IDMs is rapid because insulin production remains high when the glucose supplied by the mother is suddenly cut off. Hourly glucose checks should be done, and a level below 40 mg/dL should be reported. Early feedings with formula or breast milk or an infusion of 10% dextrose in water may be required. Once the glucose level is stable for 24 hours, the normal feeding regimen may be resumed. The infant is assessed for signs of respiratory

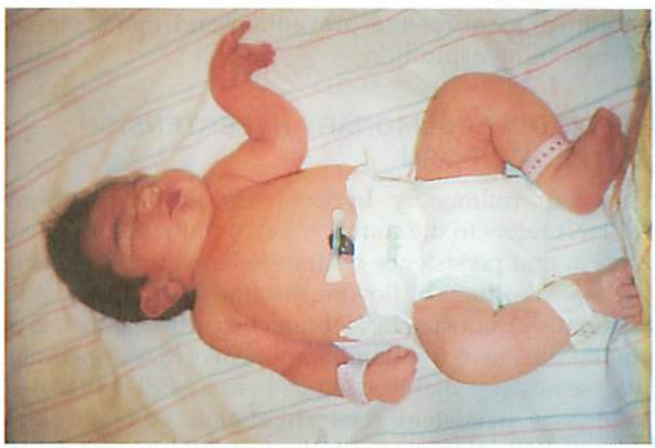


FIGURE 16-9 A newborn with macrosomia caused by maternal diabetes mellitus during pregnancy. This newborn weighed 5 kg (11 lbs) at birth. Newborns with macrosomia often have respiratory and other problems.

distress, hyperbilirubinemia, birth trauma, and anomalies. Education of the mother concerning diabetes control and self-care is essential.

NEONATAL SEPSIS

Neonatal sepsis refers to a systemic infection from bacteria in the bloodstream that occurs during the first month after birth. Newborns lack immunoglobulin M, which protects against bacteria, because that immunoglobulin does not cross the placenta from the mother to the fetus. The positive diagnosis of the infection is based on clinical symptoms and positive blood culture. The infection is usually polymicrobial (i.e., caused by more than one pathogen). Organisms responsible for neonatal infection include *Staphylococcus aureus, Staphylococcus epidermidis, Escherichia coli, Haemophilus influenzae,* and group B streptococci (GBS). Infection can result from transplacental passage of organisms, pathogens ascending from the vagina, cutaneous transmission as the fetus passes through the birth canal, environmental contamination after birth, and health care–associated transmission after birth from health care providers or invasive procedures performed in the nursery. Because of the newborn's limited immunity and inability to localize infection, it can spread rapidly into the bloodstream, and generalized sepsis can occur.

The infected newborn may demonstrate nonspecific signs, including poor feeding, vomiting, diarrhea, and lethargy; later, the newborn may show cyanosis, jaundice, and hypothermia. Because of the immaturity of the thermoregulatory center in the brain, the newborn commonly has low body temperature with an infection; however, newborns may also demonstrate temperature instability and fever. As sepsis becomes more severe, respiratory difficulty and septic shock may follow (Nursing Care Plan 16-1).

Prevention of Neonatal Sepsis

Prevention of neonatal sepsis starts prenatally with maternal screening for sexually transmitted infections (STIs). Sterile technique during delivery and aseptic technique, maintaining strict standard precautions during all hospital care, are essential in preventing neonatal sepsis. Mothers with positive cultures for GBS prenatally and during labor and delivery are treated with prophylactic antibiotic therapy to reduce the risk of neonatal sepsis. Genital lesions such as herpes require elective cesarean birth to prevent the newborn from being exposed to the virus. The prophylactic antibiotic treatment of the eyes of all newborns and appropriate umbilical cord care also help prevent neonatal sepsis. Laboratory reports are monitored. A white blood cell (WBC) count of $30{,}000/mm^3$ may be normal in the first 24 hours of life in a term newborn. A low neutrophil and high immature WBC level may indicate infection. Antibiotics such as ampicillin or gentamicin or a cephalosporin may be prescribed for 7 to 14 days. Placing the newborn in an incubator provides isolation from others and allows close observation. Home care for long-term antibiotic therapy can be initiated.

Management of the newborn includes cultures of blood, urine, stool, spinal fluid, and, in some cases, any intravenous lines. Cultures are also taken from an area with suspicious drainage, such as the eyes or umbilical stump. Nurses play a crucial role in providing education for preventive strategies and in helping families cope with their high-risk newborn.

NEWBORN WITH EFFECTS OF MATERNAL SUBSTANCE ABUSE

The woman who abuses drugs, alcohol, or other substances can deliver a newborn with varied physical and neurobehavioral manifestations. In addition, newborns may have neonatal abstinence syndrome (formerly referred to as *narcotic withdrawal*) after birth (Box 16-4). The substance-abusing mother is at risk of malnutrition, resulting in an infant who is small for gestational age, or the newborn may have congenital anomalies if the drug was taken in early pregnancy and crossed the placental barrier. The use of cocaine during pregnancy is associated with the development of abruptio placentae (Table 16-3). When drug use by the mother is suspected, a urine specimen may be collected from the infant for analysis (Skill 16-2 on p. 340). A pediatric urine collection bag may also be applied to a newborn to assess accurate intake and output when indicated.

THE NURSE AND THE FAMILY OF THE NEWBORN AT RISK

The birth of a newborn who has an anomaly, infection, or other problem is a crisis for the family. A grief reaction may occur for the loss of the anticipated "perfect baby." Parent-newborn attachment may be interrupted by incubators, intravenous lines, and ventilators. Parents may blame themselves, and self-esteem may plunge. Parents need support; recognition of the problem; and a clear explanation of the problem, treatment, and anticipated prognosis. The nurse's role is to provide support by helping the parents recognize the reality of the problem, establish trust in the health care provider, dispel misconceptions, and mobilize family support systems. The nurse must realize that some parents, although they are grateful for the expert care given to their newborn in the neonatal intensive care unit, may be jealous that the nurse is able to care for their infant and they cannot. Feelings of inferiority may influence the nurse-parent rapport. It is important to help the parents participate in the care of their newborn while providing positive support

Nursing Care Plan 16-1 Neonatal Sepsis

Scenario

A 1-day-old female newborn has an axillary temperature of 39.7° C (103.4° F). She had apnea during the first 4 hours of life and demonstrates some lethargy. Maternal history reveals premature rupture of membranes (PROMs) 18 hours before birth. Her physician orders a chest radiograph, complete blood cell count, and cultures from a large pustule on her chest area and from the umbilical cord.

Selected Nursing Diagnosis

Risk for infection related to events before, during, or after birth

Expected Outcomes	Nursing Interventions	Rationales
Causative organism(s) will be identified and treated, and disease process will resolve.	Assess for risks of infection and initiate appropriate isolation and infection control management.	Newborn's defense mechanisms are immature and overwhelmed. Maternal source of infection must be identified to prevent reinfection.
	Monitor vital signs continuously by mechanical means.	Ongoing assessment indicates early changes, making adjustment of treatment possible.
	Calculate and administer medications at the proper route, rate, time, and dose.	
	Calculate and administer electrolyte replacements.	Electrolytes are lost in the presence of sepsis.
Newborn will be protected from exposure to pathogens from hospital environment, staff, or visitors.	Clean and sterilize all equipment to be used. Change tubing, lines, or humidifiers according to facility's protocol.	Cross-contamination is minimized and controlled.
	Monitor visitors for signs of illness.	Protects health of newborn by preventing exposure to pathogens.
Newborn will be free from signs of infection.	Inspect umbilical cord stump for signs of infection.	An open wound is a potential site of infection.
	Note on newborn record if mother has history of a condition that may increase potential for newborn sepsis.	Enables planning for detection or treatment of infection.
	Assess for signs of infection such as poor feeding, poor muscle tone, pallor, increased temperature.	Temperature may be subnormal in newborn sepsis. The nurse must be alert for other signs of infection.

Selected Nursing Diagnosis

Imbalanced nutrition, less than body requirements related to poor feeding or intolerance

Expected Outcomes	Nursing Interventions	Rationales
Nutritional needs will be met and maintained.	Assess for weight loss, vomiting or diarrhea, poor sucking ability, large residual if feeding by gavage.	Caloric loss through vomiting and diarrhea or poor intake will cause weight loss.
	Initiate oral feedings as soon as possible with breast milk if appropriate.	Breast milk contains natural immunoglobulins and offers some passive immunity from mother.

Selected Nursing Diagnosis

Deficient knowledge related to infection control

Expected Outcomes	Nursing Interventions	Rationales
Parents and support system will be taught effective infection control measures.	Provide videos on infection control and supervise handwashing.	Demonstrations and videos increase understanding of techniques. Written information can be referred to when at home.
	Furnish parents with booklets, brochures, informative articles, and up-to-date references from Internet.	
Parents will state effective measures to prevent infection and manage minor illness.	Instruct mother to use clean bottle and nipple for each feeding and not store leftover formula.	Microorganisms can move from a contaminated nipple into bottle. Proper storage and use of formula preparations can prevent spoilage.
	Teach mother to consult health care provider before medicating newborn.	Use of aspirin has been linked to development of Reye's syndrome. Safe dosage of medications for neonates differs from that for adults. Neonate is at risk for toxic responses.

Critical Thinking Question

1. A 1-day-old newborn has been diagnosed as having sepsis. He is increasingly lethargic and has a poor sucking reflex and decreased oral intake. What are the priority nursing interventions?

Box 16-4 Signs Typical of Neonatal Abstinence Syndrome

RESPIRATORY DISTRESS
- Stuffy nose
- Tachypnea
- Flaring of nares
- Retractions
- Apnea

GASTROINTESTINAL DYSFUNCTION
- Diarrhea
- Vomiting
- Frantic sucking
- Poor feeder

CENTRAL NERVOUS SYSTEM
- Shrill, high-pitched cry
- Irritability
- Hypertonicity
- Tremors
- Short sleep cycles
- Occasional seizures

OTHER
- Sweating
- Fever
- Sneezing
- Yawning
- Mottled color
- Abrasions of elbows and knees

and encouragement. The nurse can use strategies to maintain parent-infant attachment whenever possible. The nurse should observe the stages of grief as parents deal with the reality (denial, anger, depression, bargaining, and acceptance) and help them toward reorganization that maintains family cohesiveness and communication. Contact with the multidisciplinary team should be maintained and community resources assessed.

DISCHARGE PLANNING AND HOME CARE

Discharge planning for the compromised newborn begins as soon as the disorder is identified. Questions about the mother's situation and home environment include: Who makes up the immediate family? Does the mother have others to provide support and care for her and the newborn? Is there access to a telephone for emergencies? Are finances available to cover necessary newborn therapy? Is a social services referral indicated?

Successful discharge and home care of the newborn with a health problem often require a multidisciplinary approach. The newborn may be transferred back from the neonatal intensive care unit to the community hospital before discharge to the home, and this transfer requires interfacility communication. Long-term care may be planned for those newborns

Table 16-3 Maternal Substance Abuse and the Newborn

PROBLEM	SYMPTOMS	INTERVENTION
Fetal alcohol syndrome (FAS): the most serious of a range of disorders caused by maternal alcohol consumption during pregnancy. Fetal alcohol spectrum disorders are a range of effects that include fetal alcohol effects (FAE), alcohol-related neurodevelopmental disorders (ARNDs), and alcohol-related birth defects (ARBDs).	Diagnosis may be confirmed by identifying fatty acid ethyl esters (FAEEs) in the meconium of the newborn. Symptoms include failure to thrive (FTT), feeding problems, CNS dysfunctions, hypotonia, speech and behavior problems, and mental retardation.	Provide quiet environment, swaddling, nutritional support, and referral for developmental monitoring.
Drug-dependent newborns: history of the mother ingesting illicit drugs during pregnancy	At birth, the newborn may manifest IUGR, jaundice (heroin and cocaine contribute to early liver maturity in the fetus and rarely show jaundice). Behavioral abnormalities and withdrawal symptoms are common.	Nursing interventions include snug swaddling, with hand near the mouth; monitoring intake and output; offering a pacifier; vertical rocking when infant is irritable; protecting skin from excoriation with the use of sheepskin or mittens; and maintaining a quiet, dimly lit environment.
Infants of tobacco-dependent mothers	Nicotine reduces the oxygen-carrying power of hemoglobin, resulting in intrauterine hypoxia and growth restriction. IUGR is common. Vasoconstriction caused by nicotine can result in placental insufficiency and prematurity with decreased Apgar scores at birth. Nicotine toxicity, including tachycardia, irritability, and poor feeding, is common.	Nurses must be alert to these signs and monitor the newborn closely.

Data from Moore, C., Jones, J., Levis, D., & Buchi, K. (2003). Prevalence of fatty acid ethyl esters in meconium specimens. *Clinical Chemistry, 49(1)*, 133–136; Goren, J., Klein, J., & Koren, G. (2006). Drugs of abuse testing in meconium. *Clinica Chimica Acta, 366(1)*, 101–111; and Plate, C., Alder, S., Jones, M., Jones, F., & Christiansen, R. (2006). Testing for fetal exposure to illicit drugs using umblical cord tissue vs meconium. *Journal of Perinatology, 26(1)*, 11–14.
CNS, central nervous system; *IUGR*, intrauterine growth restriction.

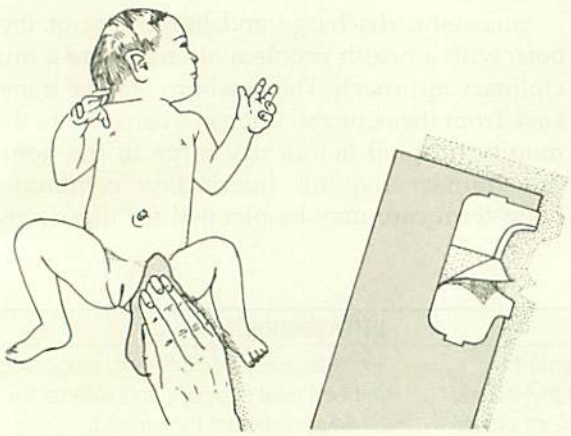
Skill 16-2 Applying a Urine Collection Bag to a Newborn

PURPOSE

To obtain a specimen for clinical laboratory assessment.

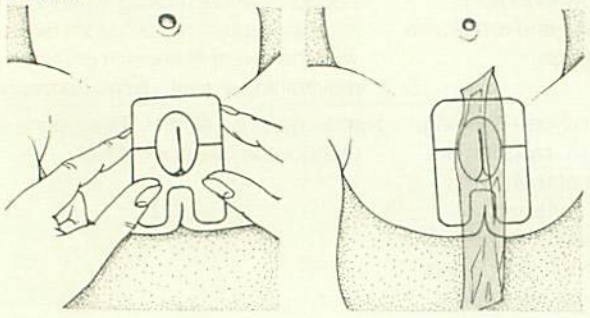
Steps

1. Prepare newborn: place in supine position and remove diaper.
2. Wash genitalia, perineum, and surrounding area; dry thoroughly. (The urine collection bag is usually a single-use, clear plastic bag with self-adhering material around the opening at the point of attachment. Adhesive will not stick to moist or oily skin, which may cause leakage of urine.)



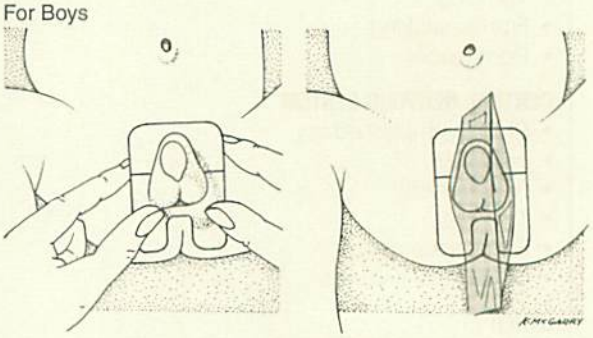
3. Remove the paper covering on the adhesive tabs of the collector bag.
4. In female newborns, fold bag in half and apply smoothly over the perineum (above the rectum), extending the tabs to the side.

For Girls

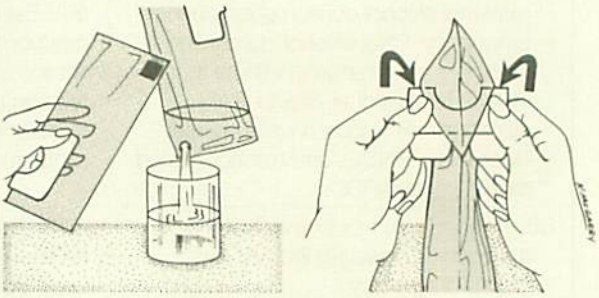


5. For the male newborn, place the penis and scrotum inside the bag before removing the tabs. Apply the posterior adhesive tabs to the perineum (above the rectum), not to the scrotum.

For Boys



6. Apply the anterior adhesive tab to cover genitalia. Make sure there are no wrinkles. Wrinkles allow openings for urine to leak out of the bag and stool to enter.
7. Carefully and loosely, replace diaper. Diaper can be cut; pull the bag through the slit. This allows bag to be visible.
8. Check frequently for urine. The urine can be aspirated with a syringe or drained from the bag.



9. For small newborns, a cotton ball can be placed inside collection bag and urine aspirated from it with a syringe.
10. Remove collection bag, and pour urine into sterile container; label and send to laboratory.
11. Clean genitalia and observe for irritation where adhesive tabs were attached. Apply clean diaper.
12. Document in medical record that the labeled urine specimen was sent to the laboratory, and record output.

with congenital malformations and some congenital infections.

Discharge of the newborn to the home setting requires parental competence. Parents must learn normal newborn care and specific information relating to their infant's medical condition. Allowing the family members to handle equipment and care for their newborn under supervision of the staff nurse helps them develop self-confidence, which is particularly important if special equipment is needed when the newborn is discharged. Follow-up care by the nurse practitioner or the pediatrician is essential. Parents must be reassured that they can call the appropriate nursing staff (high-risk nursery staff), telephone help lines (see Chapter 18), hospital or community clinics, private agencies with home care services, or other community resources when they have questions about their newborn's care.

Get Ready for the NCLEX® Examination!

Key Points

- An abnormal gene can cause a birth defect of body structure, function, or metabolism. Birth defects can also result from environmental factors. Identifying the gene involved in the defect is the first step toward developing the specific treatments.
- Inherited inborn errors of metabolism include PKU, galactosemia, and congenital hypothyroidism. The same (heel stick) blood sample can be used to test for all three of these metabolic disorders in the newborn.
- PKU is an inborn error of metabolism in which the newborn cannot process an amino acid called phenylalanine; if untreated, it can result in severe mental retardation.
- In galactosemia, the newborn has a deficiency in the enzyme galactose, resulting in the inability to convert galactose to glucose.
- Congenital hypothyroidism can result from a maternal iodine deficiency or use of antithyroid drugs by the mother during pregnancy.
- The defects involved in tetralogy of Fallot include ventricular septal defect, pulmonary stenosis, hypertrophy of the left ventricle, and an overriding aorta. Cyanosis occurs. The defect is surgically corrected.
- Noncyanotic heart defects result from the blood recirculating to the lungs.
- Hyperbilirubinemia is an abnormally high level of bilirubin in the newborn's blood. Elevated bilirubin levels can cause kernicterus, which results in mental retardation.
- Standard phototherapy is the exposure of the newborn to high-intensity light. Phototherapy helps break down and excrete elevated bilirubin in the blood. Precautions are taken during phototherapy, such as shielding the newborn's eyes for protection. Other methods of phototherapy, such as using a fiberoptic blanket, do not require covering the newborn's eyes.
- The respiratory system plays a critical role in successful adaptation to extrauterine life. Common respiratory disorders occurring after birth include RDS, MAS, PPHN, and TTN.
- Blood glucose levels less than 40 mg/dL indicate hypoglycemia in term newborns. IDMs are at risk for hypoglycemia because of high insulin production and sudden cutoff of mother's glucose supply at birth. Glucose is essential for normal brain functioning.
- Newborns of diabetic mothers are at risk for respiratory distress, congenital anomalies, hypoglycemia, polycythemia, hyperbilirubinemia, and respiratory distress.
- Neonatal sepsis is an infection occurring during the first month after birth. Bacteria are found primarily in the newborn's blood.
- The newborn with special problems, such as congenital anomalies, requires interdisciplinary care, communication with the parents, identification of needs, care and support groups for parents, and follow-up care. The nurse is often the facilitator for this team communication and is the key health care provider to give the family emotional support.
- Nursing care for newborns with neonatal abstinence syndrome includes decreasing stimuli such as noise, lights, and handling. The nurse should be nonjudgmental toward the mother and encourage her in newborn attachment and newborn care.
- Maternal nicotine intake during pregnancy causes vasoconstriction, resulting in fetal hypoxia that affects fetal development.
- Fetal alcohol spectrum disorders include FAS, caused by maternal ingestion of alcohol during pregnancy and manifested by newborn neurodevelopmental delays. Diagnosis may be confirmed by examination of fetal meconium.

Additional Learning Resources

SG Go to your Study Guide on pages 503–504 for additional Review Questions for the NCLEX® Examination, Critical Thinking Clinical Situations, and other learning activities to help you master this chapter content.

evolve Go to your Evolve website (http://evolve.elsevier.com/Leifer/maternity) for the following FREE learning resources:

- Animations
- Answer Guidelines for Critical Thinking Questions
- Answers and Rationales for Review Questions for the NCLEX® Examination
- Concept Map Creator
- Glossary with pronunciations in English and Spanish
- Patient Teaching Plans
- Skills Performance Checklists and more!

Online Resources

- www.aap.org/policy/hyperb.htm
- www.cleftline.org
- www.marchofdimes.com
- www.medem.com
- www.sbaa.org/html/sbaa_facts2.html

Review Questions for the NCLEX® Examination

1. A newborn baby is diagnosed with Turner's syndrome, which is considered a(n):
   1. Disorder of the blood
   2. Perinatal injury
   3. Inborn error of metabolism
   4. Chromosomal abnormality

2. Down syndrome is also known as:
   1. Trisomy 13
   2. Trisomy 15
   3. Trisomy 18
   4. Trisomy 21

3. Characteristically, a newborn with phenylketonuria has a(n):
   1. *Deficiency* of the enzyme necessary to convert galactose to glucose
   2. Sweet odor in body fluids
   3. Large protruding tongue
   4. Unusual musty odor to his or her urine

4. The nurse caring for a newborn with a serum bilirubin level of 14 mg/dL is aware that the primary treatment of choice is:
   1. Phototherapy
   2. Exchange transfusion
   3. Intravenous therapy
   4. Gavage feedings

5. A newborn infant diagnosed with hypoglycemia requires treatment with intravenous glucose. The nurse is aware that in order to require this treatment the infant's blood glucose must have been:
   1. Less than 40 mg/dL
   2. Between 40 and 50 mg/dL
   3. Above 50 mg/dL
   4. Between 70 and 100 mg/dL

6. Which disorder would be categorized as a malformation present at birth?
   1. Spina bifida
   2. Cystic fibrosis
   3. Hemophilia
   4. Meconium aspiration syndrome

7. Infants of diabetic mothers (IDMs) have an increased risk of which disorder(s)? *(Select all that apply.)*
   1. Respiratory distress syndrome
   2. Hypoglycemia
   3. Polycythemia
   4. Macrosomia
   5. Cephalhematoma

Critical Thinking Questions

1. A 3-day-old newborn has been placed in an incubator to treat hyperbilirubinemia. The mother states she doesn't understand why the baby is jaundiced because she is not Rh negative. What is the best response of the nurse?
2. A mother asks the nurse why the PKU blood screening test can't be performed at birth instead of waiting until the time of discharge. What is the best response of the nurse?