chapter 24

Nursing Management of the Newborn at Risk: Acquired and Congenital Newborn Conditions

KeyTERMS

alcohol-related birth defects
anencephaly
asphyxia
congenital diaphragmatic hernia
congenital heart disease
developmental dysplasia of the hip
galactosemia
hydrocephalus
hyperbilirubinemia
hypospadias
infant of a diabetic mother
kernicterus
meconium aspiration syndrome
microcephaly
myelomeningocele
neonatal abstinence syndrome
neonatal sepsis
omphalocele
respiratory distress syndrome
spina bifida

LearningOBJECTIVES

After studying the chapter content, the student should be able to accomplish the following:

1. Define the key terms.
2. Identify the most common acquired conditions affecting the newborn.
3. Describe the nursing management of a newborn experiencing respiratory distress syndrome.
4. Outline the birthing room preparation and procedures necessary to prevent meconium aspiration syndrome in the newborn at birth.
5. Identify risk factors for the development of necrotizing enterocolitis.
6. Discuss parent education for follow-up care needed by newborns with retinopathy of prematurity.
7. Discuss the impact of maternal diabetes on the newborn and care needed.

(Continued)

WOW

Courage and faith in oneself project onto others, giving them the strength to persevere.

LearningOBJECTIVES Continued

8. Describe the assessment and intervention for an infant experiencing drug withdrawal after birth.
9. Identify assessment and nursing management for newborns sustaining trauma and birth injuries.
10. Outline assessment, interventions, prevention, and management of hyperbilirubinemia in newborns.
11. Summarize the interventions appropriate for a newborn with neonatal sepsis.
12. Compare and contrast the four classifications of congenital heart disease.
13. Identify the major structural disorders affecting the gastrointestinal tract, nervous system, respiratory system, genitourinary tract, and skeletal system.
14. Describe three inborn errors of metabolism.
15. Formulate a plan of care for a newborn with an acquired or congenital condition.
16. Discuss the importance of parental participation in care of the newborn with a congenital or acquired condition, including the nurse's role in facilitating parental involvement.

Advances in prenatal and neonatal medical and nursing care throughout the industrialized world have led to a marked increase in the number of newborns who have survived a high-risk pregnancy but experience acquired or congenital conditions. These newborns are considered at risk: that is, they are susceptible to morbidity and mortality because of the acquired or congenital disorder. Several National Health Goals address the issues of acquired and congenital conditions in newborns (Healthy People 2010).

Technological and pharmacologic advances, in conjunction with standardized policies and procedures, over the past several decades have significantly improved survival rates for at-risk newborns. However, morbidity remains an important sequela. For example, some of these newborns are at risk for long-term health problems that require long-term technological support. Other newborns remain at risk for physical and developmental problems into the school years and beyond. Providing the complex care needed to maintain the child's health and well-being will have a tremendous emotional and economic impact on the family. Nurses are challenged to provide support to mothers and their families when neonatal well-being is threatened.

Acquired disorders typically occur at or soon after birth. They may result from problems or conditions experienced by the woman during her pregnancy or at birth, such as diabetes, maternal infection, or substance abuse, or conditions associated with labor and birth, such as prolonged rupture of membranes or fetal distress. There may be no identifiable cause for the disorder.

Congenital disorders are disorders present at birth, usually due to some type of malformation that occurred during the antepartal period. Congenital disorders, which typically involve a problem with inheritance, include structural anomalies (commonly referred to as birth defects), chromosomal disorders, and inborn errors of metabolism. Most congenital disorders have a complex etiology, involving many interacting genes, gene products, and social and environmental factors during organogenesis. Some alterations can be prevented or compensated for with pharmacologic, nutritional, or other types of interventions, while others cannot be changed. Only through a better understanding of the complex interplay of genetic, environmental, social, and cultural factors can these devastating and life-changing outcomes be prevented (Cleves & Hobbs, 2004).

This chapter addresses selected acquired and congenital newborn conditions. In addition, the nurse's role in assessment and intervention is discussed. Parental education and measures to help the parents cope are emphasized. Nurses play a key role in helping the parents deal with all aspects of the situation.

Acquired Disorders

Neonatal Asphyxia

Newborns normally start to breathe without assistance and often cry just after birth. By 1 minute of age, most newborns are breathing well. A newborn who fails to establish adequate, sustained respiration after birth is said to have **asphyxia.** On a physiologic level, asphyxia can be defined as impairment in gas exchange resulting in a decrease in blood oxygen levels (hypoxemia) and an excess of carbon dioxide or hypercapnia that leads to acidosis.

Incidence and Risk Factors

Asphyxia is the most common clinical insult in the perinatal period. As many as 10% of newborns require some degree of active resuscitation to stimulate breathing (Cunningham et al., 2005). According to the World Health Organization, 4 to 9 million cases of neonatal asphyxia occur annually worldwide, accounting for approximately 20% of all newborns deaths. More than a million newborns who survive asphyxia at birth develop long-term problems such as cerebral palsy, mental retardation, and speaking, hearing, visual, and learning disabilities (MNH, 2005).

HEALTHY PEOPLE 2010

National Health Goals Related to Acquired and Congenital Newborn Conditions

Objective	Significance
1. Reduce fetal and infant deaths Decrease the number of all infant deaths (within 1 year) from a baseline of 7.2/1,000 live births to 4.5/1,000 live births. Decrease the number of neonatal deaths (within the first 28 days of life) from a baseline of 4.8 to 2.9 deaths/1,000 live births. Decrease the number of post-neonatal deaths from a baseline of 2.4 to 1.2 deaths/1,000 live births. Reduce the number of deaths related to all birth defects from a baseline of 1.6 to 1.1 deaths/1,000 live births. Reduce the number of infant deaths related to congenital heart defects from a baseline of 0.53 to 0.38 deaths/1,000 live births. Reduce deaths from sudden infant death syndrome (SIDS) from a baseline of 0.72 to 0.25 deaths/1,000 live births.	Will foster early and consistent prenatal care, including education to place infants on their backs for naps and sleeping to prevent SIDS and avoidance of exposing the newborn to cigarette smoke
2. Reduce the occurrence of developmental disabilities Reduce the number of children with mental retardation from a baseline of 131 to 124 children/10,000. Reduce the number of children with cerebral palsy from a baseline of 32.2 to 31.5 children/10,000. Reduce the number of children with autism spectrum disorder.	Will promote measures for close antepartal and intrapartal monitoring of women at risk, subsequently reducing the incidence of disabilities, leading to a reduction in long-term effects and costs of care
3. Reduce the occurrence of spina bifida and other neural tube defects Reduce the number of new cases of spina bifida or other neural tube defects from a baseline of 6 to 3 new cases/10,000 live births.	Will increase awareness of the need for all women of childbearing age to take a multivitamin containing at least 400 mg of folic acid and consume foods high in folic acid
4. Reduce the occurrence of fetal alcohol syndrome	Will foster programs for at-risk groups, including adolescents, about the effects of substance abuse, especially alcohol, during pregnancy
5. Ensure appropriate newborn bloodspot screening, follow-up testing, and referral to services Ensure all newborns are screened at birth for conditions as mandated by their state-sponsored newborn screening programs. Ensure that follow-up diagnostic testing for screening positives is performed within an appropriate time period. Ensure that infants with diagnosed disorders are enrolled in appropriate service interventions within an appropriate time period	Will help in the development of protocols and procedures to ensure appropriate screening and follow-up for all newborns

DHHS, 2000; available on-line at: www.healthypeople.gov

Risk factors associated with newborn asphyxia include:

- History of maternal substance abuse
- Gestational hypertension
- Fetal distress due to hypoxia before birth
- Chronic maternal diseases such as diabetes or a heart or renal condition
- Maternal or perinatal infection
- Placental problems, such as placenta previa or abruptio placentae
- Umbilical cord problems, such as a nuchal or prolapsed cord
- Difficult or traumatic birth
- Multiple births
- Postterm newborn
- Congenital heart disease in newborn

- Maternal anesthesia or recent analgesia
- Abnormal fetal lie or presentation
- Preterm newborn (Woods, 2004)

Nursing Management

Newborn asphyxia is treatable if the perinatal team identifies and recognizes newborns who may be at risk and uses basic, effective resuscitation measures soon after birth. Prompt assessment of the newborn at birth, with immediate intervention, is essential for a newborn experiencing distress.

Assessment

The newborn with asphyxia may have hypothermia, apnea, or respiratory distress manifested by gasping respirations, grunting, nasal flaring, sternal retraction, tachypnea, hypotonia, pallor, and bradycardia (Verklan & Walden, 2004).

With failure to breathe well after birth, the newborn will develop hypoxia (too little oxygen in the cells of the body). As a result, the heart rate falls, cyanosis develops, and the newborn becomes hypotonic and unresponsive.

In addition to the clinical signs, the Apgar scores at 1 minute and 5 minutes provide valuable information. If the score is below 7 at either time, resuscitation is needed.

Several diagnostic studies also assist in identifying underlying etiologies to help plan appropriate interventions. A chest x-ray is helpful to identify any structural abnormalities that might interfere with respiration. Blood studies include cultures to rule out an infectious process, a toxicology screen to detect any maternal drugs in the newborn, and a metabolic screen to identify any metabolic conditions (Green & Wilkinson, 2004).

Nursing Interventions

Management of the newborn experiencing asphyxia includes immediate clinical assessment and resuscitation. Ensure that the equipment needed for resuscitation is readily available and in working order. Dry the newborn quickly with a warm towel and then place him or her under a radiant heater to prevent rapid heat loss through evaporation. Handling and rubbing the newborn with a dry towel is usually all that is needed to stimulate the onset of breathing. If the newborn fails to respond to stimulation, then active resuscitation is needed. Essential equipment includes a wall suction apparatus, an oxygen source, a newborn ventilation bag, endotracheal tubes (2 to 3 mm), laryngoscope, and ampules of naloxone (Narcan) with syringes and needles for administration.

The procedure for newborn resuscitation is easily remembered by the "ABCDs"—airway, breathing, circulation, and drugs (see Chapter 23, Box 23-2). Continue resuscitation until the newborn has a pulse above 100 bpm, a good cry, or good breathing efforts and a pink tongue. This last sign indicates a good oxygen supply to the brain (Woods, 2004).

Additional nursing measures for the newborn with asphyxia include the following:

- Monitor vital signs to assess the level of hypoxia.
- Check the blood glucose level and observe for signs of hypoglycemia.
- Maintain a neutral thermal environment to prevent hypothermia.
- Inform and reassure the parents about the resuscitation activities being performed.
- Offer ongoing explanations to the parents about the procedures or medications given.
- Support the parents physically and emotionally through the initial crisis and throughout the newborn's stay.
- Promote bonding by allowing the parents to hold the stimulated infant, if stable.
- Continue observation and assessment of the successfully resuscitated infant.

Transient Tachypnea of the Newborn

Most newborns make the transition from fetal to newborn life without incident. During fetal life, the lungs are filled with a serous fluid. During and after birth, this fluid must be removed and replaced with air. Passage through the birth canal during a vaginal birth compresses the thorax, which helps remove this fluid. Pulmonary circulation and the lymphatic drainage remove the remaining fluid shortly after birth. Transient tachypnea of the newborn occurs when the liquid in the lung is removed slowly or incompletely. The condition is self-limiting, usually resolving within days after birth.

Incidence and Risk Factors

Transient tachypnea of the newborn occurs in approximately 11 per 1,000 live births (Asenjo, 2003) and is commonly seen in newborns who are sedated or have been born via cesarean birth. Prolonged labor, macrosomia of the fetus, and maternal asthma and smoking have also been associated with a higher incidence of this condition (Kicklighter, 2003).

Nursing Management

Astute observation of the newborn with respiratory distress is important because transient tachypnea of the newborn is a diagnosis of exclusion. Initially it might be difficult to distinguish from respiratory distress syndrome or group B streptococcal pneumonia, since the clinical picture is similar. The symptoms of transient tachypnea rarely last more than 72 hours (Mattson & Smith, 2004). Nursing management focuses on providing adequate oxygen and determining whether these respiratory manifestations appear to be resolving or persisting.

Assessment

Newborns with transient tachypnea present within the first few hours of birth with tachypnea, expiratory grunting, retractions, labored breathing, nasal flaring, and mild

cyanosis (Asenjo, 2003). Mild to moderate respiratory distress is present by 6 hours of age, with respiratory rates as high as 100 to 140 breaths per minute (Olds et al., 2004). Breath sounds might be slightly decreased secondary to reduced air entry. The chest may appear hyperextended or barrel-shaped.

A chest x-ray usually reveals mild symmetric lung overaeration and prominent perihilar interstitial markings and streaking, which correlates with lymphatic engorgement of retained fetal fluid (Asenjo, 2003).

Nursing Interventions

Care of the newborn with transient tachypnea is mainly supportive while the retained lung fluid is reabsorbed. Supportive care includes administration of IV fluids and gavage feedings until the respiratory rate decreases enough to allow oral feedings. Supplemental oxygen under an oxygen hood to maintain adequate oxygen saturation and maintenance of a neutral thermal environment with minimal stimulation are important to minimize oxygen demand. As transient tachypnea resolves, the newborn's respiratory rate declines to 60 breaths per minute or less, oxygen requirement decreases, and the x-ray shows resolution of the perihilar streaking. The condition usually resolves within 72 hours after birth. Reassurance and progress reports to the parents are paramount in helping them cope with this crisis.

Respiratory Distress Syndrome

Despite improved survival rates and advances in perinatal care, many high-risk newborns are at risk for respiratory problems, particularly **respiratory distress syndrome** (RDS), a breathing disorder caused by lack of alveolar surfactant, which leads to decreased pulmonary compliance and increased work of breathing. Since the link between RDS and surfactant deficiency was discovered more than 30 years ago, tremendous strides have been made in understanding the pathophysiology and treatment of this disorder. The introduction of prenatal steroids to accelerate lung maturity and the development of synthetic surfactant can be credited with the dramatic improvements in the outcome of neonates with RDS (Rodriguez, 2003).

Incidence and Risk Factors

RDS affects an estimated 25,000 infants born alive in the United States annually. The incidence declines with degree of maturity at birth. It occurs in 60% of preterm newborns of less than 28 weeks' gestation, 30% of those born at 28 to 34 weeks, and less than 5% of those born after 34 weeks (ALA, 2004). Common risk factors contributing to the development of RDS include low gestational age, perinatal asphyxia regardless of gestational age, cesarean birth in the absence of labor related to the lack of thoracic squeezing, male gender, and maternal diabetes.

RDS is decreased with prolonged rupture of membranes, intrauterine growth restriction (IUGR), gestational hypertension, maternal heroin addiction, and use of prenatal corticosteroids because of the physiologic stress imposed on the fetus. Chronic stress experienced by the fetus in utero accelerates the production of surfactant before the 35th week of gestation and thus reduces the incidence of RDS at birth.

Pathophysiology

The pathophysiology of RDS relates to surfactant deficiency. Surfactant is a complex mixture of phospholipids and proteins that adheres to the alveolar surface of the lungs. It forms a coating over the inner surface of the alveoli to reduce the surface tension, thereby preventing alveolar collapse at the end of expiration. In the affected newborn, lack of surfactant results in stiff lungs and alveoli that tend to collapse, leading to diffuse atelectasis. The x-ray findings validate the pathophysiology by showing hypoaeration, underexpansion, reticulogranular ("ground glass") pattern, and decreased lung volumes (Mattson & Smith, 2004). The diagnosis of RDS is based on the clinical symptoms and abnormal x-rays.

Nursing Management

Nursing management focuses on differentiating RDS from other respiratory conditions, such as transient tachypnea or group B pneumonia, and supporting respirations to ensure adequate oxygenation. RDS usually begins at or soon after birth and tends to escalate. Continual observation of the baby's respiratory status is important in determining the underlying respiratory condition to plan appropriate treatment.

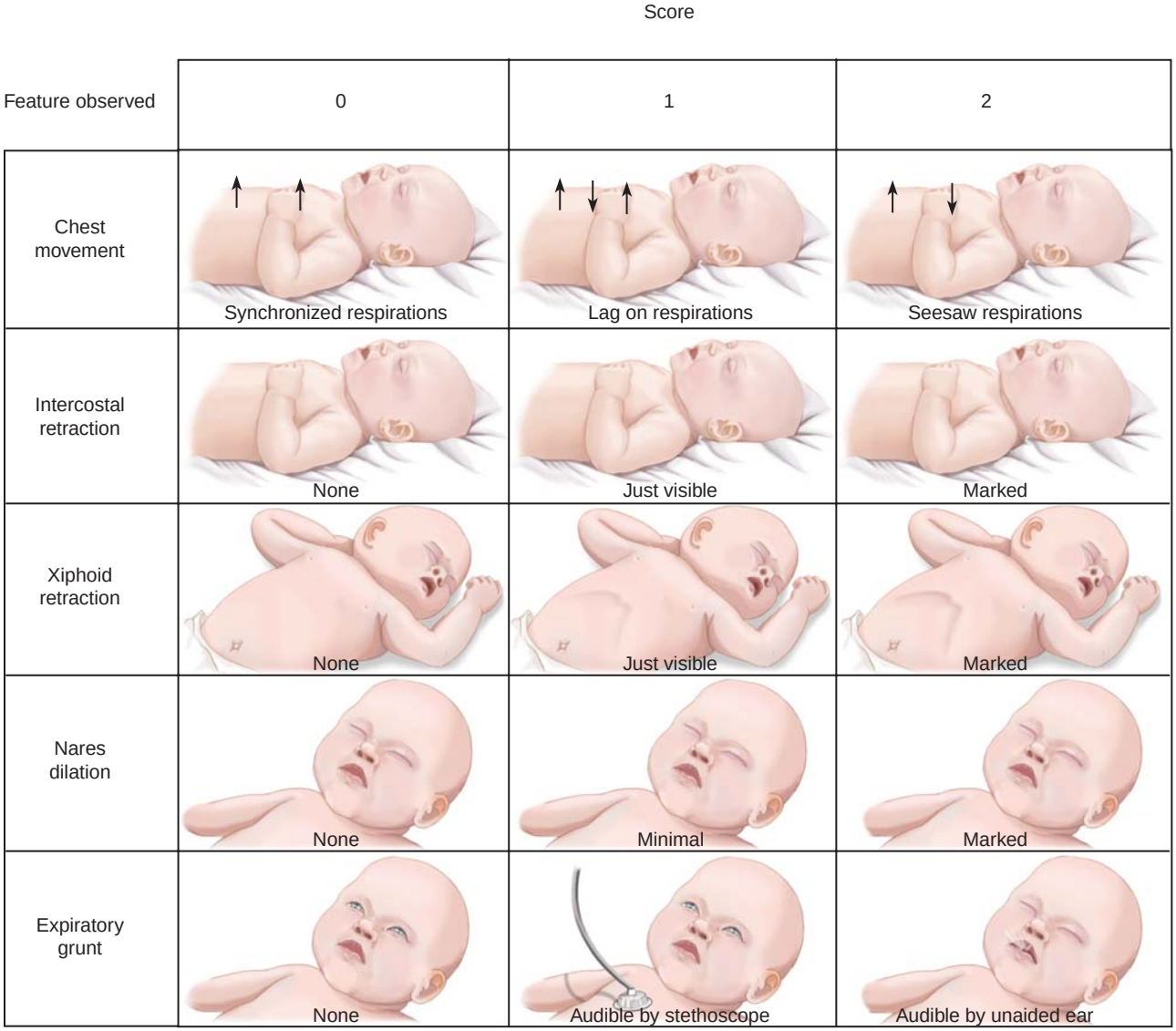
Assessment

The newborn with RDS presents at birth or within a few hours of birth with expiratory grunting respirations, nasal flaring, chest wall retractions, see-saw respirations, tachypnea with rates above 60 breaths per minute, fine inspiratory crackles on auscultation, tachycardia with rates above 150 to 180 bpm, and generalized cyanosis secondary to hypoxemia. To help determine the degree of respiratory distress, use an assessment tool such as the Silverman-Anderson index. This tool involves observation of five features, each of which is scored as 0, 1, or 2 (Fig. 24-1). The higher the score, the greater the respiratory distress. A score over 7 suggests severe respiratory distress.

If untreated, RDS will worsen. It appears to be a self-limiting disease, though, with the respiratory symptoms declining after 72 hours. This decline parallels the production of surfactant in the alveoli (Haws, 2004). The newborn needs supportive care until surfactant is produced.

Nursing Interventions

Identification of newborns at high risk for RDS is crucial because the associated atelectasis increases the work of breathing. This results in hypoxemia and acidemia, leading to vasoconstriction of the pulmonary vasculature.



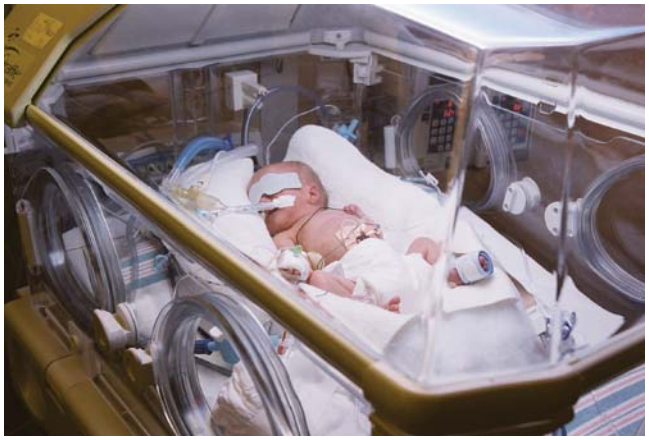
● Figure 24-1 Assessing the degree of respiratory distress. (Used with permission from Silverman, W. A. & Anderson, D. H. [1956]. A controlled clinical trial of effects of water mist on obstructive respiratory signs, death rate, and necroscopy findings among premature infants. *Pediatrics, 17*[4], 1–9.)

Subsequently, alveolar capillary circulation is limited, further inhibiting surfactant production. A vicious cycle is created, compounding the problem (Zukowsky, 2004).

Care of the newborn with RDS is primarily supportive and requires a multidisciplinary approach to obtain the best outcomes. Applying the basic principles of newborn care, such as thermoregulation, cardiovascular and nutritional support, and infection prevention, is crucial to achieve therapeutic goals, primarily reducing mortality and minimizing lung trauma. Effective therapies for established RDS include conventional mechanical ventilation, continuous positive airway pressure (CPAP), and surfactant therapy. The use of exogenous surfactant replacement therapy to stabilize the newborn's lungs until postnatal surfactant synthesis matures has become a life-saver.

Anticipate the administration of surfactant replacement therapy, prophylactically or as a rescue approach. With prophylactic administration, surfactant is given within minutes after birth through an endotracheal tube, thus providing replacement surfactant before severe RDS develops. Rescue treatment is indicated for newborns with established RDS who require mechanical ventilation and supplemental oxygen. The ideal timing of therapy has not been established: research is needed to determine which one affords better outcomes (Horbar et al., 2004).

Administer the prescribed oxygen concentration, via oxygen hood or nasal cannula. Anticipate the need for ventilator therapy, which has greatly improved in the past several years, with significant expansion of conventional and high-frequency ventilation therapies (Fig. 24-2). Recent



● Figure 24-2 A newborn with RDS receiving mechanical ventilation.

studies show no difference in outcomes for newborns who received early treatment with high-frequency oscillatory ventilation compared with those receiving conventional mechanical ventilation (Bakewell-Sachs & Blackburn, 2003). Although mechanical ventilation has improved survival rates, it is also a contributing factor to bronchopulmonary dysplasia, pulmonary hypertension, and retinopathy of prematurity (Cunningham et al., 2005).

Additional interventions to support the newborn with RDS include the following:

- Administer exogenous surfactant as ordered.
- Transfer the newborn to the neonatal intensive care unit (NICU) soon after birth.
- Continuously monitor the infant's cardiopulmonary status via invasive or noninvasive means (e.g., arterial lines or auscultation, respectively).
- Continuously monitor oxygen saturation levels.
- Closely monitor vital signs, acid–base status, and arterial blood gases.
- Administer broad-spectrum antibiotics if blood cultures are positive.
- Administer sodium bicarbonate or acetate as ordered to correct metabolic acidosis.
- Prevent or treat hypotension via fluids and pressor agents as needed.
- Prevent or treat hypoglycemia, including testing for blood glucose levels and administering dextrose as ordered.
- Cluster caretaking activities to avoid overtaxing and compromising the newborn.
- Place the newborn in the prone position to optimize respiratory status and reduce stress.
- Perform gentle suctioning to remove secretions and maintain a patent airway.
- Observe for signs and symptoms of respiratory complications such as pneumothorax.
- Assess level of consciousness to identify intraventricular hemorrhage.
- Provide sufficient calories via gavage and intravenous feedings.
- Maintain adequate hydration and assess for signs and symptoms of fluid overload.
- Provide information to the parents about treatment modalities; give thorough but simple explanations about the rationales for interventions.
- Encourage the parents to participate in care (Blackburn, 2003).

Meconium Aspiration Syndrome

Meconium is a viscous green substance composed primarily of water and other gastrointestinal secretions that can be noted in the fetal gastrointestinal tract as early as 10 to 16 weeks' gestation (Clark & Clark. 2004). It is expelled as the newborn's first stool after birth. **Meconium aspiration syndrome** occurs when the newborn inhales meconium mixed with amniotic fluid into the lungs while still in utero or on taking the first breath after birth. It is a common cause of newborn respiratory distress and can lead to severe illness. Aspiration induces airway obstruction, surfactant dysfunction, hypoxia, and chemical pneumonitis with inflammation of pulmonary tissues. In severe cases, it progresses to persistent pulmonary hypertension and death (Cunningham et al., 2005).

Incidence and Risk Factors

Meconium staining of the amniotic fluid, with the possibility of aspiration, occurs in approximately 20% of pregnancies at term (Cunningham et al., 2005). Meconium may be passed in utero secondary to hypoxic stress and aspirated in the presence of gasping or deep fetal breathing movements. Although the etiology is not well understood, the effects of meconium can be harmful to the fetus. Meconium alters the amniotic fluid by reducing antibacterial activity and subsequently increasing the risk of perinatal bacterial infection. Additionally, meconium is very irritating because it contains enzymes from the fetal pancreas.

Predisposing factors for meconium aspiration syndrome include postterm pregnancy; breech, forceps, or vacuum extraction births; prolonged or difficult labor associated with fetal distress in a term or postterm newborn; maternal hypertension or diabetes; oligohydramnios; IUGR; prolapsed cord; or acute or chronic placental insufficiency (Mattson & Smith, 2004).

Nursing Management

Nursing management focuses on ensuring adequate tissue perfusion and minimizing oxygen demand and energy expenditure. Observe for meconium-stained amniotic fluid when the maternal membranes rupture. Have a bulb syringe or suctioning equipment readily available. Proper oropharyngeal suctioning following the birth of the fetal head but before the birth of the chest can help reduce the incidence of meconium aspiration in newborns.

Assessment

Review prenatal and birth records to identify newborns who may be at high risk for meconium aspiration. Assess the amniotic fluid when the membranes rupture. Green-stained amniotic fluid suggests meconium aspiration syndrome and should be reported immediately. Also note any yellowish-green staining of the umbilical cord and nails and skin.

Other assessment findings include a barrel-shaped chest with an increased anterior-posterior (AP) chest diameter (similar to that found in a patient with chronic obstructive pulmonary disease), prolonged tachypnea, progression from mild to severe respiratory distress, intercostal retractions, end-expiratory grunting, and cyanosis (Clark & Clark, 2004). Chest x-rays show patchy, fluffy infiltrates unevenly distributed throughout the lungs, marked hyperaeration mixed with areas of atelectasis. Direct visualization of the vocal cords for meconium staining using a laryngoscope can confirm aspiration. Lung auscultation typically reveals coarse crackles and rhonchi. Arterial blood gas analysis will indicate metabolic acidosis with a low blood pH, decreased Pa_{O_2}, and increased Pa_{CO_2} (Engstrom, 2004).

Nursing Interventions

Caring for the newborn with meconium aspiration begins in the birthing unit when the birth attendant identifies meconium-stained amniotic fluid with membrane rupture during labor. Immediately, an amnioinfusion with sterile saline is administered to dilute the meconium. Upon delivery of the newborn's head, before the newborn takes the first breath, the posterior pharynx is gently suctioned to decrease the potential for aspiration. If the newborn is significantly depressed at birth, secondary clearing of the lower airways by direct tracheal suctioning may be necessary. Repeated suctioning is limited to prevent overstimulation and further depression (Hashim & Guillet, 2002). Usually the newborn is transferred to the NICU for close monitoring.

Other interventions include the following:

- Maintain a thermoneutral environment, including placing the newborn under a radiant warmer or in a warmed isolette, to prevent hypothermia.
- Minimize handling, to reduce energy expenditure and oxygen consumption that could lead to further hypoxemia and acidosis.
- Administer oxygen therapy as ordered via oxygen hood or with positive-pressure ventilation. Monitor oxygen saturation levels via pulse oximetry to evaluate the baby's response to treatment and to detect changes. Administer hyperoxygenation to dilate the pulmonary vasculature and close the ductus arteriosus or nitric oxide inhalation to decrease pulmonary vascular resistance, or use high-frequency oscillatory ventilation to increase the chance of air trapping (Haws, 2004).
- Monitor arterial blood gas results for changes and assist with measures to correct acid–base imbalances to facilitate perfusion of tissues and prevent pulmonary hypertension (Cunningham et al., 2005).
- Administer vasopressors and pulmonary vasodilators as prescribed.
- Cluster newborn care to minimize oxygen demand.
- Administer surfactant as ordered to counteract inactivation by meconium.
- Administer broad-spectrum antibiotics to treat bacterial pneumonia.
- Assist with the use of extracorporeal membrane oxygenation (ECMO), a modified type of heart–lung machine, if all else fails.
- Administer sedation to reduce oxygen consumption and energy expenditure.
- Continuously monitor the newborn's condition.
- Reassure and support the parents throughout the experience (Haws, 2004).

Persistent Pulmonary Hypertension of the Newborn

Persistent pulmonary hypertension of the newborn, previously referred to as persistent fetal circulation, is a cardiopulmonary disorder characterized by marked pulmonary hypertension that causes right-to-left extrapulmonary shunting of blood and hypoxemia. Pulmonary vascular resistance is elevated to the point that venous blood is diverted to some degree through fetal structures (i.e., the ductus arteriosus and foramen ovale) into the systemic circulation. This diversion of blood bypasses the lungs, resulting in systemic arterial hypoxemia.

Incidence and Risk Factors

Persistent pulmonary hypertension can occur idiopathically or it can be a complication of perinatal asphyxia, meconium aspiration syndrome, pneumonia, congenital heart defects, metabolic disorders such as hypoglycemia, hypothermia, hypovolemia, hyperviscosity, acute hypoxia with delayed resuscitation, sepsis, and RDS. It occurs in 2 to 6 newborns per 1,000 live births of term, near-term, or postterm infants (Steinhorn, 2004).

Nursing Management

The management of persistent pulmonary hypertension requires meticulous attention to detail, with continuous monitoring of the newborn's oxygenation and perfusion status and blood pressure. The goals of therapy include improving alveolar oxygenation, inducing metabolic alkalosis by administering sodium bicarbonate, correcting hypovolemia and hypotension with the administration of volume replacement and vasopressors, and anticipating use of ECMO when support has failed to maintain acceptable oxygenation (Steinhorn, 2004).

Assessment

A newborn with persistent pulmonary hypertension demonstrates tachypnea within 12 hours after birth. Additional findings include marked cyanosis, grunting, retractions, systolic ejection murmur, and hypotension resulting from both heart failure and persistent hypoxemia (Green & Wilkinson, 2004). An echocardiogram confirms the diagnosis.

Nursing Interventions

The condition can be prevented by instituting early and effective resuscitation and correcting acidosis and hypoxia in all compromised newborns. These compromised infants are treated in the NICU so that they can receive close monitoring and supervision and ventilatory assistance if needed.

Typically, immediate resuscitation is needed after birth and oxygen therapy is initiated. Monitor arterial blood gases frequently to evaluate the effectiveness of oxygen therapy. Respiratory support with mechanical ventilation is used frequently. Administer prescribed medications, monitor cardiopulmonary status, cluster care to reduce stimulation, and provide support and education to the parents.

Bronchopulmonary Dysplasia

Bronchopulmonary dysplasia is a chronic lung disorder of the newborn that follows a lung injury. The lung injury commonly occurs secondary to mechanical ventilation and oxygen toxicity. High inspired oxygen concentrations cause an inflammatory process in the lungs that leads to parenchymal damage (Zukowsky, 2004).

Although the etiology of the lung injury is multifactorial, it is associated with surfactant deficiency, pulmonary edema, lung immaturity, barotrauma from mechanical ventilation, and fluid overload. These newborns often need hospital care for several months after birth and home oxygen therapy after being discharged. The overall costs of treating bronchopulmonary dysplasia in the United States are estimated to be $2.4 billion annually (ALA, 2004).

Incidence and Risk Factors

Approximately 5,000 to 10,000 new cases of bronchopulmonary dysplasia occur each year. White male infants seem to be at greatest risk for developing bronchopulmonary dysplasia (ALA, 2004). Risk factors include male gender, preterm birth (<32 weeks), nutritional deficiencies, white race, excessive fluid intake during the first few days of life, presence of patent ductus arteriosus, severe RDS treated with mechanical ventilation for more than 1 week, and sepsis (Dugas et al., 2005).

Nursing Management

Bronchopulmonary dysplasia can be prevented by administering steroids to the mother in the antepartal period and exogenous surfactant to the newborn to aid in reducing the development of RDS and its severity. In addition, using high-frequency ventilation and nitric oxide helps reduce the need for respiratory support with mechanical ventilation (Lowdermilk & Perry, 2004).

Supplemental oxygen, antibiotics, and fluid restriction and diuretics to decrease fluid accumulation in the lungs are used. Intravenous feedings are given to meet the infant's nutrition needs, and physical therapy is used to improve muscle performance and to help the lungs expel mucus. Nursing management involves implementing all treatment modalities along with supporting and educating the parents throughout care.

Assessment

Although bronchopulmonary dysplasia is most common in preterm newborns, it can also occur in full-term ones who have respiratory problems during their first days of life. Thus, it is essential to assess the newborn's history related to the administration of supplemental oxygen, the length of exposure to oxygen therapy, and the use of ventilatory support.

Clinical signs and symptoms of bronchopulmonary dysplasia include tachypnea, poor weight gain related to the increased metabolic workload, tachycardia, sternal retractions, nasal flaring, and bronchospasm with abnormal breath sounds (crackles, rhonchi, and wheezes). Hypoxia, as evidenced by abnormal blood gas results, and acidosis and hypercapnia also are noted. Diagnosis is based on x-rays showing hyperinflation, infiltrates, and cardiomegaly and clinical signs.

Nursing Interventions

Nursing care includes providing continuous ventilatory and oxygen support and optimal nutrition to support growth, and administering bronchodilators, anti-inflammatory agents, and diuretics to control fluid retention. Continuously monitor the newborn's respiratory status to determine the need for continued ventilatory assistance. When the newborn is clinically stable and ready, expect to wean him or her slowly so that the baby can compensate for the changes. Supplemental oxygen may be needed after discharge from the hospital. Provide a high caloric intake to promote growth and to compensate for the calories expended due to the increased work of breathing.

Newborns with bronchopulmonary dysplasia may require continued care at home. When planning for discharge, educate the family caretaker about how to manage a chronically ill child that may be oxygen-dependent for an extended time. Also instruct the family about the safe use of oxygen in the home, including the need to notify emergency medical services and utility companies that a technology-dependent child is living in their district. In addition, initiate a social service referral to help the family access community resources (McKinney et al., 2005).

Retinopathy of Prematurity

The retina transmits visual information to the brain. Retinopathy of prematurity (ROP) is a developmental abnormality that affects the immature blood vessels of the retina. It develops in both eyes secondary to an injury such as hyperoxemia due to prolonged assistive ventilation and high oxygen exposure, acidosis, and shock. These events cause abnormal blood vessels to proliferate in the retina, resulting in scar tissue. ROP can lead to lifelong vision impairment (NEI, 2005). It can also lead to vitreous hemorrhage, retinal detachment, and blindness (Brooks, 2004).

Incidence and Risk Factors

The incidence of ROP in preterm newborns is inversely proportional to their birth weight. Of the approximately 4 million infants born in the United States annually, about 57,000 weigh 1,500 grams (2.75 lb) or less. About 14,000 to 16,000 of these infants are affected by some degree of ROP (NEI, 2005). Other risk factors include the duration of intubation and the use of oxygen therapy, intraventricular hemorrhage, multiple prenatal maternal risk factors (substance abuse, hypertension, preeclampsia, heavy smoker, or inadequate oxygen supply to placenta), and sepsis (Vision Channel, 2004).

The eye develops rapidly between 28 and 40 weeks' gestation. When the baby is born preterm, normal blood vessel development may cease. Exposure to high oxygen concentrations leads to severe retinal vasoconstriction with endothelial damage and vessel obliteration. Adhesions form, causing retinal detachment. Although the precise levels of hyperoxemia that can be sustained without causing retinopathy are not known, very immature newborns who develop respiratory distress often must be given high oxygen concentrations to maintain life (Cunningham et al., 2005).

Nursing Management

The incidence of ROP can be decreased by minimizing the risk of preterm birth through providing quality prenatal care and health counseling to all new mothers. When ROP does develop, care depends on the stage and degree of retinal findings. ROP is classified in five stages, ranging from mild (stage I) to severe (stage V). Typically, stages I and II resolve on their own and require only periodic evaluation by the ophthalmologist. For more advanced stages, surgical intervention such as laser photocoagulation and scleral buckling surgery and/or vitrectomy is performed.

Assessment

The newborn who develops ROP exhibits no signs or symptoms, so assessment involves identifying the newborn at risk. Any newborn with a birthweight of less than 1,500 g or born at less than 28 weeks' gestation should be examined by a pediatric ophthalmologist within 4 to 6 weeks after birth. Diagnosis is made by an ophthalmic examination (Kenner & Lott, 2004).

Nursing Interventions

Cautious, judicious use of oxygen is imperative in preventing this serious eye condition. Continuously assess and monitor the newborn who is receiving oxygen therapy. Cover the isolette with a blanket and dim the surrounding lights to protect the newborn's eyes.

Assist with scheduling an ophthalmic examination for the newborn and administer a mydriatic eye agent to dilate the newborn's pupils approximately 1 hour prior to the examination as ordered. During this time, take extra care to protect the newborn's eyes from bright light. If necessary, assist with the examination by holding the newborn's head. Follow-up eye examinations are scheduled every 2 to 3 weeks depending on the severity of the clinical findings at the first examination (Verklan & Walden, 2004).

Provide support to the parents. This is an extremely difficult time for them: in addition to learning to meet the needs of their preterm newborn, they must also deal with the possibility that their baby may have a condition that could lead to blindness. Consider the family's needs and provide individualized support and guidance. Provide information about the newborn's condition and treatment options. Stress the need for follow-up vision screenings, because ROP is considered a life-long disease with numerous late-onset problems.

Periventricular–Intraventricular Hemorrhage

Periventricular–intraventricular hemorrhage is defined as bleeding that usually originates in the subependymal germinal matrix region of the brain, with extension into the ventricular system (Haws, 2004). Each ventricular area contains a rich network of capillaries that are very thin and fragile and can easily rupture. It is a common problem of preterm infants, especially those born before 32 weeks. A significant number of these newborns will incur brain injury, leading to cerebral palsy, hydrocephalus, and behavioral, learning, auditory, or visual deficits in their early and later school-age years. Identifying preventive strategies to reduce the incidence of these brain insults is a national public health priority (Bloch, 2005).

Periventricular–intraventricular hemorrhage is classified according to a grading system of I to V (least severe to most severe) (Blackburn, 2003). The prognosis is guarded, depending on the grade and severity of the hemorrhage. Generally, newborns with mild hemorrhage (grades I and II) have a much better developmental outcome than those with severe hemorrhage (grades III and IV). Complications include obstructive hydrocephalus, developmental impairment, cerebral palsy, and seizure disorders (Haws, 2004).

Incidence and Risk Factors

The incidence of ventricular hemorrhage depends on the gestational age at birth. About 30% to 40% of newborns weighing 1,500 g or less or born at 30 weeks' gestation or

less will have evidence of hemorrhage. Only about 4% of term newborns show evidence of ventricular hemorrhage. Very-low-birthweight infants have the earliest onset of hemorrhage and the highest mortality rate (Cunningham et al., 2005).

The preterm newborn is at greatest risk for periventricular–intraventricular hemorrhage because the immaturity of the brain makes it more vulnerable to injury. The more preterm the newborn is, the greater likelihood of brain damage. While all areas of the brain can be injured, the periventricular area is the most vulnerable.

With a preterm birth, the fetus is suddenly transported from a well-controlled uterine environment into a highly stimulating one. This tremendous physiologic stress and shock may contribute to the rupture of periventricular capillaries and subsequent hemorrhage. Other associated risk factors include acidosis, asphyxia, unstable blood pressure, meningitis, seizures, acute blood loss, hypovolemia, respiratory distress with mechanical ventilation, intubation, apnea, hypoxia, suctioning, hyperosmolar solutions, rapid volume expansion, as well as most nursing activities that involve handling. Most hemorrhages occur in the first 72 hours after birth (Cunningham et al., 2005).

Nursing Management

Nursing management focuses on identifying the risk factors leading to hemorrhage and interventions to decrease the risk of hemorrhage. Using a developmental care environment in the NICU is helpful to minimize the risk of hemorrhage. Support for the parents to cope with the diagnosis and potential long-term sequelae is essential. The long-term neurodevelopmental outcome is determined by the severity of the bleed.

Assessment

Assessment of a newborn with periventricular–intraventricular hemorrhage varies significantly. Some newborns have no signs; in others the findings are dramatic. Signs include a sudden unexplained drop in hematocrit, pallor, poor perfusion as evidenced by respiratory distress and oxygen desaturation, lethargy, sudden deterioration in vital signs associated with shock secondary to hemorrhage, changes in level of consciousness, seizures, metabolic acidosis, glucose instability and hypotonia, bradycardia, shock, and a tense anterior fontanel (Annibale & Hill, 2003). Frequently a bleed can progress rapidly and result in shock and death. Cranial ultrasonography is the diagnostic tool of choice to detect hemorrhage.

Nursing Interventions

Prevention of preterm birth is essential in preventing periventricular–intraventricular hemorrhage. In addition, the nurse plays a major role in preventing perinatal asphyxia and birth trauma. If a preterm birth is expected, having the mother deliver at a tertiary facility with a NICU would be most appropriate.

Nursing care for periventricular–intraventricular hemorrhage is primarily supportive and includes the following:

- Correct anemia, acidosis, and hypotension with fluids and medications.
- Prevent fluctuations in blood pressure by slowly administering fluids; avoid rapid volume expansion to minimize changes in cerebral blood flow.
- Limit stimulation in the newborn's environment to reduce stress.
- Minimize handling of the newborn by clustering nursing care; reduce the newborn's exposure to noxious stimuli to avoid a fluctuation in blood pressure and energy expenditure.
- Keep the newborn in a flexed, contained position with the head elevated to prevent or minimize fluctuations in intracranial pressure.
- Provide adequate oxygenation to promote adequate tissue profusion but controlled ventilation to decrease the risk of pneumothorax.
- Assess for signs of hemorrhage, such as changes in the level of consciousness, bulging fontanel, seizures, apnea, and reduced activity level.
- Provide education and emotional support for the parents throughout the newborn's stay.
- Discuss expectations for short-term and long-term care needs with the parents.
- Promote community awareness of factors that may contribute to periventricular–intraventricular hemorrhage, such as a lack of prenatal care, maternal infection, alcohol consumption, and smoking (Bloch, 2005).

Necrotizing Enterocolitis

Necrotizing enterocolitis (NEC) is a serious gastrointestinal disease of unknown etiology in newborns. Although it is more common in preterm newborns, it can be observed in term newborns. The precise pathogenesis is unclear, but four major factors have been proposed: the presence of a pathogenic organism, a hypoxic/ischemic event, the challenge of enteral feeding, and altered enteric mucosa integrity. Currently there are no effective prevention strategies (Lin et al., 2005).

Incidence and Risk Factors

NEC occurs in up to 10% of all admissions to the NICU; approximately 90% of cases are found in preterm newborns (Bell, 2005). Predisposing risk factors are highlighted in Box 24-1.

Pathophysiology

NEC is characterized by mucosal or transmucosal necrosis of part of the intestine (Wood, 2005). Although any region of the bowel can be affected, the distal ileum and proximal colon are the regions most commonly involved. NEC usually occurs between 3 and 10 days of life.

Several factors contribute to the development of newborn NEC, but three events are typically found: perinatal

BOX 24-1

PREDISPOSING FACTORS FOR THE DEVELOPMENT OF NECROTIZING ENTEROCOLITIS

Prenatal Factors
- Preterm labor
- Prolonged rupture of membranes
- Preeclampsia
- Maternal sepsis
- Amnionitis
- Uterine hypoxia

Postnatal Factors
- Respiratory distress syndrome
- Patent ductus arteriosus
- Congenital heart disease
- Exchange transfusion
- Low birthweight
- Low Apgar scores
- Umbilical catheterization
- Hypothermia
- Gastrointestinal infection
- Hypoglycemia
- Asphyxia

hypoxia, bacterial invasion, and high-solute feedings. In perinatal hypoxia, blood is shunted from the gut to more important organs (heart and brain), resulting in ischemia and damage to the intestinal wall. Bacterial invasion follows, and high-solute feedings provide sustenance for bacteria to flourish. In an attempt to improve gastrointestinal function and reduce the risk of NEC, many neonatologists are trying enteral antibiotics, judicious administration of parenteral fluids, human milk feedings, antenatal corticosteroids, enteral probiotics (*Lactobacillus acidophilus*), and delayed or slow feedings (Bell, 2005).

Nursing Management

Nursing management focuses on being alert to the risk factors associated with NEC and meticulous observation for clinical manifestations of this potentially devastating condition. Frequently the infant has nonspecific symptoms that can be easily overlooked. The nurse schedules and assists with the various diagnostic tests ordered to evaluate the infant for this condition.

Assessment

Assessment of a newborn with NEC typically reveals abdominal distention and tenderness, bile-stained emesis, lethargy, feeding intolerance, decreased activity, respiratory distress, visible loops of bowel, metabolic acidosis, temperature instability, hypotension, grossly bloody stools, delayed gastric emptying, diarrhea, oliguria, hypoglycemia, and cyanosis (Verklan & Walden, 2004).

The diagnosis of NEC is based on clinical suspicion supported by x-rays and laboratory studies. Diagnostic findings typically indicate leukocytosis, thrombocytopenia, electrolyte imbalances, metabolic acidosis, hypoxemia, blood in stools, and disseminated intravascular coagulation (DIC). The x-rays show diffuse gaseous distention of the intestines, with air within the wall of the intestine (pneumatosis) and persistently dilated loops of bowel (Verklan & Walden, 2004).

Nursing Interventions

As soon as NEC is suspected, expect to stop oral feedings and withhold food and fluids, placing the newborn on NPO status. Institute gastric decompression as ordered with an orogastric tube attached to low intermittent suction. Administer antibiotics as ordered, because it is thought that NEC involves bacterial invasion. Give parenteral fluid therapy to replace formula or breast milk. Continue to monitor the newborn's condition via abdominal x-rays, blood tests, and arterial blood gases, as ordered.

Supportive treatment is continued as long as there is no evidence of intestinal necrosis or perforation (free air in the abdomen on x-ray). If necrosis or perforation occurs, surgery is indicated and may include laparotomy with resection of necrotic bowel and possibly creation of an ostomy (Kenner & Lott, 2004).

Key nursing interventions for the newborn with NEC include the following:

- Manage pain by administering analgesics as ordered.
- Stress infection control, with an emphasis on careful handwashing.
- Make continuous nursing assessments, including:
  - Checking for guaiac-positive stools
  - Measuring abdominal girth frequently
  - Palpating the abdomen for tenderness and rigidity
  - Auscultating bowels to assess for paralytic ileus
  - Observing for abdominal redness or shininess, which indicates peritonitis
  - Careful monitoring of intake and output
  - Coordinating laboratory and x-ray studies to monitor the newborn's status
- Maintain fluid and electrolyte balance.
- Support the parents throughout medical and/or surgical interventions by:
  - Listening to their worries and fears
  - Answering their questions honestly and hopefully
  - Teaching them about therapies and procedures
  - Encouraging interaction with the newborn through gentle touching

Infants of Diabetic Mothers

An **infant of a diabetic mother** is one born to a woman with pregestational or gestational diabetes (see Chapter 20 for additional information). The newborn of a diabetic

woman is at high risk for numerous health-related complications, especially hypoglycemia. In light of the increasing incidence of type 2 diabetes among women of childbearing age due to obesity, it is important to educate women about the potential impact of poor glycemic control on their offspring.

Impact of Diabetes on the Newborn

For more than a century, it has been known that diabetes during pregnancy can have severe adverse effects on fetal and newborn outcomes. Infants of diabetic mothers have an increased morbidity and mortality in the perinatal period. The incidence of major congenital anomalies is much greater for these newborns than for other newborns. Poor glycemic control in the first trimester, during organogenesis, is thought to be the major reason for congenital malformations. The most common types of malformations in infants of diabetic mothers involve the cardiovascular, skeletal, central nervous, gastrointestinal, and genitourinary systems; cardiac anomalies are the most common (Cleves & Hobbs, 2004).

Infants of diabetic mothers are longer and weigh more than newborns of similar gestational age. They also have increased organ weights (organomegaly) and excessive fat deposits on the shoulders and trunk, contributing to the increased overall body weight and predisposing them to shoulder dystocia. These newborns are macrosomic (an infant whose birthweight exceeds 4,500 g). These oversized newborns frequently require cesarean births for cephalofetal disproportion and are often hypoglycemic in the first few hours after birth.

Despite their increased size and weight, they may be remarkably feeble, showing behaviors similar to those of a preterm newborn. Thus, birthweight may not be a reliable criterion of maturity. Newborns of women with diabetes but without vascular complications often tend to be large for gestational age (LGA), whereas those of women with diabetes and vascular disease are usually small for gestational age (SGA).

The large size of the infant born to a diabetic mother is secondary to exposure to high levels of maternal glucose crossing the placenta into the fetal circulation. Maternal hyperglycemia acts as a fuel to stimulate increased production of fetal insulin, which in turn promotes somatic growth within the fetus. The fetus responds to these high levels by producing more insulin, which acts as a growth factor in the fetus (Olds et al., 2004). How the fetus will be affected depends on the severity, duration, and control of the diabetes in the mother.

Common Problems in Infants of Diabetic Mothers

Common problems include macrosomia, RDS, birth trauma, hypoglycemia, hypocalcemia and hypomagnesemia, polycythemia, hyperbilirubinemia, and congenital anomalies (Table 24-1).

Nursing Management

The focus of care for these infants is early detection and initiation of therapy to address potential problems (Nursing Care Plan 24-1). Care begins in the prenatal period by identifying women with diabetes and taking measures to control maternal glucose levels (see Chapter 20 for information on management of the pregnant woman with diabetes).

Treatment focuses on correcting hypoglycemia, hypocalcemia, hypomagnesemia, dehydration, and jaundice. Oxygenation and ventilation are supported as necessary.

Assessment

The infant of a diabetic mother has a characteristic appearance:

- Full rosy cheeks with a ruddy skin color
- Short neck (some describe "no-neck" appearance)
- Buffalo hump over the nape of the neck
- Massive shoulders with a full intrascapular area
- Distended upper abdomen due to organ overgrowth
- Excessive subcutaneous fat tissue, producing fat extremities (Fig. 24-3)

Be alert for hypoglycemia, which may occur immediately after birth or within an hour. Assess blood glucose levels, which should remain above 40 mg/dL. Closely assess the newborn for signs of hypoglycemia, including listlessness, hypotonia, apathy, poor feeding, apneic episodes with a drop in oxygen saturation, cyanosis, temperature instability, pallor and sweating, tremors, irritability, and seizures.

Determine baseline serum calcium, magnesium, and bilirubin levels and monitor frequently for changes (Table 24-2). Hypocalcemia is typically manifested in the first 2 to 3 days of life as a result of birth injury or a prolonged delay in parathyroid hormone production. Hypomagnesemia parallels calcium levels and is suspected only when hypocalcemia does not respond to calcium replacement therapy. Red blood cell breakdown leads to increased hematocrit and polycythemia. In addition, hyperbilirubinemia may be caused by slightly decreased extracellular fluid volume, hepatic immaturity, and birth trauma forming enclosed hemorrhages. It can appear within the first 24 hours of life (pathologic) or after 24 hours of life (physiologic).

Assess the newborn for signs of birth trauma involving the head (tense, bulging fontanels, cephalhematoma, skull fractures, and facial nerve paralysis), shoulders and extremities (posturing, paralysis), and skin (bruising). Inspect the newborn for compromised oxygenation by examining the skin for cyanosis, pallor, mottling, and sluggish capillary refill. Take the newborn's temperature frequently and provide a neutral thermal environment to prevent cold stress, which would increase the glucose utilization and contribute to the hypoglycemic state.

Table 24-1 Common Problems of Infants of Diabetic Mothers (IDMs)

Condition	Description	Effects
Macrosomia	– Newborn with an excessive birthweight; arbitrarily defined as a birthweight >4,000 g (8 lb 13 oz) to 4,500 g (9 lb 15 oz) or >90% for gestational age – Complication in 10% of all pregnancies in the United States	– Increased risk for shoulder dystocia, traumatic birth injury, birth asphyxia – Risks for newborn hypoglycemia and hypomagnesemia, polycythemia, and electrolyte disturbances – Increased maternal risk for surgical birth, postpartum hemorrhage and infection, and birth canal lacerations – Increased risk of developing type 2 diabetes later in life for both – Higher weight and accumulation of fat in childhood and a higher rate of obesity in adults
Respiratory distress syndrome (RDS)	– Cortisol-induced stimulation of lecithin/sphingomyelin (phospholipids) necessary for lung maturation is antagonized due to the high insulin environment within the fetus due to mother's hyperglycemia. – Less mature lung development than expected for gestational age – Decrease in the phospholipid phosphatidylglycerol (PG), which stabilizes surfactant, compounding risk	– Most commonly, breathing normally at birth but developing labored, grunting respiration with cough and a hoarse complaining cry within a few hours with chest retractions and varying degrees of cyanosis – IDMs with vascular disease seldom develop RDS because the chronic stress of poor intrauterine perfusion leads to increased production of steroids, which accelerates lung maturation.
Hypoglycemia	– Glucose is the major source of energy for organ function. – Typical characteristics: – Poor feedings – Jitteriness – Lethargy – High-pitched or weak cry – Apnea – Cyanosis and seizures – Some newborns asymptomatic	– Low blood glucose levels are problematic during early post-birth period due to abrupt cessation of high-glucose maternal blood supply and the continuation of insulin production by the newborn. – Limited ability to release glucagon and catecholamines, which normally stimulate glucagon breakdown and glucose release – Prolonged and untreated hypoglycemia leads to serious, long-term adverse neurologic sequelae such as learning disabilities and mental retardation.
Hypocalcemia and hypomagnesemia	Hypocalcemia (drop in calcium levels) manifested by tremors, hypotonia, apnea, high-pitched cry, and seizures due to abrupt cessation of maternal transfer of calcium to fetus primarily in third trimester and birth asphyxia Associated hypomagnesemia directly related to the maternal level before birth About half of IDMs affected	Newborn is at risk for a prolonged delay in parathyroid hormone production and cardiac dysrhythmias.

Table 24-1 Common Problems of Infants of Diabetic Mothers (IDMs) (continued)

Condition	Description	Effects
Polycythemia	– Venous hematocrit of >65% in the newborn – Increased oxygen consumption by IDM secondary to fetal hyperglycemia and hyperinsulinemia – Increased fetal erythropoiesis secondary to intrauterine hypoxia due to placental insufficiency from maternal diabetes – Hypoxic stimulation of increased red blood cell (RBC) production as compensatory mechanism	Increased viscosity, resulting in poor blood flow predisposing newborn to decreased tissue oxygenation and development of microthrombi
Hyperbilirubinemia	Usually seen within the first few days after birth, manifested by a yellow appearance of the sclera and skin Excessive red cell hemolysis necessary to break down increased RBCs in circulation due to polycythemia Resultant elevated bilirubin levels Excessive bruising secondary to birth trauma of macrosomic infants, further adding to high bilirubin levels	If untreated, high levels of unconjugated bilirubin may lead to kernicterus (neurologic syndrome that results in irreversible damage) with long-term sequelae that include cerebral palsy, sensorineural hearing loss, and mental retardation.
Congenital anomalies	– Occur in up to 10% of IDMs, accounting for 30% to 50% of perinatal deaths – Incidence is greatest among SGA newborns. – Overall, approximately three times the usual incidence of congenital anomalies compared to newborns from the non-diabetic general population	Most common anomalies: – Coarctation of the aorta – Atrial and ventricular septal defects – Transposition of the great vessels – Sacral agenesis – Hip and joint malformations – Anencephaly – Spina bifida – Caudal dysplasia – Hydrocephalus

Sources: Jazayeri & Contreras, 2005; Moore, 2004; Harris, 2004; Mattson & Smith, 2004; Lessaris, 2005; Barbour, 2003; Schaefer-Graf et al., 2003; and Johnson, 2003.

Nursing Interventions

Appropriate nursing interventions for infants of diabetic mothers include the following:

- Monitor blood glucose levels via heel stick every hour for the first 4 hours of life and then every 3 to 4 hours until stable. Document the results. Report unstable glucose values if oral feedings do not maintain and stabilize the newborn's blood glucose levels.
- Prevent hypoglycemia by providing early oral feedings with formula or breast milk at frequent intervals (every 2 to 3 hours). Feedings help to control glucose levels, reduce hematocrit, and promote bilirubin excretion.
- If glucose levels are not stabilized, initiate IV glucose infusions as ordered and monitor the infusions at the prescribed rate.
- Monitor serum calcium levels for needed supplementation (oral or IV calcium gluconate), and observe for signs and symptoms of hypocalcemia, such as tremors, jitteriness, twitching, seizures, and high-pitched cry.
- Monitor serum bilirubin levels and prepare to administer fluid therapy as ordered for needed hydration, and institute phototherapy if the newborn is over 24 hours old.
- Maintain a neutral thermal environment to avoid cold stress, which may stimulate the metabolic rate, thereby increasing the demand for glucose.

Nursing Care Plan 24-1

Overview of an Infant of a Diabetic Mother (IDM)

Jamie, a 38-year-old Hispanic woman, gave birth to a term LGA newborn weighing 10 lb. She had a history of gestational diabetes but had not received any prenatal care. She arrived at the hospital in active labor. Despite macrosomia, the newborn's Apgar scores were 8 and 9. No resuscitative measures were needed. The nursery nurse notices a quiet newborn with tremors of the extremities. A glucose level via a heel stick was 35 mg/dL. The newborn is now demonstrating signs of respiratory distress—grunting, nasal flaring, and tachypnea.

Nursing Diagnosis: Risk for injury related to hypoglycemia secondary to maternal gestational diabetes

Outcome identification and *evaluation*	Interventions with *rationales*
The newborn will exhibit adequate glucose control *as evidenced by blood glucose levels above 40 mg/dL and an absence of clinical signs of hypoglycemia.*	Monitor blood glucose levels *to detect hypoglycemia, which would be <40 mg/dL secondary to the infant's hyperinsulin state in utero.* Observe for manifestations of hypoglycemia such as pallor, tremors, jitteriness, lethargy, and poor feeding *to allow for early detection and prompt intervention to minimize the risk of complications associated with hypoglycemia.* Maintain a neutral thermal environment *to reduce heat loss through evaporation, convection, conduction, and radiation, which further depletes glycogen stores.* Monitor temperature *to prevent cold stress and use of glucose to maintain temperature.* Initiate early feedings or administer glucose supplements as ordered *to prevent hypoglycemia caused by the infant's hyperinsulin state.* Cluster infant care activities *to conserve energy to reduce use of glucose and glycogen stores.* Reduce environmental stimuli by dimming lights and speaking softly *to reduce energy needs and further utilization of glucose.*

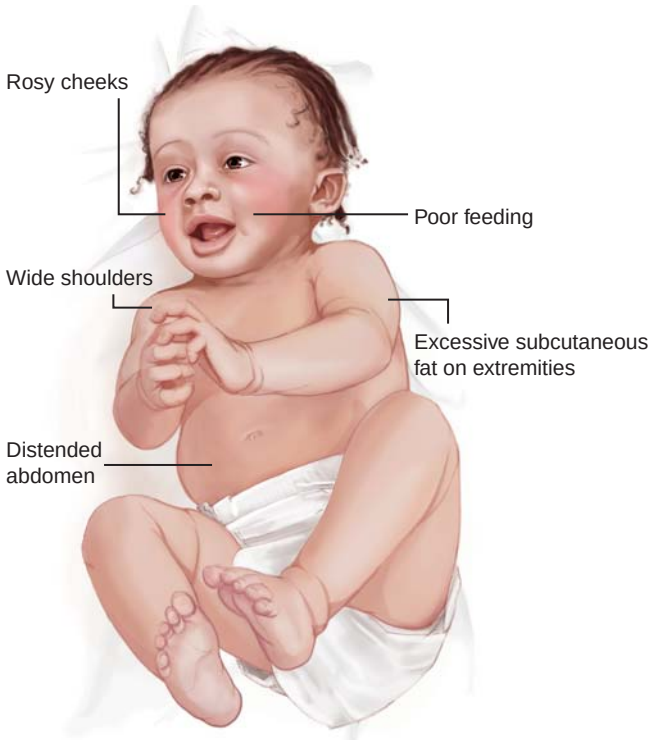
Nursing Diagnosis: Risk for impaired gas exchange related to respiratory distress secondary to fetal hyperinsulinemia inhibiting pulmonary surfactant production and delaying lung maturation

Newborn will demonstrate signs of adequate oxygenation without respiratory distress *as evidenced by respiratory rate and vital signs within acceptable parameters, absence of nasal flaring and grunting, and oxygen saturation and arterial blood gas levels within acceptable parameters.*	Assess newborn's skin *to identify cyanosis, pallor, and mottling, which may indicate compromised oxygenation.* Monitor newborn's vital signs *to establish a baseline and evaluate for changes.* Assess airway patency and suction as ordered *to promote adequate oxygen intake and maintain patency.*

Overview of an Infant of a Diabetic Mother (IDM) (continued)

Outcome identification and *evaluation*	Interventions with *rationales*
	Assess lung sounds for changes *to allow for early detection of change in status.* Monitor oxygen saturation levels *to determine adequacy of tissue perfusion.* Assess arterial blood gases *to determine presence of acidosis, hypoxemia, or hypercarbia, which would indicate hypoxia.* Administer oxygen as ordered *to increase the availability of oxygen and reduce hypoxia.* Maintain normal blood glucose levels and a neutral thermal environment and reduce excessive stimuli *to decrease oxygen consumption.*

- Provide rest periods to decrease energy demand and expenditure.
- Closely monitor the baby's respiratory status to identify signs and symptoms of respiratory distress.
- Perform a head-to-toe physical assessment to identify congenital anomalies.
- Provide support and information to the parents and family. They may erroneously interpret the newborn's large size as an indication that the newborn is free of problems. Encourage open communication and listen with empathy to the family's fears and concerns.
- Offer frequent opportunities for the parents to interact with their newborn.
- Make appropriate referrals to social services and community resources as necessary to help the family cope.



● Figure 24-3 Characteristics of an infant of a diabetic mother.

Birth Trauma

Injuries to the newborn from the forces of labor and birth are categorized as birth trauma. In the past, numerous injuries were associated with difficult births requiring external or internal version or mid- or high forceps deliveries. Today, however, cesarean births have contributed to the decline in birth trauma.

Incidence and Risk Factors

The process of birth is a blend of compression, contractions, torques, and traction. When fetal size, presentation, or neurologic immunity complicates this process, the forces of labor and birth may lead to tissue damage, edema, hemorrhage, or fracture in the newborn. Significant birth

Table 24-2 Critical Laboratory Values for Infants of Diabetic Mothers

Hypoglycemia	<40 mg/dL
Hypocalcemia	<7 mg/dL
Hypomagnesemia	<1.5 mg/dL
Hyperbilirubinemia	>12 mg/dL (term infant)
Polycythemia	>65% (venous hematocrit)

trauma accounts for fewer than 2% of neonatal deaths and stillbirths in the United States (Lynam & Verklan, 2004). Improved prenatal diagnosis and monitoring during labor have helped to reduce the incidence of birth injuries today.

Factors placing the newborn at risk for birth injury include cephalopelvic disproportion, maternal pelvic anomalies, oligohydramnios, prolonged or rapid labor, abnormal presentation (breech, face, brow), instrument-assisted extraction (vacuum or forceps), fetal prematurity, fetal macrosomia, and fetal abnormalities (Lynam & Verklan, 2004).

Types of Birth Trauma

Fractures

Fractures most often occur during breech births or shoulder dystocia in newborns with macrosomia. Midclavicular fractures are the most common type of fracture, secondary to shoulder dystocia. The newborn presents with irritability and does not move the arm on the affected side either spontaneously or when the Moro reflex is elicited. X-rays confirm the fracture. Typically, healing is rapid and uneventful. Arm motion may be limited by pinning the newborn's sleeve to the shirt. Explanation to the parents and reassurance are needed.

Loss of spontaneous arm or leg motion indicates a long-bone fracture of the humerus or femur, respectively. Usually swelling and pain accompany the limited movement. Femoral and humeral shaft fractures are usually mid-shaft and treated with splinting. Healing and complete recovery are expected within 2 to 4 weeks without incident (Laroia, 2004).

Brachial Plexus Injury

Brachial plexus injury occurs most often in large newborns, frequently with shoulder dystocia or in breech births. These injuries are relatively common, complicating between 1 in 500 and 1 in 1,000 term births (Cunningham et al., 2005). Most cases involve *Erb's palsy,* in which cervical nerves 5 and 6 are damaged, leading to paralysis of the upper portion of the arm. Injury to the upper plexus results from stretching or pulling the head away from the shoulder during a difficult birth. The involved extremity lies adducted, prone, and internally rotated. Shoulder movement is absent. Immobilization of the arm across the upper chest protects the shoulder from excessive motion. Gentle passive range-of-motion exercises are performed daily to prevent contractures.

Injury to the lower brachial plexus is less common, but *Klumpke's palsy* can occur, resulting in hand and wrist weakness. The grasp reflex is absent on assessment. The hand is placed in a neutral position and passive range-of-motion exercises are used.

Cranial Nerve Trauma

Pressure on the facial nerve before birth or from the use of obstetric forceps may cause a transient palsy to the face. It results in facial asymmetry, especially during crying. The mouth is drawn toward the unaffected side, wrinkles are deeper on the unaffected side, and the eye is persistently open on the affected side. Most newborns begin to recover within the first week, but full resolution may take several months. The open eye is protected with a patch and artificial tears are instilled frequently. Parents need instruction about how to feed the newborn, since he or she cannot close the lips around the nipple without having milk seep out. Parents also need reassurance that most facial trauma is self-limiting and resolves in a short time.

Head Trauma

Mild trauma can cause soft tissue injuries such as cephalhematoma and caput succedaneum. Greater trauma can cause depressed skull fractures. *Cephalhematoma* is a subperiosteal collection of blood secondary to the rupture of blood vessels between the skull and periosteum. The suture lines delineate its extent. Cephalhematoma typically is located on one side over the parietal bone. It occurs in 2.5% of all births and typically appears within hours after birth (Cunningham et al., 2005). Resolution occurs gradually over 2 to 3 weeks without treatment (see Chapter 18).

Caput succedaneum is a soft tissue swelling caused by edema of the head against the dilating cervix during the birth process. It is not limited by suture lines, extends across the midline, and is associated with head molding. It does not usually cause complications other than a misshaped head. The swelling from caput succedaneum is maximal at birth, rapidly decreases in size, and usually resolves over the first few days without treatment (see Chapter 18).

Subarachnoid hemorrhage is probably one of the most common types of intracranial trauma. Hypoxia/ischemia, variations in blood pressure, and the pressure exerted on the head during labor are major causes of this type of intracranial hemorrhage. Bleeding is of venous origin, and underlying contusions also may occur (Haws, 2004). Some red blood cells may appear in the cerebrospinal fluid of full-term newborns. Newborns with subarachnoid hemorrhage may present with apnea, seizures, lethargy, or abnormal findings on a neurologic examination (Becske & Jallo, 2004). Minimal handling to reduce stress is important.

Subdural hemorrhage (hematomas) occurs less often today because of improved obstetric techniques. Typically, tears of the major veins or venous sinuses overlying the cerebral hemispheres or cerebellum are the cause. Such tears are most common in newborns of primaparas, in large newborns, or after an instrumented birth. All of these conditions can produce increased pressure on the blood vessels inside the skull, leading to tears. The newborn with a subdural hemorrhage can be asymptomatic or can exhibit seizures, enlarging head size, decreased level of consciousness, or abnormal findings on a neuro-

logic examination, with hypotonia, a poor Moro reflex, or extensive retinal hemorrhages (Lynam & Verklan, 2004). A subdural hematoma can be life-threatening if it is in an inaccessible location and cannot be aspirated (Whitby et al., 2003).

Depressed skull fractures, although rare, may result from the pressure of a forceps delivery but can also occur during spontaneous or cesarean births. They also may be associated with other head trauma causing subdural bleeding, subarachnoid hemorrhage, or brain trauma (Laroia, 2004). Depressed skull fractures can be observed and palpated as depressions. Confirmation via x-ray is necessary. Neurosurgical consultation is typically needed.

Nursing Management

Recognition of trauma and birth injuries is imperative so that early treatment can be initiated. The nurse needs to complete a careful physical and neurologic assessment of every newborn admitted to the nursery to establish whether injuries exist. Assess and document symmetry of structure and function thoroughly. Be prepared to assist with scheduling diagnostic studies to confirm trauma or injuries, which will be important in determining treatment modalities.

Provide parents with a realistic picture of the situation to gain their understanding and trust. Be readily available to answer questions and teach them how to care for the newborn, including any modifications that might be necessary. Allow parents adequate time to understand the implications of the birth trauma or injury and what treatment modalities are needed, if any. Spending time with the parents and providing them with support, information, and teaching are important to allow them to make decisions and care for their newborn. Anticipate the need for community referral for ongoing follow-up and care.

ConsiderTHIS!

I admit, I had led a reckless life since I was a teen. I rebelled against my mother's authority and started smoking and doing drugs just to "check out" of my painful world. It was one big blast after another with a high and then a low. I never considered the consequences of my behavior then and never thought it would hurt anyone until I learned I was about 4 months pregnant. I convinced myself that if I cut back, everything would be fine.

Now, as I stand here in the NICU watching my tiny son struggle for air and tremble all over, I am not so convinced that I didn't hurt anyone except myself. As I witness my son fight against MY nicotine and drug addiction, my heart is heavy with guilt. I wonder how I could have thought that my troubles wouldn't become another's plight sooner or later. What must I have been thinking to isolate my addiction and not consider the impact that it would have on my mother and my son?

Thoughts: This woman honestly regrets what her addiction has done to her son as she stands watching him go through withdrawal. Her lifestyle choices do affect others, despite her previous denial. One problem with addiction is the difficulty in getting help after deciding to finally quit. There aren't enough rehab centers to deal with the large numbers needing their services and it can be difficult to get into one. What can be offered to pregnant women who abuse substances? How can nurses increase community awareness about the impact of this problem, especially during pregnancy?

Newborns of Substance-Abusing Mothers

It is generally assumed that all pregnant women want to provide a healthy environment for their unborn child and know how to avoid harmful consequences. However, for women who use substances such as drugs or alcohol, this may not be the case. Substance use during pregnancy exposes the fetus to the possibility of IUGR, prematurity, neurobehavioral and neurophysiologic dysfunction, birth defects, infections, and long-term developmental sequelae (Verklan & Walden, 2004).

It is difficult to establish the true prevalence of substance use in pregnant women: many women deny taking any nonprescribed substance because of the associated social stigma and legal implications. The National Institute on Drug Abuse (NIDA) suggests that approximately 1 in 10 infants are exposed to one or more mood-altering drugs in utero (NIDA, 2005). Drug exposure may go unrecognized in these infants, and they may be discharged from the newborn nursery at risk for a complex of medical and social problems, including abuse and neglect.

Tobacco, alcohol, and marijuana are the most commonly abused substances during pregnancy; others are highlighted in Box 24-2.

Substance abuse during pregnancy is the subject of much controversy. The timing of drug ingestion usually determines the type and severity of damage to the fetus. Frequently, the woman uses more than one substance, which compounds the problem. Nurses must be knowledgeable about the issues of substance abuse and must be alert for opportunities to identify, prevent, manage, and educate women and families about this key public health issue.

Alcohol

The consumption of alcohol in the United States is pervasive and widely accepted. Alcohol use, abuse, and addiction affect all levels of our society. If a drug or substance is sold to the public without restrictions, there is a common misconception that it is safe. However, a "safe" level of alcohol consumption during pregnancy has yet to

BOX 24-2

SUBSTANCES OFTEN ABUSED DURING PREGNANCY

Opioids
- Morphine
- Codeine
- Methadone
- Meperidine (Demerol)
- Heroin

CNS Stimulants
- Amphetamines
- Cocaine

CNS Depressants
- Barbiturates
- Diazepam (Valium)
- Sedative–hypnotics

Hallucinogens
- LSD
- Inhalants
- Glue, paint thinner, nail polish remover
- Nitrous oxide (NIDA, 2005)

be established. Therefore, the prudent choice for women who are or may become pregnant is to abstain from alcohol entirely.

The adverse effects of alcohol consumption have been recognized for centuries, but the associated pattern of fetal anomalies was not labeled until the early 1970s. The distinctive pattern identified three specific findings: growth restriction (prenatal and postnatal), craniofacial structural anomalies, and central nervous system (CNS) dysfunction. These distinctive findings were called fetal alcohol syndrome, characterized by physical and mental disorders that appear at birth and remain problematic throughout the child's life. However, there are also circumstances in which effects of prenatal alcohol exposure are apparent, but the newborn does not meet all of the criteria. In an attempt to include those who do not meet the strict criteria, the terms fetal alcohol effects, alcohol-related birth defects, and alcohol-related neurologic defects are used to describe children with a variety of problems thought to be related to alcohol consumption during pregnancy. The Institutes of Medicine coined the term fetal alcohol spectrum disorder as a way of describing the broader effects of prenatal alcohol exposure. Children with fetal alcohol syndrome are at the severe end of the spectrum (Weiner, 2005). Newborns with some but not all of the symptoms of fetal alcohol syndrome are described as having **alcohol-related birth defects.** Fetal alcohol effects may include such problems as low birthweight, developmental delays, and hyperactivity. Box 24-3 summarizes the manifestations of fetal alcohol syndrome.

Worldwide, the incidence of fetal alcohol syndrome is 1 to 3 cases per 1,000 live births, and that of fetal alcohol effects is 3 to 5 per 1,000 live births (March of Dimes, 2005). Current estimates indicate that approximately 13% of women of childbearing age are either problem drinkers or alcoholics; therefore, the number of fetuses exposed to alcohol during utero increases dramatically (March of Dimes, 2005).

Fetal alcohol syndrome is one of the most common known causes of mental retardation, and it is the only cause that is entirely preventable. The effects last a lifetime. Children with this syndrome have varying degrees of psychological and behavioral problems and often find it difficult to hold a job and live independently (CDC, 2005).

Decreasing or eliminating alcohol consumption during pregnancy is the only way to prevent fetal alcohol syndrome and fetal alcohol effects. Unfortunately, few treatment programs address the needs of pregnant women, so many newborns are exposed to alcohol in utero.

Women who are pregnant or are planning to become pregnant need to be informed of the detrimental effects

BOX 24-3

CLINICAL PICTURE OF FETAL ALCOHOL SYNDROME

- Microcephaly (head circumference <10th percentile)*
- Small palpebral (eyelid) fissures*
- Abnormally small eyes
- Maxillary hypoplasia (flattened or absent)
- Epicanthal folds (folds of skin of the upper eyelid over the eye)
- Thin upper lip*
- Missing vertical groove in median portion of upper lip*
- Short upturned nose
- Short birth length and low birthweight
- Joint and limb defects
- Altered palmar crease pattern
- Prenatal or postnatal growth ≤10th percentile*
- Congenital cardiac defects (septal defects)
- Delayed fine and gross motor development
- Poor eye–hand coordination
- Clinically significant brain abnormalities*
- Mental retardation
- Narrow forehead
- Performance substantially below expected level in cognitive or developmental functioning, executive or motor functioning, and attention or hyperactivity; social or language skills*
- Inadequate sucking reflex and poor appetite (March of Dimes, 2005)

*Diagnostic criteria for fetal alcohol syndrome.

of alcohol during pregnancy. Educate women using a nonjudgmental, culturally connected approach. There is little debate today about drinking and pregnancy—*there is no safe time to drink, nor is there a safe amount of alcohol to drink.*

Tobacco and Nicotine

The tobacco industry recently admitted that nicotine is addictive. In the United States, approximately one in four women smoke. Although the incidence of smoking in the United States is declining, more women than men now smoke. Currently, at least 11% of women in the United States smoke during pregnancy (March of Dimes, 2005). The risks of smoking, such as cancer and cardiovascular and pulmonary disease, are widely known, and smoking during pregnancy places the mother and fetus at additional risk.

Cigarette smoke contains more than 2,500 chemicals. The active constituents of cigarette smoke are nicotine, tar, carbon monoxide, and cyanide. It is not known for certain which of these chemicals are harmful to a developing fetus, but both nicotine and carbon monoxide are believed to play a role in causing adverse pregnancy outcomes. Nicotine crosses the placenta and carbon monoxide combines with hemoglobin, impairing oxygenation for the mother and the fetus. Nicotine is highly addictive and provides an almost immediate "kick" because it causes a discharge of epinephrine from the adrenal cortex. This stimulation is then followed by depression and fatigue, leading the abuser to seek more nicotine.

The fetus of a woman who smokes is at risk for low birthweight (the risk almost doubles), small-for-gestational-age status, and preterm birth. The risk for sudden infant death syndrome (SIDS) increases, as does the risk for chronic respiratory illness (MWH, 2005).

Women smoke for many reasons and are influenced by both external and internal factors. External factors include social and cultural norms and the smoking behavior of people with whom the woman lives and works. Internal factors include stress, addiction, boredom, weight control, and the need for a coping mechanism. Some groups of women are more likely than others to smoke: for example, women who are single, separated, or divorced and women with less education, lower incomes, and lower-status occupations than the norm have higher rates of smoking (CDC, 2004).

Smokers who are contemplating pregnancy are faced with a dilemma. On the one hand, they wish a healthy outcome for the fetus; however, smoking is an addiction that is difficult to break. Nurses play a major role in teaching women about healthy behaviors and providing support for smoking cessation. Assisting the woman in smoking cessation requires a thorough consideration of all the factors associated with the woman's smoking and the challenges she faces—for example, why she smokes, the stressors in her life, and her social support network. Options to explore include group smoking cessation programs, relaxation techniques, individual counseling, hypnosis, and partner-support counseling. The major motivation is the woman's desire to change.

Nurses can be instrumental in increasing the number of pregnant women who make a serious attempt to quit smoking by using the "5 A's" approach:

- *Ask:* Ask all women if they smoke and would like to quit.
- *Advise:* Encourage the use of clinically proven treatment plans.
- *Assess:* Provide motivation by discussing the 5 R's:
  - Relevance of quitting to the woman
  - Risk of continued smoking to the fetus
  - Rewards of quitting for both
  - Roadblocks to quitting
  - Repeat at every visit
- *Assist:* Help the woman to protect her fetus and newborn from the negative effects of smoking.
- *Arrange:* Schedule follow-up visits to reinforce the woman's commitment to quit.

Health-promotion activities for smoking cessation are also important, such as joining with other community organizations and private-sector partners to reduce tobacco use and improve the health of newborns (Murray & Wewers, 2004).

Marijuana

Marijuana remains the most widely used illicit psychoactive substance in the Western world and the most commonly used illicit drug in the United States (NIDA, 2005). Marijuana, derived from the *Cannabis sativa* plant, has not been shown to have a teratogenic effect on the fetus, and no consistent types of malformations have been identified. However, marijuana does have significant effects during pregnancy. Similar to tobacco smoking, the carbon monoxide in the smoke will be delivered to the fetus, and thus IUGR is common in heavy marijuana smokers (NIDA, 2005). Mothers who smoke marijuana during pregnancy risk having a smaller infant. Research has shown that infants born to mothers who smoked marijuana during their pregnancies have altered responses to visual stimuli, sleep-pattern abnormalities, photophobia, lack of motor control, hyperirritability, increased tremulousness, and a high-pitched cry, which may indicate neurologic problems in development. Long-term effects on childhood development have not been established; research continues (NIDA, 2005).

Methamphetamines

Methamphetamine is an addictive stimulant drug that releases high levels of the neurotransmitter dopamine, which stimulates brain cells, enhancing mood and body

movement. Methamphetamine is made in illegal laboratories and has a high potential for abuse and addiction. Street methamphetamine is called by many names, such as "speed," "meth," "ice," and "chalk." It can be inhaled, injected, smoked, or taken orally (NIDA, 2005). Methamphetamines are used medically to treat obesity and narcolepsy in adults and hyperkinetic children.

Methamphetamines accelerate heart and respiratory rate, elevate blood pressure, and dilate the pupils of the eyes. A secondary effect, loss of appetite, has contributed to their use in the weight-loss industry. The illicit use of amphetamines during pregnancy has received relatively little research interest because it is less common than cocaine and narcotic use.

Fetal effects from methamphetamines are similar to the effects of cocaine (see below), suggesting vasoconstriction as a mechanism. Because they suppress appetite during pregnancy, maternal malnutrition may lead to problems with fetal growth and development. Infants born to mothers dependent on amphetamines have an increased risk of preterm births and low birthweight. Also, these infants may experience symptoms of withdrawal, as manifested by dysphoria, agitation, jitters, poor weight gain, abnormal sleep patterns, poor feeding, frantic fist sucking, high-pitched cry, respiratory distress soon after birth, frequent infections, and significant lassitude (Pitts, 2004). The long-term effects of methamphetamines have not been documented.

Cocaine

Cocaine is a strong CNS stimulant that interferes with the reabsorption of dopamine, a neurotransmitter associated with pleasure and movement. Physical effects of cocaine use include constricted blood vessels, dilated pupils, and increased temperature, heart rate, and blood pressure. It can be taken orally, sublingually, intranasally, intravenously, or by inhalation (Pitts, 2004).

The abuse of cocaine has become an alarming problem during the past decade. It is estimated that up to 8 million Americans use cocaine regularly, and 30% to 40% of cocaine addicts are women (NIDA, 2005). Maternal use of cocaine during pregnancy remains a significant public health problem, particularly in urban areas of the United States and among women of low socioeconomic status (March of Dimes, 2005). Cocaine exposure is associated with premature birth and lower birthweight, but its impact on later development is less clear. It is speculated that maternal cocaine use may interfere with the infant's cognitive development, resulting in learning and memory difficulties later in life (Messinger et al., 2004). Several congenital anomalies have also been associated with maternal cocaine use, including genitourinary anomalies, cardiac and CNS defects, and "prune belly syndrome" (absence of abdominal muscles at birth) (Cunningham et al., 2005).

Cocaine is a psychomotor stimulant and when used in low doses produces an increase in pleasure, alertness, and a sense of well-being. It lowers anxiety and social inhibitions. It can be ingested in several ways: intranasally by snorting, intravenously, and by smoking. Users soon discover that if they take more cocaine or "crack," the intensity of euphoria increases (NIDA, 2005).

It can be difficult to determine the effects of cocaine on the newborn due to the likelihood that the mother using cocaine also takes other drugs. Two major side effects of cocaine use—vasoconstriction and hypertension—are responsible for most of the fetal and newborn effects. Characteristics of cocaine-exposed newborns include:

- Prematurity
- Low birthweight
- Smaller head circumference
- A piercing cry (indicative of neurologic dysfunction)
- Genitourinary tract abnormalities
- Cardiac anomalies
- Limb defects
- Ambiguous genitalia
- Poor feeding
- Poor visual and auditory responses
- Poor sleep patterns
- Decreased impulse control
- Stiff, hyperextended positioning
- Irritability and hypersensitivity (hard to console when crying)
- Inability to respond to caretaker
- Higher incidence of SIDS (Pitts, 2004)

Cocaine-exposed newborns are typically fussy, irritable, and inconsolable at times. Techniques such as swaddling, gentle rocking, using a flexed position, and offering a pacifier can help manage CNS irritation. Keeping environmental stimuli to a minimum will also help. Cocaine-exposed infants demonstrate poor coordination of sucking and swallowing, making feeding time frustrating for the newborn and caregiver alike. A calm, gentle approach combined with proper positioning and handling will increase feeding success.

Heroin

Heroin is an illegal, highly addictive opiate derived from morphine. The white or brown powder can be sniffed, smoked, or injected. The medical and social consequences of its abuse, such as HIV infection, tuberculosis, fetal effects, crime, violence, and disruption of families, have a devastating impact on society, costing billions of dollars each year (NIDA Research Report, 2005). Heroin produces a sense of euphoria and readily crosses the placenta in the pregnant woman.

Heroin addiction during pregnancy poses serious health risks to the mother and the fetus. Heroin causes

severe physical addiction. It is a CNS depressant that produces mental dullness and drowsiness. Although no teratogenic effects have been associated with its use, newborns of heroin-addicted mothers are born dependent on heroin. In addition, pregnant women who share needles are at risk for contracting hepatitis B and C and HIV, which can be transmitted to the newborn (Cunningham et al., 2005). The rates of stillbirth, IUGR, prematurity, and newborn mortality are three to seven times higher in heroin-addicted pregnant women than in women in the general population (NIDA, 2005).

The effects of heroin on the newborn include low birthweight, meconium aspiration secondary to hypoxia, a high incidence of SIDS, and delayed effects from subacute withdrawal, with symptoms such as restlessness, continual crying, agitation, sneezing, vomiting, fever, diarrhea, seizures, irritability, and poor socialization that may persist for 4 to 6 months (March of Dimes, 2005).

In general, abrupt cessation of the use of heroin or any opiate during pregnancy is not advised because intrauterine death or prematurity may result. Treatment for heroin-dependent mothers consists of methadone maintenance. Women who enroll in a methadone maintenance program often have a good outcome, including a longer gestation time and increased birthweight, compared to women who go untreated (March of Dimes, 2005). Most newborns of mothers who abuse heroin begin withdrawal within 24 to 72 hours as opposed to a week after birth for infants whose mothers have been maintained on methadone (Pitts, 2004). Treatment is supportive for the newborn experiencing heroin withdrawal.

Methadone

Methadone is a synthetic opiate narcotic that is used primarily as maintenance therapy for heroin addiction. It blocks the effects of heroin for about 24 hours. When combined with prenatal care and a comprehensive drug treatment program, many of the detrimental maternal and newborn outcomes associated with heroin abuse can improve. There is preliminary evidence that buprenorphine (Buprenex) also is safe and effective in treating heroin dependence during pregnancy, although infants exposed to methadone or buprenorphine during pregnancy typically require treatment for withdrawal symptoms (NIDA Research Report, 2005).

Although methadone poses some threat to the fetus, it is important to weigh the benefits of methadone in pregnancy against the risks associated with the continuing use of heroin. For this reason, methadone maintenance is often recommended for pregnant heroin-dependent women, and it remains the standard of care for treatment of women with narcotic addiction.

Methadone maintenance is advantageous because it provides the woman with an alternative to an illicit substance. This substance can be monitored by a medical team, which helps her to stay heroin-free. It also helps to promote compliance with prenatal care: the woman is given a prescribed dose and must return on a consistent basis to receive more. Other advantages include improved fetal and newborn growth, a reduced risk of fetal death (maternal opiate withdrawal can cause fetal death or pregnancy loss), and a reduced risk of HIV infection because the woman no longer needs to engage in the high-risk behaviors involved in obtaining illegal drugs. The woman on methadone maintenance also can breastfeed (Weiner, 2005).

Despite the significant advantages of methadone to a heroin-dependent pregnant woman, dangers to the fetus and newborn remain. In utero exposure to methadone may lead to low birthweight caused by symmetric fetal growth restriction involving weight, length, and head circumference. Withdrawal from methadone is more severe than from heroin and more prolonged, possibly lasting up to 3 weeks due to the much longer half-life of methadone. Also, seizures attributed to withdrawal are commonly severe and may not occur until 2 to 3 weeks of age, after the newborn has been discharged and is home without medical supervision. Finally, the rate of SIDS among opiate-exposed infants is three to four times higher than for the general population (Cunningham et al., 2005). Nurses play a major role in teaching mothers and caregivers to monitor the newborn for methadone withdrawal symptoms after returning home.

Neonatal Abstinence Syndrome

Newborns of women who abuse tobacco, illicit substances, caffeine, and alcohol can exhibit withdrawal behavior. Withdrawal symptoms occur in 60% of all newborns exposed to drugs (Wang, 2004). Drug dependency acquired in utero is manifested by a constellation of neurologic and physical behaviors and is known as **neonatal abstinence syndrome.** Although often treated as a single entity, neonatal abstinence syndrome is not a single pathologic condition. Manifestations of withdrawal are a function of the drug's half-life, the specific drug or combination of drugs used, dosage, route of administration, timing of drug exposure, and length of drug exposure (Engstrom, 2004). Neonatal abstinence syndrome has both medical and developmental consequences for the newborn.

The newborn's behavior often prompts the healthcare provider or nurse to suspect intrauterine drug exposure (Box 24-4). The newborn physical examination may also reveal low birthweight for gestational age or drug or alcohol-related birth defects and dysfunction.

To remember the symptoms of neonatal abstinence syndrome, use the acronym WITHDRAWAL:

W = Wakefulness: sleep duration less than 1 to 3 hours after feeding

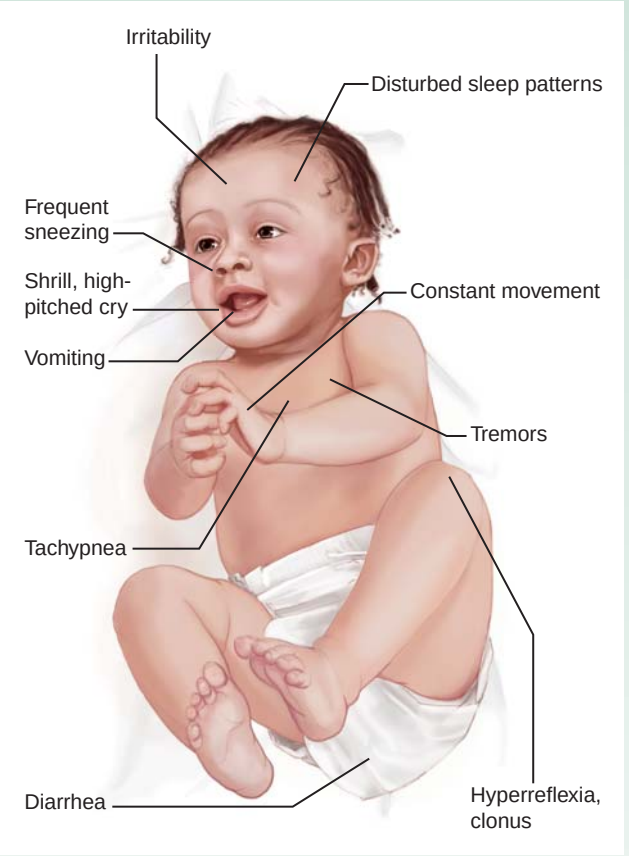
BOX 24-4

MANIFESTATIONS OF NEONATAL ABSTINENCE SYNDROME

CNS Dysfunction
- Tremors
- Generalized seizures
- Hyperactive reflexes
- Restlessness
- Hypertonic muscle tone, constant movement
- Shrill, high-pitched cry
- Disturbed sleep patterns

Metabolic, Vasomotor, and Respiratory Disturbances
- Fever
- Frequent yawning
- Mottling of the skin
- Sweating
- Frequent sneezing
- Nasal flaring
- Tachypnea >60 bpm
- Apnea

Gastrointestinal Dysfunction
- Poor feeding
- Frantic sucking or rooting
- Loose or watery stools
- Regurgitation or projectile vomiting (Belik & Al-Hamad, 2004)



I = Irritability
T = Temperature variation, tachycardia, tremors
H = Hyperactivity, high-pitched persistent cry, hyperreflexia, hypertonus
D = Diarrhea, diaphoresis, disorganized suck
R = Respiratory distress, rub marks, rhinorrhea
A = Apneic attacks, autonomic dysfunction
W = Weight loss or failure to gain weight
A = Alkalosis (respiratory)
L = Lacrimation (AAP Committee on Substance Abuse, 2005)

Diagnostic studies to help identify the severity of withdrawal include various scoring systems to rate the infant's withdrawal behaviors. Toxicology screening of the newborn's blood, urine, and meconium helps to identify the substances to which the newborn has been exposed. In general, a urine screen signifies only recent use of drugs: it can detect marijuana use up to a month earlier, cocaine use up to 96 hours earlier, heroin use 24 to 48 hours earlier, and methadone use up to 10 days earlier (Wang, 2004).

Nursing Management

Management of the substance-exposed newborn remains a major challenge to health care professionals. The major goals include providing comfort to the newborn by relieving symptoms, improving feeding and weight gain, preventing seizures, promoting mother–newborn interactions, and reducing the incidence of newborn mortality and abnormal development (Belik & Al-Hamad, 2004).

Pharmacologic treatment is warranted if conservative measures, such as swaddling and decreased environmental stimulation, are not adequate. The AAP recommends that for newborns with confirmed drug exposure, drug therapy should be indicated if the newborn has seizures, diarrhea and vomiting resulting in excessive weight loss and dehydration, poor feeding, inability to sleep, and fever unrelated to infection (AAP Committee on Substance Abuse, 2005). Common medications used in the management of newborn withdrawal include morphine, paregoric, phenobarbital, tincture of opium, methadone clonidine, chlorpromazine, and diazepam (Wang, 2004).

For a substance-abusing mother, the birth of a drug-exposed newborn is both a crisis and an opportunity. The mother may feel guilty about the newborn's condition. Many of these newborns are unresponsive and disorganized in sleeping and feeding patterns. When awake, they can be easily overstimulated and irritated. Such characteristics make parent–newborn interaction difficult and frustrating, leading to possible detachment and avoidance (Pitts, 2004). The mother may be single and a victim of physical and sexual abuse and may have a limited support system. Many of these mothers had poor parenting themselves, lack information about characteristic infant behaviors, and have unrealistic expectations about the newborn's abilities (Ballard, 2002). On the other hand, the newborn may be a powerful motivator for the mother to undergo treatment and seek recovery. Nurses can play a pivotal role in assisting her to abstain from drug use and become a good mother to her newborn.

Assessment

Several assessment tools can be used to assess a drug-exposed newborn. Figure 24-4 shows an example. Key areas to assess include:

- Maternal history to identify risk behaviors for substance abuse:
  - Previous unexplained fetal demise
  - Lack of prenatal care
  - History of missed prenatal appointments
  - Severe mood swings
  - Precipitous labor
  - Poor nutritional status
  - Abruptio placentae
  - Hypertensive episodes
  - History of drug abuse
- Laboratory test results (toxicology) to identify substances in mother and newborn
- Signs of neonatal abstinence syndrome (use the "WITHDRAWAL" acronym)
- Evidence of seizure activity and need for protective environment

Nursing Interventions

Nursing interventions focus on promoting a calming, supportive environment. Decrease stimuli by dimming the lights in the nursery, and swaddle the newborn tightly to decrease irritability behaviors. Plan activities to allow for minimal stimulation of the newborn. Use a pacifier to satisfy needs for nonnutritive sucking.

When feeding the newborn, use small amounts and position the newborn upright to prevent aspiration and to facilitate rhythmic sucking and swallowing. Breastfeeding is encouraged unless the mother is still using drugs. Monitor the newborn's weight daily to evaluate success of food intake. Assess hydration; check skin turgor and fontanels. Monitor the newborn's fluid and electrolyte and acid–base status.

In addition, implement the following interventions:

- Assess the frequency and characteristics of bowel movements.
- Monitor for any changes in condition or signs.
- Protect the neonate's elbows and knees against friction and abrasions.
- Administer ordered medications and document behavioral changes.
- Teach the mother or caretaker how to care for the newborn at home (Teaching Guidelines 24-1).
- Refer the mother to community agencies to address addiction and the infant's developmental needs (Green & Wilkinson, 2004).

The needs of the substance-exposed newborn are multiple, complex, and costly, both to the healthcare system and to society. Substance abuse takes place among people of all colors, sizes, shapes, incomes, types, and conditions. Most pregnant women are unaware of the adverse impact their substance abuse can have on the newborn.

Nurses are in a unique position to help because they interact with high-risk mothers and newborns in many settings, including the community, healthcare facilities, and family agencies. It is the responsibility of all nurses to identify, educate, counsel, and refer pregnant women with substance-abusing problems. Early, supportive, ongoing nurse care is critical to the well-being of the mother and her newborn.

Hyperbilirubinemia

Hyperbilirubinemia is a total serum bilirubin level above 5 mg/dL resulting from unconjugated bilirubin being deposited in the skin and mucous membranes (Mattson & Smith, 2004). Hyperbilirubinemia is exhibited as jaundice. Newborn jaundice is one of the most common reasons for hospital readmission. It occurs in 60% to 80% of term newborns in the first week of life and in virtually all preterm newborns (Madan et al., 2004).

Pathophysiology

Newborn jaundice results from an imbalance in the rate of bilirubin production and bilirubin elimination. This relative imbalance determines the pattern and degree of newborn hyperbilirubinemia (Dixon, 2004).

During the newborn period, a rapid transition from the intrauterine to the extrauterine pattern of bilirubin physiology occurs. Fetal unconjugated bilirubin is normally cleared by the placenta and the mother's liver in utero, so total bilirubin at birth is low. After the umbilical cord is cut, the newborn must conjugate bilirubin (convert a lipid-soluble pigment into a water-soluble pigment) in the liver on his or her own. The rate and amount of bilirubin conjugation depend on the rate of red blood cell breakdown, the bilirubin load, the maturity of the liver, and the number of albumin-binding sites (Olds et al., 2004). Bilirubin production increases after birth mainly because of a shortened

CENTRAL NERVOUS SYSTEM DISTURBANCES													
SIGNS AND SYMPTOMS	SCORE	AM							PM				
Excessive high-pitched cry	2												
Continuous high-pitched cry	3												
Sleeps <1 hour after feeding	3												
Sleeps <2 hours after feeding	2												
Sleeps <3 hours after feeding	1												
Hyperactive Moro reflex	2												
Markedly hyperactive Moro refex	3												
Mild tremors disturbed	1												
Moderate–severe tremors disturbed	2												
Mild tremors undisturbed	1												
Moderate–severe tremors undisturbed	4												
Increased muscle tone	2												
Excoloration (specify area)	1												
Myoclonic jerks	3												
Generalized convulsions	5												
METABOLIC / VASOMOTOR/RESPIRATORY DISTURBANCES													
Sweating													
Fever <101 (99–100.8°F/37.2–38.2°C)	1												
Fever <101 (38.2°C and higher)	2												
Frequent yawning (>3– 4 times/interval)	1												
Mottling	1												
Nasal stuffiness	1												
Sneezing (>3–4 times/interval)	1												
Nasal flaring	2												
Respiratory rate >60 / min	1												
Respiratory rate >60 / min, with retractions	2												
GASTROINTESTINAL DISTURBANCES													
Excessive sucking	1												
Poor feeding	2												
Regurgitation	2												
Projectile vomiting	3												
Loose stools	2												
Watery stools	3												
TOTAL SCORE													

● Figure 24-4 Neonatal abstinence scoring system. (From Cloherty, J. P. & Stark, A. R. [1998]. *Manual of neonatal care* [4th ed., pp. 26–27]. Boston: Little, Brown.)

TEACHING GUIDELINES 24-1

Caring for Your Newborn at Home

- Position your newborn with the head elevated to prevent choking.
- To aid your newborn's sucking and swallowing during feeding, position the chin downward and support it with your hand.
- Place your newborn on his or her back to sleep or nap, never on the stomach.
- Keep a bulb syringe close by to suction your newborn's mouth in case of choking.
- Cluster newborn care (bathing, feeding, dressing) to prevent overstimulation.
- If your newborn is fussy or crying, try these measures to help calm him or her:
  - Wrap your newborn snugly in a blanket and rock in rocking chair.
  - Take the baby for a ride in the car (using a newborn car seat).
  - Play soothing music and "dance" with the newborn.
  - Use a wind-up swing with music.
- To help your newborn get to sleep, try these measures:
  - Schedule a bath with a gentle massage prior to bedtime.
  - Change diaper and clothes to make the baby comfortable.
  - Feed the baby just prior to bedtime.
  - If the newborn cries when put in crib and all needs are met, allow him or her to cry.
  - Use a rocking chair to feed and sing a soft lullaby.
- Call your primary care provider if you observe withdrawal behaviors such as:
  - Slight tremors (shaking) of hands and legs
  - Stiff posture when held in your arms
  - Irritable and frequently fussy
  - High-pitched cry, excessive sucking motions
  - Erratic sleep pattern
  - Frequent yawning, nasal stuffiness, sweating
  - Prolonged feeding time needed
  - Frequent vomiting after feeding

red blood cell lifespan (70 days vs. 90 days in the adult) combined with an increased red blood cell mass. Therefore, the amount of bilirubin the newborn must deal with is large when compared to that of an adult. Additional risk factors contributing to newborn jaundice include:

- Polycythemia
- Significant bruising or cephalhematoma, which increases bilirubin production
- Infections such as TORCH (toxoplasmosis, hepatitis B, rubella, cytomegalovirus, herpes simplex virus)
- Use of drugs during labor and birth such as diazepam (Valium) or oxytocin (Pitocin)
- Prematurity
- Gestational age of 34 to 36 weeks
- Hemolysis due to ABO incompatibility or Rh isoimmunization
- Macrosomic infant of a diabetic mother
- Delayed cord clamping, which increases the erythrocyte volume
- Decreased albumin binding sites to transport unconjugated bilirubin to the liver because of acidosis
- Delayed meconium passage, which increases the amount of bilirubin that returns to the unconjugated state and can be absorbed by the intestinal mucosa
- Siblings who had significant jaundice
- Inadequate breastfeeding leading to dehydration, decreased caloric intake, weight loss, and delayed passage of meconium
- Ethnicity, such as Asian-American, Mediterranean, or Native American
- Male gender (AAP, 2004)

Bilirubin has two forms—unconjugated or indirect, which is fat-soluble and toxic to body tissues, and conjugated or direct, which is water-soluble and nontoxic. Elevated serum bilirubin levels are manifested as jaundice in the newborn. Typically the total serum bilirubin level rises over the first 3 to 5 days and then declines.

Physiologic Jaundice

Physiologic jaundice is the manifestation of the normal hyperbilirubinemia seen in newborns, appearing during the third to fourth days of life, due to the limitations and abnormalities of bilirubin metabolism. It occurs in 60% of term infants and up to 80% of preterm infants (Blackburn, 2003). Serum bilirubin levels reach up to 10 mg/dL and then decline rapidly over the first week after birth (Cunningham et al., 2005). Most newborns have been discharged by the time this jaundice peaks (at about 72 hours).

Factors that contribute to the development of physiologic jaundice include an increased bilirubin load because of relative polycythemia, a shortened red blood cell lifespan, immature hepatic uptake and conjugation process, and increased enterohepatic circulation (Holcomb, 2005).

Physiologic jaundice differs between breast-fed and bottle-fed newborns in relation to the onset of symptoms. Breast-fed newborns typically have peak bilirubin levels on the fourth day of life; levels for bottle-fed newborns usually peak on the third day of life. The rate of bilirubin decline is less rapid in breast-fed newborns compared to bottle-fed newborns (Cunningham et al., 2005).

Jaundice associated with breastfeeding presents in two distinct patterns: early-onset breastfeeding jaundice and late-onset breast milk jaundice. Early-onset breastfeeding jaundice is probably associated with ineffective breastfeeding practices because of relative caloric deprivation in the first few days of life. Decreased volume and frequency of feedings may result in mild dehydration and

the delayed passage of meconium. This delayed defecation allows enterohepatic circulation reuptake of bilirubin and an increase in the serum level of unconjugated bilirubin. To prevent this, strategies to promote early effective breastfeeding are important. The AAP Work Group on Breastfeeding (2005) recommends early and frequent breastfeeding without supplemental water or dextrose-water unless medically indicated. Early frequent feedings can provide the newborn with adequate calories and fluid volume (via colostrum) to stimulate peristalsis and passage of meconium to eliminate bilirubin.

Late-onset breast milk jaundice occurs later in the newborn period, with the bilirubin level usually peaking in the 6th to 14th day of life. Total serum bilirubin levels may be 12 to 20 mg/dL, but the levels are not considered pathologic (Sarici et al., 2004). The specific cause of late-onset breast milk jaundice is not entirely understood, but it may be related to a change in the milk composition resulting in enhanced enterohepatic circulation. Additional research is needed to determine the cause. Interrupting breastfeeding is not recommended unless bilirubin levels reach dangerous levels; if this occurs, breastfeeding is stopped for only 1 or 2 days. Substituting formula during this short break usually results in a prompt decline of bilirubin levels.

Pathologic Jaundice

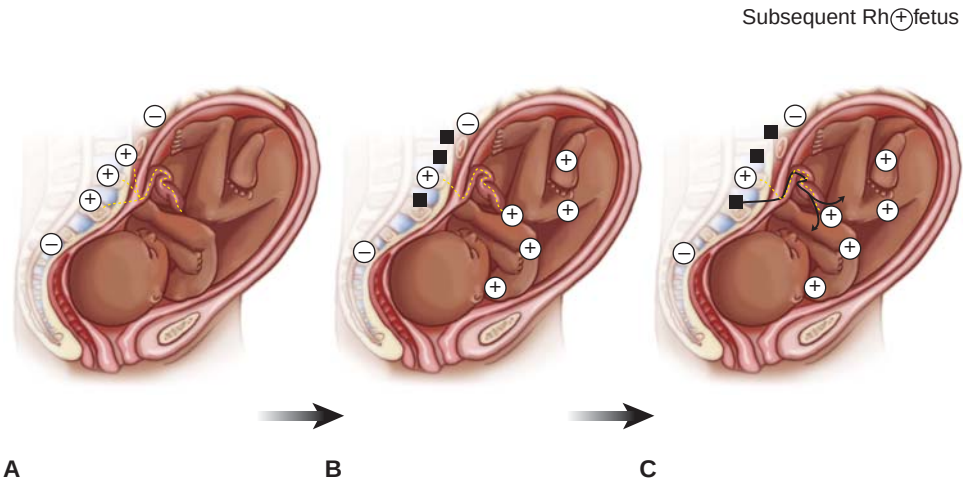
Pathologic jaundice is manifested within the first 24 hours of life when total bilirubin levels increase by more than 5 mg/dL/day and the total serum bilirubin level is higher than 17 mg/dL in a full-term infant (Ozen & Mukherjee, 2004). Conditions that alter the production, transport, uptake, metabolism, excretion, or reabsorption of bilirubin can cause pathologic jaundice in the newborn. A few conditions that contribute to red blood cell breakdown and thus higher bilirubin levels include polycythemia, blood incompatibilities, and systemic acidosis. These altered conditions can lead to high levels of unconjugated bilirubin, possibly reaching toxic levels and resulting in a severe condition called kernicterus.

Kernicterus (yellow nucleus) or bilirubin encephalopathy is a preventable neurologic disorder characterized by encephalopathy, motor abnormalities, hearing and vision loss, and death (Springer & Annibale, 2004). Neurotoxicity develops because unconjugated bilirubin has a high affinity for brain tissue, and bilirubin not bound to albumin is free to cross the blood–brain barrier and damage cells of the CNS.

In the acute stage, the newborn becomes lethargic, irritable, and hypotonic and sucks poorly. If the hyperbilirubinemia is not treated, the newborn becomes hypertonic, with arching and seizures. A high-pitched cry may be noted. These changes can occur rapidly, so all newborns must be assessed for jaundice and tested if indicated so that treatment can be initiated.

The most common condition associated with pathologic jaundice is hemolytic disease of the newborn secondary to incompatibility of blood groups of the mother and the newborn. The most frequent conditions are Rh factor and ABO incompatibilities. *Rh incompatibility* or isoimmunization develops when an Rh-negative woman who has experienced Rh isoimmunization subsequently becomes pregnant with an Rh-positive fetus. The maternal antibodies cross the placenta into the fetal circulation and begin to break down the red blood cells (Fig. 24-5). Destruction of the fetal red blood cells leads to fetal anemia and hemolytic disease of the newborn. The severity of the fetal hemolytic process depends on the level and effectiveness of anti-D antibodies and the capacity of the fetal system to remove antibody-coated cells.

Clinical manifestations of hemolytic disease of the newborn include ascites, congestive heart failure, edema, pallor, jaundice, hepatosplenomegaly, hydramnios, thick placenta, and dilation of the umbilical vein (Harrod et al., 2003). The jaundice typically manifests at birth or in the first 24 hours after birth with a rapidly rising unconjugated bilirubin level.



● Figure 24-5 Rh isoimmunization. (**A**) The Rh-negative mother is exposed to Rh-positive antigens. (**B**) Maternal antibodies form. (**C**) Rh antibodies are transferred to the fetus.

Immune hydrops, also called hydrops fetalis, is a severe form of hemolytic disease of the newborn that occurs when pathologic changes develop in the organs of the fetus secondary to severe anemia. Hydrops fetalis results from fetal hypoxia, anemia, congestive heart failure, and hypoproteinemia secondary to hepatic dysfunction. ABO and Rh incompatibilities can both cause hydrops fetalis, but Rh disease is the more common cause. Typically, hydrops is not observed until the hemoglobin drops below approximately 4 g/dL (hematocrit <15%) (Wagle & Deshpande, 2003). Fetuses with hydrops may die in utero from profound anemia and circulatory failure. The placenta is very enlarged and edematous. One sign of severe anemia and impending death is the sinusoidal fetal heart rate pattern (Cunningham et al., 2005). The newborn hydropic infant appears pale, edematous, and limp at birth and typically requires resuscitation.

The newborn with immune hydrops exhibits severe generalized edema, organ hypertrophy and enlargement, and effusion of fluid into body cavities. Intrauterine transfusions with Rh-negative, type O blood may be life-saving if done in time. The widespread administration of Rh immune globulin (RhoGAM), combined with aggressive fetal surveillance and transfusion, has reduced the incidence of hemolytic disease of the newborn.

ABO incompatibility is an immune reaction that occurs when the mother has type O blood and the fetus has type A, B, or AB blood. Although it occurs more frequently than Rh incompatibilities, it causes less severe problems and rarely results in hemolytic disease severe enough to be clinically diagnosed and treated. Enlargement of the spleen and liver may be found in newborns with ABO incompatibility, but hydrops fetalis is rare (Diehl-Jones & Askin, 2004).

Women with type O blood develop anti-A or anti-B antibodies throughout their life through foods they eat and exposure to infections. Most species of anti-A and anti-B antibodies are immunoglobin M (IgM), which cannot cross the placenta and thus cannot gain access to the fetal red blood cells. Some anti-A and anti-B antibodies from the mother may cross the placenta to the fetus during the first pregnancy and can cause hemolysis of fetal blood cells.

Clinically, the newborn may present with mild hemolysis, anemia, and hyperbilirubinemia. Very few newborns develop anemia or hepatosplenomegaly, and most often the elevated bilirubin levels respond to phototherapy after birth. Because the antibodies resulting in ABO incompatibility occur naturally, it is impossible to eliminate this type of incompatibility. Therefore, nurses need to be aware of this potential cause of hyperbilirubinemia.

Nursing Management

Nursing management of a newborn with hyperbilirubinemia requires a comprehensive approach. As members of the health care team, nurses share in the responsibility for early detection and identification, family education, proper management, and follow-up of the mother and newborn. It is important for nurses to obtain a complete, detailed history to identify factors that may place the infant at risk for hyperbilirubinemia. Nurses can help the mother to understand the diagnostic tests and treatment modalities by offering individualized teaching. Throughout the continuum of care, nurses can improve care by their presence and support.

Assessment

Review the medical record for factors that might predispose the newborn to hyperbilirubinemia. Identify maternal and fetal blood types, checking for possible incompatibilities (Table 24-3). Perform a complete physical examination.

Detect jaundice by observing the infant in a well-lit room and blanching the skin with digital pressure over a bony prominence. Inspect the eyes and mucous membranes for discoloration. Typically, jaundice begins on the head and gradually progresses to the abdomen and extremities. Jaundice in a term newborn fewer than 24 hours old is always pathologic and needs thorough investigation and prompt treatment to prevent kernicterus. Observe for pallor (anemia), excessive bruising (bleeding), and dehydration (sluggish circulation), which may

Table 24-3 Comparing Rh and ABO Incompatibility in the Newborn

Clinical Picture	Rh Incompatibility	ABO Incompatibility
First-born	Rare	Common
Later pregnancies	More severe	No increase in severity
Jaundice	Moderate to severe	Mild
Hydrops fetalis	Frequent	Rare
Anemia	Frequently severe	Rare
Ascites	Frequent	Rare
Hepatosplenomegaly	Frequent	Common

contribute to the development of jaundice and the risk for kernicterus.

Diagnostic tests that may be ordered to aid in diagnosing the condition include:

- Direct Coombs test—to identify hemolytic disease of the newborn; positive results indicate that the newborn's red blood cells have been coated with antibodies and thus are sensitized
- Blood type—to determine Rh status and any incompatibility of the newborn
- Total and direct bilirubin—establishes the diagnosis of hyperbilirubinemia
- Total serum protein—detects reduced binding capacity of albumin
- Reticulocyte count—an elevated level indicates increased hemolysis

Assist with obtaining blood specimens. Cord blood is used for hemoglobin concentration measurements; a heel stick is used for direct Coombs testing and bilirubin levels.

Nursing Interventions

For the newborn with jaundice, regardless of its etiology, phototherapy is used to convert unconjugated bilirubin to the less toxic water-soluble form that can be excreted. Phototherapy, via special lights placed above the newborn or a fiber-optic blanket placed under the newborn and wrapped around him or her, involves blue wavelengths of light to alter unconjugated bilirubin in the skin. The newborn is exposed to the lights continuously except for feeding. Only the newborn's eyes and genital area are covered to ensure exposure of the greatest surface area (Fig. 24-6).

If the total serum bilirubin level remains elevated after intensive phototherapy has been used, an exchange transfusion, the most rapid method for lowering serum bilirubin levels, may be necessary (Springer & Annibale, 2004). In the presence of hemolytic disease, severe anemia, or a rapid rise in the total serum bilirubin level, an exchange transfusion is recommended. Exchange transfusion removes the newborn's blood and replaces it with nonhemolyzed red blood cells from a donor. During the transfusion, the newborn cardiovascular status is continuously monitored because serious complications can arise, such as acid–base imbalances, infection, hypovolemia, and fluid and electrolyte imbalances. Therefore, exchange transfusion is used only as a second-line therapy after phototherapy has failed to yield results. Intensive nursing care is needed.

General nursing interventions for the newborn with jaundice related to hyperbilirubinemia include the following:

- Educate parents about jaundice and its potential risk by providing written and verbal material.
- Encourage the early initiation of feedings to prevent hypoglycemia and provide protein to maintain the albumin levels to transport bilirubin to the liver.

● Figure 24-6 Newborn receiving phototherapy.

- Ensure newborn feedings (breast or formula) every 2 to 3 hours to promote prompt emptying of bilirubin from the bowel.
- Encourage the mother to breastfeed (8 to 12 feedings per day) to prevent inadequate intake and thus dehydration.
- Supplement breast milk with formula to supply protein if bilirubin levels continue to increase with breastfeeding only.
- Show the parents how to identify newborn behaviors that might indicate rising bilirubin levels; urge them to seek treatment from their pediatrician:
  - Lethargic, sleepy, poor muscle tone, floppy
  - Poor suck, not interested in feeding
  - High-pitched cry
- Document the timing of onset of jaundice to differentiate between physiologic (>24 hours) and pathologic jaundice (<24 hours).
- Explore with the family their understanding of jaundice and treatment modalities to reduce anxiety and gain their cooperation in monitoring the infant.
- Monitor serum bilirubin levels to reduce the risk of developing severe hyperbilirubinemia.
- Emphasize appropriate follow-up with the primary care provider within 48 to 72 hours after discharge to assess jaundice status (AAP, 2004).

For the newborn receiving phototherapy, place the newborn under the lights or on the fiberoptic blanket, exposing as much skin as possible. Cover the newborn's genitals and shield the eyes to protect these areas from becoming irritated or burned. Assess the intensity of the light source to prevent burns and excoriation. Turn the newborn every 2 hours to maximize the area of exposure, removing the newborn from the lights only for feedings. Maintain a neutral thermal environment to decrease energy expenditure, and assess the newborn's neurologic status frequently.

Assess the newborn's temperature every 3 to 4 hours as indicated. Monitor fluid intake and output closely and assess daily weights for gains or losses. Check skin turgor for evidence of dehydration. With feedings, remove the newborn from the lights and remove the eye shields to allow interaction with the newborn. Encourage breast or bottle feedings every 2 to 3 hours. Follow agency policy about removing the eye shields periodically to assess the eyes for discharge or corneal irritation secondary to eye shield pressure. Typically, the eyes are assessed and eye shields removed once a shift.

Monitor stool characteristics for consistency and frequency. Unconjugated bilirubin excreted in the feces will produce a greenish appearance, and typically stools are loose. Lack of frequent green stools is a cause for concern.

Provide meticulous skin care. Assess skin surfaces and turgor frequently for dryness and irritation secondary to the dehydrating effects of phototherapy and irritation from highly acidic stool to prevent excoriation and skin breakdown (Green & Wilkinson, 2004).

The use of phototherapy can be anxiety-producing for the parents. Explain the rationale for the procedure and demonstrate techniques that the parents can use to interact with their newborn.

If an exchange transfusion is necessary, assist the physician with the procedure. Monitor the newborn's status closely before, during, and after the procedure for any changes, especially in vital signs and heart rate and rhythm.

Parents need instruction about how to assess their newborn for signs and symptoms of jaundice because physiologic jaundice may not occur until after the newborn is discharged. Additional education related to phototherapy may be necessary when home phototherapy is used (Teaching Guidelines 24-2).

TEACHING GUIDELINES 24-2

Caring for Your Newborn Receiving Home Phototherapy

- Inspect your newborn's skin, eyes, and mucous membranes for a yellow color.
- Remember that a home health nurse will come to visit and help you set up the light system.
- Keep the lights about 12 to 30 inches above your newborn.
- Cover your newborn's eyes with patches or cotton balls and gauze to protect them.
- Keep the newborn undressed except for the diaper area; fold the diaper down below the newborn's navel in the front and as far as possible in the back to expose as much skin area as possible.
- Turn your newborn every 2 hours to make sure to expose all areas of the body.
- Remove the newborn from the lights only during feeding.
- Remove the eye patches during feedings so that you can interact with your newborn.
- Record your newborn's temperature, weight, and fluid intake daily.
- Document the frequency, color, and consistency of all stools; the stools should be loose and green as the bilirubin is broken down.
- Keep the skin clean and dry to prevent irritation.
- Feed your newborn frequently, including supplemental glucose water if allowed to provide added fluid, protein, and calories.
- Rock, cuddle, or hold the newborn to promote bonding when out of the lights.
- Contact your pediatrician or home health care agency with any questions or changes, including refusing feedings, fewer than five wet diapers in one day, vomiting of complete amounts of feeding, or elevated temperature.
- Keep appointments for follow-up laboratory testing to monitor bilirubin levels.

Newborn Infections

Newborns are susceptible to infections because their immune system is immature and slow to react. The antibodies newborns received from their mother during pregnancy and from breast milk help protect them from invading organisms. However, these need time to reach optimal levels.

Pathophysiology

When a pathologic organism overcomes the newborn's defenses, infection and sepsis results. **Neonatal sepsis** is the presence of bacterial, fungal, or viral microorganisms or their toxins in blood or other tissues. Infections that have an onset within the first month of life are termed newborn infections. Exposure to a pathogenic organism, whether a virus, fungus, or bacteria, occurs and it enters the newborn's body and begins to multiply.

Etiologies and Risk Factors

Bacterial infections of the newborn affect approximately 4 out of every 1,000 live births (Stoll, 2004). Making the

diagnosis of sepsis in newborns is difficult due to its nonspecific symptoms. The mortality rate from newborn sepsis may be as high as 50% if untreated. Infection is a major cause of death during the first month of life, contributing to 13% to 15% of all neonatal deaths (Bellig & Ohning, 2004). An awareness of the myriad of risk factors associated with newborn sepsis prepares the nurse for early identification and treatment, thus reducing mortality and morbidity. Among the factors that contribute to the newborn's vulnerability to infection are poor skin integrity, invasive procedures, and exposure to numerous caregivers and an environment conducive to bacterial colonization (Aly et al., 2005).

Newborn infections are usually grouped into three classes according to their time of onset: congenital infection, acquired in utero by vertical transmission with onset before birth; early-onset infections, acquired by vertical transmission in the perinatal period, either shortly before or during birth; and late-onset infections, acquired by horizontal transmission in the nursery. As many as 80% to 90% of neonatal infections have their onset in the first 2 days of life (CDC, 2002).

Intrauterine infections occur when pathogenic organisms cross the placenta into the fetal circulatory system or ascend from the vagina and begin to multiply. The organism can reside in the amniotic fluid, as with cytomegalovirus, or can travel up from the vagina, infecting the membranes and causing them to rupture. This rupture can cause respiratory and gastrointestinal tract infections of the neonate.

Early-onset or intrapartum factors that increase the risk for infection include prolonged rupture of the membranes, urinary tract infections, preterm labor, prolonged or difficult labor, maternal fever, colonization with group B streptococci, and maternal infections. The most common organisms involved in early-onset newborn infections are *Escherichia coli,* group B streptococci, *Klebsiella pneumoniae, Listeria monocytogenes,* and other enteric gram-negative bacilli (Hoerst & Samson, 2002). Most infections during the birthing process occur when the newborn comes into contact with an infected birth canal, which can host bacteria against which the newborn cannot defend. The newborn's susceptibility to infection by exogenous organisms may be in part due to the inadequacy of physical barriers: the newborn has thin, friable skin with little subcutaneous tissue. Lack of gastric acidity may also result in easy colonization by environmental organisms. Newborns also may aspirate microorganisms during birth and develop pneumonia.

Risk factors for late-onset infection in the newborn include low birthweight, prematurity, meconium staining, need for resuscitation, birth asphyxia, and improper handwashing. Infections are more common in newborns undergoing invasive procedures such as endotracheal intubation or catheter insertion. Common pathogens implicated in causing late-onset infections include *Candida albicans,* coagulase-negative staphylococci, *Staphylococcus aureus, E. coli, Enterobacter, Klebsiella, Serratia, Pseudomonas,* and group B streptococci (Merenstein et al., 2002).

Nursing Management

Nursing management requires keen assessment skills to identify a newborn with an infection because the signs and symptoms are often subtle, such as apnea, lethargy, poor feeding, temperature instability, respiratory distress, and poor color (Thureen et al., 2005). Obtaining specimens and coordinating therapies are paramount in treating the newborn with an infection.

Assessment

Manifestations of infections in the newborn usually are nonspecific. Few newborn infections are easy to recognize. Early symptoms can be vague because of the newborn's inability to mount an inflammatory response. Often the nursery nurse reports that the newborn does not "look right." Assess the newborn for common nonspecific signs of infection:

- Hypothermia
- Pallor or duskiness
- Hypotonia
- Cyanosis
- Poor weight gain
- Irritability
- Seizures
- Jaundice
- Grunting
- Nasal flaring
- Apnea and bradycardia
- Lethargy
- Hypoglycemia
- Poor feeding (lack of interest in feeding)
- Abdominal distention (Bellig & Ohning, 2004)

Since infection can be confused with other newborn conditions, laboratory and radiographic tests are needed to confirm the presence of infection. Be prepared to coordinate the timing of the various tests and assist as necessary.

Workups for sepsis may include a complete blood count with a differential to identify anemia, leukocytosis, or leukopenia. A C-reactive protein may also be ordered to validate inflammation. An elevated C-reactive protein level is associated with tissue injury and inflammation. It is not used as a sole indicator of neonatal sepsis but can be used along with other studies to evaluate the infectious process. X-rays of the chest and abdomen are also ordered to detect infectious processes located there. Blood, cerebrospinal fluid, and urine cultures are taken to identify the location and type of infection present. Positive cultures confirm that the newborn has an infection.

Nursing Interventions

To enhance the newborn's chance of survival, early recognition and diagnosis are key. Often the diagnosis of sepsis

is based on suspicion of the presenting clinical picture. Antibiotic therapy is usually started before the laboratory results identify the infecting pathogen. Along with antibiotic therapy, circulatory, respiratory, nutritional, and developmental support is important. Antibiotic therapy is continued for 7 to 21 days if cultures are positive, or it is discontinued within 72 hours if cultures are negative. With the use of antibiotics along with early recognition and supportive care, mortality and morbidity rates have been reduced greatly.

Perinatal infections continue to be a public health problem, with severe consequences for those affected. By promoting a better understanding of newborn infections and appropriate use of therapies, nurses can lower the mortality rates associated with severe sepsis, especially with appropriate timing of interventions. The potential for nursing interventions to identify, prevent, and minimize the risk for sepsis is significant. Primary disease prevention must be a major focus for nurses. Family education plays a key role in the prevention of perinatal infections, in addition to following accepted practices in immunization.

Nurses possess the education and assessment tools to decrease the incidence of and reduce the impact of infections on women (see Chapter 20 for additional information) and their newborns by implementing measures for prevention and early recognition, including:

- Formulate a sepsis prevention plan that includes education of all members of the healthcare team on identification and treatment of sepsis.
- Screen all newborns daily for signs of sepsis.
- Monitor sepsis cases and outcomes to reinforce continued quality-improvement measures or modify current practices.
- Outline and carry out measures to prevent nosocomial infections:
  - Thorough handwashing hygiene for all staff
  - Frequent oral care and inspections of mucous membranes
  - Proper positioning and turning to prevent skin breakdown
  - Use of strict aseptic technique for all wound care
  - Frequent monitoring of invasive catheter sites for signs of infection
- Identify newborns at risk for sepsis by reviewing risk factors.
- Monitor vital sign changes and observe for subtle signs of infection.
- Monitor for signs of organ system dysfunction:
  - Cardiovascular compromise—tachycardia and hypotension
  - Respiratory compromise—respiratory distress and tachypnea
  - Renal compromise—oliguria or anuria
  - Systemic compromise—abnormal blood values
- Provide comprehensive sepsis treatment:
  - Provide circulatory support with fluids, vasopressors.
  - Provide supplemental oxygen and mechanical ventilation.
  - Obtain and assist with culture samples as requested.
  - Administer antibiotics as ordered, observing for side effects.
  - Provide for the newborn's comfort level.
  - Assess the family's educational needs and provide information as needed.

Congenital Conditions

Congenital conditions can arise from many etiologies, including single-gene disorders, chromosome aberrations, exposure to teratogens, and many sporadic conditions of unknown cause. Congenital conditions may be inherited or sporadic, isolated or multiple, apparent or hidden, gross or microscopic. They cause nearly half of all deaths in term newborns and cause long-term sequelae for many. The incidence varies according to the type of defect. When a serious anomaly is identified prenatally, the parents can decide whether or not to continue the pregnancy. When an anomaly is identified at or after birth, parents need to be informed promptly and given a realistic appraisal of the severity of the condition, the prognosis, and treatment options so that they can participate in all decisions pertaining to their child.

Congenital Heart Disease

Congenital heart disease is a structural defect involving the heart, the great vessels, or both that is present at birth (O'Toole, 2005). It is a broad term that can describe a number of abnormalities affecting the heart. One in 100 newborns in North America has congenital heart disease—about 8 to 10 of every 1,000 live births (Rempel et al., 2004). It is responsible for more deaths in the first year of life than any other birth defects (Cheffer & Rannalli, 2004). The defect may be very mild and the newborn appears healthy at birth, or it may be so severe that the newborn's life is in immediate jeopardy. Severe congenital cardiac defects usually present in the first few days or weeks of life, while the newborn's circulation is continuing to adapt to the demands of extrauterine life. Advances in diagnosis and medical and surgical interventions have led to dramatic increases in survival rates for newborns with serious heart defects.

Etiology and Risk Factors

In most cases, the exact cause of congenital heart disease is unknown. Most congenital heart defects develop during the first 8 weeks of gestation and are usually the result of genetic and environmental forces, which might include:

- Maternal alcoholism
- Maternal diabetes mellitus

- Single-gene mutation or chromosomal disorders
- Maternal exposure to x-rays
- Maternal exposure to rubella infection
- Poor maternal nutrition during pregnancy
- Maternal age over 40
- Maternal use of amphetamines
- Genetic factors (family recurrence patterns)
- Maternal metabolic disorder of phenylketonuria
- Maternal use of anticonvulsants, estrogen, progesterone, lithium, warfarin (Coumadin), or isotretinoin (Accutane) (Littleton & Engebretson, 2005)

Classification

Typically, congenital heart disease is divided into four physiologic categories based on structural abnormalities and functional alterations (Table 24-4):

- Defects causing increased pulmonary blood flow, such as atrial septal defect and ventricular septal defect
- Defects causing obstructed blood flow out of the heart, such as pulmonary or aortic stenosis
- Defects causing decreased pulmonary blood flow, such as tetralogy of Fallot
- Defects with cyanosis and increased pulmonary blood flow or mixed defects, such as truncus arteriosus or transposition of the great arteries

These four categories are more descriptive than the system used previously, which classified the disorder only as cyanotic or acyanotic. This previous classification was imprecise because some newborns with "acyanotic" defects developed cyanosis, and delayed symptoms often become apparent during infancy and early childhood. With the hemodynamic classification, the clinical picture of each grouping is more uniform and predictable (Hockenberry, 2005).

Nursing Management

Ideally, nursing management begins prenatally by reviewing the maternal history for risk factors that might predispose the newborn to a congenital heart defect. While most congenital heart defects cannot be prevented, several key areas need to be addressed to ensure the optimal health status for the woman and her fetus. For example, ensure that all women are tested prior to pregnancy for immunity to rubella so that they can be immunized if necessary. Any chronic health problems, such as diabetes, hypertension, seizures, and phenylketonuria, should be controlled and any medication or dietary adjustments should be made before attempting conception. Once pregnant, the woman should be encouraged to avoid alcohol, smoking, and the use of unprescribed drugs. Refer the woman and her partner for genetic counseling if cardiac defects are present in the family to provide the parents with a risk assessment for future offspring.

Some defects can be discovered on routine prenatal ultrasound. Therefore, stress the importance of receiving prenatal care throughout pregnancy so that appropriate interventions can be initiated early if the need arises.

After birth, when the newborn is admitted to the nursery, carefully assess the cardiovascular and respiratory systems, looking for signs and symptoms of respiratory distress, cyanosis, or congestive heart failure that might indicate a cardiac anomaly. Assess rate, rhythm, and heart sounds, reporting any abnormalities immediately. Note any signs of heart failure, including edema, diminished peripheral pulses, hepatomegaly, tachycardia, diaphoresis, respiratory distress with tachypnea, peripheral pallor, and irritability (Kenner & Lott, 2004). Assist with diagnostic testing, such as:

- Arterial blood gases to determine oxygenation levels and to differentiate lung disease from heart disease as the cause of cyanosis
- Chest x-rays to identify cardiac size, shape, and position
- Magnetic resonance imaging (MRI) to evaluate for cardiac malformations
- Electrocardiogram to detect atrial or ventricular hypertrophy and dysrhythmias
- Echocardiogram to evaluate heart anatomy and flow defects
- Blood studies to assess anemia, blood glucose, and electrolyte levels
- Catheterization to obtain data for definitive diagnosis or in preparation for cardiac surgery (Montoya & Washington, 2002)

Provide continuous monitoring of the newborn's cardiac and respiratory status, administer medications as ordered, and provide comfort measures to the newborn who will be subjected to a variety of painful procedures. Be vigilant in ensuring the newborn's comfort, since he or she cannot report or describe pain. Assist in preventing pain as much as possible, interpreting the newborn's cues suggesting pain and managing it appropriately (Pasero, 2004). Make pain assessment part of routine newborn nursing care.

Include the parents in the plan of care. Parents want to make informed decisions based on their beliefs and values and desire to make them in the best interest of their infant (Rempel et al., 2004). Nurses can play a key role in meeting the parents' needs by doing the following:

- Assess their ability to cope with the diagnosis.
- Encourage them to verbalize their feelings about the newborn's condition and treatment.
- Instruct them about the medications prescribed, including side effects and doses, and how to observe for signs and symptoms indicating heart failure.
- Educate them about the specific cardiac defect; include written information and pictures to enhance understanding.

Table 24-4 Classifications of Congenital Heart Disease

Cardiac Defect	Examples	Pathophysiology	Clinical Picture
Increased pulmonary blood flow (left-to-right shunting)	Atrial septal defect (ASD) Ventricular septal defect (VSD) Patent ductus arteriosus (PDA)	Cardiac septum communication or abnormal connection between the great arteries permits blood to flow from higher pressure (left side of heart) to lower pressure (right side of heart).	Asymptomatic or murmur, fatigue with feedings, and symptoms of CHF: pallor, cyanosis, or gray coloring, diminished peripheral pulses, edema, diaphoresis, tachypnea, and tachycardia
Decreased pulmonary blood flow	Tetralogy of Fallot (TOF) Tricuspid atresia	Pulmonary blood flow obstruction accompanied by an anatomic defect such as ASD or VSD between the right and left sides of the heart, which allows desaturated blood to shunt right to left, causing desaturated blood to enter into the systemic circulation	Cyanosis, murmur, hypoxemia, dyspnea, increased cardiac workload, and marked exercise intolerance
Obstruction to blood flow out of the heart	Pulmonary stenosis Aortic stenosis Coarctation of the aorta	A narrowing or constriction of an opening causes pressure to rise in the area behind the obstruction and a decrease in blood available for systemic perfusion	CHF, decreased cardiac output, and pump failure
Cyanotic defects with increased pulmonary blood flow or mixed defects	Transposition of the great arteries Truncus arteriosus Hypoplastic left heart syndrome	Fully saturated systemic blood flow mixes with desaturated pulmonary blood flow, causing desaturation of the systemic circulation. This leads to pulmonary congestion and a decrease in cardiac output. To support life, intervention must bring about a mixing of arterial and venous blood.	Cyanosis, CHF, ruddiness, dusky or gray color, dyspnea

CHF, congestive heart failure

Sources: Hockenberry, 2005; Verklan & Walden, 2004; Mattson & Smith, 2004; McKinney et al., 2005.

- Present an overview of the prognosis and possible interventions.
- Assist them with making decisions about treatment, and support their decisions for the newborn's care.
- Orient them to the NICU prior to surgery.
- Provide emotional support throughout care.

The parents also need clear instructions about how to monitor the newborn at home, especially if the newborn will be discharged and then brought back later so the condition can be corrected. The parents also need instructions about caring for their newborn after the defect is corrected. Educate the parents about signs and symptoms that need

to be reported, such as weight loss, poor feeding, cyanosis, breathing difficulties, irritability, increased respiratory rate, and fever. Referrals to local support groups, national organizations, and websites also are helpful. Emphasize the importance of close supervision and follow-up care.

Inborn Errors of Metabolism

Inborn errors of metabolism are genetic disorders that disrupt normal metabolic function. Most are due to a defect in an enzyme or transport protein, resulting in a blocked metabolic pathway. Clinical symptoms are manifested secondary to toxic accumulations of substances before the block. When viewed individually, inborn errors of metabolism are rare, but collectively they are responsible for significant levels of infant mortality and morbidity. Table 24-5 summarizes four common inborn errors.

A successful outcome for the affected newborn depends on early diagnosis and prompt intervention. Most inborn errors present in the newborn period with nonspecific and subtle manifestations—lethargy, hypotonia, respiratory distress, poor feeding and weight gain, vomiting, and seizures (Weiner, 2005). Identification of an inborn error of metabolism in a newborn depends largely on the awareness of the nurse and clues from the maternal history, laboratory work, and clinical examination.

Congenital CNS Structural Defects

Congenital CNS structural defects are serious malformations involving the spine (spina bifida) and brain (anencephaly). They are more commonly described as neural tube defects because they occur when the neural tube fails to close properly during early embryogenesis. The neural tube develops into the brain and spinal cord and normally closes between the 17th and 30th day of gestation. Neural tube defects develop during this first month, when most women are still unaware of their pregnancy and the embryo is estimated to be about the size of a grain of rice. In pregnancies in which the fetus has a neural tube defect, the level of alpha-fetoprotein in the amniotic fluid and maternal serum is elevated.

Neural tube defects involve abnormalities in the region-specific neural tube closure junctions with the cranial and caudal levels of the neural tube, often resulting in frank exposure of neural tissue. These defects vary in their severity, depending on the type and level of the lesion. Neural tube defects affect 0.6 per 1,000 live births in the United States, where there are approximately 3,000 pregnancies annually that are complicated by neural tube defects (Becske & Jallo, 2004). They are the second most common major congenital anomaly worldwide, behind cardiac malformations (CDC, 2004).

Neural tube defects may be either closed (covered by skin or a membrane) or open (neural tissue exposed). Some common defects are anencephaly, hydrocephalus, microcephaly, spina bifida, myelomeningocele, and meningocele. Defects that will be discussed here are hydrocephalus, microcephaly, anencephaly, and spina bifida.

A worldwide decline in neural tube defects has occurred over the past few decades as a result of prevention (preconception folic acid supplementation and monitoring of maternal serum alpha-fetoprotein levels) and use of ultrasonography and amniocentesis to identify affected fetuses (Blackburn, 2003). Despite this decline, still more infants could be born free of these birth defects if all women consumed the necessary amount of folic acid (CDC, 2004). Early prenatal diagnosis can offer parents the option for elective termination if desired.

Hydrocephalus

Hydrocephalus is an increase in cerebrospinal fluid (CSF) in the ventricles of the brain due to overproduction or impaired circulation and absorption. The term stems from the Greek words *hydor* (water) and *cephalic* (head). Normal growth of the brain is altered secondary to the increase in intracranial pressure from the CSF. Congenital hydrocephalus usually arises as a result of a malformation in the brain or an intrauterine infection (toxoplasmosis or cytomegalovirus). It occurs in about 3 or 4 per 1,000 live births (Allen & Vessey, 2004).

Hydrocephalus rarely occurs as an isolated defect; it is usually associated with spina bifida or other neural tube anomalies. Clinical manifestations include large head, widened sutures, poor feeding, bulging and tense fontanels, "setting sun" eyes, vomiting, lethargy, visible scalp veins, and irritability (Haws, 2004). Diagnosis is by computed tomography (CT) scan or MRI (Fig. 24-7).

No treatment is available that can counteract the accumulation of CSF in the brain. Therefore, surgery with the insertion of a ventricular shunt is the mainstay of treatment to relieve pressure within the cranium. Shunts are designed to maintain normal intracranial pressure by draining off excess CSF. Shunting has dramatically improved the outcome of newborns with hydrocephalus (Sgouros, 2004).

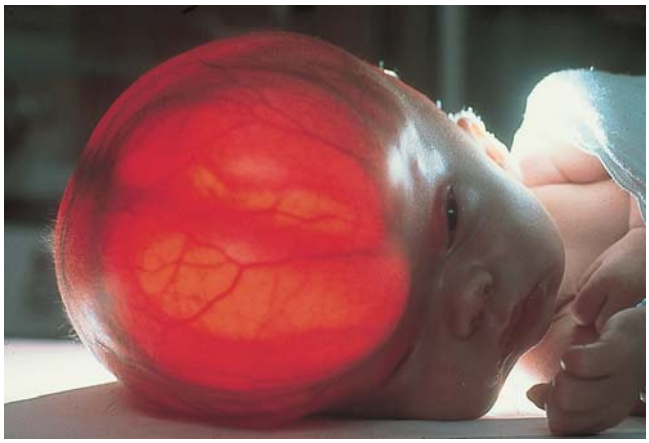
A ventriculoperitoneal shunt is inserted from the ventricle in the brain and threaded down into the peritoneal cavity to allow drainage of excess CSF. The long-term prognosis for this condition varies and depends on the patency of the shunt, the presence of other CNS anomalies and their impact on the newborn, and the quality of care the newborn receives.

Prior to shunt insertion, nursing management focuses on daily documentation of the newborn's head circumference and associated neurologic behaviors that might indicate an increase in intracranial pressure: irritability, high-pitched cry, poor feeding and sucking, vomiting, or decrease in consciousness. Gently palpate the fontanels for signs of bulging and tenseness, and palpate the suture lines for increasing separation. Protect the enlarged head to prevent skin breakdown. Handle the head gently and use a sheepskin or a waterbed or egg-crate mattress. Change the newborn's position frequently to minimize pressure.

Table 24-5 Inborn Errors of Metabolism

Condition	Incidence and Etiology	Clinical Picture	Management
Phenylketonuria (PKU)	1:15,000 live births Autosomal recessive genetic disorder caused by a deficiency of the hepatic enzyme phenylalanine hydroxylase Enzyme deficiency with subsequent accumulation of amino acid phenylalanine	Newborns appear normal at birth but by 6 months of age signs of slow mental development evident Vomiting, poor feedings, failure to thrive, overactivity, irritability, musty-smelling urine If not treated, possible mental retardation	Screening of all newborns at about 48 hours after birth to ensure adequate intake of protein Dietary restriction of phenylalanine, with regular monitoring of serum phenylalanine levels (effective when started before the first month of age) Life-long dietary restriction of phenylalanine
Maple syrup urine disease (MSUR)	1:150,000 in general population; most prevalent among the Mennonite population in Lancaster, Pennsylvania Autosomal recessive inherited disorder Enzyme metabolism of certain amino acids is affected, with the buildup of acids causing ketoacidosis.	Lethargy, poor feeding, vomiting, weight loss, seizures, shrill cry, shallow respirations, loss of reflexes, coma, sweet maple syrup odor to urine	Dialysis to remove accumulated acids Life-long low-protein diet to prevent neurologic deficits of disease
Galactosemia	1:50,000 births Autosomal recessive inherited disorder in which an enzyme needed to convert galactose to glucose is missing and newborn cannot metabolize lactose	Vomiting, hypoglycemia, liver damage, hyperbilirubinemia, poor weight gain, cataracts, frequent infections	Routine newborn screening for galactosemia is performed in the majority of states. Life-long lactose-restricted diet to prevent mental retardation, liver disease, and cataracts.
Congenital hypothyroidism	1:4,000 live births Multiple causes—absent or underdeveloped thyroid gland or biochemical defects in thyroid hormone	Large protruding tongue, slow reflexes, distended abdomen, large, open posterior fontanel, constipation, hypothermia, poor feeding, hoarse cry, dry skin, coarse hair, goiter, and jaundice. If untreated, irreversible cognitive and motor impairment. Decreased levels of thyroid hormone (T4) and elevated levels of TSH.	Newborn screening program in all states Life-long thyroid replacement hormone therapy and continued monitoring of thyroid levels and clinical response to therapy

Sources: Weiner, 2005; Verklan & Walden, 2004; Lanting et al., 2005; Hockenberry, 2005; Littleton & Engebretson, 2005



● Figure 24-7 Newborn with hydrocephalus.

Postoperatively, strictly monitor the newborn's neurologic status and behavior and report any changes that might indicate an increased intracranial pressure secondary to a blockage in the shunt. These finding may include papillary dilation (increased intracranial pressure places pressure on the oculomotor nerve, producing dilation), increasing head size, bulging fontanels, and change in level of consciousness. Assess the abdomen for distention because drainage of CSF into the abdomen can cause peritonitis. Paralytic ileus is another possible postoperative complication due to distal catheter placement (Hockenberry, 2005).

After surgery, continue to provide protective and comfort measures for the enlarged head. Position the newborn's head so that he or she does not lie on the shunt area. Educate the parents about caring for the shunt and signs and symptoms of infection or blockage. A referral for follow-up home care is appropriate.

Microcephaly

Microcephaly is a condition in which a small brain is located within a normal-sized cranium. This implies neurologic impairment. Risk factors for this anomaly include maternal viral infections (toxoplasmosis, rubella, cytomegalovirus, herpes, and syphilis), radiation exposure, diabetes, phenylketonuria, street drug exposure, and malnutrition (Verklan & Walden, 2004). Diagnosis is confirmed by a CT scan or MRI. Care is supportive since there is no known treatment to reverse the disorder. Parents need to be informed of potential cognitive impairment of their newborn. Ensure that appropriate community referrals are made to assist the parents and the child, who will have developmental delays.

Anencephaly

Anencephaly, the most severe neural tube defect, is the congenital absence of the cranial vault, with the cerebral hemispheres completely missing or reduced to small masses (O'Toole, 2003). It most commonly involves the forebrain and variable amounts of the upper brain stem, where there is no brain tissue above the brain stem. The incidence is approximately 0.2 per 1,000 live births, and both genetic and environmental insults appear to be responsible (Verklan & Walden, 2004). Anencephaly is apparent on visual inspection after birth, with exposed neural tissue without a cranium surrounding it. Prenatally, alpha-fetoprotein levels are elevated late in the first trimester. Most newborns with anencephaly are stillborn; those born alive die within a few days. Comfort measures for the newborn and support for the parents are needed as they grieve for the loss of their infant.

Spina Bifida

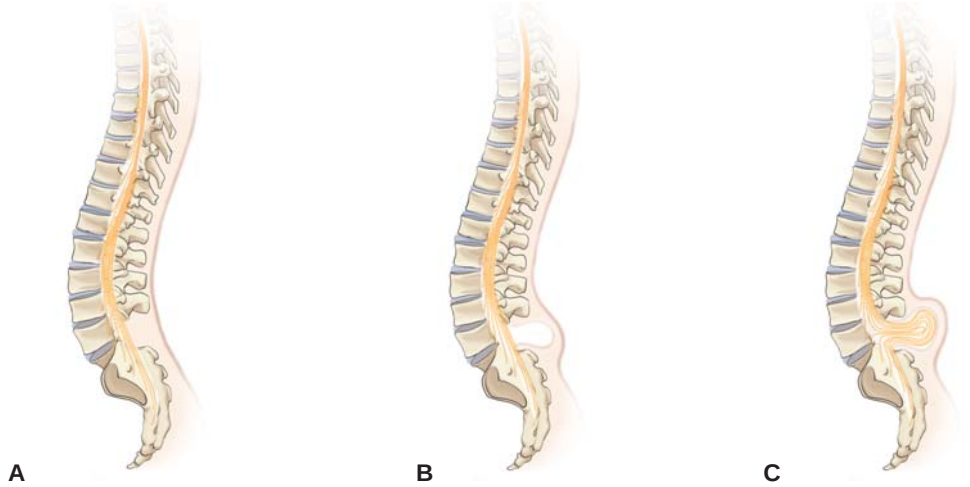
Spina bifida is a general category of caudal defects (below the level of T12) involving spinal cord tissue (Finnell et al., 2003). It is the most complex but treatable CNS abnormality that is visible at birth (Foster, 2004). Spina bifida is the leading cause of infantile paralysis in the world today; incidence rates are about 1 per 1,000 live births (Vachha & Adams, 2005). This classification includes two types of common defects: meningocele and myelomeningocele (Fig. 24-8).

A meningocele is an opening in the spine through a bony defect (spina bifida) where a herniation of the meninges and spinal fluid has protruded. The spinal cord and nerve roots do not herniate into this dorsal dural sac. Newborns with meningocele usually have normal examination findings and a covered (closed) dural sac. They typically do not have associated neurologic malformations. Surgical treatment to close the defect is usually warranted.

A **myelomeningocele** is a more severe form of spina bifida in which the spinal cord and nerve roots herniate into the sac through an opening in the spine, compromising the meninges. It is the most common form of spina bifida, accounting for 94% of cases (Foster, 2004). The incidence is 1 in 1,200 to 1,400 live births, affecting 6,000 to 11,000 newborns in the United States each year (Becske & Jallo, 2004).

This complex condition, resulting from a neurodevelopmental disruption early in gestation, affects not just the spine but also the CNS. Hydrocephalus frequently accompanies this anomaly (Ellenbogen, 2004). This protrusion is typically covered partially or completely by skin but is very fragile and may leak CSF if traumatized. Risk factors for myelomeningocele include both genetic and environmental factors:

- Celtic ancestry (highest incidence)
- Female sex (accounting for 60% to 70% of affected newborns)
- Low socioeconomic status
- Maternal diabetes
- Use of anticonvulsants (valproic acid and carbamazepine)
- Previous pregnancy with a newborn with a neural tube defect



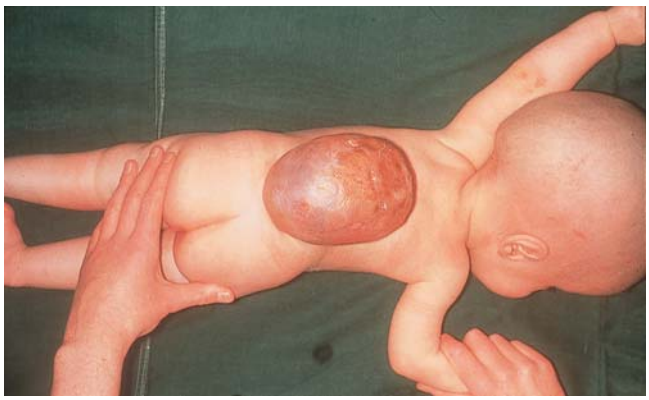
● Figure 24-8 Two common types of spina bifida. (**A**) Normal spinal cord. (**B**) Meningocele. (**C**) Myelomeningocele.

- Maternal obesity
- Maternal malnutrition
- Low folic acid intake (Cunningham et al., 2005)

Myelomeningoceles can arise at any point along the vertebral column, but they most commonly occur in the lower lumbar or sacral regions, causing neurologic deficits below the level of the defect. Paralysis, bladder and bowel incontinence, and hydrocephalus are the most common complications (Fig. 24-9). Surgical repair as soon as possible, usually within 72 hours after birth, helps prevent infection and preserve neurologic function (Ellenbogen, 2004).

Nursing management for a newborn with myelomeningocele involves the following actions:

- Use strict aseptic technique when caring for the defect to prevent infection.
- Avoid hypothermia: heat can be lost through the defect opening, placing the newborn at increased risk for cold stress.
- Avoid trauma to the sac (to prevent leakage of CSF or damage to the nerve tissue) through prone or side-lying positioning.



● Figure 24-9 Newborn with myelomeningocele and hydrocephalus.

- Apply a sterile dressing or protective covering over the sac to prevent rupture and drying, with frequent changes to prevent the dressing from adhering to the defect.
- Frequently monitor the sac for signs of oozing fluid or drainage.
- Meticulously clean the genital area to avoid contamination of the sac.
- Assess movement and sensation below the defect.
- Measure head circumference daily to observe for hydrocephalus.
- Provide support and information to help the parents cope.
- Allow the parents to vent their feelings.
- Make referrals to support groups.
- Encourage open discussions regarding the baby's prognosis and long-term care.
- Involve the parents in the newborn's care.

Respiratory System Structural Anomalies

Two structural anomalies of the respiratory system, choanal atresia and congenital diaphragmatic hernia, can be life-threatening.

Choanal Atresia

Choanal atresia is an uncommon congenital malformation of the upper airway involving a narrowing of the nasal airway by membranous or bony tissue. It typically presents with other anomalies involving the heart and CNS. It occurs in 1 in 8,000 live births, with a female preponderance (Dave, 2005). This structural anomaly can result in significant respiratory distress in the newborn. If the nasal airway is completely obstructed, death from asphyxia may occur at birth.

During attempted inspiration, the tongue is pulled to the palate and obstruction of the oral airway results. If the newborn cries and takes a breath through the mouth, the airway obstruction is momentarily relieved. When the

crying stops, however, the mouth closes and the cycle of obstruction is repeated (Tewfik & Hagr, 2005).

The cause of this congenital defect is unknown, but it is thought to result from persistence of the membrane between the nasal and oral spaces during fetal development. This defect may be unilateral or bilateral and is often associated with other congenital anomalies.

Failure to pass a suction catheter through the nose into the pharynx is highly suggestive of choanal atresia. The diagnosis can be confirmed with a CT scan. Other signs include respiratory distress, cyanosis unless newborn is crying, and inability to suck and breathe simultaneously. Surgery to remove the obstruction and establish a patent airway is needed. Full recovery is the usual outcome.

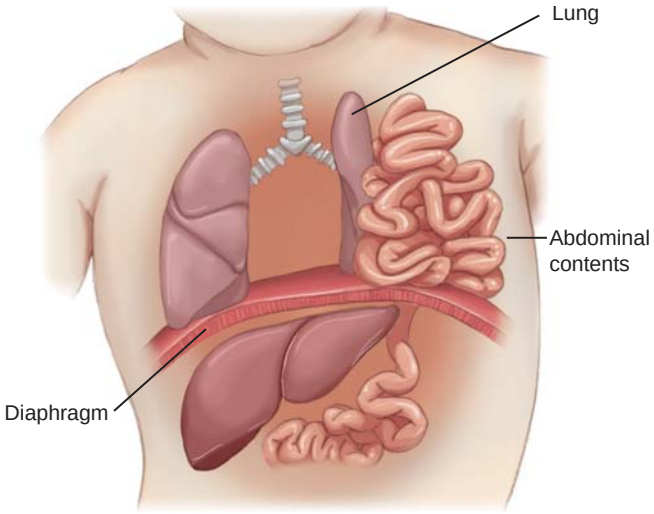
Congenital Diaphragmatic Hernia

Congenital diaphragmatic hernia is a rare disorder (1 in 3,000 newborns) that frequently presents with significant respiratory distress in the immediate newborn period (Tesselaar et al., 2004). The abdominal contents are herniated into the thoracic cavity through a defect in the diaphragm. It is thought that the diaphragm failed to close properly during early embryonic development. The timing of the herniation and the amount of abdominal contents in the thoracic cavity greatly influence the clinical picture at birth and the survival rate (Cheffer & Rannalli, 2004).

Most hernias (85%) involve the left hemidiaphragm (Mattson & Smith, 2004). Prenatal diagnosis is possible through ultrasound. This diagnosis should be considered when hydramnios is present. The presence of the abdominal contents in the chest compresses the lung, leads to pulmonary hypoplasia, and promotes persistent pulmonary hypertension in the newborn (Haws, 2004).

Associated anomalies include congenital cardiac defects, genital or renal anomalies, neural tube defects, choanal atresia, or chromosomal anomalies, such as trisomy 13 and 18. The survival rate of newborns with a diaphragmatic hernia varies, but overall the rate has remained at about 50% for nearly half a century despite advances in neonatal intensive care, anesthesia, and surgery (Golombek, 2002) (Fig. 24-10).

Affected newborns present with profound respiratory distress because at least one of the lungs cannot expand or may not have fully developed, resulting in persistent pulmonary hypertension shortly after birth; aggressive resuscitation is needed. Other clinical features include absent breath sounds on the affected side of the chest, heart sounds displaced to the right, bowel sounds noted in the chest, barrel chest and a scaphoid-shaped abdomen, and cyanosis. Diagnosis is made by chest x-ray, which reveals an air-filled bowel in the chest cavity (Hockenberry, 2005). Surgery to correct the defect is needed.



● Figure 24-10 Congenital diaphragmatic hernia. Note how some of the abdominal contents enters the thoracic cavity, subsequently compressing the lung.

Nursing care related to the care of the affected newborn includes the following:

- Assist with endotracheal intubation and positive-pressure ventilation to aid in lung expansion and improvement of ventilation.
- Position the newborn on the affected side with the head and chest elevated to promote normal lung expansion.
- Assist with placement of an orogastric tube for gastric decompression.
- Monitor ventilatory pressures to prevent pneumothorax.
- Monitor vital signs, weight, urinary output, and serum electrolytes to identify changes early.
- Maintain thermoregulation to prevent cold stress.
- Anticipate use of extracorporeal membrane oxygenation (ECMO) or high-frequency oscillatory ventilation if the newborn's condition does not stabilize.
- Maintain nothing by mouth (NPO) status to prevent aspiration.
- Administer inotropics to support systemic blood pressure.
- Administer surfactant, steroids, and inhaled nitric oxide as ordered to correct hypoxia and acid–base imbalance.
- Assist with insertion of a chest tube in the event of a pneumothorax.
- Monitor oxygen saturation levels to evaluate systemic perfusion status.
- Minimize environmental stimuli to reduce agitation and oxygen demand.
- Provide supportive positioning with a rolled blanket to promote comfort.

- Provide the parents with continuing updates about the newborn's condition.
- Encourage the parents to see and touch the infant frequently to promote bonding.
- Recognize and react to newborn clues (Mattson & Smith 2004).

Gastrointestinal System Structural Anomalies

Gastrointestinal anomalies that disrupt facial structures are found in about 1% (or 1 million) of the infants born worldwide each year. The most common of these is cleft lip and palate, a complex condition due to multiple genetic and environmental factors (Murray & Schuttem, 2004). Other gastrointestinal system anomalies include esophageal atresia, tracheoesophageal fistula, omphalocele, gastroschisis, and imperforate anus.

Cleft Lip and Palate

A cleft lip involves a congenital fissure or longitudinal opening in the lip; a cleft palate involves a congenital fissure or longitudinal opening in the roof of the mouth. The defect may be limited to the outer flesh of the upper lip or it may extend back through the midline of the upper jaw through the roof of the palate. It may occur as a single defect or part of a syndrome of anomalies. It can be unilateral or bilateral. Unilateral cleft lip occurs more commonly on the left side. Bilateral cleft lip is usually accompanied by a cleft palate (O'Toole, 2005). Cleft palate can range from a cleft in the uvula to a complete cleft in the soft and hard palates that can be unilateral, bilateral, or in the midline (Fig. 24-11).

Cleft lip and palate is the most common craniofacial birth defect. It is more common in white and Asian males. In addition to immediate feeding difficulties, infants with cleft lip and palate may have problems with dentition, language acquisition, and hearing (Pelchat et al., 2004).

Risk factors for this anomaly include maternal use of phenytoin (Dilantin), alcohol, retinoic acid (Accutane), and cigarette smoking. In addition, a family history of cleft lip or palate increases the incidence (Mattson & Smith, 2004).

The diagnosis of cleft lip is readily apparent at birth. Treatment is surgical repair between the ages of 6 to 12 weeks. Successful surgery often leaves only a thin scar on the upper lip. The outcome of surgery depends on the severity of the defect: children with more severe cases will need additional surgery in stages (Lowdermilk & Perry, 2004).

Repairing the facial anomaly as soon as possible is important to facilitate bonding between the newborn and the parents and to improve nutritional status. Milk flow during feeding requires negative pressure and sucking pressure. Newborns with cleft lip and palate have feeding difficulties because they cannot generate a negative pressure in the mouth to facilitate sucking (Tolarova, 2004). Specialized nipples, bottles, and feeders are available to help meet the nutritional needs of infants with this anomaly.

Surgical correction for cleft palate is done around 6 to 18 months of age to allow for developmental growth to occur. A plastic palate guard to form a synthetic palate may need to be used to allow for introduction of solid foods and to prevent aspiration in the interim.

Caring for a newborn with a cleft lip and palate includes the following:

- Feed the infant in an upright position to prevent aspiration.
- Assess for adequate achievement of suction during feeding.
- Position the newborn on sides in an infant seat after feeding.
- Burp the infant frequently to reduce the risk for vomiting and aspiration; burp him or her in the sitting position on your lap to prevent trauma to the mouth on your shoulder.
- Limit feeding sessions to avoid poor weight gain due to fatigue.
- Use high-calorie formula to improve caloric intake.
- Be alert for bonding problems; encourage the parents to express their feelings about this visible anomaly.
- Encourage parental interaction and involvement with the newborn.
- Plan for discharge as soon as the parents feel comfortable with infant care.
- Show the family photos taken before and after surgical repair in other babies.

● Figure 24-11 The newborn with a cleft lip. Note that the defect may extend up through the roof of the palate.

- Allow the parents to vent their frustrations about feeding problems.
- Outline treatment modalities and explain the staging of surgical interventions.
- Model nurturing behaviors when interacting with the infant.
- Offer information and make appropriate referrals for community support and counseling as needed (Kenner & Lott, 2004).

Esophageal Atresia and Tracheoesophageal Fistula

Esophageal atresia and tracheoesophageal fistula are gastrointestinal anomalies in which the esophagus and trachea do not separate normally during embryonic development. Esophageal atresia refers to a congenitally interrupted esophagus where the proximal and distal ends do not communicate: the upper esophageal segment ends in a blind pouch and the lower segment ends a variable distance above the diaphragm (Fig. 24-12). Tracheoesophageal fistula is an abnormal communication between the trachea and esophagus. When associated with esophageal atresia, the fistula most commonly occurs between the distal esophageal segment and the trachea. The incidence of esophageal atresia is 1 per 3,000 to 4,500 live births (Blair & Konkin, 2004).

Several types of esophageal atresia exist, but the most common anomaly is a fistula between the distal esophagus and the trachea, which occurs in 86% of newborns with an esophageal defect. Esophageal atresia and tracheoesophageal fistula are thought to be the result of incomplete separation of the lung bed from the foregut during early fetal development. A large percentage of these newborns have other congenital anomalies involving the vertebra, renal, heart, musculoskeletal, and gastrointestinal systems (Haws, 2004); most have several anomalies.

The first sign of esophageal atresia may be hydramnios in the mother, because the fetus cannot swallow and absorb amniotic fluid in utero, leading to accumulation. The second sign soon after birth is copious, frothy bubbles of mucus in the mouth and nose accompanied by drooling. Abdominal distention develops as air builds up in the stomach. A gastric tube cannot be inserted beyond a certain point since the esophagus ends in a blind pouch. The newborn may have rattling respirations, excessive salivation and drooling, and "the three C's" (coughing, choking, and cyanosis) if feeding is attempted. The presence of a fistula increases the risk of respiratory complications such as pneumonitis and atelectasis due to aspiration of food and secretions (Kronemer & Snyder, 2004).

Diagnosis is made by x-ray: a gastric tube appears coiled in the upper esophageal pouch, and air in the gastrointestinal tract indicates the presence of a fistula (Verklan & Walden, 2004). Once a diagnosis of esophageal atresia is established, preparations for surgery are made if the newborn is stable.

Preoperative care focuses on the following:

- Prevent aspiration by elevating the head of the bed 30 to 45 degrees to prevent reflux.
- Maintain NPO status.
- Monitoring parenteral IV fluid infusions.
- Assess and maintain the patency of the orogastric tube; monitor the functioning of the tube, which is attached to low continuous suction; and avoid irrigation of the tube to prevent aspiration.
- Assist with diagnostic studies to rule out other anomalies.
- Use comfort measures to minimize crying and prevent respiratory distress.
- Inform the parents about the rationales for the aspiration prevention measures.
- Document frequent observations of the newborn's condition (Haws, 2004).

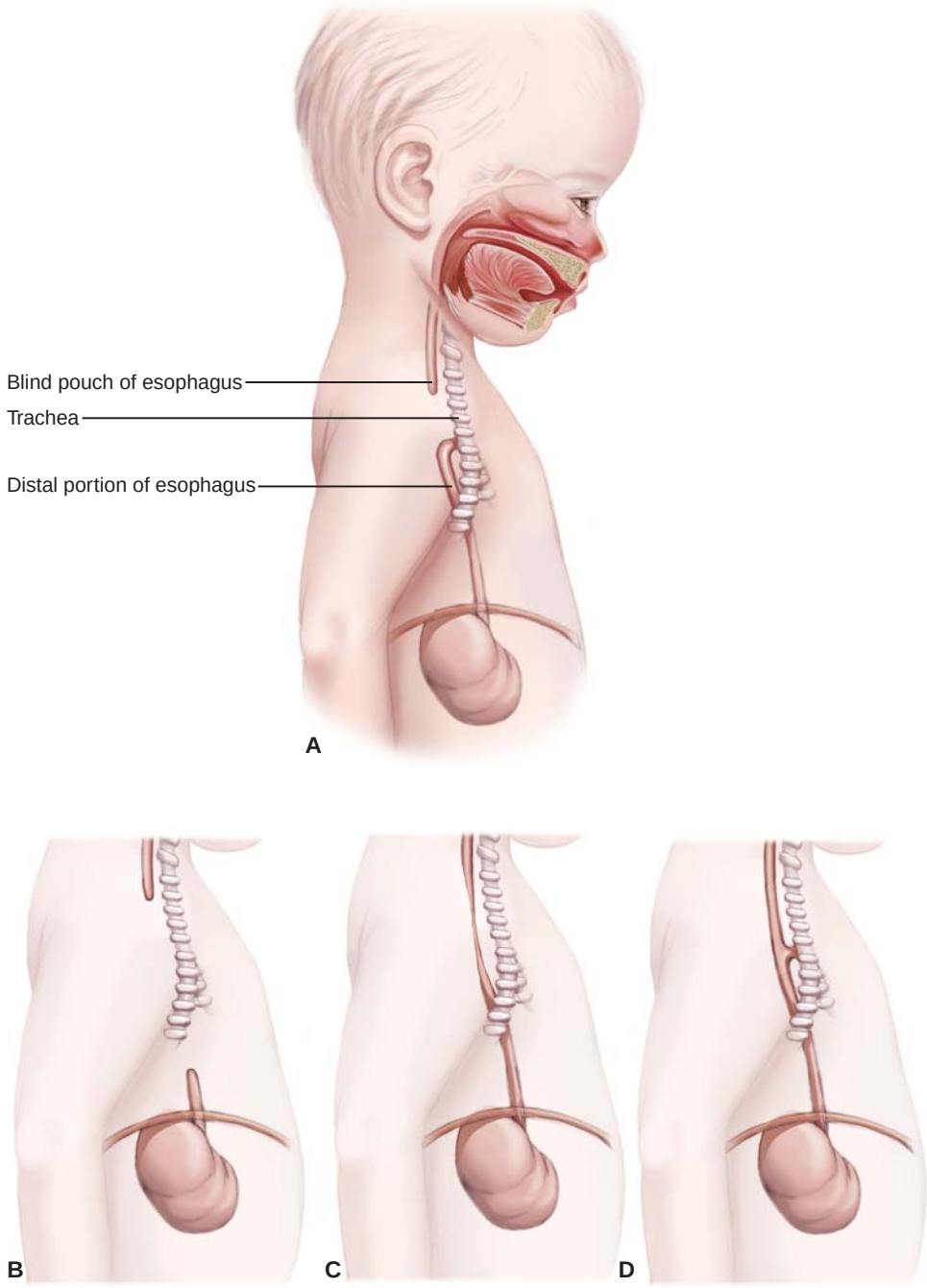
Surgery consists of closing the fistula and joining the two esophageal segments. Postoperative care involves close observation of all systems to identify any complications. Total parenteral nutrition and antibiotics are commonly used until the esophageal anastomosis is proven intact and patent. Then oral feedings are usually started within a week after surgery (Verklan & Walden, 2004). Keep the parents informed of their newborn's condition and progress. Demonstrate and reinforce all teaching prior to discharge.

Omphalocele and Gastroschisis

Omphalocele and gastroschisis are congenital anomalies of the anterior abdominal wall. An **omphalocele** is a defect of the umbilical ring that allows evisceration of abdominal contents into an external peritoneal sac. Defects vary in size; they may be limited to bowel loops or may include the entire gastrointestinal tract and liver (Fig. 24-13). Bowel malrotation is common, but the displaced organs are usually normal. Omphaloceles are associated with other anomalies in more than 70% of the cases. This anomaly is usually detected during routine prenatal ultrasound of the fetus or during investigation of an increased alpha-fetoprotein level (Khan & Thomas, 2004).

Gastroschisis is a herniation of abdominal contents through an abdominal wall defect, usually to the left or right of the umbilicus (Haws, 2004). Gastroschisis differs from omphalocele in that there is no peritoneal sac protecting the herniated organs, and thus exposure to amniotic fluid makes them thickened, edematous, and inflamed (Verklan & Walden, 2004). Gastroschisis is associated with significant newborn mortality and morbidity rates. Despite surgical correction, feeding intolerance, failure to thrive, and prolonged hospital stays occur in nearly all newborns with this anomaly (Laughon et al., 2003).

Factors associated with high-risk pregnancies, such as maternal illness and infection, drug use, smoking, and

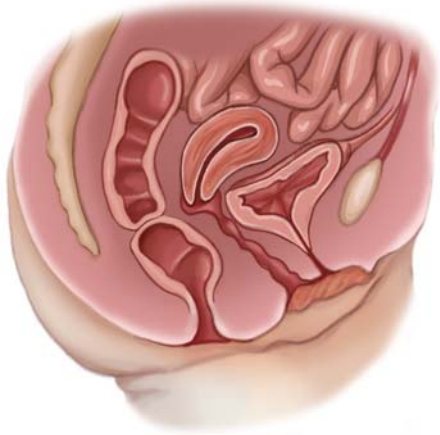


● Figure 24-12 Esophageal atresia and tracheoesophageal fistula. (**A**) The most common type of esophageal atresia, in which the esophagus ends in a blind pouch and a fistula connects the trachea with the distal portion of the esophagus. (**B**) The upper and distal portions of the esophagus end in a blind pouch. (**C**) The esophagus is one segment, but a portion of it is narrowed. (**D**) The upper portion of the esophagus connects to the trachea via a fistula.

genetic abnormalities, are also associated with omphalocele and gastroschisis. These factors contribute to placental insufficiency and the birth of a small-for-gestational-age or preterm newborn, the populations in which both of these abdominal defects most commonly occur. The combined incidence of both congenital abdominal wall anomalies is 1 in 2,000 births (Glasser, 2003).

Nursing care of newborns with omphalocele or gastroschisis focuses on preventing hypothermia, maintaining perfusion to the eviscerated abdominal contents by minimizing fluid loss, and protecting the exposed abdominal contents from trauma and infection. These objectives can be accomplished by placing the infant in a sterile drawstring bowel bag that maintains a sterile environment for the exposed contents, allows visualization, reduces heat and moisture loss, and allows heat from radiant warmers to reach the newborn. The newborn is placed feet-first into the bag and the drawstring is secured around the torso (Lockridge et al., 2002). Strict sterile technique is necessary to prevent contamination of the exposed abdominal

● Figure 24-13 Omphalocele in a newborn. Note the large, protruding sac.



● Figure 24-14 Imperforate anus in which the rectum ends in a blind pouch.

contents. An orogastric tube attached to low suction is used to prevent intestinal distention. Intravenous therapy is administered to maintain fluid and electrolyte balance and provide a route for antibiotic therapy. Monitor the newborn's fluid status frequently. Closely observe the exposed bowel for vascular compromise, such as changes in color or a decrease in temperature, and report immediately.

Surgical repair of both defects occurs after initial stabilization and comprehensive evaluation for any other anomalies. It may have to occur in stages, depending on the defect. Postoperative care involves providing pain management, monitoring respiratory and cardiac status, monitoring intake and output, assessing for vascular compromise, maintaining the orogastric tube to suction, documenting the amount and color of drainage, and administering ordered medications and treatments (Haws, 2004).

Throughout the entire time after birth, the parents need continued support and progress reports on their newborn. Encourage the parents to touch the newborn and participate in care if possible. Because of the nature of this defect, bonding opportunities will be limited initially, but visiting should be permitted and strongly encouraged. Provide information about the defect, treatment modalities, prognosis, and home care instructions.

Imperforate Anus

An imperforate anus is a gastrointestinal system malformation of the anorectal area that may occur in several forms. The rectum may end in a blind pouch that does not connect to the colon or it may have fistulas (openings) between the rectum and the perineum, the vagina in girls or the urethra in boys (Fig. 24-14). The malformations occur during early fetal development and are associated with anomalies in other body systems.

Imperforate anus occurs in about 1 of every 5,000 live births (Hart, 2005). The defect can be further classified as a high or low type, depending on its level. The level significantly influences the outcome in terms of fecal continence as well as management (Molmenti, 2004).

Surgical intervention is needed for both high and low types of imperforate anus. Surgery for a high type of defect involves a colostomy in the newborn period, with corrective surgery performed in stages to allow for growth. Surgery for the low type of anomaly, which frequently includes a fistula, involves closure of the fistula, creation of an anal opening, and repositioning of the rectal pouch into the anal opening. A major challenge for either type of surgical repair is finding, using, or creating adequate nerve and muscle structures around the rectum to provide for normal evacuation.

Preoperatively, nursing care focuses on maintaining NPO status and gastric decompression and administering intravenous therapy and antibiotic therapy as ordered. Provide a full explanation of the defect, surgical options, potential complications, typical postoperative course, and long-term care needed to the parents. Make sure they are aware of the available treatment modalities.

Postoperative care includes providing pain relief, maintaining NPO status and gastric decompression until normal bowel function is restored, and providing colostomy care if applicable.

Genitourinary System Structural Anomalies

Although genitourinary structural anomalies typically are not life-threatening, they do pose problems. When a newborn has a structural anomaly involving the genitourinary system, parents begin to think about continence issues and the reproductive ability of their offspring. Discussing long-term outcomes immediately after the defect is diagnosed is often difficult. The nurse must provide continued support and teaching and give honest answers to the parents' questions. Developing a therapeutic relationship

with the parents is important to allay their anxieties and fears during this stressful period.

Hypospadias

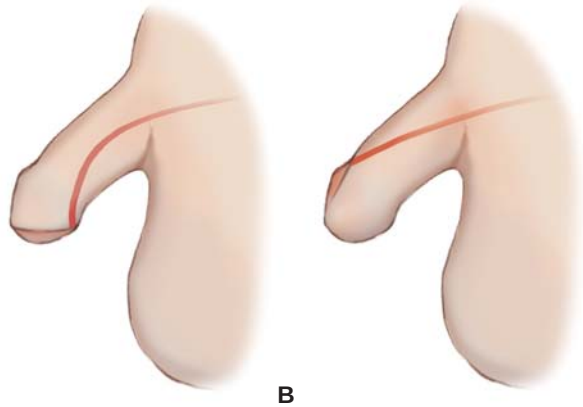
Hypospadias involves an abnormal positioning of the urinary meatus on the underside of the penis (Fig. 24-15A). The degree of hypospadias depends on the location of the opening. It is often accompanied by a downward bowing of the penis (chordee), which can lead to urination and erection problems in adulthood (CDC, 2004).

Hypospadias is a relatively common birth defect that occurs in approximately 1 of every 300 male births in the United States (Gatti & Kirsch, 2003). The malformation is the result of incomplete fusion of the urethral folds, which usually occurs between 9 and 12 weeks of gestation (Porter et al., 2005). The cause is unknown, but it is thought to be of multifactorial inheritance, because it occasionally occurs in more than one male in the same family.

Hypospadias can be corrected surgically. Depending on the severity, the correction can be completed in one or more procedures with good results. Surgical intervention should be completed during the first year of life to prevent any body image problems in the child.

Epispadias

Epispadias is a rare congenital genitourinary defect occurring in 1 of 117,000 male births and 1 of 484,000 female births (Gilbert, 2004). The condition is usually diagnosed at birth or shortly thereafter. In boys with epispadias, the urethra generally opens on the top or side rather than the tip of the penis. In females, the urinary meatus is located between the clitoris and the labia. This anomaly often occurs in conjunction with exstrophy of the bladder (McKinney et al., 2005). Surgical correction is necessary, and affected male newborns should not be circumcised (Lowdermilk & Perry 2004; see Fig. 24-15B).



● Figure 24-15 Genitourinary tract structural anomalies. (**A**) Hypospadias. (**B**) Epispadias.

Bladder Exstrophy

In bladder exstrophy, the bladder protrudes onto the abdominal wall because the abdominal wall failed to close during embryonic development. Wide separation of the rectus muscles and the symphysis pubis accompanies this defect. Virtually all affected male infants have associated epispadias. The upper urinary tract is usually normal. The incidence is approximately 1 in 24,000 to 40,000 live births (Botwinski, 2004).

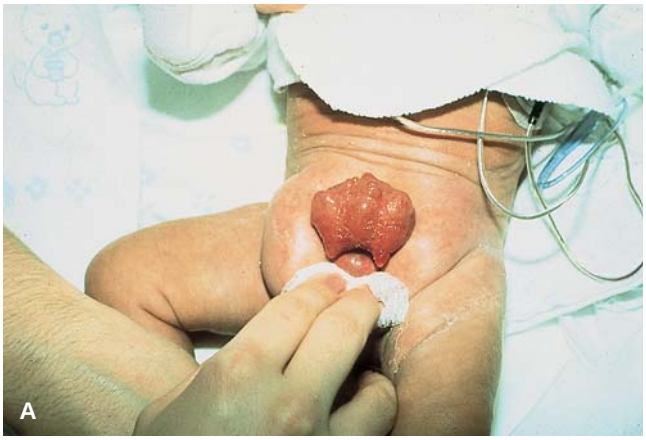
Treatment is surgical reconstruction performed in several stages. Goals of therapy include restoring urinary continence, preserving renal function, and reconstructing functional and cosmetically acceptable genitalia. Initial bladder closure is completed within 48 hours of birth, with epispadias repair taking place at the same time if possible. Surgery to reconstruct the bladder neck and reimplant the ureters is performed at about 2 to 3 years of age. Some children require permanent urinary diversion because a functional bladder cannot be reconstructed (Blaivas, 2004) (Fig. 24-16).

Nursing care for the newborn with bladder exstrophy includes the following activities:

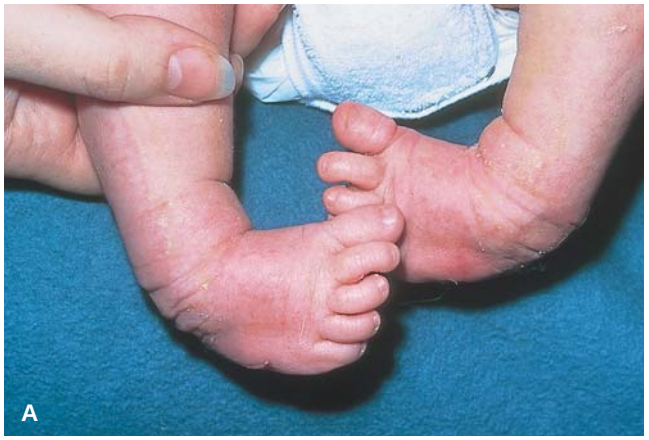
- Identify the genitourinary defect at birth so that immediate treatment can be provided.
- Cover the exposed bladder with a sterile clear non adherent dressing to prevent hypothermia and infection.
- Irrigate the bladder surface with sterile saline after each diaper change to prevent infection.
- Assist with insertion and monitoring of a suprapubic catheter to drain the bladder and prevent obstruction.
- Administer antibiotic therapy as ordered to prevent infection.
- Schedule diagnostic tests to assess for additional anomalies.
- Assess the newborn frequently for any signs of infection.
- Maintain modified Bryant traction for immobilization after surgery.
- Administer antispasmodics, analgesics, and sedatives as ordered to prevent bladder spasm and provide comfort.
- Educate the parents about the care of the urinary catheter at home if applicable.
- Support the parents throughout.
- Promote bonding by encouraging the parents to visit and touch the newborn.
- Refer the parents to a support group to enhance their coping ability.
- Be a therapeutic listener to the family (Verklan & Walden, 2004).

Musculoskeletal System Structural Anomalies

Clubfoot and developmental dysplasia of the hip, two common congenital anomalies of the musculoskeletal system, can hamper the child's ability to become mobile.



● Figure 24-16 Bladder exstrophy. (**A**) Before surgical correction. (**B**) After surgery.



● Figure 24-17 Clubfoot deformity. (**A**) Initial appearance. (**B**) Application of cast.

These anomalies can be identified with a careful examination at birth or soon after and need early intervention. Treatment is typically successful.

Congenital Clubfoot

Clubfoot, or talipes equinovarus, is a congenital deformity that typically has four components: inversion and adduction of the forefoot, inversion of the heel and hindfoot, limitation of extension of the ankle and subtalar joint, and internal rotation of the leg (Gore & Spencer, 2004). Reducing or eliminating all of the components of the deformity is the goal to ensure that the newborn has a functional, mobile, painless foot that does not require the use of special or modified shoes (Faulks & Luther, 2005).

The incidence of clubfoot is approximately 1 case per 1,000 live births in the United States. It is bilateral in about half of the cases and affects boys slightly more often than girls (Patel & Herzenberg, 2005) (Fig. 24-17). Clubfoot is a complex, multifactorial deformity with genetic and intrauterine factors. Heredity and race seem to factor into the incidence, but the means of transmission and the etiology are unknown. Most newborns who have clubfoot have no identifiable genetic, syndromal, or extrinsic cause.

On examination, the foot appears "down and in." It is smaller, with a flexible, softer heel because of the hypoplastic calcaneus. The heel is internally rotated, making the soles of the feet face each other when the deformity occurs bilaterally.

Clubfoot can be classified into extrinsic (supple) type, which is essentially a severe positional or soft tissue deformity, or intrinsic (rigid) type, where manual reduction is not possible. The type of clubfoot deformity determines the treatment course. Treatment for the extrinsic (supple) type consists of serial casting, followed by maintenance splinting. Treatment for the intrinsic (rigid) type includes initial casting followed by surgery. It is generally agreed that the initial treatment should be nonsurgical and started soon after birth.

Treatment for either type starts with serial casting, which is needed due to the rapid growth of the newborn. Casts initially are changed weekly and are applied until the deformity responds and is fully corrected. If serial casting is not successful in correcting the deformity, sur-

gical intervention is necessary between 4 and 9 months of age (Morcuenda et al., 2004).

Nursing management focuses on education, anticipatory guidance, and pain management. Educate the parents about their newborn's condition and the treatment protocol to reduce their anxiety, and provide reassurance that the clubfoot is not painful and will not hinder the child's development. Discuss challenges associated with sleep, play, and dressing. Inform them that slight modifications will be necessary to accommodate the plaster casts. Review positioning, bathing, and skin care along with pain management when new casts are applied. Stress the need to provide a calm, quiet environment to promote relaxation and sleep for their newborn.

Developmental Dysplasia of the Hip

Developmental dysplasia of the hip (DDH) involves abnormal growth or development of the hip that results in instability. This includes hips that are unstable, subluxated, or dislocated (luxated) or have a malformed acetabulum. This instability allows the femoral head to become easily displaced from the acetabulum. Typically, the newborn with DDH is otherwise healthy, usually without any other deformities.

The etiology of DDH is not clear, but associated factors include racial background (Native Americans), genetic transmission (runs in families), intrauterine positioning (breech), sex (female), oligohydramnios, birth order (first-born), and postnatal infant-carrying positions (swaddling, which forces the hips to be adducted). The incidence of hip instability is about 10 per 1,000 live births (McCarthy, 2005).

DDH is frequently not identified during the newborn examination. Newborns require careful evaluation for hip dysplasia at subsequent visits throughout the first year. Two maneuvers are used to assess hip instability in the newborn: Ortolani and Barlow (see Chapter 18 for more information on performing these maneuvers). Ortolani's maneuver elicits the sensation of the dislocated hip reducing; Barlow's maneuver detects the unstable hip dislocating from the acetabulum. Additional physical signs of DDH include an symmetric number of skin folds on the thigh or buttock, an apparent or true short leg, and limited hip abduction (Fig. 24-18; Hernandez & Glass, 2005).

Treatment is started as soon as DDH is identified to bring about a more favorable outcome. If a positive Ortolani or Barlow sign is found on the newborn examination, the newborn is referred to an orthopaedist. The goal of treatment is to relocate the femoral head in the acetabulum to facilitate normal growth and development. The Pavlik harness is the most widely used device; it prevents adduction while allowing flexion and abduction to accomplish the treatment goal (Fig. 24-19). The harness is worn continuously until the hip is stable, which make take several months. If harnessing is not successful, surgery is necessary.

Nursing care related to DDH starts with recognition of the disorder and early reporting to the health care provider. Early diagnosis is the crucial aspect; education also is key. Teach the parents how to care for their newborn while in the harness during treatment. Proper fit and adjustments for growth are essential for successful treatment. Frequent clinical assessment on an outpatient basis is needed to monitor progress. Through education, the nurse can be very effective in helping the parents to stay compliant with treatment.

KEY CONCEPTS

- Asphyxia, the most common clinical insult in the perinatal period, results in brain injury and may lead to mental retardation, cerebral palsy, or seizures.

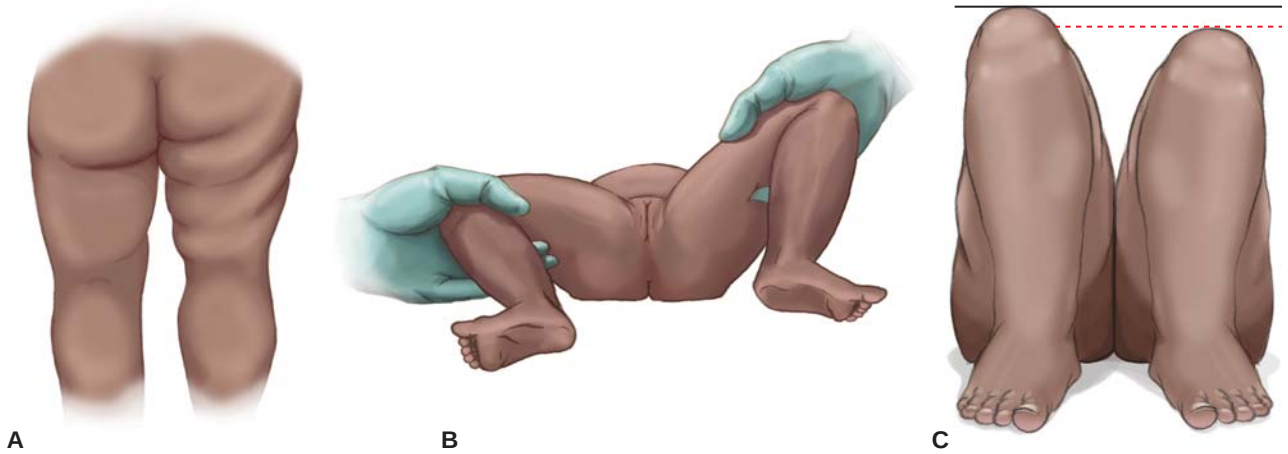
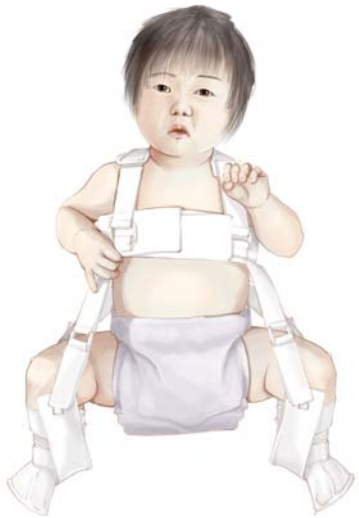


Figure 24-18 Characteristics of developmental dysplasia of the hip. (**A**) Asymmetric number of skin folds on the thigh or buttock. (**B**) Limited hip abduction. (**C**) Appearance of short leg.



● Figure 24-19 The Pavlik harness to treat developmental dysplasia of the hip.

- Transient tachypnea of the newborn occurs when the liquid in the lung is removed slowly or incompletely.
- Common risk factors for respiratory distress syndrome (RDS) include young gestational age, perinatal asphyxia regardless of gestational age, cesarean birth in the absence of labor (related to the lack of thoracic squeeze), male gender, and maternal diabetes.
- Meconium aspiration has three major pulmonary effects: airway obstruction, surfactant dysfunction, and chemical pneumonitis.
- The management of persistent pulmonary hypertension of the newborn requires meticulous attention to detail, with continuous monitoring of oxygenation, blood pressure, and perfusion.
- Bronchopulmonary dysplasia is a newborn lung disease that follows a lung injury secondary to mechanical ventilation and oxygen toxicity.
- Retinopathy of prematurity (ROP) is a developmental abnormality that affects the immature vasculature of the retina: abnormal growth of blood vessels (neovascularization) takes place within the retina and vitreous.
- Periventricular/intraventricular hemorrhage is bleeding that usually originates in the subependymal germinal matrix region of the brain with extension into the ventricular system.
- Necrotizing enterocolitis (NEC) is a serious gastrointestinal disease of unknown etiology in newborns that can result in necrosis of a segment of the bowel.
- Infants of diabetic mothers are at risk for malformations most frequently involving the cardiovascular, skeletal, central nervous, gastrointestinal, and genitourinary systems; cardiac anomalies are the most common.
- Factors that place the newborn at risk for birth trauma include cephalopelvic disproportion, maternal pelvic anomalies, oligohydramnios, prolonged or rapid labor, abnormal presentation, fetal prematurity, fetal macrosomia, and fetal abnormalities.
- Women who use drugs during their pregnancy expose their unborn child to the possibility of intrauterine growth restriction, prematurity, neurobehavioral and neurophysiologic dysfunction, birth defects, infections, and long-term developmental sequelae.
- Newborns of women who abuse tobacco, illicit substances, caffeine, and alcohol can exhibit withdrawal behavior.
- Physiologic jaundice is a common, normal newborn phenomenon that appears during the second or third day of life and then declines over the first week after birth. Pathologic jaundice is manifested within the first 24 hours of life when total bilirubin levels increase by more than 5 mg/dL/day and the total serum bilirubin level is higher than 17 mg/dL in a full-term infant.
- Newborn infections are usually classified according to the time of onset and grouped into three categories: congenital infection, acquired in utero by vertical transmission with onset before birth; early-onset neonatal infections, acquired by vertical transmission in the perinatal period, either shortly before or during birth; and late-onset neonatal infections, acquired by horizontal transmission in the nursery.
- There are several ways to classify pathogens that cause neonatal sepsis; typically three categories are used—bacterial, TORCH, and fungal.
- Congenital conditions, often referred to as birth defects, do not follow a recognized classical Mendelian inheritance pattern. Most have a complex etiology, involving many interacting genes, gene products, and social and environmental factors that may lead to structural malformations.
- Congenital heart disease is commonly classified physiologically as defects that result in increased pulmonary blood flow, defects that result in decreased pulmonary blood flow, defects that cause obstruction to blood flow out of the heart, and defects with cyanosis and increased pulmonary blood flow or mixed defects.
- Inborn errors of metabolism are genetic disorders that disrupt normal metabolic function. They are individually rare but collectively are responsible for significant levels of infant mortality and morbidity.
- Congenital structural anomalies may be inherited or sporadic, isolated or multiple, apparent or hidden, gross or microscopic. They cause nearly half of all deaths in term newborns and cause long-term sequelae for many.
- A worldwide decline in neural tube defects has occurred over the past few decades due to improved prevention secondary to preconception folic acid supplementation, maternal serum alpha-fetoprotein

monitoring, and use of ultrasonography and amniocentesis.

- The birth of an infant who has a genetic condition, a syndrome, or a structural congenital anomaly is often a shocking and traumatic experience for the parents. Nurses play a primary role in setting the stage for the acceptance and adjustment process through their interaction with parents.

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Web Resources

AHRQ's Tobacco Pathfinder: **www.ahrq.gov**
American Cleft Palate Association: **www.cleftline.org**
American Diabetes Association: **www.diabetes.org**
American Society of Plastic Surgeons: **www.plasticsurgery.org**
Anencephaly Support Foundation: **www.asfhelp.com**
Association for Bladder Exstrophy Community: **www.bladderexstrophy.com**
Association of Retinopathy of Prematurity: **www.ropard.org**
Birth Defects for Children: **www.birthdefects.org**
Centers for Disease Control and Prevention: **www.cdc.gov**
Congenital Heart Defects: **www.congenitalheartdefects.com**
Esophageal Atresia/Tracheoesophageal Fistula Family Support Connection: **www.eatef.org**
Maple Syrup Urine Disease Family Support Group: **www.msud-support.org**
March of Dimes Birth Defects Foundation: **www.marchofdimes.com**
Narcotics Anonymous: **www.na.org**
National Association for Continence: **www.nafc.org**
National Center on Birth Defects and Developmental Disabilities: **www.cdc.gov/ncbddd/fas**
National Clearinghouse for Alcohol and Drug Abuse Information: **www.health.org**
National Eye Institute: **www.nei.nih.gov**
National Institute on Alcohol Abuse and Alcoholism: **www.niaaa.nih.gov**
National Organization for Rare Disorders (NORD): **www.rarediseases.org**
National Organization on Fetal Alcohol Syndrome: **www.nofas.org**
National Women's Health Information Center: **www.4woman.gov**
Neonatal Network: **www.neonatalnetwork.com**
Parental Guide for Developmentally Supportive Care: **www.comeunity.com/premature/baby/supportive-care.html**
Parents of Galactosemic Children, Inc.: **www.galactosemia.org**
Partnership for a Drug-Free America: **www.drugfreeamerica.org**
Physical and Developmental Environment of the High-Risk Infant: **www.med.usf.edu/~tsinger**
Safe Motherhood Initiative: **www.safemotherhood.org**
SHARE Pregnancy & Infant Loss Support, Inc.: **www.nationalshareoffice.com**
Spina Bifida Association of America: **www.sbaa.org**
Substance Abuse & Mental Health Services: **www.findtreatment.samhsa.gov**

ChapterWORKSHEET

MULTIPLE CHOICE QUESTIONS

1. Which findings would lead the nurse to suspect newborn respiratory distress?
   a. Abdominal distention
   b. Acrocyanosis
   c. Depressed fontanels
   d. Nasal flaring
2. When assessing the substance-exposed newborn, the nurse would expect to find:
   a. Calm facial appearance
   b. Daily weight gain
   c. Increasing irritability
   d. Feeding and sleeping well
3. Which newborn condition might be overlooked if there is inadequate ingestion of protein prior to screening?
   a. Congenital hypothyroidism
   b. Sickle cell anemia
   c. Cystic fibrosis
   d. Phenylketonuria (PKU)
4. A newborn with tracheoesophageal fistula is likely to present with which assessment finding?
   a. Subnormal temperature
   b. Absent Moro reflex
   c. Inability to swallow
   d. Foamy bubbles or drooling from mouth

CRITICAL THINKING EXERCISES

1. As the nursery nurse, you receive a newborn from the labor and birth suite and place him under the radiant warmer. The nurse who gives you report states that the mother couldn't remember when her membranes broke before labor and that she ran a fever during labor for the past few hours. The Apgar scores were good, but the newborn seemed lethargic. As you begin your assessment, you note that she is pale and floppy and has a subnormal temperature; heart rate is 180 bpm and respiratory rate is 70 breaths per minute.
   a. What in the mother's history should raise a red flag to the nurse?
   b. What condition is this neonate at high risk for?
   c. What interventions are appropriate for this condition?
2. Terry, a day-old baby girl, is very fretful, and calming measures don't seem to work. As the nursery nurse you notice that she is losing weight and her formula intake is poor, even though she is manifesting hungry behavior. The mother received no prenatal care and denied drug use, but her drug screen was positive for heroin.
   a. What additional information do you need to obtain from the mother?
   b. What additional laboratory work might be needed for Terry?
   c. What specific measures need to be made for her ongoing care?
3. A baby girl weighing 7.5 pounds was born after a gestation of 41 weeks by cesarean section. She is brought to the nursery. On your initial assessment you find a floppy infant with short stature, dry, brittle hair, a short, thick neck, dull-appearing facial features, a thick, protruding tongue, and a hoarse-sounding cry.
   a. What is your impression of this newborn?
   b. What laboratory studies and results would you anticipate?
   c. What explanation could be offered to the parents concerning this condition?

STUDY ACTIVITIES

1. Arrange for a tour of a regional NICU to see the nurse's role in caring for sick neonates. Ask the nurse to give a quick history of each newborn's condition. Was the nurse's role like you imagined? What was your impression of the NICU, and how would you describe it to expectant parents?
2. Select a website from the list at the end of the chapter. What kind of information is given? How helpful would it be for parents with an infant diagnosed with a specific condition?
3. Contact a local childbirth educator and ask permission to present a brief informational session to expectant parents at a natural childbirth class on the importance of newborn screening tests. What should be your "take-home message" to them?
4. A herniation of a newborn's abdominal contents present at birth describes _____.
5. An abnormal opening between the ventricles of the heart, a common cardiac defect found in infants of diabetic mothers, is _____.