CHAPTER 10

The High-Risk Newborn and Family

Debbie Fraser Askin and David Wilson

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**GENERAL MANAGEMENT OF HIGH-RISK NEWBORNS**

**IDENTIFICATION OF HIGH-RISK NEWBORNS**

The high-risk neonate is defined as a newborn, regardless of gestational age or birth weight, who has a greater-than-average chance of morbidity or mortality, usually because of conditions or circumstances superimposed on the normal course of events associated with birth and the adjustment to extraterine existence. The high-risk period begins at the time of viability (the gestational age at which survival outside the uterus is believed to be possible, or as early as 23 weeks of gestation) up to 28 days after birth and includes threats to life and health that occur during the prenatal, perinatal, and postnatal periods.

There has been increased interest in late-preterm infants of 34 to 36 weeks of gestation who may receive the same treatment as term infants. Wang, Dorer, Fleming, and colleagues (2004) emphasize that late-preterm infants often experience similar morbidities to preterm infants: respiratory distress, hypoglycemia requiring treatment, temperature instability, poor feeding, jaundice, and discharge delays as a result of illness. Therefore assessment and prompt intervention in life-threatening perinatal emergencies often make the difference between a favorable outcome and a lifetime of disability. The nurse in the newborn nursery is familiar with the characteristics of neonates and recognizes the significance of serious deviations from expected observations. When providers can anticipate the need for specialized care and plan for it, the probability of successful outcome is increased.

**Late-Preterm Infant**

Within the past two decades, several significant changes have occurred in neonatal care. Early postpartum discharge for term and preterm infants gained popularity as health care institutions attempted to cut health care costs. Another change occurred in newborn care, as infants who appeared to be “near” term began to be treated much like term infants, thus avoiding the costs of neonatal intensive care for infants who appeared to be healthy. Experts have recommended that infants born between 34 and 36/7 weeks of gestation be referred to as late-preterm infants rather than near-term infants (Engle, 2006; Engle, Tomashek, Wallman, et al, 2007). Late-preterm infants may be able to make an effective transition to extraterine life; however, such infants, by nature of their limited gestation, remain at risk for problems related to feeding, neurodevelopment, thermoregulation, hypoglycemia, hyperbilirubinemia, sepsis, and respiratory function (Bakewell-Sachs, 2007; Darcy, 2009). In one study children born at 34 to 36 weeks were more than three times as likely as children born at term to be diagnosed with cerebral palsy (CP) (Petrini, Dias, McCormick, et al, 2009). It is now estimated that late-preterm infants represent 70% of the total preterm infant population and that the mortality rate for this group is significantly higher than that of term infants (7.9 versus 2.4 per 1000 live births, respectively) (Tomashes, Shapiro-Mendoza, Davidoff, et al, 2007). Because late-preterm infants’ birth weights often range from 2000 to 2500 g (4.4 to 5.5 lb) and they appear relatively mature in comparison to smaller preterm infants, they may be cared for in the same manner as healthy term infants, while risk factors for late-preterm infants are overlooked. Late-preterm infants are often discharged early from the birth institution and have a significantly higher rate of rehospitalization than term infants (Escobar, Clark, and Greene, 2006). Discussions regarding high-risk infants in this chapter also refer to late-preterm infants who are experiencing a delayed transition to extraterine life.

The Association of Women’s Health, Obstetric and Neonatal Nurses has published the Late Preterm Infant Assessment Guide (Askin, Bakewell-Sachs, Medoff-Cooper, et al, 2007) for the education of perinatal nurses regarding the late-preterm infant’s risk factors and appropriate care and follow-up care (Table 10-1).

**Classification of High-Risk Newborns**

High-risk infants are most often classified according to birth weight, gestational age, and predominant pathophysiologic problems. The more common problems related to physiologic status are closely associated with the infant’s state of maturity and usually involve chemical disturbances (e.g., hypoglycemia, hypocalcemia) and consequences of immature organs and systems (e.g., hyperbilirubinemia, respiratory distress, hypothermia). Box 10-1 outlines specific terminology describing the developmental status of the newborn.

Formerly, weight at birth reflected a reasonably accurate estimation of gestational age; that is, if an infant’s birth weight...
This chapter.


*This is not an exhaustive list of nursing interventions; additional interventions include those discussed under the care of the high-risk infant in this chapter.

exceeded 2500 g (5.5 lb), the infant was considered to be mature. However, accumulated data have shown that intraterine growth rates are not the same for all infants and that other factors (e.g., heredity, placental insufficiency, and maternal disease) influence intraterine growth and the infant’s birth weight. From these data a more definitive and meaningful classification system that encompasses birth weight, gestational age, and neonatal outcome has been developed. It has also been determined that the lowest perinatal mortality occurs in the infant who weighs between 3000 and 4000 g (6.4 and 8.8 lb) and whose gestational age is more than 36 weeks and less than 42 weeks (Walsh and Fanaroff, 2006). (See Fig. 8-2 for size comparison of newborn infants.)

Many perinatal problems can be anticipated before delivery. Prenatal testing and labor monitoring have reduced the incidence of perinatal mortality, and specialized care of the distressed newborn is improving the survival rate. If the infant is likely to require special therapy at or soon after birth, plans can be made for the delivery to take place at a hospital with the facilities to provide such care. This eliminates delay in initiating needed care and averts some of the hazards associated with transporting the sick newborn. Prenatal evaluation of fetal wellbeing and advanced surgical and anesthetic techniques have made intraterine treatment of certain pathologic conditions possible, thus enhancing the neonate’s chances for survival (Reed and Blumer, 2006).

### TABLE 10-1 LATE-PRETERM INFANT ASSESSMENT AND INTERVENTIONS

<table>
<thead>
<tr>
<th>RISK FACTORS</th>
<th>ASSESSMENT</th>
<th>INTERVENTIONS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory distress</td>
<td>Assess for cardinal signs of respiratory distress (nasal flaring, grunting, tachypnea, central cyanosis, retractions) and presence of apnea, especially during feedings.</td>
<td>Perform gestational age assessment. Observe for signs of respiratory distress; monitor oxygenation by pulse oximetry; provide supplemental oxygen judiciously.</td>
</tr>
<tr>
<td>Thermal instability</td>
<td>Monitor axillary temperature every 30 min immediately postpartum until stable; thereafter every 1-4 hr depending on gestational age and ability to maintain thermal stability.</td>
<td>Provide skin-to-skin care in immediate postpartum period for stable infant. Implement measures to avoid excess heat loss (adjust environmental temperature, avoid drafts). Bathe only after thermal stability has been maintained for 1 hr. Initiate early feedings of human milk or formula. Avoid dextrose water or water feedings. Provide IV dextrose as necessary for hypoglycemia.</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Monitor for signs and symptoms of hypoglycemia. Assess feeding ability (latch-on, nipple-feeding). Assess thermal stability and signs and symptoms of respiratory distress. Monitor bedside glucose in infants with additional risk factors (IDM, prolonged labor, respiratory distress, poor feeding).</td>
<td>Monitor transcutaneous bilirubin and note risk zone on hour-specific nomogram (see Fig. 8-6).</td>
</tr>
<tr>
<td>Jaundice</td>
<td>Observe for jaundice in first 24 hr. Evaluate maternal-fetal history for additional risk factors that may cause increased hemolysis and circulating levels of unconjugated bilirubin (Rh, ABO, spherocytosis, bruising). Assess feeding method, voiding and stooling patterns.</td>
<td>Initiate early feedings (human milk or formula). Ensure maternal knowledge of feeding method and signs of inadequate feeding (sleepiness, lethargy, color changes during feeding, apnea during feeding, decreased or absent urine output).</td>
</tr>
<tr>
<td>Feeding problems</td>
<td>Assess suck-swallow and breathing. Assess for respiratory distress, hypoglycemia, thermal stability. Assess latch-on, maternal comfort with feeding method. Determine weight loss (should be ≤10% of birth weight).</td>
<td>Perform newborn screening, including hearing test. Implement individualized developmental care. Encourage parents to keep follow-up appointments with primary care provider for evaluation of growth and development (including cognitive function and achievement of appropriate milestones).</td>
</tr>
<tr>
<td>Neurodevelopmental problems</td>
<td>Assess for respiratory distress, neonatal jaundice, hypoglycemia, and thermal instability. Assess neurodevelopmental status. Assess for seizure activity.</td>
<td>Use Standard Precautions, especially hand washing between infants and contact with surfaces that may harbor bacteria (e.g., keyboards, telephones). Maintain thermal stability. Administer hepatitis B vaccine. Encourage breast-feeding and assist mother-baby pair with breast-feeding. Encourage parents to decrease infant exposure to respiratory viruses postdischarge and obtain vaccines as appropriate to prevent development of respiratory viruses (e.g., influenza).</td>
</tr>
<tr>
<td>Infection</td>
<td>Evaluate maternal-fetal history for risk factors that may contribute to neonatal sepsis. Assess for signs and symptoms of neonatal infection.</td>
<td>Use Standard Precautions, especially hand washing between infants and contact with surfaces that may harbor bacteria (e.g., keyboards, telephones). Maintain thermal stability. Administer hepatitis B vaccine. Encourage breast-feeding and assist mother-baby pair with breast-feeding. Encourage parents to decrease infant exposure to respiratory viruses postdischarge and obtain vaccines as appropriate to prevent development of respiratory viruses (e.g., influenza).</td>
</tr>
</tbody>
</table>
heat warmers that provide a constant source of warmth and allow maximum access to the infant. Most important, advances in intensive care have created a need for highly skilled personnel trained in the art of neonatal intensive care.

The diversity of special care needs requires that the unit be arranged for graduated care of the infant population. There should be adequate facilities and skilled personnel to provide one-to-one nursing care for each seriously ill infant, as well as a means for graduation to one-to-three or one-to-four nursing care in a quieter area where infants require less intensive care until they are ready to be discharged to home. Family-centered care and a relatively quiet environment are often difficult to provide in a busy neonatal intensive care unit (NICU); therefore some units have developed step-down units and single-room units where high-risk infants may be observed by skilled staff. Such areas are designed for family-centered care along with appropriate neurodevelopmental care.

**Organization of Services**

The most efficient organization of services is a regionalized system of facilities within a designated geographic area. Neonatal intensive care facilities may provide three prescribed levels of care with special equipment, skilled personnel, and ancillary services concentrated in a centralized institution (American Academy of Pediatrics and American College of Obstetricians and Gynecologists, 2007):

- **Level I** facility—Provides management of normal maternal and newborn care.
- **Level II**A facility—Provides a full range of maternity and newborn care and can provide care to infants born at more than 32 weeks of gestation and weighing more than 1500 g (3.3 lb) who are moderately ill with problems that are expected to resolve rapidly and who are not anticipated to need subspecialty care; or who are convalescing after intensive care.
- **Level II**B facility—In addition to the above, can provide mechanical ventilation for up to 24 hours and can provide continuous positive airway pressure (CPAP).
- **Level III** facility—Neonatal intensive care
  - Level IIIA units provide care for infants with birth weight of more than 1000 g (2.2 lb) and gestational age of more than 28 weeks. Life support is limited to conventional mechanical ventilation.
  - Level IIIB units can provide care for extremely low–birth-weight (ELBW) infants with technology including high-frequency ventilation and inhaled nitric oxide, on-site access to pediatric medical subspecialists, and advanced diagnostic imaging and pediatric surgery available.
  - Level IIIC units have the capabilities of a level IIIB NICU and, in addition, offer extracorporeal membrane oxygenation (ECMO) and surgical repair of serious congenital cardiac malformations.

**Transporting High-Risk Newborns**

When an at-risk infant is identified or anticipated, arrangements are made for care in the intensive care facility. The uterus is the ideal transport unit for the infant with anticipated difficulties; therefore, whenever possible, take the mother where special care is available for her delivery.
Some infants develop difficulties after a seemingly normal pregnancy and uncomplicated labor. Because it is impossible to always predict when infants will require intensive care, a coordinated system is needed to ensure them an optimum opportunity for survival. Each hospital that delivers infants should be able to provide for appropriate neonatal stabilization and arrange for transport to a tertiary care facility. The infant must be kept warm, be adequately oxygenated (including intubation if indicated), have vital signs and oxygen saturation monitored, and, when indicated, receive an intravenous (IV) infusion. The infant is transported in a specially designed incubator unit that contains a complete life-support system and other emergency equipment that can be carried by ambulance, van, plane, or helicopter.

The transport team may consist of one or more of the highly trained persons from the NICU: a neonatologist (or a fellow in neonatology), a neonatal nurse practitioner, a respiratory therapist, and one or more nurses. The professional assigned to accompany the infant must be constantly alert to every change in the infant’s condition and able to intervene appropriately. The neonate who must be moved from one place to another within the hospital (e.g., to surgery, or from delivery room to nursery) is transported in an incubator or radiant warmer and accompanied by the necessary personnel and equipment.

### NURSING CARE OF HIGH-RISK NEWBORN

Because the majority of infants admitted to intensive care facilities are born before the estimated date of delivery, this chapter focuses primarily on the preterm infant. (See p. 344 for a description of the characteristics of preterm infants.) The incidence of neonatal complications (e.g., respiratory distress and hypoglycemia) is highest in this group, and often other high-risk factors (e.g., sepsis and congenital malformations) are found in association with prematurity. This chapter discusses nursing problems encountered in the intensive care nursery, then considers common complications. Nursing care of high-risk infants with more serious disorders is examined in relation to specific high-risk conditions.

### ASSESSMENT

At birth the newborn is given a cursory yet thorough assessment to determine any apparent problems and identify those that demand immediate attention. This examination is primarily concerned with the evaluation of cardiopulmonary and neurologic functions. The assessment includes the assignment of an Apgar score (see Chapter 8) and an evaluation for any obvious congenital anomalies or evidence of neonatal distress. The infant is stabilized and evaluated before being transported to the NICU for therapy and more extensive assessment. (See Clinical Assessment of Gestational Age, Chapter 8.)

A thorough, systematic physical assessment is an essential component in the care of the high-risk infant (see Nursing Care Guidelines box). Subtle changes in feeding behavior, activity, color, oxygen saturation (S\textsubscript{PO}$_2$), or vital signs often indicate an underlying problem. The preterm infant, especially the ELBW infant, is not able to withstand prolonged physiologic stress and may die within minutes of exhibiting abnormal symptoms if the underlying pathologic process is not corrected. The alert nurse is aware of subtle changes and reacts promptly to implement interventions that promote optimum function in the high-risk neonate. The nurse notes changes in the infant’s status through ongoing observations of the infant’s adaptation to the extrauterine environment.

Observational assessments of the high-risk infant are made according to the infant’s acuity (seriousness of condition); the critically ill infant requires close observation and assessment of respiratory function, including continuous pulse oximetry, electrolytes, and blood gases. Accurate documentation of the infant’s status is an integral component of nursing care. With the aid of continuous, sophisticated cardiopulmonary monitoring, nursing assessments and daily care can be coordinated to allow for minimum handling of the infant (especially the very low–birth-weight [VLBW] or ELBW infant) to decrease the effects of environmental stress.

### MONITORING PHYSIOLOGIC DATA

Most neonates under intensive observation are placed in a controlled thermal environment and monitored for heart rate, respiratory activity, and temperature. The monitoring devices are equipped with an alarm system that indicates when the vital signs are above or below preset limits. However, a “hands on” assessment, including auscultation of heart tones and breath sounds, is essential.

The placement of electrodes may be challenging because of the lack of flat areas on the neonate’s chest, the limited space for alternating sites, the size of the electrodes, and irritation from the adhesive. Hydrogel electrodes are gentler on the skin and are easily removed by lifting an edge from the skin and moistening it with plain water to release the adhesive (Lund and Durand, 2006). If the same electrode is reapplied to the skin, rinse the hydrogel with plain water to remove accumulated sodium from perspiration, which can eventually irritate the skin. It is important to follow the manufacturer’s directions for care and handling of electrodes to avoid malfunction or burns to sensitive skin.

Monitor blood pressure routinely in the sick neonate by either internal or external means. Direct recording with arterial catheters is often used but carries the risks inherent in any procedure in which a catheter is introduced into an artery. An umbilical venous catheter may also be used to monitor the neonate’s central venous pressure. Oscillometry (Dinamap) or Doppler transcutaneous apparatus is a simple, effective means for detecting alterations in systemic blood pressure (hypotension or hypertension). Table 10-2 lists normal blood pressure ranges for healthy preterm infants. Infants who have birth asphyxia, have low Apgar scores, or are mechanically ventilated have lower systolic and diastolic pressures.

In the NICU frequent laboratory examinations and their interpretation are integral parts of the ongoing assessment of infants’ progress. The nurse keeps accurate intake and output records on all acutely ill infants. An accurate output can be obtained by collecting urine in a plastic urine collection bag specifically made for preterm infants (see Urine Specimens, Chapter 27) or by weighing the diapers, which is the simplest and least traumatic means of measuring urinary output. The
**Physical Assessment**

**General Assessment**
Using electronic scale, weigh daily or as the baby’s condition dictates. Measure length and head circumference periodically. Describe general body shape and size, posture at rest, ease of breathing, presence and location of edema. Describe any apparent deformities. Describe any signs of distress (e.g., poor color, mottling, hypotonia).

**Respiratory Assessment**
Describe shape of chest (concave), symmetry, chest tubes, or other deviations. Describe use of accessory muscles: nasal flaring or substernal, intercostal, or suprasternal retractions. Determine respiratory rate and regularity. Auscultate and describe breath sounds: stridor, crackles, wheezing, diminished sounds, areas of absence of sound, grunting, stridor, diminished air entry, equality of breath sounds. Determine whether suctioning is needed.

Determine oxygen saturation by pulse oximetry and partial pressure of oxygen and carbon dioxide by transcutaneous oxygen (tcPO2) and transcutaneous carbon dioxide (tcPCO2).

**Cardiovascular Assessment**
Determine heart rate and rhythm. Describe heart sounds, including any murmurs. Determine the point of maximum intensity (PMI), the point at which the heart-beat sounds and palpates loudest (a change in the PMI may indicate a mediastinal shift). Describe infant’s color (abnormalities may be of cardiac, respiratory, or hematopoietic origin): cyanosis, pallor, plethora, jaundice, mottling. Assess color of mucous membranes and lips. Determine blood pressure. Indicate extremity used and cuff size. Describe peripheral pulses, capillary refill (<2 to 3 seconds), peripheral perfusion (mottling). Describe monitors, their parameters, and whether alarms are in “on” position.

**Gastrointestinal Assessment**
Determine presence of abdominal distention: increase in circumference, shiny skin, evidence of abdominal wall erythema, visible peristalsis, visible loops of bowel, status of umbilicus. Determine any signs of regurgitation and time related to feeding; character and amount of residue if gavage fed, if nasogastric tube in place, describe type of suction, drainage (color, consistency, pH, guaiac).

**Skin Assessment**
Determine axillary temperature. Determine relationship to environmental temperature. Describe color of mucous membranes and lips. Describe any rash, skin lesion, or birthmarks. Describe texture and turgor of skin: dry, smooth, flaky, peeling, etc. Determine any discoloration, reddened area, signs of irritation, blisters, abrasions, or denuded areas, especially where monitoring equipment, infusions, or other apparatus come in contact with skin; also check and note any skin preparation used (e.g., povidone-iodine). Determine texture and turgor of skin: dry, smooth, flaky, peeling, etc. Describe any rash, skin lesion, or birthmarks. Determine whether intravenous infusion catheter or needle is in place, and observe for signs of infiltration. Describe parenteral infusion lines: location, type (arterial, venous, peripheral, umbilical, central, peripherally inserted central catheter); type of infusion (medication, saline, dextrose, electrolytes, lipids, total parenteral nutrition); type of infusion pump and rate of flow; type of catheter or needle; and appearance of insertion site.

**Genitourinary Assessment**
Describe any abnormalities of genitalia. Describe urine amount (as determined by weight), color, pH, labstick findings, and specific gravity (to screen for adequacy of hydration). Check weight (the most accurate measure for assessment of hydration).

**Neurologic-Musculoskeletal Assessment**
Describe infant’s movements (random, purposeful, jittery, twitching, spontaneous, elicited); level of activity with stimulation; evaluate based on gestational age. Describe infant’s position or attitude: flexed, extended. Describe reflexes observed: Moro, sucking, Babinski, plantar, and other age-appropriate reflexes. Determine level of response and consolability. Determine changes in head circumference (if indicated); size and tension of fontanelles, suture lines. Determine pupillary responses in infant at or above 32 weeks of gestation.

**Temperature**
Determine axillary temperature. Determine relationship to environmental temperature.

Blood examinations are a necessary part of the ongoing assessment and monitoring of the sick newborn’s progress. The tests most often performed are blood glucose, bilirubin, electrolytes, calcium, hematocrit, and blood gases. Samples may be obtained by heel stick; venipuncture; arterial puncture; or an indwelling catheter in an umbilical vein, umbilical artery, or peripheral artery. (See Atraumatic Care box, Heel Punctures, in Chapter 8.) In one study, the use of an automated incision device for heel blood sampling resulted in the need for fewer heel pokes, less bruising of both the foot and the leg, and less inflammation than manual lancets (Vertanen, Fellman, Brommels, et al, 2001). When skilled phlebotomists are available, venipuncture for blood collections may be preferred. A Cochrane review comparing heel punctures to venipuncture

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**NURSING CARE GUIDELINES**

**Physical Assessment**

<table>
<thead>
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<td>Describe shape of chest (concave), symmetry, chest tubes, or other deviations. Describe use of accessory muscles: nasal flaring or substernal, intercostal, or suprasternal retractions. Determine respiratory rate and regularity. Auscultate and describe breath sounds: stridor, crackles, wheezing, diminished sounds, areas of absence of sound, grunting, stridor, diminished air entry, equality of breath sounds. Determine whether suctioning is needed. Describe ambient oxygen and method of delivery; if intubated, describe size of tube, type of ventilator and settings, and method of securing tube. Determine oxygen saturation by pulse oximetry and partial pressure of oxygen and carbon dioxide by transcutaneous oxygen (tcPO2) and transcutaneous carbon dioxide (tcPCO2).</td>
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</tr>
<tr>
<td>Temperature</td>
<td>Determine axillary temperature. Determine relationship to environmental temperature.</td>
</tr>
</tbody>
</table>

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**Nursing Tip**

When small volumes of urine are measured, superabsorbent disposable diapers, especially when kept closed, give more accurate volume measurements than cloth diapers because they are less affected by evaporative losses.
found that infants receiving a venipuncture for blood collection demonstrated less pain response than those receiving a heel lance and that use of venipuncture reduced the need for repeated heel punctures (Shah and Ohlsson, 2007a).

When numerous blood samples must be drawn, it is important to maintain an accurate record of the amount of blood being removed, especially in ELBW and VLBW infants, who cannot afford to lose blood during the acute phase of their illness.

When infants require close monitoring of oxygenation, pulse oximetry, a noninvasive measurement of the saturation or percent of oxygen in the hemoglobin, is typically used. Although used less frequently than pulse oximetry, some situations warrant the monitoring of transcutaneous oxygen (tcPO₂) and carbon dioxide (tcPCO₂). The nurse notes changes in oxygenation (or other aspects being monitored) associated with handling and adjusts the infant’s care accordingly. The frequency of taking vital signs depends on the infant’s acuity level and response to handling.

**Safety Measures**

The increased sophistication of supportive technology, including delivery systems, monitors, ventilator devices, and warmers, is both boon and bane. Although built-in safety systems and better engineering have made these devices more reliable and easier to use, our increasing reliance on them carries with it the additional risks of electrical biohazards and inaccurate function. Additionally, untrained or inexperienced operators confer an extra element of risk. Parents need instruction regarding safety precautions and observations. They are usually uncomfortable around the equipment and atmosphere of an intensive care unit and therefore appreciate an explanation of the purposes and functions of the devices and pertinent safety aspects.

Although most NICUs are closed units, parents must also learn about specific safety measures designed to prevent neonatal abduction. Most institutions have their own protocols for preventing such an occurrence. (See Protect from Infection and Injury, Chapter 8.)

**Respiratory Support**

The primary objective in the care of high-risk infants is to establish and maintain respiration. Many infants require supplemental oxygen and assisted ventilation. All infants require appropriate positioning to ensure an open airway and to maximize oxygenation and ventilation. Oxygen therapy is provided on the basis of the infant’s requirements and illness (see Respiratory Distress Syndrome, p. 347, and Oxygen Therapy, p. 352).

**Thermoregulation**

Concurrent with the establishment of respiration, the most crucial need of the low-birth-weight (LBW) infant is provision of external warmth. Prevention of heat loss in the distressed infant is absolutely essential for survival, and maintaining a neutral thermal environment is a challenging aspect of neonatal intensive nursing care. Heat production is a complicated process that involves the cardiovascular, neurologic, and metabolic systems, and the immature neonate has all the problems related to heat production that are faced by the full-term infant. (See Thermoregulation, Chapter 8.) However, LBW infants are placed at further disadvantage by a number of additional problems. They have an even smaller muscle mass and fewer deposits of brown fat for producing heat, lack insulating subcutaneous fat, and have poor reflex control of skin capillaries.

**Pathophysiology**

The immature neonate, unable to increase activity and lacking a shivering response, produces heat primarily through increased metabolic processes. Some heat continues to be generated by liver, heart, brain, and skeletal muscles, but the major source of increased heat production during cold stress is **nonshivering thermogenesis**. Norepinephrine, secreted by the sympathetic nerve endings in response to chilling, stimulates fat metabolism in the richly vascularized brown adipose tissue to produce internal heat, which is then conducted through the blood to surface tissues. A significant increase in metabolism requires increased oxygen consumption.

The consequences of cold stress that pose additional hazards to the neonate are (1) hypoxia, (2) metabolic acidosis, and (3) hypoglycemia. Increased metabolism in response to chilling creates a compensatory increase in oxygen and calorie consumption.

Norepinephrine, released in response to cold stress, causes pulmonary vasoconstriction, which further reduces the effectiveness of pulmonary ventilation. This decrease in oxygen intake diminishes the supply available for glucose metabolism. As a result, glucose is broken down by an alternate, hypoxic pathway (anaerobic glycolysis) that generates increased lactic acid. This, together with acid end-products of brown fat metabolism, contributes to the acidotic state. Anaerobic metabolism dissipates glycogen at a greatly increased rate over aerobic metabolism, thus precipitating hypoglycemia. This condition is especially marked when glycogen stores are diminished at birth and caloric intake is inadequate after birth.

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**TABLE 10-2 BLOOD PRESSURE RANGES IN DIFFERENT WEIGHT GROUPS OF HEALTHY PRETERM INFANTS**

<table>
<thead>
<tr>
<th>BIRTH WEIGHT</th>
<th>SYSTOLIC PRESSURE (mm Hg)</th>
<th>DIASTOLIC PRESSURE (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>501-750 g (1.1-1.6 lb)</td>
<td>50-62</td>
<td>26-36</td>
</tr>
<tr>
<td>751-1000 g (1.6-2.2 lb)</td>
<td>48-59</td>
<td>23-36</td>
</tr>
<tr>
<td>1001-1250 g (2.2-2.7 lb)</td>
<td>48-61</td>
<td>26-35</td>
</tr>
<tr>
<td>1251-1500 g (2.7-3.3 lb)</td>
<td>46-56</td>
<td>23-33</td>
</tr>
<tr>
<td>1501-1750 g (3.3-3.8 lb)</td>
<td>46-58</td>
<td>23-33</td>
</tr>
<tr>
<td>1751-2000 g (3.8-4.4 lb)</td>
<td>48-61</td>
<td>24-35</td>
</tr>
</tbody>
</table>


*Defined as infants without a history of maternal hypertension, Apgar scores of <3 at 1 min and <6 at 5 min, pneumothorax, hematocrit 0.32, serum pH 7.1, use of dopamine, infusion of erythrocytes or colloid, mechanical ventilation, or cardiopulmonary resuscitation.
Maintaining Thermoneutrality

To delay or prevent the effects of cold stress, at-risk newborns are placed in a heated environment immediately after birth, where they remain until they are able to independently maintain **thermal stability**—the capacity to balance heat production and conservation and heat dissipation. Because overheating produces an increase in oxygen and calorie consumption, the infant is also jeopardized in a hyperthermic environment. A **neutral thermal environment** is one that permits the infant to maintain a normal core temperature with minimum oxygen consumption and calorie expenditure. Studies indicate that optimum thermoneutrality cannot be predicted for every high-risk infant’s needs (Blackburn, 2007; Blake and Murray, 2006).

VLBW and ELBW infants, with thin skin and almost no subcutaneous fat, can control body heat loss or gain only within a limited range of environmental temperatures. In these infants heat loss from radiation, evaporation, and **transepidermal water loss** is three to five times greater than in larger infants, and a decrease in body temperature is associated with an increase in mortality.

The three primary methods for maintaining a neutral thermal environment are the use of an incubator, a radiant warming panel, and an open bassinet with cotton blankets. The healthy, full-term infant dressed and under blankets can maintain a stable temperature within a wider range of environmental temperatures; however, the infant requiring close observation or treatments such as phototherapy may need to be cared for in an incubator or under radiant heat (Fig. 10-1). The incubator should always be prewarmed before placing an infant in it. The use of double-walled incubators significantly improves the infant’s ability to maintain a desirable temperature and reduces energy expenditure related to heat regulation. The incubator should be located in a large, quiet, and draft-free area. Inside or outside the incubator, head coverings are effective in preventing heat loss. A fabric-insulated cap is more effective than one fashioned from stockinette (Blackburn, 2007).

An effective means for maintaining the desired range of temperature in the infant is the use of an automatically controlled (servocontrolled) incubator. The mechanism, when set at the upper and lower limits of the desired circulating air temperature range, adjusts automatically in response to signals from a thermal sensor attached to the abdominal skin. If the infant’s temperature drops, the warming device is triggered to increase heat output. The servocontrol is usually set to a desired skin temperature between 36° and 36.5° C (96.8° and 97.7° F) (Blake and Murray, 2006).

Convective heat loss occurs when infants are exposed to increased air flow velocity and turbulence (e.g., drafts from doors, ventilation system, opening and closing incubator ports, and side panels). The infant being cared for in a radiant warmer also experiences convective heat losses in response to ventilation drafts and traffic flow around the bed; these losses may be partially countered with plastic wrap placed directly on the infant’s body or stretched over the side guards of the warmer unit (Fig. 10-2). Oxygen or any source of air, such as an oxygen mask or tube, should not blow directly on the infant’s face. Oxygen concentrated around the head, such as that supplied to a hood, must be warmed and humidified.

Radiant heat loss is one of the greatest threats to temperature regulation in the incubator, since the temperature of circulating air within has no influence on heat loss to cooler surfaces without, such as windows, walls, or a lower nursery temperature. Such losses can be effectively reduced with the use of double-walled incubators; the infant radiates heat to the inner wall, which is surrounded by the warmed incubator air. The use of a cloth incubator cover further reduces radiant heat loss and provides some protection from exterior light sources.

A high-humidity atmosphere contributes to body temperature maintenance by reducing evaporative heat loss. Humidity is provided in some incubators by circulating air over a heated water reservoir, which has the additional advantage of decreasing heat loss by convection as the air flows over the infant. The water reservoir in older model incubators was often a source of water-borne bacteria, resulting in the need for frequent water changes. Newer technologies such as ultrasonic nebulizers may...
reduce the risk of such infections. Follow manufacturer’s recommendations in determining the frequency of water changes. The recommended humidity is 50% to 65%; higher humidity and a warmer environment are recommended for VLBW and ELBW infants.

A number of “microenvironments” may be used with the VLBW and ELBW infant to minimize evaporative and insensible water losses (IWLs). These include items such as bubble wrap blankets, humidified reservoirs for incubators, humidified tents, humidified Plexiglas boxes with plastic wrap coverings, polyethylene bags, and plastic wrap blankets. In cold-stressed infants, heat shields may be inappropriate because they may block heat from reaching the infant. The use of emollient cream to prevent transepidermal water loss has been used; however, this therapy has increased the risk of infection with coagulase-negative staphylococcus, and in preterm infants weighing 750 g or less, it should be used with caution (Association of Women’s Health, Obstetric and Neonatal Nurses, 2007).

The nurse can reduce conductive heat loss by warming all items that come in direct contact with the infant, such as scales, radiographic film, blankets, and the hands of caregivers. For example, the nurse can store blankets in a warming unit ready for use and place a freestanding warming unit or a heat lamp over a scale before weighing an infant.

Although the open radiant warmer unit allows easier access to the infant, there is an inherent increase in evaporative water loss (and evaporative heat loss) from the skin, especially in ELBW and VLBW infants. Transepidermal water losses, a form of IWL, may be increased by as much as 50% to 200%, thus predisposing the infant to dehydration; daily fluid requirements are generally increased to compensate for such losses. The use of plastic wrap over the ELBW or VLBW infant in a radiant warmer will help reduce IWL and convective losses.

The infant being cared for in a radiant warmer is kept warm using the servocontrol method. Air temperature manual control should not be used because of the danger of overheating the infant. A reflective aluminum temperature probe cover is used to allow proper function of the servocontrol heating unit. Traditionally, the temperature probe is placed over a nonbony, well-perfused tissue area such as the abdomen, flank, or back. In general, the probe site is changed when the infant’s position is changed to prevent the probe from coming in contact with the bed surface and potentially trapping heat at the probe site, causing an abnormal ambient temperature. Blackburn, De Paul, Loan, and colleagues (2001) found that abdominal and back skin temperatures varied considerably based on the infant’s position and the probe position; when infants were positioned prone and the probe was on the abdomen, the skin temperature rose. The researchers concluded that changing probe sites with repositioning may result in unstable body temperatures, that a consistent method of probe placement is needed, and that placement of the probe on the lateral abdomen may allow for frequent position changes (supine and prone) without the difficulties that occur when the infant lies on the probe.

The use of sterile cloth or disposable drapes also blocks radiant heat waves in a radiant warmer; during such procedures the use of a warmed blanket under the infant is appropriate. Clothing an infant on servocontrol in an incubator or radiant warmer is not recommended; head covering and foot covering (socks or booties) may be used with discretion.

Prolonged exposure to cold stress in the sick or preterm infant, particularly the ELBW or VLBW infant, may have disastrous results from which recovery may not be possible. Thermoregulation measures in the labor and delivery area and during transport to the NICU are essential. The use of a plastic bag or plastic wrap; careful drying; prewarming of equipment such as scales, stethoscopes, and incubators; and prompt placement of the VLBW or ELBW newborn in a proper heat source are essential for the prevention of further morbidity.

Hyperthermia may cause equally untoward effects because high-risk infants typically have a limited ability to perspire, thus decreasing heat dissipation. In high-risk neonates hyperthermia is usually a result of overheating rather than hypermetabolism. Therefore knowledge of proper care and use of external heating devices, such as radiant warmers or incubators, is as important as knowing the conditions for which they are being used.

Protection from Infection

Protection from infection is an integral part of all newborn care, but preterm and sick neonates are particularly susceptible. Thorough, meticulous, and frequent hand washing is the foundation of a preventive program. This includes all persons who come in contact with infants and their equipment. After handling another infant or equipment, no one should ever touch an infant without first washing hands.

Personnel with infectious disorders are either barred from the unit until they are no longer infectious or are required to wear suitable shields, such as masks or gloves, to reduce the likelihood of contamination. Standard Precautions as a method of infection control are instituted in all nursery areas to protect the infants and staff. (See Chapter 27.)

Readmission of infants from home or admission of infants delivered in unsterile conditions or infants suspected of having communicable illnesses is handled per institutional protocol. Such infants should at least be initially physically isolated from other highly susceptible high-risk infants. (See American Academy of Pediatrics and American College of Obstetricians and Gynecologists [2007] for further infection control recommendations, including nursery care of infants with specific communicable diseases.)

Hydration

High-risk infants often receive supplemental parenteral fluids to supply additional calories, electrolytes, and/or water. Adequate hydration is particularly important in preterm infants because their extracellular water content is higher (70% in full-term infants and up to 90% in preterm infants), their body surface area is larger in comparison to their weight, and the capacity for osmotic diuresis is limited in their underdeveloped kidneys. Therefore these infants are highly vulnerable to fluid depletion.

Parenteral fluids may be given to the high-risk neonate via several routes depending on the nature of the illness, the duration and type of fluid therapy, and unit preference. Common routes of fluid infusion include peripheral, peripherally inserted central venous (or percutaneous central venous), surgically inserted central venous or arterial, and, at times, umbilical venous or umbilical arterial catheterization. The preferred sites
for peripheral IV infusions in neonates are the peripheral veins on the dorsal surfaces of the hands or feet. Alternative sites are scalp veins and antecubital veins. Special precautions and frequent observations (at least once every hour) must accompany the use of peripheral lines with hypertonic solutions (dextrose 10% to 12%) and parenteral hyperalimentation solutions. In many neonatal centers the percutaneous central venous catheter, also commonly called the peripherally inserted central venous catheter, is used for IV hydration therapy and medication administration because of less expense and decreased neonatal trauma, and because of the ease of insertion (Bradshaw, Turner, and Pierce, 2006).

In most facilities NICU nurses insert peripheral IV catheters and maintain the infusions. IV fluids must always be delivered by continuous infusion pumps that deliver minute volumes at a preset flow rate. Secure the catheter to the skin with transparent tape or a specialized IV dressing, taking care not to cause undue pressure from the needle hub and tubing. Because ELBW and VLBW infants are highly vulnerable to any fluid shifts, infusion rates are carefully regulated and checked hourly to prevent tissue damage from extravasation, fluid overload, or dehydration (Kerr, Starbuck, and Block, 2006). Pulmonary edema, congestive heart failure, patent ductus arteriosus (PDA), and intraventricular hemorrhage (IVH) may occur with fluid overload. Dehydration may cause electrolyte disturbances (particularly sodium), with potentially serious central nervous system (CNS) effects.

！！！NURSING ALERT

Nurses should be constantly alert for signs of infiltration (e.g., redness, edema, or color change of tissue; blanching at site) and for signs of overhydration (weight gain of >30 g/24 hr [0.07 lb], periorbital edema, tachypnea, tachycardia, and crackles on lung auscultation).

Small, fragile peripheral blood vessels are subject to rupture and subsequent infiltration. This situation is compounded by the use of infusion pumps that continue to infuse fluid into surrounding tissues. Observations are especially important when using hypertonic solutions (calcium, sodium bicarbonate, parenteral hyperalimentation) and IV drugs (antibiotics and vasoactive drugs such as dopamine and dobutamine), which can cause serious tissue damage. With flexible catheters and small IV catheter shields, arm boards and limb restraints are usually unnecessary. If used, restraints should be checked frequently to ensure that no harm to the patient’s extremity occurs and that peripheral circulation is adequate.

Infants who are ELBW, tachypneic, receiving phototherapy, or in a radiant warmer have increased IWL that require appropriate fluid adjustments. Nurses must monitor fluid status by taking daily (or more frequent) weights; accurately monitoring intake and output of all fluids, including medications and blood products; monitoring urine specific gravity as well as urine glucose and protein; and evaluating serum electrolyte levels. ELBW infants often require more frequent monitoring of these parameters because of their excessive transpeddermal fluid loss, immature renal function, and propensity to dehydration or overhydration. Intolerance of even dextrose 5% is not uncommon in the ELBW infant, with subsequent glycosuria and osmotic diuresis. Alterations in behavior, alertness, or activity level in these infants receiving IV fluids may signal an electrolyte imbalance, hypoglycemia, or hyperglycemia. The nurse is also observant for tremors or seizures in the VLBW or ELBW infant, since these may be a sign of hyponatremia or hypernatremia.

A common problem observed in infants who have an umbilical arterial catheter in place is vasoconstriction of peripheral vessels, which can seriously impair circulation. The response is triggered by arterial vasospasm caused by the presence of the catheter, the infusion of fluids, or injection of medication. Blanching of the buttocks, genitalia, or legs or feet is an indication of vasospasm. The problem is recognized promptly and reported to the practitioner. The nurse must also observe for signs of thrombi in infants with umbilical venous or arterial lines. The precipitation of microthrombi in the vascular bed with the use of such catheters is commonly manifested by a sudden bluish discoloration seen in the toes, called “cath toes.” The problem is promptly reported to the practitioner because failure to alleviate the pathologic condition may result in permanent injury to the toes, foot, or leg.

Circulatory effects are observed first in the toes but may extend to include the legs and buttocks. The toes first flush and then turn a mulberry color; if the condition is not corrected, there may be serious complications involving the loss of a limb. The infant with an umbilical venous or arterial catheter should also be observed closely for catheter dislodgment and subsequent bleeding or hemorrhage; urinary output, renal function, and gastrointestinal function are also evaluated in these infants. Although the intent of such catheters is to effectively deliver IV fluids (and sometimes medications) and to obtain arterial blood gas samples, they are not without inherent complications.

Nutrition

Optimum nutrition is critical in the management of ELBW, VLBW, and LBW preterm infants, but difficulties arise in providing for their nutritional needs. The various mechanisms for ingestion and digestion of foods are not fully developed. The more immature the infant, the greater the problem.

Physiologic Characteristics

The preterm infant’s need for rapid growth and daily maintenance must be met in the presence of several anatomic and physiologic disabilities. Although infants demonstrate some sucking and swallowing activities before birth, coordination of these mechanisms does not occur until approximately 32 to 34 weeks of gestation, and they are not fully synchronized until 36 to 37 weeks. Initial sucking is not accompanied by swallowing, and esophageal contractions are uncoordinated. As infants mature, the suck-swallow pattern develops but is slow and inefficient, and these reflexes may easily become exhausted.

As with most full-term infants, preterm infants have poor muscle tone in the area of the lower esophageal (cardiac) sphincter. This causes milk in the stomach to be easily regurgitated into the esophagus, where it can trigger the chemoreceptors and cause apnea (vagal stimulation) and bradycardia and increase the risk of aspiration. The stomach has a limited capac-
Nutritional Needs

The demand for nutrients in LBW infants is much higher than that in larger infants, and individual infants vary in activity level, ease of achieving basal energy expenditure, thermoneutrality, physical condition, and efficacy of nutrient absorption. The American Academy of Pediatrics, Committee on Nutrition (2009a), recommends an energy intake of 105 to 130 kcal/kg/day (taken enterally) for most preterm infants to achieve a satisfactory growth rate. It is estimated that for a daily weight gain of 15 g/kg, a caloric expenditure of 45 to 67 kcal/kg above the maintenance expenditure of 50 kcal/kg (Table 10-3) would be required (American Academy of Pediatrics, 2009a). Thus the amount of calories required for optimum growth in sick and VLBW infants is significantly higher than in their healthy full-term counterparts; the challenge of providing adequate calories for extrauterine growth in the preterm infant with limited capability to ingest and absorb nutrients is an important part of nursing care for this population.

Table 10-3 shows the caloric requirements of healthy, growing preterm infants at 3 to 4 weeks of age. The energy requirements for sick and VLBW infants remains unknown; estimates are an intake of up to 105 to 115 kcal/kg/day, including a protein intake of 3 g/kg/day, for the ELBW infant (American Academy of Pediatrics, 2009a). Because most of the nutritional stores are accumulated in the final months of gestation, preterm infants also have low stores of calcium, iron, phosphorus, proteins, and vitamins A and C.

The infant’s size and condition determine the amount and method of feeding. Nutrition can be provided by either the parenteral or enteral route or by a combination of the two.

Total parenteral nutritional support of acutely ill infants may be accomplished with commercially available IV solutions specifically designed to meet the infant’s nutritional needs, including protein, amino acids, trace minerals, vitamins, carbohydrates (dextrose), and fat (lipid emulsion). Early protein intake (on day 1 of life) is also important in optimizing growth in LBW infants (Stephens, Walden, Gargus, et al, 2009). Daily monitoring of weight, electrolytes, renal function, calcium, and hydration status is carried out to ensure adequate therapy. As important as nutrition is the maintenance of adequate serum glucose homeostasis in sick preterm infants, who may depend on exogenous glucose sources for several days or weeks. Cornblath and Ichord (2000) recommend that in sick preterm infants an operational threshold blood glucose value of 45 to 50 mg/dl (2.6 to 2.8 mmol/L) be maintained.

Studies have revealed benefits to the early introduction of small amounts of enteral feedings in metabolically stable preterm infants (Hay, 2008). These minimum enteral feedings (MEFs; trophic feedings, gastrointestinal [GI] priming) have been shown to simulate the infant’s GI tract, preventing mucusosal atrophy and subsequent enteral feeding difficulties. They have also been shown to reduce the risk of sepsis. MEFs with as little as 0.1 to 4 ml/kg formula or breast milk may be given by gavage as early as day one of life or as soon as the infant is medically stable. In the past early introduction of milk feedings was thought to increase the risk of a devastating intestinal complication, necrotizing enterocolitis (NEC). NEC occurs more frequently in preterm infants, but the etiology of the disease remains unclear. No increased incidence of NEC in those VLBW infants given MEF has been found (Terrin, Passariello, Canani, et al, 2009; Berseth, Bisquera, and Paje, 2003).

A Cochrane review showed that infants receiving trophic feedings versus no feedings had an overall reduction in the number of days to full feedings and a shorter length of stay (Tyson and Kennedy, 2005). However, the researchers suggested that there was insufficient evidence to conclude that trophic feedings would indeed prevent NEC.

Controversy still exists regarding the type of enteral feeding that best meets the nutritional needs of LBW infants. The predominant view supports the use of milk from an infant’s own mother. Alternatively, if breast milk is not available, commercial formulas designed specifically to meet the needs of small preterm infants that provide for adequate growth and metabolic stability can be used (Table 10-4). Prepared formulas have the advantage of allowing more concentrated feedings.

A number of studies regarding the effects of long-chain polyunsaturated fatty acids on cognitive development, visual acuity, and physical growth in full-term and preterm infants have prompted formula companies to add docosahexaenoic acid (DHA) and arachidonic acid (AA) to their infant formulas. AA and DHA are in human milk, and their presence has been

### TABLE 10-3

<table>
<thead>
<tr>
<th>ENERGY EXPENDITURE</th>
<th>AVERAGE ESTIMATION (kcal/kg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total energy used</td>
<td>40-60</td>
</tr>
<tr>
<td>Resting metabolic rate</td>
<td>40-50*</td>
</tr>
<tr>
<td>Activity</td>
<td>0-5*</td>
</tr>
<tr>
<td>Thermoregulation</td>
<td>0-5*</td>
</tr>
<tr>
<td>Energy synthesis</td>
<td>15†</td>
</tr>
<tr>
<td>Stored energy</td>
<td>20-301†</td>
</tr>
<tr>
<td>Stool loss (energy)</td>
<td>15†</td>
</tr>
<tr>
<td>Energy intake</td>
<td>90-120†</td>
</tr>
</tbody>
</table>

*Energy required for maintenance.
†Energy expenditure for growth.

TABLE 10-4  PRETERM INFANT FORMULAS, HUMAN MILK FORTIFIERS, AND CALORIC ADDITIVES (DIET MODIFIERS)

<table>
<thead>
<tr>
<th>FORMULA (MANUFACTURER)</th>
<th>COMMENTS AND NUTRITIONAL CONSIDERATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>EnfaCare (Mead Johnson)</td>
<td>22 kcal/fluid oz; iron fortified; contains nucleotides, AA and DHA; liquid</td>
</tr>
<tr>
<td>Similac NeoSure (Ross)</td>
<td>22 kcal/fluid oz; iron fortified; contains nucleotides; liquid; contains DHA and AA</td>
</tr>
<tr>
<td>Enfamil Premature with iron (Mead Johnson)</td>
<td>Available in 24 kcal/fluid oz; contains AA and DHA; liquid</td>
</tr>
<tr>
<td>Similac Special Care 24 (Ross); with Iron; High Protein; and Low Iron</td>
<td>24 kcal/fluid oz; iron fortified; liquid</td>
</tr>
<tr>
<td>Similac Special Care with Iron 20, 24, and 30 (Ross)</td>
<td>Powder; add to human milk—do not use as separate formula</td>
</tr>
<tr>
<td>Enfamil Human Milk Fortifier (Mead Johnson)</td>
<td>Powder; add to human milk; do not use as separate formula; fortification in excess of 1 package per 25 ml human milk not recommended</td>
</tr>
<tr>
<td>Similac Human Milk Fortifier (Ross)</td>
<td>Powder; add to human milk—do not use as separate formula; liquid</td>
</tr>
<tr>
<td>Polycose (Ross)</td>
<td>Powdered, mostly long-chain fructose; fat from MCT oil; contains DHA and AA</td>
</tr>
<tr>
<td>Portagen (Mead Johnson)</td>
<td>Powder; 87% fat from MCT oil; not recommended as infant formula; indicated for use in children with</td>
</tr>
<tr>
<td></td>
<td>inadequate nucleotides</td>
</tr>
</tbody>
</table>

AA, Arachidonic acid; DHA, docosahexaenoic acid.

The high-risk newborn and family

Assumed to lead to an increase in cognitive development in human milk–fed infants compared with infants fed a formula without these fatty acids (Gregory, 2004). One meta-analysis of four clinical trials demonstrated no clinically significant developmental benefits to supplementation of formula with AA and DHA in term and preterm infants at 18 months of age (Beyerlein, Hadders-Algra, Kennedy, et al, 2009).

Milk produced by mothers whose infants are born before term contains higher concentrations of protein, sodium, chloride, and immunoglobulin A (IgA). Thus mothers appear to be the preferred source of milk for their preterm infants. Growth factors, hormones, prolactin, calcitonin, thyroxine, steroids, and taurine (an essential amino acid) are also in human milk. The milk produced by mothers for their infants changes in content over the first 30 days postnatally, at which time it is similar to full-term human milk. Preterm infants who received human milk during their hospitalization demonstrated better intellectual performance scores at 7 ½ to 8 years of age compared with children who received formula (Schanler, 2001). Improved psychomotor development at 18 months has also been observed in preterm infants fed donor human milk compared with formula-fed preterm infants. Despite its benefits, LBW infants (<1500 g [3.3 lb]) who are exclusively fed unfortified human milk demonstrate decreased growth rates and nutritional deficiencies even beyond the hospitalization period. These infants often have inadequacies of calcium, phosphorus, protein, sodium, vitamins, and energy (Schanler, 2001). Specially designed supplements for human milk have been developed to address these deficits. Preterm infants fed fortified human milk (FHM) have shorter hospital stays and less infection and NEC than infants given preterm formulas. Fortifiers are commercially available, usually as a liquid or powder containing protein; carbohydrate; calcium; phosphorus; magnesium; sodium; and varied amounts of zinc, copper, and vitamins. Because fortifiers do not contain sufficient iron, an exogenous source must be administered after enteral feeding. Fortifiers should be added to milk as close as possible to feeding time, and FHM should be refrigerated until it is used.

The antinfectious attributes of human milk provide additional advantages for preterm infants. Secretory IgA concentration is higher in the milk from mothers of preterm infants than in the milk from mothers of full-term infants. IgA is important in the control of bacteria in the intestinal tract, where it inhibits adherence and proliferation of bacteria on epithelial surfaces. Additional protection from infection is provided by leukocytes, lactoferrin, and lysozyme, all of which are in human milk. Recent research suggests that administration of probiotics, live microbial supplements, decreases the incidence of NEC by normalizing intestinal flora, reducing intestinal permeability, and reducing gut inflammation (Alfaeieh, Anabrees, and Bassler, 2009; Deshpande, Rao, and Pathole, 2007). NEC has been shown in several studies to be higher in formula-fed infants than in preterm infants fed human milk. Another report suggests that severity of NEC is lessened and the prevalence of intestinal perforation lowered when preterm infants are fed human milk (Schanler, 2001).

Preterm infants exclusively fed human milk have demonstrated significantly decreased NEC, fewer positive blood cultures, and decreased need for antibiotics. In one study infants fed human milk also received more skin-to-skin (STS) contact with their mothers and shorter hospital stays. Schanler (2001) suggests that STS contact might potentially stimulate the enteromammary immune system to produce specific antibodies against nosocomial pathogens in the nursery. Gastric emptying is improved with human milk feedings for preterm infants, primarily because of increased intestinal lactase and possibly decreased intestinal permeability. Finally, the psychologic advantages the mother gets from using her own milk cannot be overlooked.

For those infants who cannot be breast-fed but who also cannot receive except on human milk, banked donor milk is important. Because of the antiinfective and growth-promoting properties of human milk, as well as its superior nutrition, donor milk is used in many NICUs for preterm or sick infants when the mother’s own milk is not available (American Academy of Pediatrics, 2005). Unprocessed human milk from unscreened donors is not recommended because of the risk of transmission of infectious agents (American Academy of Pediatrics, 2005).

The Human Milk Banking Association of North America (HMBANA; www.hmbana.org) has established guidelines for
the operation of donor human milk banks (Human Milk Banking Association, 2008). Donor milk banks collect, screen, process (pasteurize), and distribute milk donated by breastfeeding mothers who are feeding their own infants and pumping a few extra ounces each day for the milk bank. All donors are screened both by interview and serologically for communicable diseases. Donor milk is stored frozen until it is heat processed to kill potential pathogens (bacteria and viruses), and then it is refrozen for storage until it is dispensed for use. The heat processing adds a level of protection for the recipient that is not possible with any other donor tissue or organ. Milk is dispensed only by prescription. A per-ounce fee is charged by the bank for processing, but the HMBANA guidelines prohibit payment to donors.

Although the timing of the first feeding has been controversial, most authorities now believe that early feeding (provided that the infant is medically stable) reduces the incidence of complicating factors such as hypoglycemia and dehydration and reduces the degree of hyperbilirubinemia. The feeding regimen used varies in different units. One strategy for the prevention of NEC that has been supported by research is the use of standardized feeding protocols. A meta-analysis of six studies found a significant reduction in NEC in infants fed by a standard protocol that included cautious advancement in feeding volumes (Patole and de Klerk, 2005).

Feeding tolerance and feeding success are not entirely the same concept. Feeding tolerance is evaluated by the following: (1) soft abdomen; (2) absence of abdominal distention or visible bowel loops on the skin surface; (3) minimum or no aspirated gastric residual; (4) presence of bowel sounds; (5) usual frequency, color, and consistency of stools; (6) minimum or no spitting up or vomiting; (7) infant’s continued interest in feeding; and (8) consistent behavior pattern. Successful oral feeding should be safe, functional, and pleasurable. Feeding success can be measured by an infant’s ability to (1) participate in feeding with energy, (2) coordinate sucking and swallowing with adequate pauses for breathing, (3) maintain vital signs and oxygenation within normal limits, (4) maintain normal muscle tone in face and body, (5) complete feeding in about 20 to 25 minutes, (6) manage a liquid bolus with minimum or no loss of liquid from mouth, (7) sustain alertness for feeding, (8) maintain strength and endurance for entire feeding, and (9) measure appropriate-for-age on standard growth curve. A preterm infant’s success with feeding is first measured in terms of safety and functionality. Nurturing by holding close, but not socializing, during a feeding creates a warm and pleasurable experience. Later, after the infant is a competent feeder, socialization will enrich both parents’ and infant’s mealtime enjoyment.

**Gavage Feeding**

Gavage feeding is a safe means of meeting the nutritional requirements of infants who are not yet ready to feed orally. These infants are usually too weak to suck effectively, are unable to coordinate swallowing, or lack a gag reflex. A Cochrane review found that infants less than 1500 g (3.3 lb) fed by continuous tube-feeding took longer to reach full oral feeds than those fed intermittently; however, there was no difference in somatic growth or in the incidence of NEC (Premji and Chessell, 2003). Intermittent gavage feeding is used as an energy-conserving technique for infants learning to nipple-feed who become excessively tired, listless, or cyanotic.

A size 3.5, 5, 6, or 8 French feeding tube is usually used to instill the feeding, and the usual methods for determining correct placement are used. (See Chapter 27 for technique.) Although the more relaxed cardiac sphincter makes passage of the tube easier, the heart rate and blood pressure may change in response to vagal stimulation. The procedure is best accomplished when an infant is in a prone or a right side-lying position with the head slightly elevated. Small flexible nasogastric tubes (3.5 and 5 French) may be maintained as an indwelling feeding tube and used for prolonged periods without complications of intermittent removal and insertion.

The stomach is aspirated, the contents measured, and the aspirate returned as part of the feeding. However, this practice may vary, depending on circumstances and individual unit protocol.

### NURSING ALERT

An increase in gastric residuals, abdominal distention, bilious vomiting, temperature instability, apneic episodes, and bradycardia may indicate early NEC and should be called to the attention of the practitioner.

The feeding is allowed to flow by gravity, and the length of time varies. This procedure is not used as a timesaving method for the nurse. Complications of indwelling tubes include the obstructed nares, mucous plugs, purulent rhinitis, epistaxis, infection, and possible stomach perforation.

The infant may be held during gavage feedings by the caregiver or parent. Also, nonnutritive sucking (NNS) on a pacifier helps infants associate the sucking with the feeling of satiety. A Cochrane review of NNS demonstrated a significant reduction in length of stay in preterm infants receiving an NNS intervention. Other positive outcomes of NNS included enhanced transition from tube- to bottle-feeding and better bottle-feeding performance (Pinelli and Symington, 2005).

### Oral Feeding

Vigorous infants can be fed orally with little difficulty, whereas compromised preterm infants require alternative methods. The amount to be fed is determined largely by the infant’s weight gain and tolerance of previous feeding and is increased by small increments until a satisfactory caloric intake is ensured.

The rate of increase that is well tolerated varies from one infant to another, and determining this rate is often a nursing responsibility. Preterm infants require more time and patience to feed than full-term infants, and the oropharyngeal mechanism may be stressed by an attempt to feed too rapidly. It is important not to tire the infants or overtax their capacity to retain the feedings. When infants require a prolonged time (>30 minutes) to complete a feeding, gavage feeding may be considered for the next time.

The decision regarding when to start breast- or bottle-feeding is somewhat controversial. In many cases the decision is based on an evaluation of the infant’s developmental maturity, weight, activity level, respiratory status (absence of apnea...
and adequate oxygen saturation levels), and sucking capabilities. Infant behavioral organizational skills, such as the ability to maintain a quiet alert state and display engagement cues, also influence the preterm infant’s successful transition to oral feedings (Thoyre, Shaker, and Pridham, 2005). When infants are unable to tolerate breast- or bottle-feedings, intermittent feedings by gavage begin until they gain enough strength and coordination to use the nipple.

**NURSING ALERT**

Poor feeding behaviors such as apnea, bradycardia, cyanosis, pallor, and decreased oxygen saturation in any infant who has previously fed well may indicate an underlying illness in the preterm infant.

Although the nurse’s role in relation to feeding depends on the institution, the following are suggested nursing responsibilities: (1) recognize feeding readiness cues; (2) identify feeding behaviors typical of preterm infants; (3) understand the infant’s history and current medical condition; (4) consider environment, behavioral state, time of day, nipple type, and positioning; (5) understand rationale for different facilitation techniques and use appropriately; (6) evaluate feeding ability and tolerance; (7) identify infants with poor progress, structural defects, or abnormal feeding patterns who would benefit from specific therapy; and (8) play a supportive role for mothers who choose to breast-feed.

A developmental approach to feeding considers the individual infant’s readiness rather than initiating feedings based on weight and age. Feeding readiness is determined by each infant’s medical status, energy level, ability to sustain a brief quiet alert state, gag reflex (demonstrated with gavage tube insertion), spontaneous rooting and sucking behaviors, and functional sucking reflex (Hunter, 2001).

Oral feeding within a developmental framework involves three steps (Thoyre, Shaker, and Pridham, 2005):

1. Assessing individual physiologic, motor, and state behaviors during feeding
2. Individualizing the feeding plan based on specific infant cues
3. Fostering parental skill and confidence with feeding

The goal of feeding must be well understood. A key concept is recognizing the difference between a successful feeding (volume and time) and a successful feeder (infant ability and enjoyment). This is the difference between task and developmental feeding techniques. Planning the progression and nature of feedings requires close monitoring and careful documentation. Baseline assessment data are collected before each feeding and observed during and after the feeding to make a comparative evaluation of feeding success. Assessment is ongoing throughout the feeding, and facilitation techniques are chosen based on the individual infant’s responses to improve the chance for feeding success and tolerance (Nye, 2008). Feeding stress and performance (Box 10-2) are evaluated and documented. Planning is done in collaboration with the health care team and family before the next feeding to determine appropriate strategies for the infant. Box 10-3 gives examples of ways to facilitate feeding.

**BOX 10-2 FEEDING STRESS CUES**

<table>
<thead>
<tr>
<th>State Organization and Endurance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased arousal</td>
</tr>
<tr>
<td>Awake but no energy</td>
</tr>
<tr>
<td>Irritable</td>
</tr>
<tr>
<td>Fatigues quickly within first 5 minutes</td>
</tr>
</tbody>
</table>

**Physiologic**

- Tachypnea
- Nasal flaring, retractions (increased work of breathing)
- Decreased oxygen saturation
- Apnea, bradycardia
- Color change to dusky or pale

**Oral-Motor**

- Unable to control fluid bolus (milk leaking out of mouth)
- High-pitched sounds
- Gulping
- Coughing, choking
- Multiple swallows without pausing for breath


**BOX 10-3 FEEDING FACILITATION TECHNIQUES FOR PRETERM INFANTS**

**Environment**

- Prepare calm, quiet area with dim lighting and no distractions.
- Ensure restful environment between feedings.

**Direct Care**

- Avoid trial oral feedings after stressful procedures.
- Choose slightly firm nipple with slower flow.
- Gently arouse to alert state.
- Swaddle in gentle flexion with infant’s hands midline and toward face.
- Support positioning with infant cradled close to body in semiprayer or upright position, with neck in neutral to slightly flexed position.
- Continuously observe physiologic, behavioral, and oral-motor functioning.
- Provide adequate breathing and rest periods for infants who cannot pace themselves by gently removing nipple or, if that is too stressful, tipping bottle gently downward to drain milk from nipple.
- Provide firm but gentle jaw and cheek support for problems with latching onto nipple (weak seal, loss of milk bolus).
- Institute “developmental burping” on shoulder with postural support and gentle back rubbing in an upward motion to stimulate burp.
- Recognize infant’s limits and when to stop feeding.
- Use gavage for the rest of the feeding as needed.
- Schedule plenty of undisturbed rest between feedings.

**Family Support and Education**

- Model appropriate feeding techniques.
- Provide opportunity for feeding.
- Educate on infant cues and how to measure feeding success.


**Breast-Feeding**

The American Academy of Pediatrics (2005) recommends human milk as the preferred food for all infants, including sick newborns and preterm infants (with rare exceptions). The academy recognizes that the choice of what to feed is the
parents’ prerogative but advises that providers give parents complete and accurate information on the benefits and methods of breast-feeding so they can make an informed decision. Barriers to initiation and continuation of breast-feeding include physician indifference, misinformation, lack of prenatal education about breast-feeding, distracting hospital policies, lack of follow-up, working mother, unsupportive work environment, lack of support from family or society, hospital discharge packs with formula or coupons for formula, and media portrayal of bottle-feeding.

Studies indicate that even small preterm infants are able to breast-feed if they have adequate sucking and swallowing reflexes and no other contraindications, such as respiratory complications or concurrent illness (Dougherty and Luther, 2008; Morton, 2002). Mothers who wish to breast-feed their preterm infants should pump their breasts until their infants are sufficiently stable to tolerate breast-feeding. Appropriate guidelines for the storage of expressed mother’s milk should be followed to decrease the risk of milk contamination and destruction of its beneficial properties. (See Chapter 12.)

Preterm infants may be able to successfully breast-feed earlier than previously believed (28 to 36 weeks). In addition, preterm infants who are breast-fed rather than bottle-fed demonstrate fewer oxygen desaturation episodes; an absence of bradycardia; warmer skin temperature; and better coordination of breathing, sucking, and swallowing (Gardner, Snell, and Lawrence, 2006). The nurse should carefully evaluate the preterm infant for readiness to breast-feed, including assessment of behavioral state, ability to maintain body temperature outside an artificial heat source, respiratory status, and readiness to suckle at the mother’s breast. The latter may be accomplished with NNS at the breast during STS (or kangaroo) contact so the mother and newborn can become accustomed to each other (Gardner, Snell, and Lawrence, 2006). Nasal cannula oxygen may also be provided during breast-feeding if the infant requires it.

Time, patience, and dedication on the part of the mother and the nursing staff are necessary to help infants breast-feed. The process starts slowly, beginning with one oral feeding daily and gradually increasing the feedings as the infant tolerates them. Supplementary bottle-feeding is inefficient because the infant expends energy and calories to feed twice. Feeding more often and/or supplementing with gavage feeding is more energy and calorie efficient. Breast-feeding the preterm infant often requires additional guidance by a lactation consultant and continued support and encouragement by the nursing staff. In addition, postdischarge breast-feeding often requires further guidance, counseling, and support.

Social support for the mother is a major influence on the decision to breast-feed. To be effective advocates for mothers of all ethnicities, nurses must understand the cultural aspects that influence, whether positively or negatively, breast-feeding choices (McCarterspaulding, 2009; Gill, 2009). African-American women, for example, identify prenatal health care providers and friends as influential in decisions regarding breast-feeding. They tend to breast-feed less than women from other cultures and should be provided with appropriate information on breast-feeding by health care providers. Breast-feeding materials are available from organizations such as La Leche League International.*

**Nipple-Feeding**

The infant is positioned in the feeder’s arms or placed semi-upright in the lap (Fig. 10-3) and is held with the back curved slightly to simulate the position assumed naturally by most full-term newborns. Stroking the infant’s lips, cheeks, and tongue before feeding helps promote oral sensitivity.

Hill, Kurkowski, and Garcia (2000) used cheek and jaw support for preterm infants between 32 and 34 weeks of gestation to facilitate feeding. Supported infants had fewer and shorter pauses during feeding and had higher postfeeding oxygen saturations than infants not receiving oral support. The groups did not differ in terms of oxygen saturation, heart rate, and respiratory rate during feeding, indicating the technique is as safe as traditional feeding techniques. This technique uses the

*PO Box 4079, Schaumburg, IL 60018; 847-519-9585 (order department); www.lli.org. In Canada, La Leche League Canada, PO Box 700, Winchester, ON KOC 2K0; 613-774-4900; www.lalecheleaguecanada.ca.
thumb and index finger to provide gentle pressure (inward and forward) on the cheeks and the third finger to lift and stabilize the jaw under the mandible where the base of the tongue resides.

Bottle-feedings continue if infants are able to tolerate the feedings and take the required amount. The infant is best fed when fully alert. Drowsy infants feed more slowly, and liquid is more likely to fill the relaxed pharynx before the infant swallows, causing choking. It is believed that many digestive powers require signal stimulation to respond. Some preterm infants respond more slowly than full-term infants; therefore the feeding interval and amount are individualized. Preterm infants are often slow feeders and require patience, frequent rest periods, and burping (or bubbling).

A key ingredient for success is choosing an appropriate nipple. The nipple used should be relatively firm and stable. Although a high-flow, pliable nipple requires less energy to use, it may provide a flow rate that is too rapid for some preterm infants to manage without risk of aspiration. A firmer nipple facilitates a more “cupped” tongue configuration and allows for a more controlled, manageable flow rate.

Prodding techniques to encourage sucking can increase the risk of aspiration, especially if inadequate breathing opportunities are not provided. The preterm infant has difficulty managing rapid or continuous milk flow with suck, swallow, and breathing coordination when the nipple is manipulated frequently by twisting or turning; the bottle is moved up and down or in and out of the mouth; or the infant’s jaw is moved up and down (not the same as cheek and jaw support). The infant will try to continue to suck or swallow at the risk of physiologic and behavioral consequences.

Research by Law-Morstatt, Judd, Snyder, and colleagues (2003) has demonstrated that a paced bottle-feeding protocol that was structured to limit the length of sucking bursts and lengthen the duration of swallowing and breathing resulted in earlier emergence of organized sucking patterns than traditional approaches to feeding. Similar findings emerged from work by Fucile, Gisel, and Lau (2005), who found that a systematic protocol of oral motor stimulation resulted in enhanced tongue and jaw muscle strength and coordination.

Feeding Resistance
Any feeding technique that bypasses the mouth precludes the opportunity for the affected child to practice sucking and swallowing, or the opportunity to experience normal hunger and satiation cycles. Infants may demonstrate aversion to oral feedings by such behaviors as averting the head to the presentation of the nipple, extruding the nipple by tongue thrust, gagging, or even vomiting.

Developmental delays have occurred in perceptual-motor performance among infants with feeding refusal as measured by standard tests, although intellectual function remains within normal limits. Other observations include disinterest in or active resistance to oral play, diminished spontaneity and motivation, and shallow interpersonal relationships, probably related to the absence of some early incorporative patterns of normal oral experiences. The longer the period of nonoral feeding, the more severe the feeding problems, especially if this period occurs during a time when the infant progresses from reflexive to learned and voluntary feeding actions. During infancy the mouth is the primary instrument for reception of stimulation and pleasure.

Infants identified as being at risk for feeding resistance should receive regular oral stimulation based on the child’s developmental level. Those who exhibit feeding aversion should begin a stimulation program to overcome resistance and acquire the ability to take nourishment by the oral route. Because management requires long-term commitment, successful implementation of a plan for oral stimulation depends on maximum parental involvement and promotion of primary nursing. Key components and interventions are in Box 10-4.

Skin Care
The skin of preterm infants is characteristically immature. Because of its increased sensitivity and fragility, no alkaline-based soap that might destroy the “acid mantle” of the skin is used. The increased permeability of the skin facilitates absorption of ingredients. All skin products (e.g., alcohol or povidone-iodine) are used with caution. The skin is rinsed with water afterward because these substances may cause severe irritation and chemical burns in LBW infants.

The skin is easily excoriated and denuded; therefore take care to avoid damage to the delicate structure. The total skin is thinner than that of full-term infants and lacks rete pegs, appendages that anchor the epidermis to the dermis. Therefore there is less cohesion between the thinner skin layers. Adhesives used after heel sticks or to secure monitoring equipment or IV infusion...
sions may excoriate the skin or adhere to the skin surface so well that the epidermis can be separated from the dermis and pulled away with the tape. The use of a zinc oxide–based tape such as Hy-Tape is encouraged to minimize epidermal stripping; the tape is flexible, waterproof, and, washable. The use of skin barriers protects healthy skin and helps excoriated skin heal.

Use scissors very carefully to remove dressings or tape from the extremities of very small and immature infants because it is easy to snip off tiny extremities or nick loosely attached skin. Avoid solvents to remove tape because they tend to dry and burn the delicate skin. Guidelines for skin care are given in the Nursing Care Guidelines box.

### NURSING CARE GUIDELINES

#### Neonatal Skin Care

**General Skin Care**

**Assessment**
Assess skin once each shift for redness, dryness, flaking, scaling, rashes, lesions, excoriation, or breakdown. Consider using a validated skin assessment tool such as the Neonatal Skin Condition Score (Lund and Osborne, 2004). Identify those infants at increased risk for skin breakdown.

Evaluate and report abnormal skin findings and analyze for possible causation. Intervene according to interpretation of findings or physician order.

**Bathing**

**Initial bath**
Assess for stable temperature a minimum of 2 to 4 hours before first bath. Use cleansing agents with neutral pH or minimum dyes or perfume, in water. Do not completely remove vernix caesa.

Bathe preterm infant (<32 weeks of gestation) in sterile water alone.

**Routine**
Decrease frequency of baths to every second or third day by daily cleansing of eye, oral and diaper areas, and pressure points. Do not completely remove vernix calcs. Use cleanser or soaps no more than two or three times a week. Avoid rubbing skin during bathing or drying. Immense stable infants fully (except head) in an appropriate-sized tub. Use swaddled immersion bathing technique: slow unwrapping after gently lower into water for sensitive, but stable, infants needing assistance with motor system reactivity.

**Emollients**
Follow hospital protocol or consider the following:
- Apply emollient as needed for dry, flaking skin.
- Use only emollients without perfumes, preservatives, or dyes.

**Adhesives**
Decrease use as much as possible. Use transparent semipermeable adhesive dressings to secure intravenous lines, catheters, and central lines. Use hydrogel electrodes. Consider using pectin or hydrocolloid barriers beneath adhesives to protect skin.

Secure pulse oximeter probe or electrodes with elasticized dressing material (carefully avoid restricting blood flow).

Do not use adhesive remover, solvents, and bonding agents. Avoid removing adhesives for at least 24 hours after application. Adhesive removal can be facilitated using water, mineral oil, or petrolatum. Remove adhesives or skin barriers slowly, supporting the skin underneath with one hand and gently peeling away the product from the skin with the other hand.

**Antiseptic Agents**
Apply before invasive procedures. Evaluate the risks and benefits of any antiseptic agent. Chlorhexidine gluconate and 10% povidone-iodine have both been shown to reduce skin bacterial counts in newborns. Povidone-iodine may be absorbed systemically. Avoid use of alcohol.

**Transdermal Water Loss**
Minimize transdermal water loss and heat loss in small preterm infants (<30 weeks of gestation) by measuring ambient humidity during first weeks of life and considering an increase in humidity to 70% for the first week of life by using one or more of the following options or hospital guidelines:
- Transparent dressings
- Servocontrolled humidifying incubator
- Supplemental conductive heat sources such as heated mattresses
- Polyethylene coverings (but avoid having plastic wraps in contact with skin surfaces for long periods)

**Skin Breakdown**

**Prevention**
Decrease pressure from externally applied forces using water, air, or gel mattresses, sheepskin, or cotton bedding. Provide adequate nutrition, including protein, fat, and zinc.

Apply transparent adhesive dressings to protect arms, elbows, and knees from friction injury. Use tracheostomy and gastrostomy dressings for drainage and relief of pressure from tracheostomy or gastrostomy tube (Hydrosorb or Lyfoam). Use emollient in the diaper area (groin and thighs) to reduce urine irritation.

**Treating Skin Breakdown**
Irrigate wound every 4 to 6 hours with warm half-strength normal saline using a 30 ml or larger syringe and 20-gauge Teflon catheter.

Culture wound and treat if signs of infection are present (excessive redness, swelling, pain on touch, heat, or resistance to healing).

Use petrolatum-based ointments for uninfected wounds. Apply hydrogel with or without antibacterial or antifungal ointments (as ordered) for infected wounds (may need to moisten before removal). Use hydrocolloid for deep, uninfected wounds (leave in place for 5 to 7 days) as an ostomy barrier and to improve appliance adhesion; warm barrier in hand for several minutes to soften before applying to skin. Avoid use of antiseptic solutions for wound cleansing (used for intact skin only).

**Treating Diaper Dermatitis**
Maintain clean, dry skin; use absorbent diapers and change often. If mild irritation occurs, use petrolatum barrier. For developing dermatitis, apply a generous quantity of zinc-oxide barrier. For severe dermatitis, identify cause and treat (e.g., frequent stooling from spina bifida, severe opiate withdrawal, or malabsorption syndrome).

Treat Candida alibicans with antifungal ointment or cream. Avoid talcum powders and antibiotic ointments. (See Care of the Umbilicus and Circumcision, Chapter 8.)

**Other Skin Care Concerns**

**Use of Substances on Skin**
Evaluate all substances that come in contact with infant’s skin. Before using any topical agent, analyze components of preparation and:
- Use sparingly and only when necessary.
- Confine use to smallest possible area.
- Whenever possible and appropriate, wash off with water.
- Monitor infant carefully for signs of toxicity and systemic effects.

**Use of Thermal Devices**
Avoid heat lamps because of increased potential for burns. If needed, measure actual temperature of exposed skin every 15 minutes. When using heating pads (Aqua-K pads):
During skin assessment of preterm infants, nurses are alert to the subtle signs that indicate zinc deficiency, a common problem in these infants. Breakdown usually occurs in the areas around the mouth, buttocks, fingers, and toes. In VLBW infants it may also occur in the creases of the neck, wrists, and ankles and around wounds. Zinc deficiency is most likely to appear in infants with sepsis, those experiencing nasogastric losses, or those who have had surgery. Report suspicious lesions to the practitioner so that zinc supplements can be prescribed. In most preterm infants the skin barrier properties resemble those of the term infant by 2 to 3 weeks postnatal age, regardless of gestational age at birth.

Although no studies comparing the effectiveness of different commercially available neonatal bedding have been done, a number of products are useful in minimizing skin problems. The Nursing Care Guidelines box gives general information about bedding. Particularly vulnerable areas of the skin, such as bony prominences, can be protected with transparent dressings. Gel pads or mattresses can also be used to prevent pressure ulcers (Association of Women’s Health, Obstetric and Neonatal Nurses, 2007).

Skin injuries have been reported during use of phototherapy blankets. Caution is warranted in using these products with extremely preterm infants or infants with birth trauma, poorly perfused skin, or hypotension. Manufacturers of phototherapy blankets recommend the following during therapy: monitor skin color, observe for rashes or excoriation, keep skin clean with warm water, promptly clean perineum after stooling, reposition every 2 hours, carefully monitor cleanliness and skin integrity, and avoid direct contact of blanket with infant’s skin.

**Administration of Medications**

Administration of therapeutic agents, such as drugs, ointments, IV infusions, and oxygen, requires judicious handling and meticulous attention to detail. The computation, preparation, and administration of drugs in minute amounts often require collaboration between nurses, physicians, and pharmacists to reduce the chance for error. In addition, the immaturity of an infant’s detoxification mechanisms and inability to demonstrate symptoms of toxicity (e.g., signs of auditory nerve involvement from ototoxic drugs such as gentamycin) complicate drug therapy and require that nurses be particularly alert for signs of adverse reaction. (See Administration of Medication, Chapter 27.)

Nurses should be aware of the hazards of administering bacteriostatic and hyperosmolar solutions to infants. Benzyl alcohol, a common preservative in bacteriostatic water and saline, is toxic to newborns and should not be used to flush IV catheters or to dilute or reconstitute medications. It is recommended that medications with preservatives such as benzyl alcohol be avoided. Nurses must read labels carefully to detect the presence of preservatives in any medication administered to an infant.

Hyperosmolar solutions present a potential danger to preterm infants. Hyperosmolar solutions given orally to infants can produce clinical, physiologic, and morphologic alterations, the most serious of which is NEC. Oral or parenteral medications should be sufficiently diluted to prevent complications related to hyperosmolality.

Take caution to reduce adverse effects of medication administration in preterm infants. Strategies to heighten awareness and decrease unnecessary morbidity in such infants include having two registered nurses double check the dosages of potentially lethal medications (high-risk medications) and providing calculators in neonatal units to perform dosage calculations, double check unit dose medications, and check medications that are reconstituted by the nurse. Some other precautions are to have readily available quick-reference guides to weight-specific medication doses and appropriate dosages for preterm infants and to provide tables with medication cross-reactivity and incompatibility. One NICU developed a distinct neonatal formulary to reduce medication errors. Another strategy was to develop computerized guidelines for managing dose ranges based on the neonate’s most recent weight so medications ordered outside the appropriate dose range could be reevaluated by the pharmacist and practitioner. This concept was also applied to computer-generated emergency medication sheets, which are updated weekly (Lucas, 2004). Information technology (e.g., computerized practitioner order entry and clinical participation by a clinical pharmacist)
is available to reduce medication errors, yet this technology does not provide the entire solution (Taylor, Loan, Kamara, et al, 2008). The human factor involved in many root-cause analyses for medication errors involves systems and the humans involved in those systems. Nurses, physicians, and pharmacists are at times affected by internal and external environmental factors that lead to a medication error: excessive workload; distractions such as a monitor alarm or questions asked during medication administration; boredom; work hours (nighttime or daytime); lack of updated, consistent drug information; ambiguous drug labeling; and dosage calculation errors (Lefrak, 2002). The variables involved are numerous and multifaceted yet can be decreased by simple cautionary measures, extensive education, and verification of medication orders written.

**Developmental Outcome**

Neonatal intensive care and rapid improvements in technology are associated with improved survival of critically ill newborn and preterm infants. Survival rates have increased to 93% for VLBW infants (1001 to 1500 g [2.2 to 3.3 lb]), 85% for ELBW infants (751 to 1000 g [1.6 to 2.2 lb]), and about 50% for VLWB infants (1001 to 1500 g [2.2 to 3.3 lb]), 85% for ELBW and preterm infants. Survival rates have increased to 93% for those survivors of early gestation and LBW when emphasis is placed on providing the finest medical and nursing care within a developmentally supportive framework. This philosophy requires caregivers to evaluate their own knowledge, skills, and attitudes and expand their thinking beyond the traditional medical and nursing models of care.

**Developmental Assessment**

One approach to NICU care is based on Als’s (1982) **synactive theory of infant development**, which provides a framework for understanding the preterm infant’s development. The model proposes a systematic method for observing NICU infants to collect information concerning each infant’s competencies, vulnerabilities, and thresholds. This information forms the basis for planning individualized care appropriate for a particular infant (Table 10-5). The major assumption of this model is that infants, even ELBW infants, can communicate through physical and behavioral responses that provide us with the best information for planning their care. Communication by the infant is seen through three subsystems of function (autonomic, motor, and state) that can be readily observed in the clinical setting during rest, care, or procedures and during recovery from care or procedures. Responses by an infant’s autonomic (physiologic), motor, and state systems to the environment, physical care, or procedures help the nurse make necessary adjustments in technique to optimize the infant’s stability and function.

**Individualized developmental care** has had numerous positive effects on medical and neurobehavioral outcomes in high-risk newborn infants. Findings noted in a randomized controlled trial of developmental care for 92 preterm infants weighing less than 1250 g (2.7 lb) included shorter duration of parenteral feeding and time to full oral feeding; decreased time in intensive care and in hospital; lower incidence of NEC; improved weight, length, and head circumferences; enhanced autonomic, motor, state, attention, and self-regulatory function; and lowered family stress (Als, Gilkerson, Duffy, et al, 2003). In contrast, the Cochrane review done by Symington and Pinelli (2006) notes that, because of the inclusion of multiple interventions, it is difficult to ascribe specific benefits to the implementation of developmental care.

Because each infant is unique, supportive developmental care requires ongoing data collection of moment-by-moment responses and flexible care to address the infant’s cues. For example, an infant who demonstrates altered vital signs and even apnea after being weighed might benefit from swaddled weighing to support the infant’s competence and organization during a stressful procedure.

Knowledge of behavioral assessment and infant development assists the nurse in providing care that supports each infant’s ongoing function in a manner consistent with current evidence. Nurses have the greatest impact on the daily routine experienced by their tiny patients. The CNS is undergoing rapid and significant change during the preterm infant’s stay in the NICU. This vulnerable period of brain growth, differentiation, and organization is combined with the challenge of developing in environmental conditions that are not typical for the fetus and newborn (Blackburn, 2007). Brain organization peaks from about 20 weeks of gestation to several years after birth. The product of this complex process is establishment of an elaborate circuitry unique to the human brain.

**Behavioral State Organization**

Traditional nursing placed emphasis on interpreting physiologic data as the basis of caregiving. Developmentally support-
CHAPTER 10  The High-Risk Newborn and Family

TABLE 10-5  SYNACTIVE THEORY OF DEVELOPMENT: NEUROBEHAVIORAL SUBSYSTEMS

<table>
<thead>
<tr>
<th>SUBSYSTEM</th>
<th>SIGNS OF STRESS</th>
<th>SIGNS OF STABILITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic</td>
<td>Physiologic instability</td>
<td>Physiologic stability</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Tachypnea, pauses, gasping, sighing</td>
<td>Smooth, stable respirations; regular rate and pattern</td>
</tr>
<tr>
<td>Color</td>
<td>Mottled, flushed, dusky, pale or gray</td>
<td>Pink, stable color</td>
</tr>
<tr>
<td>Visceral</td>
<td>Hiccups, gagging, choking, spitting up, grunting and straining as if having a bowel movement; coughing, sneezing, yawning</td>
<td>Absence of hiccups, gagging, spitting up, etc.</td>
</tr>
<tr>
<td>Autonomic</td>
<td>Tremors, startles, twitches</td>
<td>Absence of tremors, startles, twitches</td>
</tr>
<tr>
<td>Motor</td>
<td>Fluctuating tone, lack of control over movement, activity, and posture</td>
<td>Consistent tone; controlled or improved movement, activity, and posture</td>
</tr>
<tr>
<td>Flaccidity</td>
<td>Low tone in trunk; limp, floppy upper and lower extremities; limp, drooping jaw (gape face)</td>
<td>Tone consistent and appropriate for postmenstrual age</td>
</tr>
<tr>
<td>Hypertonicity</td>
<td>Arm or leg extensions, arm(s) outstretched with fingers splayed in salute gesture, fingers stiffly outstretched, trunk arching, neck hyperextended</td>
<td>Well-maintained posture</td>
</tr>
<tr>
<td>Hyperflexion activity</td>
<td>Trunk hyperflexion, hyperflexion of extremities, fisting; squirming; frantic diffuse activity or little or no activity or responsiveness</td>
<td>Successful motor strategies for self-regulation (see Self-Regulation below)</td>
</tr>
<tr>
<td>State</td>
<td>Disorganized quality to state behaviors, including range of available states, maintenance of state control, and transition from one state to another</td>
<td>Easy-to-read state behaviors that are maintained; calm, focused alertness, well-modulated sleep</td>
</tr>
<tr>
<td>Sleep</td>
<td>Whispering sounds, facial twitching, irregular respirations, fussing, grimming, restless appearance</td>
<td>Clear, well-defined sleep states; periods of quiet, restful sleep</td>
</tr>
<tr>
<td>Awake</td>
<td>Glazed unfocused look, staring, worried or in pain expression, hyperalert or panicked appearance, eye roving, crying, &quot;cry-face,&quot; actively avverting gaze or closing eyes, irritability, prolonged awake periods, inconsolability, frenzy</td>
<td>Alert with bright, shiny eyes; focused attention on object or person; animated expression (e.g., cheek softening, frowning, &quot;ooh-face,&quot; &quot;cooing, smiling)</td>
</tr>
<tr>
<td>Other state-related behaviors and attention-interaction</td>
<td>Efforts to attend to and interact with environmental stimulation eliciting signs of stress and disorganized subsystem functioning</td>
<td>Responsive to auditory, visual, and social stimuli</td>
</tr>
<tr>
<td>Autonomic</td>
<td>Physiologic instability of varying degrees with autonomic, respiratory, color, and visceral responses</td>
<td>Responsiveness to stimuli well maintained and prolonged</td>
</tr>
<tr>
<td>Motor</td>
<td>Fluctuating tone, increased motor activity, progressively frantic diffuse activity if stimulation continues</td>
<td>Actively seeking auditory stimulation, minimum motor activity</td>
</tr>
<tr>
<td>State</td>
<td>Roving eyes, gaze averted, glaze-unfocused look or worried, panicked expression; weak cry; cry-face; irritability</td>
<td>Bright, shiny-eyed, alert, and attentive expression</td>
</tr>
<tr>
<td>Other state-related behaviors and attention-interaction</td>
<td>Efforts to attend to and interact with environmental stimulation eliciting signs of stress and disorganized subsystem functioning</td>
<td>Responsive to auditory, visual, and social stimuli</td>
</tr>
<tr>
<td>Self-regulation</td>
<td>Infant’s efforts to achieve, maintain, or regain a balanced, stable, and relaxed state of subsystem functioning and integration. Success of these efforts will vary among infants depending on maturity, available self-regulatory skills, and overall subsystem organization. Examples of self-regulatory strategies include:</td>
<td></td>
</tr>
<tr>
<td>Facial—Foot bracing against a boundary or blanket nest, hand holding, clasping hands together, hand to mouth or face, grasping blanket, tubing, tucking trunk, sucking, position changes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>State—Lowering state from high arousal to quiet alert or sleep state; releasing energy by rhythmic, robust crying; focused attention and orientation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Facilitation by caregivers</td>
<td>Environmental modifications or developmental care techniques to aid infant’s own self-regulatory abilities when environmental challenges exceed infant’s capabilities.</td>
<td></td>
</tr>
</tbody>
</table>


Intensive care uses both physiologic and behavioral information to better understand the needs of infants in the NICU setting. Behavioral states are highly individualized and formed by experience, maturation, circadian rhythms, and genetic inheritance. The emerging availability and regulation of arousal states mark a balancing of CNS inhibitory and excitatory processes that affect attention states and also mark executive functions (prefrontal cortex) that influence information processing, learning, and socialization. State organization has been described as a gating mechanism that protects the cortex from overstimulation and promotes coordination between attentional, executive, and sensory cortical systems.
TABLE 10-6 AROUSAL STATES

<table>
<thead>
<tr>
<th>STATE</th>
<th>DESCRIPTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep sleep</td>
<td>Regular breathing; eyes closed with no movement of eyes under lid; relaxed face; little or no movement or activity except for possible startle response</td>
</tr>
<tr>
<td>Active sleep</td>
<td>Sometimes called light sleep; may see rapid eye movements under closed lids, low activity level, breathing regular or irregular, occasional sighing or smiling</td>
</tr>
<tr>
<td>Drowsy</td>
<td>Eyes open or closed, unfocused expression; activity level varied</td>
</tr>
<tr>
<td>Quiet awake</td>
<td>Different qualities of alerting</td>
</tr>
<tr>
<td>Robust</td>
<td>Bright, shiny appearance to eyes; focused attention; minimum motor activity</td>
</tr>
<tr>
<td>Low level</td>
<td>Dull or unfocused eyes; little energy; appears to look through object or caregiver</td>
</tr>
<tr>
<td>Hyperalert</td>
<td>Wide eyes, panicked expression; may fixate on object or caregiver intensely and have trouble breaking away</td>
</tr>
<tr>
<td>Active awake</td>
<td>Active; eyes open or closed; fussy but not crying robustly</td>
</tr>
<tr>
<td>Crying</td>
<td>Highest level of arousal; agitated, rhythmic, and robust crying</td>
</tr>
</tbody>
</table>

Modified from Als H: Manual for the naturalistic observation of newborn behavior, Newborn Individualized Developmental Care and Assessment Program (NIDCAP), Boston, 1995, Harvard Medical School.

Infant responsiveness to environmental stimuli depends on the quality, amount, and availability of particular states of arousal. States can be organized into five levels of arousal (Table 10-6). Transitional states such as drowsiness are not considered true states but are in-between levels of arousal in which the infant either moves toward wakefulness or back into sleep.

Distinct sleep and awake states are observable in infants between 25 and 27 weeks (Holditch-Davis and Blackburn, 2007). Young preterm infants spend 70% or more of their time in active sleep. Developmental maturation for the young preterm infant is seen by a decrease in the amount of active sleep with an increase in quiet sleep, awake periods, and crying. Around 30 to 32 weeks, quiet alert states with some focused attention can occur. Before 28 weeks, attempts to attend to stimuli may have physiologic consequences for the immature infant (Blackburn, 1998). Responsiveness to sound and touch is greater during active or light (rapid eye movement [REM]) sleep, resulting in longer periods of vulnerability to sleep disturbance (Holditch-Davis, 1998). Maturation continues throughout the first year of life. By 6 months, the amount of quiet sleep is greater than that of active sleep. By 1 year, infants usually sleep 10 to 12 hours at night and take one or two naps during the day. Preterm infants generally sleep for shorter periods at night and awaken more frequently than full-term infants. Other maturational changes include organization of the standard sleep cycle and electroencephalogram sleep patterns comparable to those of adults. Neurologic insults, severity of illness, hyperbilirubinemia, and prenatal exposure to drugs can alter behavioral state patterns.

Physiologic parameters vary depending on level of arousal. Heart rate is higher during waking periods but more variable during active sleep. Blood pressure is higher during wakefulness. Cerebral blood flow is greater during active sleep (greater during quiet sleep in full-term infants). Respiratory rates fluctuate more and are higher in active sleep. Arterial oxygen and carbon dioxide levels are lower in active sleep than in quiet sleep or awake states. Hypoventilation and poorly coordinated chest wall and abdominal movements are reported during active sleep. Apneic pauses of less than 20 seconds are more frequent in active than quiet sleep in preterm infants.

Nursing care should be timed to the responsiveness of the infant as much as possible to optimize the development of sleep organization and enhance alerting as it emerges. Sensory stimulation can influence behavioral state as seen by either increased or decreased infant arousal when presented with a stimulus and its removal; the type of stimulus (e.g., loud bell or soft lullaby) also is a factor. The quality of each state, its duration, and the movement between states provide information concerning how well organized the state is and how much state control the infant has. Protection of sleep is an important goal for both the preterm and full-term infant. Environmental modifications and timing of care to provide longer episodes of undisturbed sleep should be planned into care.

Nurses can also support transitions between states. Gentle arousal to wakefulness by soft speech or gentle touch before caregiving is preferable to the traditional model in which care is begun without warning and with abrupt disruption of sleep. Slow movements and gentle handling support quiet alerting or return to sleep without periods of arousal after care is over. Nurses should facilitate return to sleep or interact with a quietly alert infant after care events.

An infant’s state of arousal allows for communication of responses that are valuable for individualized caregiving. Observing state patterns and individual responses of infants, nurses can better know their patients and support behavioral state organization. The nurse can also share this knowledge with parents to foster intimacy with the child.

**Sensory System**

Research with animal fetuses and infants has shown that atypical sensory experiences, whether overstimulating or depriving, can modify the developing brain (Glass, 2005; Lickliter, 2000a). In fact, much of the cerebral cortex is associated with the sensory system. Most sensory systems develop prenatally and are capable of functioning before birth. Onset of function of each sensory system proceeds in the same order for each individual (i.e., tactile, vestibular, gustatory-olfactory, auditory, and visual). The visual system becomes functional after birth. Sensory input provided before the stimulation would typically occur has been shown to interfere with perceptual and behavioral development (Lickliter, 2000b).

The normal experience for the preterm infant is within the womb and for the full-term infant is the home environment with a few primary caregivers. These environments are vastly different from the NICU. The NICU experience for the high-risk infant is made up of external conditions and interactions with caregivers. Often that experience is overstimulating to later-developing sensory systems (i.e., auditory and visual) and understimulating to earlier ones (i.e., tactile, vestibular, gustatory-olfactory). Alterations in the sensory environment may have developmental consequences. It is important for the nurse...
to consider the following while caring for high-risk infants: (1) timing of stimulation in relation to the infant’s current developmental stage, (2) amount of stimulation provided or denied, (3) type of stimulation, and (4) the infant’s response to the stimulation (Lickliter, 2000b).

By the age of viability, infants in the NICU have sophisticated perioral sensation and perceive pressure, pain, and temperature (Als, 1999). Touch in the NICU frequently involves routine, sometimes impersonal, caregiving and procedures that are either intrusive or painful. Even nonpainful care has been associated with adverse responses in preterm infants.

Preterm infants demonstrate cry expression, grimacing, and knee and leg flexion during major reposition changes. Hypoxemia, associated with nonpainful or routine caregiving activities such as suctioning, repositioning, taking vital signs, changing diapers, and removing electrodes, has been reported (Glass, 2005). Other physiologic changes involve blood pressure, heart rate, and respiratory rhythm and rate (Glass, 2005; Browne, 2000; Peters, 1999). Nursing activities that are painful or especially intrusive, such as needle puncture, suctioning, and chest physiotherapy, have resulted in acute decreases in SaO2 and behavioral state changes in preterm infants ranging from 23 to 37 weeks of gestation (Zahr and Balian, 1995). Increased motor activity, agitation, crying, and startle reflex have also been described as negative behavioral responses to touch (Browne, 2000).

Touch is the first sensory system to develop and forms the basis for early communication between infants and caregivers. In particular, touch is a powerful means of emotional exchange for parents and infants. Positioning and handling techniques promote comfort and minimize stress, while creating a balance between nurturing care and necessary interventions.

**Therapeutic Handling**

Using the developmental model of supportive care, the nurse closely monitors physiologic and behavioral signs to promote organization and well-being of high-risk infants during handling (Box 10-5). The type, timing, and amount of handling are carefully considered in terms of the infant’s current age, condition, vulnerabilities, thresholds for stress, and capabilities. Because touch can be disruptive to maturing sleep-wake states, avoid waking an infant for care or nurturing. Sleep deprivation may affect secretion of growth hormone and interfere with growth and development (Hunter, 2001).

Respectful approach before touching an infant allows more time for transition and adaptation from being alone to being handled. The nurse can use the infant’s own cues to determine optimum times for caregiving rather than following a rigid schedule. The best time for care is when an infant is awake. If the care or procedure cannot be postponed, softly calling an infant by name and then gently placing a hand on the body signals care is beginning and avoids the abrupt interruption that frequently precedes caregiving. Abrupt transitions can disrupt even organized functioning of an infant’s autonomic, motor, and state subsystems.

Infants who are unable to maintain a gently flexed position during repositioning or care procedures may benefit from containment. Gently holding the infant’s arms and legs in a tucked, flexed position close to the body can be accomplished with hands or blanket swaddling. **Facilitated tucking** before ET suctioning was shown to decrease physiologic and behavioral distress in preterm infants as young as 23 weeks of gestational age (Ward-Larson, Horn, and Gosnell, 2004). Blanket swaddling and nesting or containment decreased physiologic and behavioral stress during routine care procedures such as bathing, weighing, and heel lance (Byers, 2003).

Because repositioning has been associated with significant physiologic distress in immature infants, avoid sudden postural changes. Slow turning while containing the infant’s extremities in a gently tucked, midline position may reduce the impact of this procedure.

Stroking preterm infants who are not physiologically stable has been reported to result in behavioral signs of distress such as gasping, grunting, gaze aversion, and decreased tcPo2 levels. Some infants experience apnea and bradycardia during massage or tactile-kinesthetic stimulation (Glass, 2005). Other researchers report positive benefits of gentle human touch, including heart rate and oxygen saturation stability (Modcrinc-Talbott, Harrison, Groer, et al, 2003). Individual infants show varied responses to tactile intervention, further supporting the need for close monitoring of behavioral and physiologic parameters.

Investigators have reported positive benefits of massage on stable, growing, preterm infants. Beachy (2003) found that, when infant massage therapy is properly applied to stable preterm infants, they respond with increased weight gain, enhanced developmental scores, and shortened hospital stays. Parents of the preterm infant also benefit because infant massage enhances bonding with their child and increases confidence in their parenting skills. Dieter, Field, Hernandez-Reif, and colleagues (2003) studied 16 preterm infants (mean gestational age of 30 weeks) and found that after 5 days of receiving massage therapy, infants in the treatment group averaged a 53% greater weight gain and spent less time sleeping than control infants.

**BOX 10-5  CONSIDERATIONS FOR TACTILE INTERVENTIONS IN THE NEONATAL INTENSIVE CARE UNIT**

- Modify all handling and touch so that it is supportive and calming.
- Consider sleep-wake states and behavioral cues to determine optimum times for handling and touch.
- Adjust handling and touch based on continual observation of the infant’s autonomic and behavioral responses.
- Ensure appropriate touch opportunities for parents aside from routine caregiving.
- Encourage parents to be primary providers of social touch.
- Avoid using massage with vulnerable high-risk infants (e.g., medically unstable, low-birth-weight infants less than 32 weeks of gestation; easily disorganized, low-threshold infants; chronically ill infants with chronic lung disease or cardiac disorders known to display physiologic and behavioral disorganization).
- Assist parents in identifying the most appropriate type of touch and handling for their infant.
- Teach infant cues to parents for monitoring responses to handling and touch.
- Weigh the risks and benefits for any tactile intervention.
Vickers, Ohlsson, Lacy, and colleagues (2004) evaluated literature relevant to infant massage, gentle touch, and gentle human touch. The researchers concluded that available studies, although demonstrating advantages to massage for preterm infants, lack sound methodologic bases on which firm recommendations can be made to advocate wide-scale use of this intervention.

**Kangaroo care**, or STS holding, has been advocated for fostering neurobehavioral development and supporting parent-infant intimacy and attachment. STS contact is maintained with the diaper-clad infant resting prone and semiupright on the bare chest of either parent, who encloses the infant in his or her own clothing to maintain temperature stability. Kangaroo care is reported to reduce incidence of severe illness and nosocomial infection, support breast-feeding duration until discharge, improve maternal satisfaction (Conde-Agudelo, Diaz-Rossello, and Belizan, 2000) and parental interaction (Feldman, Eidelman, Sirota, et al, 2002), and accelerate neurologic maturation (Feldman and Eidelman, 2003). Hurst, Valentine, Renfro, and colleagues (1997) reported a significant increase in milk volume in mothers providing kangaroo care to stable, ventilated, LBW infants (mean 27.7 weeks of gestation) compared with a control group. Others have reported advantages that include maintenance of skin temperature, reduction of apnea and bradycardia, stable tcPo2 level, increased frequency and duration of quiet sleep, less time crying, and lower activity levels during kangaroo care (Roberts, Paynter, and McEwan, 2000; Byers, 2003). Kangaroo care has been successfully initiated in stable preterm infants who weigh less than 1000 g (2.2 lb) (Neu, Browne, and Vojir, 2000).

However, in a study of preterm infants (mean gestational age of 29 weeks), kangaroo care was associated with a significant increase in bradycardia, less regular breathing, and hypoxemia. Temperatures increased (as measured rectally) every 2 hours (Bohnhorst, Heyne, Peter, et al, 2001). Although adverse effects are not usually associated with kangaroo care, monitoring to avoid potential harmful effects should include cardiorespiratory parameters, body temperature, and oxygenation.

Infants appear to be most vulnerable during the transfer from bed to parent and back to bed when kangaroo care is provided. Handling and repositioning necessary to prepare and move an infant into the STS position may result in similar disorganizing responses as previously described with routine nursing care.

**Therapeutic Positioning**

The American Academy of Pediatrics (2005) recommends the supine sleeping position for healthy infants in the first year of life as a preventive measure for sudden infant death syndrome (SIDS). Prone sleeping has decreased from more than 70% to about 13% in the United States since the guidelines were published in 1992. SIDS is the third highest cause of infant death after the neonatal period (28 days); the rate has decreased by more than 50% with the advent of supine sleeping. (See Sudden Infant Death Syndrome, Chapter 13.)

Parents of infants in the NICU should be educated on the safe sleeping position at home as part of discharge instructions. Supportive positioning in the NICU for acutely ill or recovering infants may look different from the academy’s recommenda-

tions, depending on each infant’s changing clinical condition, maturation, and readiness for the supine sleeping position and minimum bedding. It is important for staff to realize that routine care practices in the NICU may serve as a model for parents who, without proper instruction, may reproduce the environment and care techniques at home. Position and bedding choices in the unit, such as prone positioning, nests, and sheepskin, may be lethal for infants who have been discharged home.

In infants in the NICU are at increased risk for acquiring position-related deformities for a variety of reasons. Illness, weakness, low tone, immature motor control, the effects of gravity, and treatments such as ECMO or sedation are a few of the factors associated with prolonged immobility or decreased spontaneous movement (Hunter, 2001). Common position-related deformities include:

- Hyperabduction and flexion of the arms, causing upper extremity external rotation, resulting in a persistent W positioning of the arms; can interfere with later midline skills that form the foundation for feeding, crawling, reaching, and midline play with objects (Vaire-Douret, Ennouri, Ijad, et al, 2004)
- Lower extremity external rotation deformities occurring when the trunk and pelvis are flat on the mattress, causing extreme hip abduction and outward rotation of the lower limbs, or the frog-leg appearance (Downs, Edwards, McCormick, et al, 1991)
- Neck extension and arching posture often observed in infants pulling away from ET tubes or nasal prongs during mechanical ventilation or nasal CPAP
- Motor asymmetries reported in preterm infants at 32 weeks of gestation or who are small for gestational age, occurring more often than in full-term infants even after 4 months corrected age (Samsom and de Groot, 2000, 2001)

**Therapeutic positioning** reduces the potential for acquired positional deformities that can affect motor development, play skills, attractiveness, and social attachment (Monterosso, Kristjanson, Cole, et al, 2003). Positioning can affect stability and comfort, and each infant must be observed for the effects of any position or repositioning. A position may also need to be adapted to accommodate necessary medical equipment or particular conditions, such as myelomeningocele, where the supine position is contraindicated before surgical repair of the defect. Deciding on which position and supportive aids to use requires the caregiver to consider the medical and developmental risks and benefits unique to a specific infant and situation (see Nursing Care Guidelines box).

The goal of therapeutic positioning for preterm and high-risk infants is to provide adequate support and containment as indicated to sustain flexed and midline postures, in an attempt to minimize positional deformities and assist infants in remaining calm and organized (Hunter, 2001).

The supine position requires support for the weak or immature infant. Because this position can create the most disorganization, make the position comfortable using positioning aids or blanket boundaries that support the head, trunk, and extremities according to the general positioning principles.

Although the prone position may appear to be the easiest to maintain, mistakes are often made with infants who are unable
NURSING CARE GUIDELINES
General Considerations for Positioning

- Neutral or slightly flexed neck
- Gently rounded shoulders (no flattened posture against bed as in supine or prone positions)
- Elbows flexed
- Hands to face or midline as position allows
- Trunk slightly rounded with pelvic tilt
- Hips partially flexed and adducted to near midline (not medial or neutral alignment) and knee flexion (no frog leg or externally rotated hips flat against bed)
- Lower boundary secured for foot bracing


Fig. 10-4 Preterm infant slowly and gently transitioned to prone position on prone roll designed with stockinette-covered foam cut to individual specifications to prevent flattening of shoulders and pelvis against mattress and to support stable breathing base for the infant. (Courtesy Paul Vincent Kuntz, Halbouty Premature Nursery, Texas Children’s Hospital, Houston.)

Fig. 10-5 Preterm infant positioned on prone roll. (Courtesy Paul Vincent Kuntz, Halbouty Premature Nursery, Texas Children’s Hospital, Houston.)

to sustain rounded shoulders, trunk, and pelvis without assistance. Use of a postural support roll has been shown to prevent scapular-humeral tightness and shoulder retraction commonly seen as a result of this position (Figs. 10-4 and 10-5) (Monterosso, Kristjanson, Cole, et al, 2003).

Auditory Environment
The auditory system of the human fetus is mature enough for sound to produce physiologic effects as early as 23 weeks of gestation (Graven, 2000). Physical and behavioral responses to sudden, loud NICU noise have been observed in preterm and full-term infants (Philbin and Klaas, 2000; Bremmer, Byers, and Kiehl, 2003). Physiologic changes include apnea and bradycardia; fluctuations in heart and respiratory rates, blood pressure, and oxygen saturation; and changes in sleep-wake states (Philbin and Klaas, 2000, Bremmer, Byers, and Kiehl, 2003). These data demonstrate that infants in the NICU are capable of perceiving and responding to sounds around them.

The primary auditory environment in fetal life is made up of the maternal voice, respirations, heartbeat, and intestinal sounds. Soon after birth, newborn infants demonstrate preference for their own mother’s voice and the language heard in utero (Gerhardt and Abrams, 2000). The acoustic environment of most NICUs is vastly different from the uterine and home milieu. Currently no data are available on the effects of long-term exposure to NICU noise levels. Of serious concern is the increased risk of sensorineural hearing loss (Cristobal and Oghalai, 2008) and language delays in infants born prematurely (Pietz, Peter, Graf, et al, 2004). NICU noise may interfere with developing auditory pathways and mask socially relevant sounds of the human voice necessary for language development.

Maintaining recommended sound levels (<45 dB) in the NICU may provide some or all of the following benefits: (1) increased physiologic stability, (2) improved growth, (3) more natural and consistent neurosensory maturation, (4) enhanced parent-infant interaction and subsequent attachment, and (5) fewer speech and language difficulties (Graven, 2000).

Visual Environment
Vision is the least mature of the newborn’s senses. The preterm infant’s eyes undergo significant maturation and differentiation of the retina and its connections to the visual cortex that typically occur in utero during the last trimester of pregnancy (Glass, 2005; Hunter, 2001). A current concern is that early, intense visual stimulation for preterm infants could adversely affect visual pathways and alter the developmental course for other sensory systems.

Visual function in preterm infants is more limited than that in full-term infants who, although limited in ability to focus (accommodation to near and far distances) and discriminate (acuity), will actively explore the environment. Preterm infants are less responsive to visual stimulation and have less acuity and accommodation than full-term infants. The ability to visually attend emerges around 30 to 32 weeks, and the infant may become stressed if the visual stimulus is intense and prolonged. Strong visual stimulation such as high-contrast black and white patterns can evoke an obligatory staring response by the immature infant who is unable to break away from it. This behavior is neither appropriate nor desired.

A variety of lighting conditions exist for NICUs from continuous 24-hour illumination, continuous dim lighting, day-night cycled lighting, or unpredictable periods of light-dark depending on staff or situations. Ambient lighting in some NICUs is reported to range from 40 to 150 foot-candles (ft-c) during the day with levels over 1500 ft-c if sunlight is added (Glass, 2005; Blackburn, 1998). These levels drop dramatically at night if light is cycled to reported levels of 5 to 9 ft-c (Fielder and Merrick, 2000).
Staff needs to carefully consider the impact of visual stimuli on the NICU infant. For preterm infants whose visual system is undergoing maturation, it is probably more prudent to provide stimulation to the earlier-developing senses first and minimize the impact of the NICU visual milieu. As attention and alerting emerge, the most appropriate visual stimulus is likely to be the human face, especially that of the parent. Box 10-6 provides some suggested approaches to visual stimulation in the NICU (Glass, 2005; Hunter, 2001; Mirmiran and Ariagno, 2000).

Facilitating Parent-Infant Relationships

Because of their physiologic instability, preterm infants are immediately separated from their mothers and surrounded by a complex, impenetrable barrier of glass windows, mechanical equipment, and special caregivers. Increasing evidence indicates that the emotional separation that accompanies the physical separation of mothers and infants interferes with the normal maternal-infant attachment process, discussed in Chapter 8. Maternal attachment is a cumulative process that begins before conception, strengthens by significant events during pregnancy, and matures through maternal-infant contact during the neonatal period.

When an infant is sick, the necessary physical separation appears to be accompanied by an emotional estrangement in the parents, which may seriously damage the capacity for parenting their infant. This detachment is further hampered by the tenuous nature of the infant’s condition. When survival is in doubt, parents may be reluctant to establish a relationship with their infant. They prepare themselves for the infant’s death while continuing to hope for recovery. This anticipatory grief (see Chapter 23) and hesitancy to embark on a relationship are evidenced by behaviors such as delay in giving the infant a name, reluctance to visit the nursery (or focusing on equipment and treatments rather than on their infant when they do visit), and hesitancy to touch or handle the infant when given the opportunity.

Comprehensive management of high-risk newborns includes encouraging and facilitating parental involvement rather than isolating parents from their infant and associated care (Box 10-7). This is particularly important in relation to mothers; to reduce the effects of physical separation, mothers are united with their newborns at the earliest opportunity (Fig. 10-6).

Preparing the parents to see their infant for the first time is a nursing responsibility. Before the first visit, the nurse prepares parents for their infant’s appearance, the equipment attached to the child, and the general atmosphere of the unit. The initial encounter with the intensive care unit is stressful, and the frightening array of people, equipment, and activity is likely to be overwhelming. A book of photographs or pamphlets describing the NICU environment (infants in incubators or under radiant warmers, monitors, mechanical ventilators, and IV equipment) provides a useful and nonthreatening introduction to the NICU.

**BOX 10-6 VISUAL STIMULATION: CONSIDERATIONS FOR INFANTS**

- Decrease ambient light levels by dimming lights or using incubator covers for lower-birth-weight and lower-gestational-age infants.
- Facilitate eye opening and visual attention in older preterm and term infants by dimming overhead lights.
- Direct procedure lights toward the necessary visual field and away from infants’ eyes when performing tasks that require visual acuity such as intravenous catheter insertion.
- Shield infants’ eyes from bright procedure lights or full ambient lighting as needed during examinations, treatments, or procedures.
- Avoid placing a cloth over the face or using eye patches that provide tactile irritation unless necessary for phototherapy or special circumstances.
- Ensure eye patches are securely in place during phototherapy.
- Introduce day-night cycling of lighting in the neonatal intensive care unit and intermediate nursery before discharge.
- Consider the human face the most appropriate visual stimulation in early infancy.
- Avoid leaving visual stimuli in the beds of infants who cannot escape from it.
- Provide appropriate visual stimuli or toys for recovering full-term or older infants.

**Fig. 10-6** Encouraging interaction of mother and her preterm infant in the intensive care unit facilitates mother-infant attachment process. (Courtesy E. Jacobs, Texas Children’s Hospital, Houston.)

**BOX 10-7 PSYCHOLOGIC TASKS OF PARENTS OF A HIGH-RISK INFANT**

- Work through the events surrounding labor and delivery.
- Acknowledge that the infant’s life is endangered and begin the anticipatory grieving process.
- Recognize and confront feelings of inadequacy and guilt in not delivering a healthy child.
- Adapt to the neonatal intensive care environment.
- Resume parental relationships with the sick infant and initiate the caregiving role.
- Prepare to take the infant home.

Encourage parents to visit their infant as soon as possible. Even if they saw the infant at the time of transport or shortly after birth, the infant may have changed considerably, especially if there are a number of medical and equipment requirements associated with the infant’s hospitalization. At the bedside the nurse should explain the function of each piece of equipment and the role it plays in facilitating recovery. Explanations may often need to be patiently repeated because parents’ anxiety over the infant’s condition and the surroundings may prevent them from really “hearing” what is said. When possible, some items related to therapy can be removed; for example, phototherapy can be temporarily discontinued and eye patches removed to permit eye-to-eye contact.

Parents appreciate the support of a nurse during the initial visit with their infant, but they may also want some time alone with the infant. It is important during the early visits to emphasize the positive aspects of their infant’s behavior and development so that parents can focus on their infant as an individual rather than on the equipment that surrounds the child. For example, the nurse may describe the infant’s spontaneous behaviors during care, such as grasp, sucking, and movement, or make comments about the infant’s biologic functions. Most institutions promote family-centered care and have open visiting policies so that parents and siblings can visit as often as they wish.

Parents vary greatly in the degree to which they are able to interact with their infant. Some may wish to touch or hold their infant during the first visit, whereas others may not feel comfortable enough to even enter the nursery. Parents may not be receptive to early and extended infant contact because they need time to adjust to the impact of an infant with birth problems and must be helped to grieve before they can accept their infant.

The parents’ inability to focus on their infant is a clue for the nurse to assist the parents in expressing feelings of guilt, anxiety, helplessness, inadequacy, anger, and ambivalence. Nurses can help parents deal with these distressing feelings and recognize that they are normal responses shared by other parents. It is important to point out and reinforce the positive aspects of parents’ behavior and interactions with their infant.

WARD’s (2001) research of parental needs in the NICU demonstrates the importance of nurses providing accurate information to the parent regarding treatment plan and procedures; answering parents’ questions honestly; actively listening to parents’ concerns, fears, and expectations; and helping parents understand infant responses to hospitalization. WARD’s (2001) research of parental needs in the NICU demonstrates the importance of nurses providing accurate information to the parent regarding treatment plan and procedures; answering parents’ questions honestly; actively listening to parents’ concerns, fears, and expectations; and helping parents understand infant responses to hospitalization.

Most parents feel shaky and insecure about initiating interaction with their infant. Nurses can sense parents’ level of readiness and offer encouragement in these initial efforts. Parents of preterm infants follow the same acquaintance process as do parents of full-term infants. They may quickly proceed through the process or may require several days, or even weeks, to complete it. Parents begin by touching their infant’s extremities with their fingertips and poking the infant tenderly, and then proceed to caresses and fondling (Fig. 10-7). Touching is the first act of communication between parents and child. Parents need to be prepared for their infant’s exaggerated and generalized startle responses to touch so that they will not interpret these as negative reactions to their overtures. It may be necessary to limit tactile stimuli when the infant is critically ill and labile, but the nurse can offer other options, such as speaking softly or sitting at the bedside.

Parents of acutely ill preterm infants may express feelings of helplessness and lack of control. Involving the parent in some type of caregiving activity, no matter how minor it may seem to the nurse, enables the parent to take on a more active role. Examples of such caregiving for the acutely ill infant who cannot be held and is seemingly not responding positively include moistening the infant’s lips with a small amount of sterile water on a cotton-tipped swab or slipping the diaper under from under the infant when it is wet or soiled.

The nurse encourages parents to bring in clothes, a toy, a stuffed animal, or a family snapshot for their infant, and the nurse can help parents set goals for themselves and for the infant. Parents may become involved by reading a children’s storybook or nursery rhymes in a soft, soothing voice. The nurse encourages parents to visit at times when they can become involved in their infant’s care (Figs. 10-8 and 10-9).

Throughout the parent-infant acquaintance process, the nurse listens carefully to what the parents say to assess their concerns and their progress toward incorporating their infant into their lives. The manner in which parents refer to their infant and the questions they ask reveal their worries and feelings and can serve as valuable clues to future relationships with the infant. The alert nurse is attuned to these subtle indications of parents’ needs, which provide guidelines for nursing intervention. Open all that the parents need is reassurance that they will have the nurse’s support during caregiving activities and that the behaviors about which they are concerned are normal reactions and will disappear as the infant matures (e.g., an exaggerated Moro reflex or inability to coordinate swallowing).

Parents need guidance in their relationships with their infant and assistance in their efforts to meet their infant’s physical and developmental needs. The nursing staff must help parents understand that their preterm infant offers few behavioral rewards and show them how to accept small rewards from their infant. They need reassurance that avoidance behaviors are not a reflection on their parenting skills. Teach parents to recognize
amount of different information given to parents and often instills confidence that, although the parents cannot be at their infant’s bedside 24 hours a day, they can call competent and caring nurses to inquire about the infant’s status. Periodic parent conferences involving the primary practitioner, primary nurse, and associate primary nurse serve to clarify misunderstandings or problems related to the infant’s condition. Other members of the NICU team, such as the perinatal social worker, lactation consultant, discharge coordinator, or surgeon, may become involved as necessary.

siblings

In the past, concerns about sibling visitation in the NICU focused on fears of infection and disruption of nursing routines. These fears have not been substantiated, and sibling visits should be a part of the normal operation of NICUs (Fig. 10-10).

The birth of a preterm infant is a difficult time for siblings, who rely on the support of understanding parents. When the happy anticipation changes to sadness, worry, and altered routines, siblings are bewildered and deprived of their parents’ attention. They know something is wrong, but they do not completely understand what it is. Concern about the negative effects on visiting siblings of seeing the ill newborn has not been confirmed. Children have not hesitated to approach or touch the infant, and children less than 5 years of age have been less reluctant than older children. In addition, no measurable differences were found between previsit and postvisit behaviors.

The potential benefits of sibling visits must be weighed against the negatives of exposing the child to the NICU environment. Children must be prepared for the unfamiliar NICU atmosphere, but contact with the infant appears to have a positive effect on siblings by helping them deal with the reality rather than the bizarre fantasies that are characteristic of young children. Such visits also help bond the family as a unit.

Support Groups

Parents need to feel they are not alone. Parent support groups have been of immeasurable value to families of infants in the NICU. Some groups consist of parents who have infants in the hospital and share the same anxieties and concerns. Other
groups include parents who have had infants in the NICU and who have dealt with the crisis effectively. The groups are usually under the leadership of a staff person and may involve physicians, nurses, and social workers, but it is the parents who can offer other parents something that no one else can provide.

Family Support America* (formerly Family Resource Coalition) is a North American network of family support programs designed to help families of preterm infants. An excellent resource for parents of preterm infants is the book by J. Zaichkain, Newborn Intensive Care: What Every Parent Needs to Know (2009). This resource has technical and anecdotal information regarding different problems facing preterm infants, common treatments and therapies, preparation for home discharge, and home care for the preterm infant.

Discharge Planning and Home Care

Parents become apprehensive and excited as the time for discharge approaches. They have many concerns and insecurities regarding the care of their infant. They fear the child may still be in danger, that they will be unable to recognize signs of distress or illness, and that the infant may not be ready for discharge. Nurses need to begin early to assist parents in acquiring or increasing their skills in the care of their infant. Appropriate instruction must be provided and sufficient time allowed for the family to assimilate the information and learn the continuing special care requirements. Where rooming-in or other live-in arrangements are available, parents can stay for a few days and nights and assume the care of their infant under the supervision and support of the nursery staff.

There should be appropriate medical and nursing follow-up care, including developmental follow-up, and referrals to services that can benefit the family. Parents of preterm infants should also receive information about immunizations, including respiratory syncytial virus (RSV) prophylaxis, as well as other discharge planning information. Home health agencies provide nursing supervision, counseling, and referrals for nursing visits. With early discharge, many hospital-based home health care agencies become involved in the follow-up monitoring and care of the NICU "graduate" in the home. For an infant being discharged with equipment such as an oxygen tank, apnea monitor, or even a ventilator, discharge planning requires multidisciplinary, collaborative practice to ensure that the family has not only the appropriate resources but also the available assistance for dealing with the infant’s needs. Many communities have organized support groups, including those discussed previously, those designed for parents of infants who require special care because of specific defects or disabilities, and those for parents of multiple births. (See Chapter 3.)

Car seat safety is an essential aspect of discharge planning. It is recommended that infants less than 37 weeks of gestation have a period of observation in an appropriate car seat to monitor for possible apnea, bradycardia, and decreased SaO2 (Bull, Engle, and American Academy of Pediatrics, 2009). One study found that mean oxygen saturation levels decreased from 97% to 94% for both term and preterm infants who were observed in their car seats; 12% of the preterm infants in the study had significant apneic or bradycardic events in their car seats (Merchant, Worwa, Porter, et al, 2001). Despite this evidence, a Cochrane review of predischarge car seat testing failed to find evidence that such testing prevents morbidity or mortality (Pilley and McGuire, 2006) (see Family-Centered Care box).

FAMILY-CENTERED CARE

Preterm and Late-Preterm Infant Car Seat Evaluation

The American Academy of Pediatrics recommends that infants born before 37 weeks of gestation be evaluated for apnea, bradycardia, and oxygen desaturation episodes before hospital discharge. The academy suggests that facilities develop policies for implementing an evaluation program; however, few evidence-based practice recommendations have been published to date delineating specific requirements for such a program. Based on the available evidence, a Cochrane review of predischarge car seat testing identified the following recommendations for providing a car seat evaluation of infants born before 37 weeks of gestation:

- Use the parents’ car seat for the evaluation.
- Perform the evaluation 1 to 7 days before the infant’s anticipated discharge.
- Secure infant in car seat per guidelines using blanket rolls on side.
- Set pulse oximeter low alarm at 88% (arbitrary).
- Set heart rate low alarm limit at 80 beats/min and apnea alarm at 20 seconds (cardiorespiratory monitor).
- Leave the infant undisturbed in car seat for 90 to 120 minutes or until the infant reaches term age.
- Document infant’s tolerance to car seat evaluation.
- An episode of desaturation, bradycardia, or apnea (≥ 220 seconds) constitutes a failure, and evaluation by the practitioner must occur before discharge.
- Repeat the test after 24 hours once modifications are made to the car seat, car bed, or infant’s position in either restraint system.
- It is recommended that a certified car seat technician place the infant in the car seat (or car bed) if a failure occurs (see National Highway Traffic Safety Administration website [www.nhtsa.dot.gov] for car seat inspection station). The technician will demonstrate appropriate positioning of the infant in the restraint device to the parents and have the parents do a return demonstration.
- Document the intervention, the infant’s tolerance, and the parents’ return demonstration.


Several car seat models can be adapted for small infants with the placement of blanket rolls on each side of the infant, but never behind, to support the head and trunk. For adequate support without slumping, the seat back-to-crotch strap distance must be 14 cm (5.5 inches) or less. A small rolled blanket may be placed between the crotch strap and the infant to reduce slouching. If the child’s head drops forward because the position of the seat is upright, a roll cloth or blanket may be placed in the vehicle seat crease and under the safety base so the infant reclines at no more than a 45-degree angle. A car seat restraint

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* Family Support America: www.familysupportamerica.org

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without a shield is recommended; if the infant needs to be supine, a crash-tested car bed may be used (Bull, Engle, and American Academy of Pediatrics, 2009).

The rear-facing position provides support for the head, neck, and back, thereby reducing the stress to the neck and spinal cord in a vehicle crash. It is recommended that, before discharge from the hospital, the preterm infant have an evaluation in the designated car seat restraint by a staff member who is knowledgeable in car seat restraint positioning. Parents should learn how to properly restrain the child in the car seat for safe transportation (Bull, Engle, and American Academy of Pediatrics, 2009).

Additional guidelines are available from the American Academy of Pediatrics, including a videotape for the safe transportation of preterm and LBW infants. (See Chapter 12 for a discussion of infant car restraints and American Academy of Pediatrics website [www.aap.org] for a list of appropriate car seats for infants.)

An important part of discharge planning and care of the preterm infant is nutrition for continued growth; thus choice of feeding must be carefully addressed. Human milk should be fortified according to the infant’s corrected age and physiologic needs. An enriched postdischarge formula (usually 22 kcal/oz) has been recommended for preterm infants born at less than 36 weeks to meet appropriate growth standards (see Table 10–4) (Lucas, Fewtrell, Morley, et al, 2001; Carver, Wu, Hall, et al, 2001). However, a Cochrane review of studies examining growth in preterm infants fed an enriched postdischarge formula did not find strong evidence of enhanced growth and development compared with infants fed standard formula (Henderson, Fahey, and McGuire, 2007).

The term vulnerable child syndrome is applied to physically healthy children who are perceived by their parents to be at high risk for medical or developmental problems. The syndrome has been observed in parents of children who had an illness or injury from which they had not been expected to recover. The family continues to perceive the child as fragile, vulnerable, and “different” and as having needs that warrant special status in the family, which adversely affects the child’s and family’s behavior. The parents may lack confidence in their parenting ability that persists beyond the illness. The parents may also become overly indulgent and have difficulty setting limits, resulting in interference with normal development. Consequently, the child becomes dependent, demanding, and out of control. Overprotection and frequent visits to the health care provider are characteristic.

Problems that may arise in the high-risk newborn include overfeeding, underfeeding, feeding resistance, aversion to human touch or interaction, and difficulty separating the child from the parent. To help parents deal with the stress of home care for the infant, nurses can help families discuss their fears and anxieties, which are exaggerated in parents of preterm infants, and encourage them to create a normal routine in caring for the infant. Parents need to learn the normal developmental delays expected of formerly preterm infants and the importance of setting disciplinary limits and schedules. Continued explanations and clarification of the infant’s true health status and ongoing support of the parents’ efforts are important aspects of follow-up care.

### Neonatal Loss

The precarious nature of many high-risk infants makes death a real and ever-present possibility. Although infant mortality has been reduced sharply with improved technology, the mortality rate is still greatest during the neonatal period. Nurses in the NICU are the persons who must prepare the parents for an inevitable death and facilitate a family’s grieving process after an expected or unexpected death.

The loss of an infant has special meaning for the grieving parents. It represents a loss of a part of themselves (especially for mothers), a loss of the potential for immortality that offspring represent, and the loss of the dream child that has been fantasized throughout the pregnancy. The parents have a sense of emptiness and failure. In addition, when an infant has lived for such a short time, they may have few, if any, pleasant memories to serve as a basis for the identification and idealization that are part of the resolution of a loss.

To help parents understand that the death is a reality, it is important that they be given the opportunity to hold their infant before death and, if possible, be present at the time of death so their infant can die in their arms if they choose.

Parents should have the opportunity to actually “parent” the infant in any manner they wish or are able to before and after the death. This may include seeing, touching, holding, caressing, and talking to their infant privately. The parents may also wish to bathe and dress the infant. If parents are hesitant to see their dead infant, it is advisable to keep the body in the unit for a few hours, since many parents change their minds after the initial shock of the death.

Parents may need to see and hold the infant more than once—the first time to say “hello” and the last time to say “good-bye.” If parents wish to see the infant after the body has been taken to the morgue, the infant should be retrieved, wrapped in a blanket, rewarmed in a radiant warmer, and taken to the mother’s room or other private place. The nurse should plan to stay with the parents but also provide them an opportunity for private time alone with their dead infant if they wish. Individual grief responses of the mother and father should be recognized and handled appropriately. Gender differences and cultural and religious beliefs will affect the parents’ grief responses (Dyer, 2005).

Some units have implemented a hospice approach for families with infants for whom the decision has been made not to prolong life and who are receiving only palliative care. A special “family” room is set aside and contains all supportive equipment needed for the care of the infant. It also provides a home-like atmosphere for the family. All hospice services are available to the family, and the infant remains under the care and supervision of a primary nurse on the NICU staff. (See Chapter 23, End-of-Life Care, for further discussion of hospice care.)

A photograph of the infant taken before or after death is highly desirable. Parents may wish to have a special family portrait taken with the infant and other family members. This often helps personalize the experience and make it more tangible. The parents may not wish to see the photograph at the time of death, but the chance to refer to it later will help to make their infant seem more real, which is a part of the normal grieving process. A photograph of their infant being held by the hand...
or touched by an adult offers a more positive image than a morgue type of photograph. Many NICUs have a bereavement or memory packet made up for the grieving parents, which may include the infant’s handprints and footprints, a lock of hair, the bedside name card, and, if appropriate to the family’s religious beliefs, a certificate of baptism. The photographs and other personal effects of the deceased infant were perceived as critically important in the grieving process by one group of parents in a survey. Parents often indicated that the photographs were helpful in remembering the infant’s actual appearance during this stressful period (Anderson, 2001; Gold, Dalton, and Schwenk, 2007). Naming the deceased infant is an important step in the grieving process. Some parents hesitate to give the newborn a name that had been chosen during the pregnancy for their special “baby.” However, it helps to have a tangible person for whom to grieve. In supporting parental responses to the loss of a child, care providers must understand and respect the cultural and religious practices.

A primary nurse who is familiar to the family should be present during the discussion about the dead or dying infant. A Resolve Through Sharing or bereavement counselor is often involved in helping the family through this difficult period. The nurse should talk with parents openly and honestly about funeral arrangements because few of them have had experience with this aspect of death. Many funeral homes now offer inexpensive arrangements for these special cases. Someone from the NICU should take the responsibility for acquiring this information. It is often helpful to parents for the NICU to have a list of local funeral homes, services offered, and prices. Families need to be informed of the options available, but a funeral is preferable because the ritual provides an opportunity for parents to feel the support of friends and relatives. A clergyman of the appropriate faith may be notified if the parents wish. Issues regarding an autopsy or organ donation (when appropriate) are approached in a multidisciplinary fashion (primary practitioner and primary nurse) with respect, tact, and consideration of the family’s wishes. (See also “Grief and Perinatal Loss” in Merenstein and Gardner, 2006.)

Before the parents leave the hospital, the nurse should provide them with the telephone number of the unit (if they do not have it) and invite them to call any time they have any further questions. Many intensive care units make it a point to contact the parents after a neonatal death to assess the parents’ coping mechanisms, evaluate the grieving process, and provide support as needed. Several organizations are available to offer support and understanding to families who have lost a newborn, including the Compassionate Friends* and Aiding Mothers and Fathers Experiencing Neonatal Death (AMEND).† (See Chapter 23 for further discussion of the family and the grieving process.)

Nurses who care for critically ill infants also experience grief. NICU nurses may feel helpless and sorrowful. It is important that such grief be allowed and that nurses attend the funeral or memorial service as a part of working through the grieving process. Nurses may fear that showing emotion is unprofessional and that the expression of grief demonstrates “loss of control”; these fears are unfounded. Studies have demonstrated that to continue to be effective managers and providers of care, nurses must be allowed to grieve and support each other through the process (Jansen, 2003).

Education regarding bereavement, end-of-life care, and culturally sensitive care of families and their dying infants may help nurses comfort families during this stressful period (Engler, Cusson, Brockett, et al, 2004).

Baptism

Many Christian parents wish to have their child baptized if death is anticipated or is a decided possibility. Whenever possible, it is most desirable that a representative of the parents’ faith (e.g., a Roman Catholic priest or a Protestant minister) perform such a ritual. When death is imminent, a nurse or a physician can perform the baptism by simply pouring water on the infant’s forehead (a medicine dropper is a convenient means) while repeating the words, “I baptize you in the name of the Father and of the Son and of the Holy Spirit.” This includes an infant of any gestational age, particularly when the parents are Roman Catholic.

When the faith of the parents is uncertain, a conditional baptism can be carried out by saying, “If you are capable of receiving baptism, I baptize you in the name of the Father and of the Son and of the Holy Spirit.” The fact of the baptism is recorded in the infant’s chart. Parents are informed at the first opportunity.

### HIGH-RISK CONDITIONS RELATED TO DYSMATURITY

#### PRETERM INFANTS

Prematurity accounts for the largest number of admissions to an NICU. The immaturity not only places infants at risk for neonatal complications (e.g., hyperbilirubinemia and RDS, which has the highest incidence in preterm infants), but may also predispose the infant to problems that persist into adulthood (e.g., learning disabilities, growth deficiencies, asthma).

**Etiology**

A variety of maternal and pregnancy-related complications increase the risk of preterm delivery; however, the actual cause of prematurity is not known in most instances. The incidence of prematurity is lowest in the middle to high socioeconomic classes, in which pregnant women are generally in good health, are well nourished, and receive prompt and comprehensive prenatal care. The incidence is highest in the lower socioeconomic classes, in which a combination of deleterious circumstances is present. Other factors, such as multiple pregnancies, pregnancy-induced hypertension, and placental problems that interrupt the normal course of gestation before completion of fetal development, are responsible for a large number of preterm births.

The outlook for preterm infants is largely, but not entirely, related to the state of physiologic and anatomic immaturity of
the various organs and systems at the time of birth. Infants at term have advanced to a state of maturity sufficient to allow a successful transition to the extraterine environment. Infants born prematurely must make the same adjustments but with functional immaturity proportional to the stage of development reached at the time of birth. The degree to which infants are prepared for extraterine life can be predicted to some extent by estimated gestational age. (See Clinical Assessment of Gestational Age, Chapter 8.) An understanding of prenatal development provides some concept of the status of the systems, at various stages of development, that must cope with functional changes that occur with birth.

**Characteristics**

Preterm infants have a number of distinct characteristics at various stages of development. Identification of these characteristics provides valuable clues to the gestational age and hence to the physiologic capabilities. The general, outward physical appearance changes as the fetus progresses to maturity. Characteristics of skin, general posture and tone, distribution of hair, and amount of subcutaneous fat provide clues to a newborn’s physical development. Observation of spontaneous, active movements and response to stimulation and passive movement contributes to the assessment of neurologic status. The appraisal is made as soon as possible after admission to the nursery because much of the observation and management of infants depend on this information.

On inspection, preterm infants are very small and appear scrawny because they lack or have only minimum subcutaneous fat deposits and have a proportionately large head in relation to the body, which reflects the cephalocaudal direction of growth. The skin is bright pink (often translucent, depending on the degree of immaturity), smooth, and shiny (may be edematous), with small blood vessels clearly visible underneath the thin epidermis. The fine lanugo is abundant over the body (depending on gestational age) but is sparse, fine, and fuzzy on the head. The ear cartilage is soft and pliable, and the soles and palms have minimum creases, resulting in a smooth appearance. The bones of the skull and the ribs feel soft, and before 26 weeks the eyes may be fused. Male infants have few scrotal rugae, and the testes are undescended; the labia minora and clitoris are prominent in females. Fig. 10-11 compares the features of full-term and preterm infants.

In contrast to full-term infants’ overall attitude of flexion and continuous activity, preterm infants are inactive and listless. The extremities maintain an attitude of extension and remain in any position in which they are placed. Physiologically immature, many preterm infants are unable to maintain body temperature, have limited ability to excrete solutes in the urine, and have increased susceptibility to infection. A pliable thorax, immature lung tissue, and an immature regulatory center lead to periodic breathing, hypoventilation, and frequent periods of apnea. These infants are more susceptible to biochemical alterations such as hyperbilirubinemia and hypoglycemia (see Chapter 9), and they have a higher extracellular water content that renders them more vulnerable to fluid and electrolyte imbalance. Preterm infants exchange fully half their extracellular fluid volume every 24 hours compared with one seventh of the volume turnover in adults.

The soft cranium is subject to characteristic unintentional deformation (dolichocephaly) caused by positioning from one side to the other on a mattress. The head looks disproportionately longer from front to back, is flattened on both sides, and lacks the usual convexity seen at the temporal and parietal areas. This positional molding is often a concern to parents and may influence their perception of the infant’s attractiveness and their responsiveness to the infant. Frequent repositioning of the infant and positioning on a gel mattress can reduce or minimize cranial molding.

Late-preterm infants may not have the immature appearance so commonly observed in preterm infants born at a lower gestational age (<34 weeks’ gestation). However, late-preterm infants are at risk for the development of some of the same physiologic adaptation problems as their preterm counterparts: respiratory distress syndrome, hyperbilirubinemia, thermoregulation difficulties, hypoglycemia, and feeding problems.

**Therapeutic Management**

When delivery of a preterm infant is anticipated, the intensive care nursery is alerted and a team approach implemented. Ideally, a neonatologist or a neonatal nurse practitioner, a staff nurse, and a respiratory therapist are present for the delivery. Infants who do not require resuscitation are immediately transferred in a heated incubator to the NICU, where they are weighed and where IV access, oxygen therapy, and other therapeutic interventions are initiated as needed. Resuscitation is conducted in the delivery area until infants can be safely transported to the NICU. Ongoing care is described elsewhere in the chapter.

**Nursing Care Management**

As with therapeutic management, individualize nursing care for each infant. See appropriate discussions under Nursing Care of High-Risk Newborns for details of care.

**POSTTERM INFANTS**

Infants born of a gestation that extends beyond 42 weeks as calculated from the mother’s last menstrual period (or by gestational age assessment) are postterm, or postmature, regardless of birth weight. This constitutes 3.5% to 15% of all pregnancies. The cause of delayed birth is unknown. Some infants are appropriate for gestational age but show the characteristics of progressive placental dysfunction. These infants, often called postterm infants, display the characteristics of infants who are 1 to 3 weeks of age, such as absence of lanugo, little if any vernix caseosa, abundant scalp hair, and long fingernails. The skin is often cracked, parchmentlike, and desquamating. A common finding in postterm infants is a wasted physical appearance that reflects intrauterine nutritional deprivation. Depletion of subcutaneous fat gives them a thin, elongated appearance. The little vernix caseosa that remains in the skinfolds may be stained a deep yellow or green, which is usually an indication of meconium in the amniotic fluid.

Fetal and neonatal mortality increase significantly in postterm infants compared with those born at term. They are especially prone to fetal distress associated with the decreasing efficiency of the placenta, macrosomia, and meconium aspira-
CHAPTER 10  The High-Risk Newborn and Family

CLINICAL EVALUATION

PRETERM  TERM

**Posture**—The preterm infant lies in a “relaxed attitude,” limbs more extended; the body size is small, and the head may appear somewhat larger in proportion to the body size. The term infant has more subcutaneous fat tissue and rests in a more flexed attitude.

**Ear**—The preterm infant’s ear cartilages are poorly developed, and the ear may fold easily; the hair is fine and feathery, and lanugo may cover the back and face. The mature infant’s ear cartilages are well formed, and the hair is more likely to form firm, separate strands.

**Sole**—The sole of the foot of the preterm infant appears more turgid and may have only fine wrinkles. The mature infant’s sole (foot) is well and deeply creased.

**Female genitalia**—The preterm female infant’s clitoris is prominent, and labia majora are poorly developed and gaping. The mature female infant’s labia majora are fully developed, and the clitoris is not as prominent.

**Male genitalia**—The preterm male infant’s scrotum is undeveloped and not pendulous; minimal rugae are present, and the testes may be in the inguinal canals or in the abdominal cavity. The term male infant’s scrotum is well developed, pendulous, and rugated, and the testes are well down in the scrotal sac.

**Scarf sign**—The preterm infant’s elbow may be easily brought across the chest with little or no resistance. The mature infant’s elbow may be brought to the midline of the chest, resisting attempts to bring the elbow past the midline.

*Fig. 10-11* Clinical and neurologic examinations comparing preterm and full-term infants. (Data from Pierog SH, Ferrara A: *Medical care of the sick newborn*, ed 2, St Louis, 1976, Mosby; photos courtesy Paul Vincent Kuntz, Texas Children’s Hospital, Houston.)
tion syndrome (MAS). The greatest risk occurs during the stresses of labor and delivery, particularly in infants of primigravida, or women delivering their first child. Induction of labor is usually recommended when infants are significantly overdue.

**HiGH risk related to disturbed respiratory function**

**APNEA OF PREMATUREITY**

Characteristically, preterm infants are periodic breathers. They have periods of rapid respiration separated by periods of very slow breathing, and often short periods with no visible or audible respirations. Apnea is primarily an extension of this periodic breathing and can be defined as a lapse of spontaneous breathing for 20 or more seconds, or shorter pauses accompanied by hypotonia, bradycardia, or color change (Stokowski, 2005).

Apnea of prematurity (AOP) is a common phenomenon in the preterm infant. Rarely observed in full-term infants, apneic spells increase in prevalence the younger the gestational age. Approximately one third of infants less than 33 weeks of gestation and more than half of apparently healthy infants less than 30 weeks of gestation have apneic spells (Stokowski, 2005). Apnea usually resolves as the infant approaches 37 weeks postmenstrual age.

AOP may be further classified according to origin. The three recognized types are (1) central apnea, an absence of diaphragmatic and other respiratory muscle function that causes a lack of respiratory effort and occurs when the CNS does not transmit signals to the respiratory muscles; (2) obstructive apnea, when air flow ceases because of upper airway obstruction, yet chest or abdominal wall movement is present; and (3) mixed apnea, a combination of central and obstructive apnea and the most common form of apnea seen in preterm infants (Poblano, Marquez, and Hernandez, 2006).

**Pathophysiology**

AOP reflects the immature and poorly refined neurologic and chemical respiratory control mechanisms in preterm infants. These infants are not as responsive to hypercapnia and hypoxemia, and their neurons have fewer dendritic associations than those of more mature infants. The respiratory reflexes of these infants are significantly less mature, which may be a contributing factor in the etiology. Overall weakness of the muscles of the thorax, diaphragm, and upper airway may also contribute to apneic episodes in the preterm infant. In addition, apnea is characteristically observed during periods of REM sleep. A variety of factors, including infection, intracranial hemorrhage (ICH), or PDA, can make apnea worse. Secondary causes of apnea should be investigated in infants with new-onset apnea or when there is a significant change in the frequency or severity of apneic episodes. Apnea in full-term infants should always be considered secondary and the cause investigated.

**Clinical Manifestations**

Factors that contribute to apnea in preterm neonates should be investigated and treated. Apnea can be anticipated in infants with a variety of conditions (Box 10-8); conversely, one of these disorders may be suspected in infants with persistent apneic spells. Although apnea is an expected event in preterm neonates, it should not be designated as being benign until all other causes have been ruled out. The observation of apnea is a reason to screen for any of the causes listed in Box 10-8.
If begun early, gentle tactile stimulation (e.g., rubbing the back or chest gently, turning the infant to a supine position) will stop most apneic spells. If tactile stimulation fails to re-institute respiration, flow-by oxygen and suctioning of the nose and mouth may be required. If breathing does not begin, the chin is raised gently to open the airway, and sufficient pressure is applied with a resuscitation mask and bag to lift the rib cage. The infant is never shaken. After breathing is restored, the infant is assessed for possible precipitating factors, such as unstable temperature, abdominal distention (if not observed earlier), and supplemental oxygen (if any) being delivered before the episode. The use of pulse oximetry has helped detect the onset of an apneic episode.

It is important that nurses maintain a careful record of episodes of apnea, including the number of apneic spells, the infant’s appearance during and after the episode, and whether the infant self-reverses or whether tactile stimulation or other measures are needed to restore breathing. Subsequent investigation into the possible cause of the apneic episode is vital to the care of the preterm infant because it may signal an underlying condition such as sepsis or NEC.

Persistent and repeated periods of apnea may be treated by mechanical ventilation or CPAP. Various methods devised to provide an intermittent stimulus for breathing, such as oscillating beds and water beds, have achieved variable success in the treatment of AOP.

**RESPIRATORY DISTRESS SYNDROME**

RDS refers to a condition of surfactant deficiency and physiologic immaturity of the thorax. The terms respiratory distress syndrome and hyaline membrane disease are most often applied to this severe lung disorder. It is seen almost exclusively in preterm infants but may also be associated with multifetal pregnancies, infants of diabetic mothers, cesarean section delivery, delivery before 37 weeks of gestation, precipitous delivery, cold stress, asphyxia, and a history of previous RDS (Dudell and Stoll, 2007). The disorder is rarely observed in drug-exposed infants or infants who have been subjected to chronic intrauterine stress (e.g., maternal preclampsia or hypertension). Respiratory distress of a nonpulmonary origin in neonates may also be caused by sepsis, cardiac defects (structural or functional), exposure to cold, airway obstruction (atresia), IVH, hypoglycemia, metabolic acidosis, acute blood loss, and drugs. Pneumonia in the neonatal period is respiratory distress caused by bacterial or viral agents and may occur alone or as a complication of RDS.

**Pathophysiology**

Preterm infants are born before the lungs are fully prepared to serve as efficient organs for gas exchange. This appears to be a

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**BOX 10-8 POSSIBLE CAUSES OF APNEA OF PREMATURITY**

- Prematurity
- Airway obstruction with mucus or milk, or poor positioning
- Anemia, polycythemia
- Dehydration
- Cooling or overheating
- Hypoxemia
- Hypercapnia or hypocapnia
- Hypoglycemia
- Hypocalcemia
- Hypoponatremia
- Sepsis, meningitis
- Seizures
- Increased vagal tone (in response to suctioning nasopharynx, gavage tube insertion, reflux of gastric contents, endotracheal intubation)
- Central nervous system depression from pharmacologic agents
- Intraventricular hemorrhage
- Patent ductus arteriosus, congestive heart failure
- Depression following maternal obstetric sedation
- Respiratory distress as a result of pneumonia, inborn errors of metabolism such as hyperammonemia, congenital defects of the upper airways

**Therapeutic Management**

Administration of caffeine is often effective in reducing the frequency of primary apnea-bradycardia spells in newborns. Caffeine acts as a CNS stimulant to breathing. Neonates receiving caffeine must be closely observed for symptoms of toxicity. Caffeine has come to the forefront of pharmacologic therapy for AOP because it has fewer side effects than previously used aminophylline or theophylline, requires dosing once daily, has more predictable plasma concentrations, has slower elimination, and has a wider therapeutic range (trough, 5 to 20 mcg/ml). Caffeine citrate (Cafcit) has been approved for use in preterm infants with AOP. It is available in injectable and oral form. Weight and urinary output should be closely monitored because caffeine acts as a mild diuretic.

**NURSING ALERT**

When the alarm sounds, infants are first assessed for color and for presence of respiration. If they display the usual color and respirations, the nurse should investigate possible causes of a false alarm, such as faulty lead placement, detached or disconnected leads, improper alarm setting, or mechanical failure.

Persistent and repeated periods of apnea may be treated by mechanical ventilation or CPAP. Various methods devised to provide an intermittent stimulus for breathing, such as oscillating beds and water beds, have achieved variable success in the treatment of AOP.

**RESPIRATORY DISTRESS SYNDROME**

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**Pathophysiology**

Preterm infants are born before the lungs are fully prepared to serve as efficient organs for gas exchange. This appears to be a
critical factor in the development of RDS. RDS results from a combination of structural and functional immaturity of the lungs.

Because the final unfolding of the alveolar septa, which increases the surface area of the lungs, occurs during the last trimester of pregnancy, preterm infants are born with numerous underdeveloped and many uninflatable alveoli. In addition, the fetal chest wall is highly compliant because of the predominance of cartilage rather than bone; and the diaphragm, the dominant respiratory muscle, is prone to fatigue.

Functionally, the fetal lungs are deficient in surfactant, a surface-active phospholipid secreted by type II cells in the alveolar epithelium. Surfactant is first produced at about 24 weeks of gestational age, but the type II cells in the lung do not fully mature until about 36 weeks of gestation (Fig. 10-12). Acting much like a detergent, this substance reduces the surface tension of fluids that line the alveoli and respiratory passages, resulting in uniform expansion and maintenance of lung expansion at low intraalveolar pressure. Immature development of these functions produces consequences that seriously compromise respiratory efficiency. Deficient surfactant production causes unequal inflation of alveoli on inspiration and the collapse of alveoli on end expiration. Without surfactant, infants are unable to keep their lungs inflated and therefore exert a great deal of effort to reexpand the alveoli with each breath. It has been estimated that each breath requires as much negative pressure (60 to 75 cm H₂O) as the initial lung expansion at birth. With increasing exhaustion they are able to open fewer and fewer alveoli. This inability to maintain lung expansion produces widespread atelectasis.

In the absence of alveolar stability (normal functional residual capacity) and with progressive atelectasis, pulmonary vascular resistance (PVR) increases, whereas with normal lung expansion it would decrease. Consequently, there is hypoperfusion to the lung tissue, with a decrease in effective pulmonary blood flow. The increase in PVR causes partial reversion to the
fetal circulation, with a right-to-left shunting of blood through the persisting fetal communications—the ductus arteriosus and foramen ovale.

Inadequate pulmonary perfusion and ventilation produce hypoxemia and hypercapnia. Pulmonary arterioles, with their thick muscular layer, are markedly reactive to diminished oxygen concentration. Thus a decrease in oxygen tension causes vasospasm in the pulmonary arterioles that is further enhanced by a decrease in blood pH. This vasoconstriction contributes to a marked increase in PVR. In normal ventilation with increased oxygen concentration, the ductus arteriosus constricts and the pulmonary vessels dilate to decrease PVR (Fig. 10-13).

Prolonged hypoxemia activates anaerobic glycolysis, which produces increased amounts of lactic acid. An increase in lactic acid causes metabolic acidosis; inability of the atelectatic lungs to blow off excess carbon dioxide produces respiratory acidosis. Lowered pH causes further vasoconstriction. With deficient pulmonary circulation and alveolar perfusion, \( P_{\text{ao}} \) continues to fall, pH falls, and the materials needed for surfactant production are not circulated to the alveoli.

Pulmonary edema observed in the early stages of RDS also contributes to impaired gas exchange. Factors believed to facilitate this fluid accumulation in the lungs include renal immaturity or insufficiency resulting from hypoxemia, high fluid intake and PDA, left ventricular dysfunction associated with papillary muscle necrosis, low serum protein concentration and low colloid osmotic pressure, increased alveolar surface tension that enhances the shift of interstitial fluid to alveolar spaces, oxygen toxicity, and high plasma vasopressin.

Pulmonary interstitial emphysema (PIE) may develop in preterm infants with RDS and immature lungs as a result of overdistention of distal airways. This condition further complicates adequate oxygenation in the immature airways (see Air Leak Syndromes, p. 358).

Deficiencies in other systems contribute to respiratory distress. For example, a high threshold of the respiratory center to afferent stimuli and weak or absent gag and cough reflexes reflect the immaturity of the nervous system. In addition, the persistence of fetal hemoglobin, so beneficial in prenatal existence, may place the infant at a disadvantage during respiratory distress. Although the binding power of fetal hemoglobin for oxygen is much greater than that of adult hemoglobin, this increased affinity also causes less oxygen to be released to the tissues at normal oxygen tension. In the newborn the arterial oxygen concentration must fall to a lower level for bound oxygen to be released from fetal hemoglobin.

A **hyaline membrane** is formed as hypoxemia and the increased pulmonary vascular pressure cause transudation of fluid into the alveoli. Necrotic cells from damaged alveoli plus the fibrin in the transudate form a membranous layer that lines the alveoli and inhibits gas exchange. The hyaline membrane contributes to respiratory difficulties by greatly diminishing lung distensibility, or compliance, the elastic quality of lung tissue that permits expansion in response to a given amount of applied pressure during inspiration. Affected lungs are stiffer and require far more pressure than do normal lungs to achieve an equal amount of expansion. Table 10-7 summarizes the major factors that produce RDS in immature infants.

**Clinical Manifestations**

Infants with RDS can develop respiratory distress either acutely or over a period of hours, depending on the acuity of pulmonary immaturity, associated illness factors, and gestational maturity. The observable signs produced by the pulmonary

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**Table 10-7: Major Factors in Respiratory Distress Syndrome**

<table>
<thead>
<tr>
<th>CAUSE</th>
<th>EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased pulmonary vascular resistance</td>
<td>Alveolar collapse; atelectasis; increased difficulty breathing</td>
</tr>
<tr>
<td>Impaired gas exchange</td>
<td>Hypoxemia and hypercapnia with respiratory acidosis</td>
</tr>
<tr>
<td>Increased transudation of fluid into lungs</td>
<td>Hypoperfusion of pulmonary circulation</td>
</tr>
<tr>
<td>Hyponerved pressure (with hypoxemia)</td>
<td>Tissue hypoxia and metabolic acidosis</td>
</tr>
<tr>
<td>Hyaline membrane formation; impaired gas exchange</td>
<td>Increased surface tension of alveoli (surfactant deficiency)</td>
</tr>
</tbody>
</table>
changes usually begin to appear in infants who apparently achieve normal breathing and color soon after birth. In a matter of a few hours, breathing gradually becomes more rapid (>60 breaths/min). Infants may display retractions—suprasternal or substernal, and supracostal, substernal, or intercostal—which result from a compliant chest wall. Weak chest wall muscles and the highly cartilaginous rib structure produce an abnormally elastic rib cage, resulting in indrawing, or retraction, of the skin between the ribs. During this early period the infant’s color may remain satisfactory, and auscultation reveals air entry. Some of the criteria for evaluating respiratory distress in infants are illustrated in Fig. 10-14.

Within a few hours, respiratory distress becomes more obvious. The respiratory rate continues to increase (to 80 to 120 breaths/min), and breathing becomes more labored. It is significant to note that infants increase the rate rather than the depth of respiration when in distress. Subternal retractions become more pronounced as the diaphragm works hard in an attempt to fill collapsed air sacs. Fine inspiratory crackles can be heard over both lungs, and there is an audible expiratory grunt. This grunting, a useful mechanism observed in the earlier stages of RDS, serves to increase end-expiratory pressure in the lungs, thus maintaining alveolar expansion and allowing gas exchange for an additional brief period. Flaring of the nares is also a sign that accompanies tachypnea, grunting, and retractions in respiratory distress. Central cyanosis (a bluish discoloration of oral mucous membranes and generalized body cyanosis) is a late and serious sign of respiratory distress. Initially supplemental oxygen may eliminate cyanosis. The use of pulse oximetry and arterial blood gas sampling obviates the necessity for dependence on color to determine oxygen requirements. Severe RDS is often associated with a shocklike state, as manifested by diminished cardiac inflow and low arterial blood pressure. As a result of extreme pulmonary immaturity, decreased glycogen stores, and lack of accessory muscles, the ELBW and VLBW infant may have severe RDS at birth, bypassing the aforementioned steps in the development of RDS.

Infants with RDS who are treated with exogenous surfactant have a good chance for recovery. Complications of RDS include those described as complications of positive pressure ventilation (see p. 353). Associated complications (of prematurity and RDS) include PDA and congestive heart failure, retinopathy of prematurity, IVH, BPD, NEC, and neurologic sequelae.

Diagnostic Evaluation

Laboratory data are nonspecific, and the abnormalities observed are identical to those observed in numerous biochemical abnormalities of the newborn (i.e., the findings of hypoxemia, hypercapnia, and acidosis). Specific tests are used to determine complicating factors, such as blood glucose (to test for hypoglycemia), blood gas measurements for serum pH (to test for acidosis), and PaO₂ (to test for hypoxia). Pulse oximetry is an important component for determining hypoxia. Other special examinations may be used to diagnose or rule out complications.

Radiographic findings characteristic of RDS include (1) a diffuse granular pattern over both lung fields that resembles ground glass and represents alveolar atelectasis and (2) dark streaks, or air bronchograms, within the ground glass areas that represent dilated, air-filled bronchioles. It is important to distinguish between RDS and pneumonia in infants with respiratory distress.

Prenatal Diagnosis

Fetal lung maturity depends on gestational age and maternal illnesses. Problems such as maternal diabetes delay fetal lung
maturation, whereas fetuses exposed to chronic stress (intrauterine growth restriction [IUGR], drug exposure) often have more mature lungs. Antenatal administration of glucocorticoids enhances fetal lung maturity, especially when combined with postnatal surfactant administration (Hintz, Poole, Wright, et al, 2005; Baud, 2004).

Functional maturity of the fetal lung can be determined by using surfactant phospholipids in amniotic fluid as indicators of maturity. The most commonly tested is the lecithin/sphingomyelin (L/S) ratio, which represents the relationship between these two lipids during gestation. Phospholipids are synthesized by fetal alveolar cells, and the concentrations in amniotic fluid change during gestation. Initially there is more sphingomyelin, but at approximately 32 to 33 weeks the concentrations become equal; sphingomyelin then diminishes and lecithin increases significantly until the fetus has developed sufficient surfactant to maintain alveolar stability at approximately 35 weeks. An L/S ratio of 2 : 1 in nondiabetic mothers indicates virtually no risk of RDS.

Other key surfactant compounds (also phospholipids) that are needed to stabilize surfactant are phosphatidylcholine (PC) and phosphatidylglycerol (PG). Without these compounds, lecithin is not functional as a surfactant. Concentrations of PC parallel those of lecithin, peaking at 35 weeks and then gradually decreasing. At 36 weeks PG appears in amniotic fluid and increases until term. By measuring these phospholipids—L/S ratio, PC, and PG—the clinician can estimate the maturity of the lungs with a high degree of accuracy. Other, less commonly used methods have been devised to provide rapid, inexpensive, and accurate measures of lung maturity. These include the “shake” or “bubble” test, in which stable foam or bubbles form when amniotic fluid is shaken in the presence of ethanol, and the tap test, in which abundant bubbles appear in a test tube of amniotic fluid with 6N-hydrochloric acid and diethyl ether.

Another test currently being used to evaluate fetal lung maturity is the TDx Fetal Lung Maturity (FLM) assay, which determines PG levels in amniotic fluid or neonatal tracheal aspirates. The FLM test is faster than L/S ratio determination (< 1 hour versus 4 to 5 hours) and is reported to predict the absence of RDS with greater accuracy; a level of 50 or more is predictive of fetal lung maturity (Fantz, Powell, Karon, et al, 2002). TDx FLM may also be used in the postnatal period to determine the presence of RDS as a result of surfactant deficiency by collecting tracheal aspirate samples (Parvin, Kaplan, Chapman, et al, 2005).

Lamellar bodies, representing the storage form of surfactant, are found in amniotic fluid in increasing quantities with the advancement of gestational age and lung maturity. A quantitative count of lamellar bodies has been reported to be as accurate as the L/S ratio in determining fetal lung maturity. The count can be obtained faster than the L/S ratio, thus making it clinically appealing (Neerhof, Dohnal, Ashwood, et al, 2001; Wijnberger, Huisjes, Voorbij, et al, 2001).

**Therapeutic Management**

The treatment of RDS includes all the general measures required for any preterm infant, as well as those instituted to correct imbalances. The supportive measures most crucial to a favorable outcome are (1) maintain adequate ventilation and oxygenation with an oxygen hood, continuous positive airway pressure (CPAP), or mechanical ventilation; (2) maintain acid-base balance; (3) maintain a neutral thermal environment; (4) maintain adequate tissue perfusion and oxygenation; (5) prevent hypotension; and (6) maintain adequate hydration and electrolyte status. Nipple and gavage feedings are avoided in any situation that creates a marked increase in respiratory rate because of the greater hazards of aspiration.

**QUALITY PATIENT OUTCOMES:** Neonatal RDS

- Room air or oxygen saturation ≥ 90%
- Respiratory rate < 60 breaths/min
- Blood pH ≥ 7.35

**Surfactant**

The administration of exogenous surfactant to preterm neonates with RDS has become an accepted and common therapy in most neonatal centers worldwide. Numerous clinical trials involving the administration of exogenous surfactant to infants with or at high risk for RDS demonstrate improvements in blood gas values and ventilator settings, decreased incidence of pulmonary air leaks, decreased deaths from RDS, and an overall decreased infant mortality rate (Halliday, 2003; Soll, 2000, American Academy of Pediatrics, 2008). Exogenous surfactant comes from a natural source (e.g., porcine or bovine) or from the production of artificial surfactant. Commercially available surfactant products include beractant (Survanta), a bovine surfactant; and poractant alfa (Curosurf), a porcine surfactant.

Studies have shown mixed results in comparing one surfactant product with another. One study found fewer complications and earlier improvement with natural (versus synthetic) surfactant use (Soll and Blanco, 2001). Moya, Gadzinowski, Bencalari, and colleagues (2005) found that an investigational synthetic surfactant, lucinactant, mimics the action of human surfactant protein-B (SP-B), and it was more effective than beractant and colfosceril palmate at reducing the RDS-related mortality rates by 14 days of life. BPD was significantly less common in infants at 36 weeks postmenstrual age who had received lucinactant.

Additional benefits of surfactant replacement therapy include decreased oxygen requirements and mean airway pressure (MAP) within hours of administration and an overall decrease in the incidence of pulmonary air leaks. To date, long-term improvement in the decrease of BPD and IVH has not been evidenced in all surfactant clinical trials.

Complications seen with surfactant administration include pulmonary hemorrhage and mucus plugging. Additional studies investigating the potential benefits of surfactant in infants with meconium aspiration found a reduction in the severity of respiratory illness and subsequent requirement of ECMO support (El Shahed, Dargaville, Ohlsson, et al, 2007). Other studies continue to investigate the potential benefits of exogenous surfactant for the treatment of infectious pneumonia and lung hypoplasia concomitant with congenital diaphragmatic hernia (Wiswell, 2001). Acute RDS/ALI may also respond favorably to surfactant administration (see Acute Respiratory Distress Syndrome/Acute Lung Injury, Chapter 32). Surfactant may be administered at birth as a prophylactic treatment of...
RDS or later in the course of RDS as a rescue treatment. Studies found improved clinical outcomes and fewer adverse effects when surfactant is administered prophylactically to infants at risk for developing RDS (American Academy of Pediatrics, 2008). Surfactant is administered via the ET tube directly into the infant’s trachea (Fig. 10-15); the exact number of doses (single versus multiple) that is most effective has yet to be determined.

Nursing responsibilities with surfactant administration include assistance in the delivery of the product, collection and monitoring of arterial blood gases, scrupulous monitoring of oxygenation, and assessment of the infant’s tolerance of the procedure. Once surfactant is absorbed, there is usually an increase in respiratory compliance that requires adjustment of ventilator settings to decrease MAP and prevent overinflation or hyperoxemia. Suctioning is usually delayed for an hour or so (depending on the type of surfactant, delivery system, and unit protocol) to allow maximum effects to occur. Current research is investigating the possibility of delivering an aerosolized surfactant. This method would decrease the problems associated with current delivery systems (contamination of the airway, interruption of mechanical ventilation, and loss of the drug in the ET tubing from reflux).

**Oxygen Therapy**

The goals of oxygen therapy are to provide adequate oxygen to the tissues, prevent lactic acid accumulation resulting from hypoxia, and, at the same time, avoid the potentially negative effects of oxygen toxicity. Numerous methods have been devised to improve oxygenation (Table 10-8). All require that the gas be warmed and humidified before entering the respiratory tract. If the infant does not require ventilatory assistance, oxygen can be given via a plastic hood placed over the head to supply variable concentrations of humidified oxygen. (See Oxygen Therapy, Chapter 31.) If oxygen saturation of blood cannot be maintained at a satisfactory level and the carbon dioxide level \(\text{Paco}_2\) rises, infants will require ventilatory assistance.

**Table 10-8: Common Methods for Assisted Ventilation in Neonatal Respiratory Distress**

<table>
<thead>
<tr>
<th>METHOD</th>
<th>DESCRIPTION</th>
<th>HOW PROVIDED</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conventional Methods</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continuous positive airway pressure (CPAP)</td>
<td>Provides constant distending pressure to airway in spontaneously breathing infant</td>
<td>Nasal prongs</td>
</tr>
<tr>
<td>Positive end-expiratory pressure (PEEP)*</td>
<td>Provides increased end-expiratory pressure during expiration and between mandatory breaths, which prevents alveolar collapse; maintains residual airway pressure</td>
<td>Endotracheal intubation and either volume-limited or pressure-limited ventilator</td>
</tr>
<tr>
<td>Intermittent mandatory ventilation (IMV)*</td>
<td>Allows infant to breathe spontaneously at own rate but provides mechanical cycled respirations and pressure at regular preset intervals</td>
<td>Endotracheal intubation and ventilator</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation (SIMV)</td>
<td>Mechanically delivers breaths synchronized to onset of spontaneous patient breaths; uses assist/control mode to facilitate full inspiratory synchrony; involves signal detection of onset of spontaneous respiration from abdominal movement, thoracic impedance, and airway pressure or flow changes</td>
<td>Patient-triggered infant ventilator with signal detector and assist/control mode; endotracheal tube</td>
</tr>
<tr>
<td>Volume guarantee ventilation</td>
<td>Delivers predetermined volume of gas using inspiratory pressure that varies according to infant’s lung compliance (often used in conjunction with SIMV)</td>
<td>Volume guarantee ventilator with flow sensor; endotracheal tube</td>
</tr>
<tr>
<td><strong>Alternative Methods</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High-frequency oscillatory ventilation (HFOV)</td>
<td>Application of high-frequency, low-volume, sine-wave flow oscillations to airway at rates between 480 and 1200 breaths/min</td>
<td>Variable-speed piston pump (or loudspeaker, fluidic oscillator); endotracheal tube</td>
</tr>
<tr>
<td>High-frequency jet ventilation (HFJV)</td>
<td>Uses separate, parallel, low-compliant circuit and injector port to deliver small pulses or jets of fresh gas deep into airway at rates between 250 and 900 breaths/min</td>
<td>May be used alone or with low-rate IMV; endotracheal tube</td>
</tr>
</tbody>
</table>

*Also referred to as conventional ventilation (versus HFOV).
Oxygen should be administered judiciously to preterm infants being stabilized in labor and delivery and for oxygenation maintenance in the NICU. Much attention has focused recently on high oxygen concentration and the effect of free oxygen radicals on the development of conditions such as NEC, BPD, and ROP. The current Neonatal Resuscitation Program guidelines recommend the use of oxygen concentrations between 21% and 100% in order to achieve an oxygen saturation of approximately 90% (American Academy of Pediatrics and American Heart Association, 2006). Finer and Leone (2009) recommend oxygen delivery to maintain an SpO2 of 83% to 93% in preterm infants to decrease morbidity and mortality associated with liberal oxygen usage and high oxygen concentrations. Research indicates that optimal target ranges for maintaining adequate oxygenation while preventing ROP and BPD or other conditions is as yet unknown (Askie, Henderson-Smart, and Ko, 2009).

CPAP, the application of 3 to 8 cm H2O (positive) pressure to the airway, uses the infant’s spontaneous respiration to improve oxygenation by helping prevent alveolar collapse and increasing diffusion time. CPAP may be delivered via fitted face mask, nasal prongs, or an ET tube (Fig. 10-16). Ventilation with CPAP is done entirely by the infant. If oxygenation is not improved and the infant requires assisted ventilation, intermittent mandatory ventilation (IMV) is used with positive end-expiratory pressure (PEEP). This allows infants to breathe at their own rate but provides positive pressure with end-expiratory pressure to prevent alveolar collapse and overcome airway resistance. Additional components involved in IMV are peak inspiratory pressure (PIP) and rate (number of breaths per minute). The PIP is the maximum amount of positive pressure applied to the infant on inspiration. The total amount of pressure transmitted to the airway throughout an entire respiratory cycle is called the mean airway pressure (MAP). Increasing MAP in infants with severe RDS correlates positively with improved oxygenation by maintaining functional residual capacity and overcoming the resistive forces of the atelectatic lung. The MAP is affected by changes in the PEEP, PIP, and inspiratory/expiratory ratio. Although MAP is now recognized as the major determinant of oxygenation, this does not imply that simply increasing MAP alone will automatically improve oxygenation (Wood, 2003).

Improved technology has made available to preterm or sick neonates a form of mechanical ventilatory assistance previously used in adults: synchronized intermittent mandatory ventilation (SIMV). With this method breaths delivered by the ventilator are synchronized to the onset of spontaneous infant breaths. The net effect is to produce full respiratory synchrony rather than asynchronous respiratory efforts (commonly called “fighting the ventilator”) that are believed to significantly impede the ability to adequately oxygenate infants without sedation or muscle paralysis. With SIMV, the operator sets the number of breaths per minute delivered by the ventilator, and the patient may breathe spontaneously between mechanical breaths. In the “assist-control” mode a mechanical breath is delivered each time a spontaneous respiration is detected; the “control” mode includes the delivery of a mechanical breath at a regular rate if the patient fails to initiate a spontaneous respiration. Additional benefits of SIMV are improved oxygenation, decreased incidence of pulmonary air leaks (pneumothorax), and decreased time on mechanical ventilation (Greenough, Dimitriou, Prendergast, et al, 2008).

If adequate oxygenation cannot be maintained and hypercarbia persists, infants may benefit from one of the two high-frequency ventilation (HFV) modalities. HFV delivers gas at very rapid rates to provide adequate minute volumes using lower proximal airway pressures by way of high-frequency oscillatory ventilation (HFOV) or high-frequency jet ventilation (HFJV). HFV was initially recommended for intractable respiratory failure, especially for infants with pulmonary air leaks and PIE. More recently, many clinicians are recommending earlier use of HFOV to prevent volutrauma to the lungs of very preterm infants (Ventre and Arnold, 2004).

Volutrauma is believed to be a key factor in the development of BPD. Courtney, Durand, Asselin, and colleagues (2002) reported that HFOV was associated with improved survival and a decreased need for supplemental oxygen at 36 weeks of postmenstrual age. HFJV is most often used in the treatment of full-term infants with meconium aspiration, persistent pulmonary hypertension, or air leak syndromes. The Cochrane review of HFJV use in preterm infants with RDS reports a similar benefit to that of HFOV in terms of pulmonary outcomes but cautions that sufficient studies have not been done to recommend the use of HFJV in preterm infants (Bhuta and Henderson-Smart, 2000).

**Complications of Positive Pressure Ventilation**

Although lifesaving, mechanical ventilation is not without hazards. Positive pressure introduced by mechanical apparatus has caused an increased incidence of air leaks that produce complications, such as PIE, pneumothorax, and pneumomediatinum (see p. 358). The avoidance of intubation and mechanical ventilation reduces the incidence of BPD (Verder, Bohlin, Kamper, et al, 2009). Other complications directly related to positive pressure include various problems associated with intubation, such as nasal, tracheal, or pharyngeal perforation; stenosis; inflammation; palatal grooves; subglottic stenosis; tube obstruction; and infection.
Nitric Oxide
Inhaled nitric oxide (NO) has emerged as a significant treatment modality for neonates with conditions that cause persistent pulmonary hypertension, pulmonary vasconstriction, and subsequent acidosis and severe hypoxia. Infants with conditions such as MAS, pneumonia, sepsis, and congenital diaphragmatic hernia with pulmonary hypoplasia often require intervention in an attempt to reverse pulmonary hypertension. NO is a colorless, highly diffusible gas that causes smooth muscle relaxation and reduces pulmonary vasconstriction and subsequent pulmonary hypertension when inhaled into the lungs. NO may be administered through the ventilator circuit and blended with oxygen. It attaches readily to hemoglobin and is thus deactivated so that systemic vasculature is not affected. NO is toxic in large quantities, but the amount required to induce pulmonary vasculature relaxation (6 to 20 ppm) is well below toxic levels.

Studies of term and near-term infants being treated with NO for respiratory failure have been positive (Finer and Barrington, 2006). In many cases reversal of persistent pulmonary hypertension of the newborn (PPHN) without ECMO has been achieved in infants with MAS, RDS, perioperative congenital heart disease, and sepsis (Konduri, 2004). One exception is the study of newborns with congenital diaphragmatic hernia who required ECMO after NO and whose morbidity and mortality were not significantly improved with inhaled NO (Field, 2005; Finer and Barrington, 2006). Surfactant replacement therapy may be performed in combination with inhaled NO therapy in infants with inadequate pulmonary maturity. Nursing care of the infant receiving inhaled NO is the same as for the newborn with PPHN; continuous assessment of respiratory status and response to treatment is essential.

The use of NO for preterm infants remains controversial. Some studies have proposed a role for NO in the treatment of RDS and respiratory failure in these infants, whereas others suggest no benefit (Barrington and Finer, 2006; Field, 2005; Hascoet, Fresson, Claris, et al, 2005; Mercier, Olivier, Loron, et al, 2009).

Medical Therapies
The treatment of the infant with RDS requires the establishment of one or more IV lines to maintain hydration and nutrition, monitor arterial blood gases, and administer medications. Systemic antibiotics may be administered during the acute phase if sepsis is suspected (see Sepsis, p. 362). The administration of morphine or fentanyl for pain and sedation is individualized according to the infant’s response to illness. Caffeine may be administered to treat apnea and to prepare for weaning VLBW and ELBW infants from mechanical ventilation. Inotropes such as dopamine and dobutamine may be required to support the infant’s systemic blood pressure and maintain effective cardiac output during the acute phase of illness.

Prevention
The most successful approach to prevention of RDS is prevention of preterm delivery, especially elective early delivery and cesarean section. Improved methods for assessing the maturity of the fetal lung by amniocentesis, although not a routine procedure, allow a reasonable prediction of adequate surfactant formation (see Diagnostic Evaluation, p. 350). Because estimation of a delivery date can be miscalculated by as much as 1 month, such tests are particularly valuable when scheduling an elective cesarean section. Studies indicate that the combination of maternal glucocorticoid administration before delivery and surfactant administration postnatally has a synergistic effect on neonatal lungs, with the net result being a decrease in infant mortality, incidence of IVH, pulmonary air leaks, and problems with PIE and RDS (Dudell and Stoll, 2007; Halliday, 2005).

Nursing Care Management
Care of infants with RDS involves all the observations and interventions previously described for high-risk infants. In addition, the nurse is concerned with the complex problems related to respiratory therapy and the constant threat of hypoxemia and acidosis that complicates the care of patients in respiratory difficulty.

The respiratory therapist, an important member of the neonatal intensive care team, is often responsible for maintenance and regulation of respiratory equipment. Nevertheless, nurses should understand the equipment and be able to recognize when it is not functioning correctly. The most essential nursing function is to observe and assess the infant’s response to therapy. Continuous monitoring and close observation are mandatory because an infant’s status can change rapidly and because oxygen concentration and ventilation parameters are mandatory. Changes in oxygen concentration are based on these observations. The nurse determines the amount of oxygen administered, expressed as the fraction of inspired air (FiO₂), on an individual basis according to pulse oximetry and/or direct or indirect measurement of arterial oxygen concentration. Capillary samples collected from the heel (see Chapter 27 for procedure) are useful for pH and PacO₂ determinations but not for oxygenation status. Continuous pulse oximetry readings are recorded at least hourly or more often as required. Blood sampling is performed after ventilator changes for the acutely ill infant and thereafter when clinically indicated.

In infants with RDS who are acutely ill or extremely preterm, an umbilical arterial catheter (UAC) may be used to draw arterial blood for monitoring oxygenation. This method, although initially invasive and therefore performed by the practitioner with sterile precautions, allows for blood sampling without repeated peripheral arterial punctures. The catheter is inserted via one of the umbilical arteries to the premeasured desired position (either at the level of the diaphragm, T6-10, or between L3-4) and rests in the descending aorta. Continuous arterial pressure monitoring may be carried out with an "in-line" transducer. Practices vary regarding medication administration via a UAC. The nurse is aware of the potential hazards associated with these catheters (infection, hemorrhage, thrombus formation and subsequent vessel occlusion, arterial vasospasm) and implements monitoring and observation strategies to promptly intervene should complications occur (see Hydration, p. 322). An umbilical venous catheter (UVC) may be used separately or in conjunction with the UAC, depending on the severity of the...
infant’s illness, the fluid requirements, and preferred medical practice.

Mucus may collect in the respiratory tract as a result of the infant’s pulmonary condition. Secretions interfere with gas flow and may obstruct the passages, including the ET tube. Suctioning should occur only when necessary and should be based on individual infant assessment, which includes auscultation of the chest, evidence of decreased oxygenation, excess moisture in the ET tube, or increased infant irritability. When nasopharyngeal passages, the trachea, or the ET tube is being suctioned, insert the catheter gently but quickly; intermittent suction is applied as the catheter is withdrawn. It is imperative that the catheter obstruct the airway for no more than 5 seconds, since continuous suction removes air from the lungs along with the mucus. It is recommended that, where possible, an in-line suction device be used on infants who are acutely ill and who do not tolerate any procedure without profound decreases in oxygen saturation, blood pressure, and heart rate. The purpose of suctioning an artificial airway is to maintain patency of that airway, not the bronchi. Suction applied beyond the ET tube can cause traumatic lesions of the trachea.

Research indicates that suctioning to a point where the catheter meets resistance and is then withdrawn causes trauma to the tracheobronchial wall. To remove secretions without damage to the tracheobronchial mucosa, the suction catheter is premeasured and inserted to a predetermined depth to avoid extension beyond the ET tube. The practice of suctioning patients on mechanical ventilation has undergone close scrutiny in recent years; further studies are needed to validate this practice and to determine the best methods for maintaining a patent airway without compromising the patient’s well-being.

The most advantageous positions for facilitating an infant’s open airway are with the infant on the side with the head supported in alignment by a small folded blanket or with the infant on the back, positioned to keep the neck slightly extended. With the head in the “sniffing” position, the trachea is opened to its maximum; hyperextension reduces the tracheal diameter in neonates (see Therapeutic Positioning, p. 336). The pulse oximeter is observed before, during, and after suctioning to provide an ongoing assessment of oxygenation status and to prevent hypoxemia.

**NURSING ALERT**

Suctioning is not an innocuous procedure; it may cause bronchospasm, bradycardia because of vagal nerve stimulation, hypoxia, and increased intracranial pressure, predisposing the infant to IVH. It should never be carried out on a routine basis. Improper suctioning technique can also cause infection, airway damage, or even pneumothorax.

Inspection of the skin is part of routine infant assessment. Position changes and the use of gel mattresses are helpful in guarding against skin breakdown.

Mouth care is especially important when infants are receiving nothing by mouth, and the problem is often aggravated by the drying effect of oxygen therapy. The nurse can prevent drying and cracking by good oral hygiene using sterile water. Irritation to the nares or mouth that occurs from appliances used to administer oxygen may be reduced by the use of a water-soluble ointment (see Skin Care, p. 329; see Nursing Care Plan).

Nursing care of an infant with RDS is demanding. Pay meticulous attention to subtle changes in the infant’s oxygenation status. The importance of attention to detail cannot be overemphasized, particularly in regard to medication administration.

**MECONIUM ASPIRATION SYNDROME**

Meconium aspiration occurs when a fetus has been subjected to asphyxia or other intrauterine stress that causes relaxation of the anal sphincter and passage of meconium into the amniotic fluid. The majority of meconium aspiration occurs with the first breath. However, a severely compromised fetus may aspirate in utero. At delivery of the chest and initiation of the first breath, infants inhale fluid and meconium into the nasooropharynx (Fig. 10-17).

**Pathophysiology**

MAS involves the passage of meconium in utero as a result of hypoxic stress. It occurs primarily in full-term and postterm infants but has been reported in infants at less than 37 weeks of gestation. Once the fetus ingests meconium, any gasping activity occurring as a result of intrauterine stress may cause the rather sticky and tenacious substance to become aspirated into the lower airways. The net results are partial airway obstruction, air trapping, hyperinflation distal to the obstruction, and atelectasis caused by surfactant deactivation. A “ball-valve” situation exists wherein gas flows into the lungs on inspiration but is trapped there on exhalation as a result of the small airway diameter. As the infant struggles to take in more air (air hunger), even more meconium may be aspirated. Hyperinflation, hypoxemia, and acidemia result in increased PVR.

In turn, shunting of blood through the ductus arteriosus (right to left) occurs because of increased resistance to blood flow through the pulmonary arteries (and to the lungs), leading
to further hypoxemia and acidosis. Ductal shunting increases with hypoxia; some blood may enter the left atrium (LA) from the right atrium (RA) via the foramen ovale because there is a net decrease in blood returning to the LA via the pulmonary venous system, thus preventing closure of the foramen ovale. This pathologic process is essentially persistence of the fetal circulation, or PPHN, which is discussed later in this chapter. The air trapping of MAS causes overdistention of the alveoli and often air leaks. There is evidence that meconium contributes to the destruction of surfactant, thus increasing surface tension and further predisposing the alveoli to decreased functional capacity.

**Clinical Manifestations**

Infants who have released meconium in utero for some time before birth are stained from green meconium stools (those with more recent meconium passage may not be stained), tachypneic, hypoxic, and often depressed at birth. They develop expiratory grunting, nasal flaring, and retractions similar to those experienced by infants with RDS. They may initially be cyanotic or pale as well as tachypneic, and they may demonstrate the classic barrel chest from hyperinflation. The infants are often stressed, hypothermic, hypoglycemic, and hypocalcemic. Severe meconium aspiration progresses rapidly to respiratory failure. These infants exhibit profound respiratory distress with gasping, ineffective ventilations; marked cyanosis and pallor; and hypotonia.

**Diagnostic Evaluation**

At birth, meconium can often be visualized via laryngoscopy in the respiratory passages and vocal cords. Chest radiographs show uneven distribution of patchy infiltrates, air trapping, hyperexpansion, and atelectasis. Air leaks may be seen as the illness progresses. Oxygenation will be poor, as evidenced by pulse oximetry and arterial blood gases. These infants may quickly develop metabolic and respiratory acidosis. Echocardiography assists in the diagnosis of right-to-left shunting of blood away from the pulmonary system.

**Therapeutic Management**

Prevention of meconium aspiration begins with suctioning the mouth, nose, and posterior pharynx just after the head is

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### NURSING CARE PLAN

**The High-Risk Infant with Respiratory Distress**

<table>
<thead>
<tr>
<th>NURSING DIAGNOSIS</th>
<th>PATIENT OUTCOMES</th>
<th>NURSING INTERVENTIONS</th>
<th>RATIONALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ineffective Breathing Pattern related to pulmonary, neurologic, vascular, alveolar, and muscular immaturity</td>
<td>High-risk infant will maintain patent airway and ventilatory status adequate for oxygenation.</td>
<td>Position to facilitate airway expansion and prevent collection of secretions (prone position may be preferred initially in preterm infant to increase chest expansion and oxygenation).</td>
<td>To allow oxygen entry into bronchial tree and alveoli</td>
</tr>
<tr>
<td>Child’s/Family’s Defining Characteristics (Subjective and Objective Data)</td>
<td>The Following NOC Concepts Apply to These Outcomes</td>
<td>Closely monitor for deviations from desired breathing pattern—pulse oximetry, arterial blood gases, clinical signs of poor oxygenation, grunting, nasal flaring, apnea, tachypnea, retractions, cyanosis.</td>
<td>To facilitate proper oxygenation by implementing appropriate therapy such as supplemental oxygen, mechanical ventilation, or change of position</td>
</tr>
<tr>
<td>Decreased inspiratory and expiratory pressure</td>
<td>Respiratory Status: Ventilation</td>
<td>Monitor vital signs for change in condition or status such as decreased cardiac output (poor perfusion, mottling, deteriorating ventilation status).</td>
<td>To implement appropriate therapy such as suctioning, supplemental oxygen, or vasopressor drugs</td>
</tr>
<tr>
<td>Decreased minute ventilation</td>
<td>Respiratory Status: Gas Exchange</td>
<td>Assist with exogenous surfactant administration and monitor patient tolerance or change in status.</td>
<td>To increase alveolar expansion and enhance oxygen–carbon dioxide exchange</td>
</tr>
<tr>
<td>Use of accessory muscles to breathe</td>
<td>Tissue Perfusion: Pulmonary</td>
<td>Suction oropharynx, nasopharynx, trachea, or endotracheal tube only as necessary and based on respiratory assessment.</td>
<td>To remove secretions that may interfere with adequate ventilation and oxygenation</td>
</tr>
<tr>
<td>Grunting</td>
<td>The Following NIC Concepts Apply to These Interventions</td>
<td>Perform gentle chest percussion, vibration, and postural drainage based on assessed need and infant tolerance.</td>
<td>To facilitate drainage and removal of secretions</td>
</tr>
<tr>
<td>Apnea</td>
<td>Vital Signs Monitoring</td>
<td></td>
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<tr>
<td>Tachypnea</td>
<td>Newborn Monitoring</td>
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<tr>
<td>Altered chest excursion</td>
<td>Acid-Base Management</td>
<td></td>
<td></td>
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<tr>
<td>Respiratory rate: &lt;20 or &gt;60 breaths/ min</td>
<td>Airway Management</td>
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<td></td>
<td>Chest Physiotherapy</td>
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<td></td>
<td>Oxygen Therapy</td>
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<td></td>
<td>Airway Suctioning</td>
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<td>Energy Management</td>
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<tr>
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<td>Respiratory Monitoring</td>
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</tbody>
</table>

Continued
### NURSING DIAGNOSIS

**Ineffective Thermoregulation related to immature neurologic and metabolic temperature control**

**Child’s/Family’s Defining Characteristics (Subjective and Objective Data)**
- Reduction in body temperature below normal range
- Slow capillary refill
- Cool skin
- Increased respiratory rate
- Tachycardia

**Patient Outcomes**
- Infant will maintain stable body temperature (specify range for age).

**The Following NOC Concept Applies to These Outcomes**
- Thermoregulation: Newborn

**NURSING INTERVENTIONS**
- Place newborn in a thermally controlled incubator or radiant warmer.
- Place knitted or cloth cap on head.
- Use environmental controls for decreasing body heat loss—plastic heat shield, increased ambient temperature, servocontrol on warmer or incubator.
- Monitor core temperature as often as necessary or per unit protocol.

**Rationale**
- To control environmental temperature and keep infant’s temperature stable
- To prevent heat loss from exposed scalp
- To regulate body temperature within acceptable range and minimize heat loss
- To detect necessity for environmental temperature regulation and to determine infant’s response to environmental thermoregulation

**Additional NIC Concepts Apply to These Interventions**
- Environmental Management
- Hypothermia Treatment

**Ineffective Breath sounds related to immaturity**

**Child’s/Family’s Defining Characteristics (Subjective and Objective Data)**
- Risk Factors
- Separation
- Preterm infant
- Physical barriers

**Patient Outcomes**
- Parent(s) will form emotional bond or attachment with newborn.

**The Following NOC Concept Applies to These Outcomes**
- Parent/Infant/Child Attachment

**NURSING INTERVENTIONS**
- Encourage parent(s) to hold and make eye contact with newborn as physical status allows.
- Encourage parent-newborn skin-to-skin contact in delivery room as condition of newborn allows.
- Explain to parents the newborn’s illness in simple terms and expectations for recovery.
- Encourage parents to name newborn.
- Encourage parent participation in newborn care activities such as touching infant, expressing and storing maternal breast milk, and talking to infant.

**Rationale**
- To minimize effects of physical separation from newborn
- To facilitate parent-infant interaction that is meaningful and comforting
- To enhance parental knowledge and decrease potential fear of unknown regarding infant’s survival and recovery
- To provide child individual identity
- To facilitate parental involvement in attaining the role of parents and decrease feelings of hopelessness

Additional nursing diagnoses that may be applicable to high-risk newborn with respiratory distress: Imbalanced Nutrition: Less Than Body Requirements; Risk for Imbalanced Body Temperature; Impaired Spontaneous Ventilation; Impaired Gas Exchange; and Ineffective Airway Clearance.
delivered while the chest is still compressed in the birth canal. After delivery, the need for tracheal suctioning is based on infant assessment. Infants who are vigorous with strong, stable respiratory effort, good muscle tone, and heart rate greater than 100 beats/min should not undergo tracheal suctioning but should be closely monitored (Kattwinkel, 2006). On the other hand, infants who demonstrate poor respiratory effort, low heart rate, and poor tone should be rapidly intubated, suctioned appropriately, and resuscitated according to clinical status after suctioning. These management protocols have been supported by ongoing research (Kabbur, Herson, Zaremba, et al, 2005).

Infants with respiratory distress are admitted to the NICU. Management of MAS consists of ventilatory support, exogenous surfactant administration, IV fluids, systemic antibiotics, and in some cases inotropes. Because these infants are prone to development of persistent pulmonary hypertension, they should be supported to maintain normal pH, carbon dioxide, and oxygen levels; they may be candidates for ECMO therapy, HFV, or NO (see Nitric Oxide, p. 354, and Persistent Pulmonary Hypertension of the Newborn, p. 359). Complications are managed symptomatically or as described under the specific disorder.

**QUALITY PATIENT OUTCOMES:** MAS
- Room air oxygen saturation ≥90%
- Maintains arterial/venous pH ≥7.35

**Nursing Care Management**

Nursing care management is the same as for any high-risk neonate (see nursing care in sections on oxygen therapy, persistent pulmonary hypertension, and other complications).

**AIR LEAK SYNDROMES**

Extraneous air syndromes, extraalveolar air accumulation, and air leaks are names applied to various clinically recognized disorders produced as a result of alveolar rupture and subsequent escape of air to tissues in which air is not normally present. Extraneous air collection (1) may occur spontaneously in normal neonates, (2) can result from congenital renal or pulmonary malformations, and (3) often complicates underlying respiratory disease and its therapy (e.g., positive pressure ventilation, especially when high distending pressures are required).

After alveolar rupture, air often vents directly into the pleural space to create a pneumothorax. Air may vent into the perivascular interstitium, a condition called pulmonary interstitial emphysema (PIE). PIE may be seen on radiographs as early as 2 to 3 hours after birth in ELBW and VLBW infants with severe RDS. Localized PIE may resolve by itself or may be a precursor to pneumothorax. HFV has been reported to improve the outcome in infants with PIE (Korones, 2003). Air can dissect along the perivascular sheaths to eventually enter the mediastinum and cause pneumomediastinum. More extensive leaks involve the pericardium (manifested as pneumopericardium) or emphysema in the cervical, subcutaneous, or retroperitoneal soft tissues.

**Clinical Manifestations**

Spontaneous pneumothorax usually occurs during the first few breaths after birth, primarily in full-term or postterm infants, and is evident by the gradual onset of symptoms of respiratory distress after arrival in the nursery. Use of positive pressure ventilation in resuscitation may cause air leaks. Mechanical positive pressure ventilation may contribute to an increase in the incidence of air leaks; however, in some cases, such as in extreme prematurity and meconium aspiration, air leaks may not be altogether preventable. The nurse suspects an air leak on the basis of respiratory manifestations and a shift in location of maximum intensity of heart sounds and absent or diminished breath sounds (although breath sounds may not be altered because of the small diameter of the chest and auscultation of referred breath sounds).

A tension pneumothorax occurs more frequently in infants requiring ventilatory assistance. In preterm infants being mechanically ventilated, an air leak may be demonstrated by hypotension, bradycardia, decreased or absent breath sounds unilaterally, decreased oxygenation (by pulse oximetry), and cyanosis, none of which responds to efforts for oxygenation (a resuscitation bag connected to the ET tube and provision of manual ventilations). There may also be chest asymmetry, altered cardiac sounds (diminished, shifted, or muffled), a palpable liver and spleen, and subcutaneous emphysema. Infants on HFV may demonstrate an air leak by a sudden decrease in systemic pressure or an absence of chest movement (because of difficulty in auscultation of the chest with such modalities). The otherwise healthy full-term infant may exhibit only mild to moderate signs of respiratory distress. The presence of an air leak has been identified as contributing to the risk of an adverse developmental outcome in preterm infants (Laptook, O’Shea, Shankaran, et al, 2005).

**NURSING ALERT**

Early manifestations of pneumothorax include tachypnea, restlessness and irritability, lethargy, grunting, nasal flaring, and retractions. Pneumothorax during ventilatory assistance is evident from abrupt and profound duskytimes or cyanosis; significant declines in heart rate, arterial blood pressure, and pulse pressure; and poor peripheral perfusion.

**Therapeutic Management**

Diagnosis is confirmed by transillumination of the chest with a fiberoptic light and/or radiographic examination. In symptomatic infants, treatment is urgent. Evacuation of trapped air is accomplished by chest tube insertion into the pleural space through a small chest incision. The chest tube is then attached to continuous water-seal drainage. A dry suction control drainage system not requiring water is also available. In situations requiring infant transport, a pocket-sized Heimlich valve may be used until an appropriate drainage system can be established; the valve is not effective when fluid drainage is required. Needle aspiration serves as an emergency measure until a chest tube can be inserted. Pneumomediastinum seldom requires treatment, but pneumopericardium is managed by needle aspiration or pericardial tube drainage. The full-term newborn with a small tension pneumothorax may require only oxygen therapy.
and IV nutrition for a brief period if respiratory distress is not severe.

Nursing Care Management
The most important nursing function, which is most effective for early detection, is close observation for the possibility of an air leak in susceptible infants. Nurses maintain a high level of suspicion in (1) infants with RDS with or without positive pressure ventilation, (2) infants with meconium-stained amniotic fluid or MAS, (3) infants with radiographic evidence of interstitial or lobar emphysema, (4) infants who required resuscitation at birth, or (5) infants receiving CPAP or positive pressure ventilation. For infants at risk, needle aspiration equipment (30-ml syringe, three-way stopcock, and 23- to 25-gauge needles) should be at the bedside for emergency use.

The general nursing care of the infant with an extraneous air syndrome is the same as for all high-risk neonates. Respiratory management is similar to that for infants with RDS. Assessing breath sounds frequently, monitoring the efficacy of gas exchange, and regulating oxygen therapy according to the infant’s needs are vital nursing functions. Attention to pain management with the procedure is vital in these preverbal and significantly stressed infants.

PERSISTENT PULMONARY HYPERTENSION OF THE NEWBORN

PPHN, formerly known as persistent fetal circulation, is a condition in which affected infants display severe pulmonary hypertension, with pulmonary artery pressure levels equal to or greater than systemic pressure, and large right-to-left shunts through both the foramen ovale and the ductus arteriosus. PPHN is a group of disorders having varied causes yet common presenting features and may be classified according to causative etiology (Konduri and Kim, 2009). Because full development of pulmonary arterial musculature occurs late in gestation, PPHN is primarily a condition of late-preterm, full-term, or postterm infants, many of whom were products of complicated pregnancies or deliveries. The condition is often associated with aspiration (especially meconium aspiration), congenital diaaphragmatic hernia with severe respiratory distress, cold stress, respiratory distress (e.g., RDS or pneumonia), and septicemia (group B streptococci [GBS]). PPHN is believed to be precipitated by perinatal factors, such as perinatal asphyxia, that cause or contribute to constriction of the pulmonary vasculature.

PPHN can be either primary or secondary. Primary PPHN occurs when the pulmonary vascular system fails to open with the initial respiration at birth; secondary PPHN results from hypoxic stress that increases PVR and causes a return to fetal cardiopulmonary circulation. PPHN is most commonly observed in infants at 35 to 44 weeks of gestation who have a history of perinatal asphyxia, metabolic acidosis, or sepsis and respiratory distress within the first 24 hours. The infants become hypoxic and display marked cyanosis, tachypnea with grunting and retractions, and decreased peripheral perfusion. A loud pulmonary component of the second heart sound and, sometimes, a systolic ejection murmur are present. Diagnosis is established from clinical signs and diagnostic tests, including chest radiography, electrocardiogram, and echocardiography.

Therapeutic Management
Early recognition and management of conditions that contribute to or cause hypoxia and pulmonary vascular vasoconstriction are the primary goals in the prevention of PPHN. Additional treatment includes careful fluid regulation and evaluation of intravascular fluid volume. Supplemental oxygen reduces hypoxia and decreases pulmonary vasoconstriction. Assisted ventilation, often by HFV, is required if hypoxia is severe. Vasodilators, such as sildenafil (a phosphodiesterase [PDE5] inhibitor) or epoprostenol (prostacyclin), are sometimes prescribed to decrease PVR, thereby avoiding ECMO and NO. Sildenafil administered in intravenous or oral form has been shown to reduce PVR (hypertension) in neonates and improve oxygenation, but further controlled clinical trials are needed (Latini, Del Vecchio, De Felice, et al, 2008; Shah and Ohlsson, 2007b; Steinborn, Kinsella, Pierce, et al, 2009).

Additional drug therapy used in the management of PPHN includes judicious use of sodium bicarbonate to maintain appropriate acid-base balance; volume expanders such as normal saline or lactated Ringer solution; and the vasopressors dopamine, dobutamine, and nitroprusside to increase systemic vascular resistance (Konduri, 2004). The use of inhaled NO has been successfully used to reverse pulmonary vascular vasoconstriction and is often attempted before other therapies such as ECMO (see Nitric Oxide, p. 354).

Another approach to management of infants with pulmonary complications is the use of ECMO with a modified heart-lung machine. Blood is shunted from a catheter in the right atrium or right internal jugular vein by gravity to a servoregulated roller pump; pumped through a membrane lung and a small heat exchanger; and returned to the systemic circulation via a major artery, such as the carotid artery, to the aortic arch. A venovenous approach (femoral vein) may be used, thus avoiding the need to ligate the carotid artery. ECMO provides oxygen to the circulation and allows the lungs to rest. The goal of ECMO is to “buy time” for the severely injured lung to heal while effectively oxygenating major organ systems, including the brain, heart, kidneys, and lungs (Fig. 10-18).

ECMO is labor intensive and thus expensive. Technical malfunctions may occur, requiring frequent monitoring of the equipment and the patient’s response to treatment. Typically,
two nurses, or a nurse and a perfusionist, are required as minimum staffing for the ECMO patient; more staff, including a respiratory therapist, are required in the acute phase. ECMO requires heparinization of the blood and blood circuit; for this reason it is not used in infants at less than 35 weeks of gestation, who are prone to intraventricular hemorrhage. Bleeding is one of the major complications associated with ECMO. Overall the need for ECMO has decreased with increased use of exogenous surfactant, inhaled NO, and HFV for neonatal hypoxic respiratory failure (Konduri and Kim, 2009).

**Nursing Care Management**

The nursing care for PPHT is the same as for infants with severe respiratory difficulties and infants supported by mechanical ventilation and cardiovascular support. The infant with PPHT is often the sickest on the unit, depending on the causative factors and reaction to treatment. Because handling for any reason causes a decrease in arterial oxygen concentration, the nurse must weigh the stresses imposed by routine care against the risk of iatrogenic hypoxia. It is important to decrease noxious stimuli that cause hypoxia and to use clustered nursing interventions that keep nonsedated infants calm. Continuous monitoring of oxygenation, temperature, central venous pressure, vital signs, blood pressure, and acid-base balance decreases the need for physical manipulation and disturbance. Infants are further assessed for response to treatments, including IV therapy, fluids, electrolytes, and exogenous glucose.

**BRONCHOPULMONARY DYSPLASIA**

BPD, sometimes referred to as chronic lung disease, is a pathologic process that develops primarily in ELBW and VLBW infants with RDS. BPD may also develop in infants with MAS, persistent pulmonary hypertension, pneumonia, and cyanotic heart disease. Infants who develop BPD are at risk for frequent hospitalization because of their borderline respiratory reserve, hyperactive airway, and increased susceptibility to respiratory infection.

Mild BPD is operationally defined the need for supplemental oxygen for 28 days or more but room air by 36 weeks corrected gestational age or at discharge. Moderate BPD is defined by the need for oxygen for 28 days or more and less than 30% oxygen at 36 weeks corrected gestational age. With severe BPD infants require more than 30% oxygen at 36 weeks corrected gestational age (Bhandari and Bhandari, 2009). An inverse relationship between incidence of BPD and birth weight is emphasized in the Vermont-Oxford Network report, which reported a 60% incidence for ELBW infants (501 to 750 g [1.1 to 1.6 lb]) versus 21% for infants weighing 1001 to 1250 g (2.2 to 2.7 lb). Risk factors for BPD include assisted ventilation, oxygen administration, prenatal and postnatal (nosocomial) infections, FPA, and fluid imbalance (Askin and Diehl-Jones, 2009a).

The more severe form of BPD usually begins with severe respiratory failure secondary to RDS or with pneumonia requiring mechanical ventilation with high airway pressure and oxygen supplementation during the first few days of life (Bancalari, 2001). Since the advent of antenatal glucocorticoid therapy, surfactant replacement, and new ventilator strategies, a “new” BPD is emerging (Greenough, 2008). These infants experience a milder initial respiratory course but continue to require ventilatory support or oxygen supplementation and show radiographic pulmonary changes characteristic of BPD.

**Pathophysiology**

The pathogenesis of BPD is complex and multifactorial. BPD begins with the immature lung that undergoes an initial injury leading to a chronic inflammatory process that results in recurrent injury and abnormal healing (Askin and Diehl-Jones, 2009a). A variety of mechanisms have been related to the initial injury: (1) prenatal infection (inflammatory process before birth), (2) mechanical ventilation (volutrauma, intubation), (3) supplemental oxygen (oxygen-derived free radicals), (4) increased pulmonary blood flow from PDA, or (5) postnatal infection.

The pulmonary changes are characterized by interstitial edema and epithelial swelling followed by thickening and fibrotic proliferation of the alveolar walls and squamous metaplasia of the bronchiolar epithelium. Areas of atelectasis and cystlike foci of hyperaeration are visible on radiographs between 10 and 20 days of life and persist for weeks; however, some infants may not demonstrate cystic foci. In addition, ciliary activity is paralyzed by high oxygen concentrations that interfere with the ability to clear the lung of mucus, thus aggravating airway obstruction and atelectasis. As the infant’s lungs begin healing, the process is altered, possibly by continuous high oxygenation, inadequate nutrition, or vitamin E deficiency, resulting in decreased surface for oxygen and carbon dioxide exchange. The overall results of this process are hypercarbia, hypoxemia, and subsequent inability to wean successfully from oxygen.

As survival of immature preterm infants (<28 weeks of gestation) increases, the occurrence of BPD also increases.

In addition to BPD, other diseases associated with similar radiographic findings include congenital heart disease and viral pneumonia caused by cytomegalovirus. There are no laboratory alterations that confirm a diagnosis; diagnosis is made on the basis of radiographic findings, oxygen therapy or positive pressure ventilation after 28 days, signs of respiratory distress, and a history of requiring mechanical ventilation in the first week of life for more than 3 days.

**Therapeutic Management**

The first approach to management is prevention of the disorder in susceptible infants. Despite previous theorization that surfactant administration to preterm infants would eradicate BPD, studies so far have failed to show a significant decrease of BPD in infants less than 30 weeks receiving surfactant for prophylaxis or rescue (American Academy of Pediatrics, 2008). To reduce the risk of volutrauma when positive pressure ventilation is being used, maintain the lowest PIP necessary to obtain adequate ventilation, and use the lowest level of inspired oxygen to maintain adequate oxygenation. HFOV has been beneficial in reducing the risk of BPD, as has the administration of vitamin A (Shah, 2003). Fluid administration is carefully controlled and restricted. Drug or surgical intervention is indicated when there is significant shunting of blood through the PDA.

No specific treatment exists for BPD except to maintain adequate arterial blood gases with the administration of oxygen and to avoid progression of the disease. Corticosteroid (dexa-
methasone) therapy has been shown to benefit infants with BPD by decreasing the pulmonary inflammatory response and improving oxygenation and gas exchange, resulting in earlier weaning from mechanical assistance. However, with complications such as sepsis, hypertension, and hyperglycemia and an overall lack of decreased mortality in such infants, this therapy remains controversial. Other adverse effects of this long-acting, potent glucocorticoid have been reported: growth restriction, GI hemorrhage, and cardiomyopathy. In light of studies that show an increased incidence of periventricular leukomalacia, neuromotor abnormalities, CP, decreased cerebral cortical gray matter volume, and adverse long-term neurologic outcome, the benefits of this treatment may not outweigh the risks (American Academy of Pediatrics and Canadian Paediatric Society, 2002; Halliday, Ehrenkranz, and Doyle, 2009). Ongoing research is being done to determine whether an alternative dosing regimen of postnatal steroids can reduce the incidence of BPD without associated neurodevelopmental side effects (Onland, Offringa, De Jaegere, et al, 2009).

Weaning infants from oxygen is difficult and must be accomplished gradually. These infants do not tolerate excessive or even normal amounts of fluid well and have a tendency to accumulate interstitial fluid in the lungs, which aggravates the condition. Oral diuretics are used to control interstitial fluid. Nebulized or metered dose inhaler bronchodilators (albuterol) and inhaled steroids may be effective and promote improvement in infants with BPD. (See also Asthma, Chapter 32.) Oral electrolyte supplements are given to replace those lost with concurrent oral diuretics and renal water losses.

Growth and development are often delayed in infants with BPD, which is related in part to the difficulties in providing adequate nutrition and in part to the lack of normal sensory stimulation because of prolonged hospitalization. Children with BPD have metabolic needs far greater than those of the average infant. This can create a problem for the caregiver, who must meet the goals of adequate nutrition while avoiding overhydration, especially if the child is ill, eats poorly, or has cardiopulmonary instability. The infant may be further compromised by gastroesophageal reflux, a frequent complication in preterm infants. (See Chapter 33.) Adequate intake of protein is particularly important in preventing postnatal growth failure in LBW infants. Protein supplements may be necessary to ensure adequate intake.

Osteopenia may occur in infants with BPD and in preterm infants, with higher incidence among the infants with BPD, presumably because of low calcium and vitamin D intake secondary to the calcicuric effects of diuretic therapy. Dietary supplementation with human milk fortifier, calcium and phosphorus, and vitamin D has reduced the incidence of osteopenia in preterm infants.

RSV prophylaxis with the monoclonal antibody palivizumab (Synagis) is effective in diminishing the complications of RSV. RSV is a common cause for hospitalization and death in growing preterm neonates, including those with BPD. Palivizumab is given intramuscularly to high-risk infants, does not interfere with immunizations, and has few side effects. Palivizumab is administered in a dose of 15 mg/kg once a month, usually beginning in October and ending in May. (See Respiratory Syncytial Virus, Chapter 32.)

**Prognosis**

Reports vary regarding the mortality rate for BPD. The hospital stay is often long because of the infant’s need for supplemental oxygen, although home oxygen therapy provides selected infants the opportunity for discharge. A nasal cannula is an acceptable way to administer oxygen for the dependent infant to promote development of motor and social skills. Long-term problems seen in older children who had BPD as infants include growth failure, airway hyperreactivity, hyperexpansion, increased incidence of respiratory infections, and airway obstruction. A significant proportion of deaths occur after discharge from the hospital.

An 8-year follow-up study comparing the outcomes of preterm infants with BPD, preterm infants without BPD, and full-term infants without BPD found that BPD and duration on oxygen have long-term adverse effects on cognitive and academic achievement above and beyond the effects of VLBW alone. After controlling for birth weight and neurologic complications, BPD was associated with lower IQs; poorer perceptual organization, attention, and motor skills; reduced school achievement; and greater participation in special education such as physical and speech-language therapies (Short, Klein, Lewis, et al, 2003; Anderson and Doyle, 2008).

**Nursing Care Management**

Infants with BPD expend considerable energy in their efforts to breathe; therefore it is important that they receive plenty of opportunities for rest and additional calories. Growth records provide clues to the need for change in their diets, and some infants require nutritional supplements. Because these infants tire easily and because large quantities of formula might compromise respiration, small, frequent feedings are better tolerated. Reducing environmental stimuli and subsequent hypoxia is an important aspect in the care of these infants. Close attention to the infant’s behavioral cues is important in the older infant with BPD because these cues may signal carbon dioxide retention.

Adequate hydration is extremely important because a large amount of fluid are lost through respiration, and secretions must be thinned sufficiently to facilitate removal by suctioning. However, because BPD increases lung permeability, many infants are subject to pulmonary edema and require fluid restriction. Nurses must be alert to signs of both overhydration and underhydration, such as changes in weight, electrolytes, output measurements, and urine specific gravity and signs of edema.

Because the growing infant with BPD has a restricted fluid intake, has higher than average caloric requirements, and often requires many oral medications, the nurse is challenged by the complexity of care involved. Infants with BPD may become difficult or maladaptive feeders if they are aware of hunger yet compromised by not being able to eat fast enough to satiate that hunger because of the increased labor of breathing. Individualized nursing care aimed at decreasing oxygenation requirements during feedings, decreasing environmental stimuli, fortifying feedings, and providing more contact with a primary caregiver may facilitate the infant’s care. Feeding schedules should be individualized as much as possible. Oral medications...
that taste bad to the infant may be given at times separate from feedings to ensure that feeding time is pleasant. Adjustments to overall fluid administration requirements are made, taking into account that the oral medications are also fluids. Regurgitated medications and feedings need to be dealt with in regard to fluid and caloric needs and the amount of absorption of medication that occurs before emesis.

Parents are extremely anxious regarding the prognosis when their infant has BPD. In addition, the lengthy hospitalization interferes with parent-child relationships and deprives the infant of appropriate parental contact and stimulation. Nurses should encourage the parents to visit the infant and become involved in the routine care. The parents need to be informed regarding medical care, equipment, and procedures related to their infant and taught procedures such as suctioning and chest physiotherapy.

The older infant with BPD should have normal nurturing and developmental opportunities appropriate to the infant’s condition and abilities. Careful monitoring of physiologic and behavioral systems during any activity is necessary so that activity can be stopped before the infant becomes irritable or tired. Opportunities out of bed in an infant seat or on a floor mat with a nurse or physical or play therapist provide one-on-one interaction that can enhance the infant’s experience of the world and people.

Irritability has been associated with infants who have BPD, making their care often challenging and frustrating (see Developmental Outcome, p. 332). Some strategies to facilitate infant coping during prolonged hospitalization include (1) decreased number of unfamiliar caregivers, (2) increased access to parents, (3) predictability in schedule and caregivers, (4) consistency of care routines and practices, (5) pleasurable opportunities for play and socialization within physical tolerance, (6) adequate nutrition, and (7) uninterrupted rest cycles with diurnal variation to facilitate biologic rhythms. Parental involvement is critical because they are the one constant for the infant.

**Home Care**

Because the availability of home cardiac and apnea monitors and home oxygen therapy has increased, many infants with BPD can be discharged when they are gaining weight and their oxygen need is low. Home care is desirable to promote parent-infant bonding, minimize health care costs, and prevent nosocomial infections. Preparation for home care requires education and considerable reassurance. (See Chapter 25.) Management of home monitoring equipment and home oxygen therapy is stress provoking, but most families become comfortable with the machinery while their infant is still in the hospital. Families need reminders about their infant’s increased risk of infection and about limiting contact with persons who have respiratory tract infections. Because of their minimum respiratory reserve, even a minor illness can threaten these infants.

Some infants are discharged with a tracheostomy on oxygen supplementation or home ventilators. Discharge teaching and home care nursing (minimum of 2 weeks to several months) is crucial to these infants’ safe and successful transition into the community and home setting. Parents need to learn how to advocate for appropriate home care and supplies in anticipation of future needs.

Because of the high mortality rate in the first year, parents should learn cardiopulmonary resuscitation and how to manage any other emergency that might be anticipated for their infant. Helping families cope with their anxieties and reassuring them of their ability to manage the care of their infant are important nursing functions. Parents need follow-up visits in the home and the comfort of knowing that help is only a telephone call away.

**HIGH RISK RELATED TO INFECTIOUS PROCESSES**

**SEPSIS**

Sepsis, or septicemia, refers to a generalized bacterial infection in the bloodstream. Neonates are highly susceptible to infection as a result of diminished nonspecific (inflammatory) and specific (humoral) immunity, such as impaired phagocytosis, delayed chemotactic response, minimum or absent IgA and immunoglobulin M (IgM), and decreased complement levels. Because of the infant’s poor response to pathogenic agents, there is usually no local inflammatory reaction at the portal of entry to signal an infection, and the resulting symptoms tend to be vague and nonspecific. Consequently, diagnosis and treatment may be delayed.

Although the mortality from sepsis has diminished, the incidence has not. Nursery epidemics are not infrequent, and the high-risk infant has a four times greater chance of developing septicemia than does the normal neonate. The frequency of infection is almost twice as great in male infants as in females and also carries a higher mortality for males. Other factors increasing the risk of infection are prematurity, congenital anomalies or acquired injuries that disrupt the skin or mucous membranes, invasive procedures such as placement of IV lines and ET tubes, administration of total parenteral nutrition, and nosocomial exposure to a number of pathogens in the NICU. Thorough hand washing is the single most important infection control measure in the NICU. Proper handling of formula and supplies such as syringes and gavage tubes is also vital to prevent infection.

Breast-feeding has a protective effect against infection and should be promoted for all newborns. It is of particular benefit to the high-risk neonate. Colostrum contains agglutinins that are effective against gram-negative bacteria. Human milk contains large quantities of IgA and iron-binding protein that exert a bacteriostatic effect on Escherichia coli. Human milk also contains macrophages and lymphocytes that promote a local inflammatory reaction.

**Pathophysiology**

The premature withdrawal of the placental barrier leaves infants vulnerable to most common viral, bacterial, fungal, and parasitic infections. Immune substances, primarily immunoglobulin G (IgG), are normally acquired from the maternal system and stored in fetal tissues during the final weeks of gestation to provide newborns with passive immunity to a variety of infectious agents. Early birth interrupts this transplacental transmission; thus preterm infants have a low level of circulating IgG;
the concentrations of immune substances directly relate to the length of gestation. IgA, which plays a role in defense against viral infections, and IgM, with properties that are most efficient in dealing with gram-negative organisms, are not transferred to the fetus, which leaves the infant highly vulnerable to invasion by these organisms.

Defense mechanisms of neonates are further hampered by a low level of complement, diminished opsonization ability, monocyte dysfunction, and a reduced number and inefficient function of circulating leukocytes. Furthermore, these leukocytes with diminished motility and phagocytic capacity are unable to concentrate their limited numbers selectively at the site of infection. In addition, a hypofunctioning adrenal gland contributes only a meager antiinflammatory response. Consequently, these deficiencies permit rapid invasion, spread, and multiplication of organisms. An immature gut mucosal barrier further predisposes the preterm infant to bacteria, which may easily cross the mucosa into the bloodstream.

**Sources of Infection**

Sepsis in the neonatal period can be acquired prenatally across the placenta from the maternal bloodstream or during labor from ingestion or aspiration of infected amniotic fluid. Prolonged rupture of the membranes always presents a risk for maternal-fetal transfer of pathogenic organisms. In utero transplacental transfer can occur with a variety of organisms and viruses such as cytomegalovirus, toxoplasmosis, and *Treponema pallidum* (syphilis), which cross the placental barrier during the latter half of pregnancy.

Early-onset sepsis (<3 days after birth) is acquired in the perinatal period. Infection can occur from direct contact with organisms from the maternal GI and genitourinary tracts. Organisms associated with early-onset infection include GBS, *E. coli*, and other gram-negative enteric organisms. Despite the development of maternal screening and prophylaxis, infection rates for early-onset GBS infection remain at approximately 0.3 per 1000 live births (Centers for Disease Control and Prevention, 2007). *E. coli*, which may be present in the vagina, accounts for approximately half of all cases of sepsis caused by gram-negative organisms. GBS is an extremely virulent organism in neonates, with a high (50%) death rate in affected infants. Other bacteria noted to cause early-onset infection include *Haemophilus influenzae*, *Citrobacter* and *Enterobacter* organisms, coagulase-negative staphylococci, and *Streptococcus viridans* (Stoll, Hansen, Higgins, et al, 2005). Other pathogens that are harbored in the vagina and may infect the infant include gonococci, *Candida albicans*, herpes simplex virus (type II), and chlamydia.

Late-onset sepsis (1 to 3 weeks after birth) is primarily nosocomial, and the offending organisms are usually staphylococci, *Klebsiella* organisms, enterococci, *E. coli*, and *Pseudomonas* or *Candida* species (Stoll, 2007). Coagulase-negative staphylococci, considered to be primarily a contaminant in older children and adults, is commonly found to be the cause of septicemia in ELBW and VLBW infants. Bacterial invasion can occur through sites such as the umbilical stump; the skin; mucous membranes of the eye, nose, pharynx, and ear; and internal systems such as the respiratory, nervous, urinary, and GI systems.

Postnatal infection is acquired by cross-contamination from other infants, personnel, or objects in the environment. Bacteria, such as *Klebsiella* and *Pseudomonas* organisms, that are commonly called “water bugs” (because they are able to grow in water) are found in water supplies; humidifying apparatus; sink drains; suction machines; most respiratory equipment; and indwelling venous and arterial catheters used for infusions, blood sampling, and monitoring vital signs. These organisms are often transmitted by personnel from person to person or object to person by poor hand washing and inadequate housecleaning.

Neonatal sepsis is most common in the infant at risk, particularly the preterm infant or the infant born after a difficult or traumatic labor and delivery, who is least capable of resisting such bacterial invasion.

**Clinical Manifestations**

A few neonatal infections (e.g., pyoderma, conjunctivitis, omphalitis, and mastitis) are easy to recognize. However, systemic infections are characterized by subtle, vague, nonspecific, and almost imperceptible physical signs. Often the only complaint concerns an infant’s “failure to do well,” not looking “right,” or nonspecific respiratory distress. Rarely is there any indication of a local inflammatory response, which would suggest the portal of entry into the bloodstream. The presence of bacteria is indicated by a specific characteristic. For example, *Pseudomonas* organisms produce necrotic purplish skin lesions, and group B β-hemolytic streptococci usually result in severe respiratory distress, periods of apnea, and a chest radiograph similar to that of RDS.

All body systems tend to show some indication of sepsis, although often little correlation exists between the manifestations and the etiologic factors involved. For example, seizures and fever, a universal feature of infection in older children, may be absent in neonates. It is usually the nursing observation of subtle changes in appearance and behavior that leads to the detection of infection. The nonspecific, early signs are hypothermia and changes in color, tone, activity, and feeding behavior. In addition, sudden episodes of apnea and unexplained oxygen desaturation (hypoxia) may signal an infection. Significantly, similar signs may be manifestations of a number of clinical conditions unrelated to sepsis, such as hypoglycemia, hypocalcemia, heroin withdrawal, or a CNS disorder.

Preterm infants, particularly ELBW and VLBW infants, are highly susceptible to early sepsis and pneumonia occurring concurrently with RDS, since preterm delivery has been increasingly shown to be associated with a maternal bacterial pathogen. ELBW and VLBW infants are also highly susceptible to fungal and viral infections. Investigation for such agents should begin when sepsis is suspected in this population. Because meningitis is a common sequela of sepsis, the neonate is evaluated for bacterial growth in cerebrospinal fluid (CSF). Clinical signs of neonatal meningitis, particularly in VLBW infants, may not have typical features of older infants. Clinical signs that may indicate possible neonatal sepsis are listed in Box 10-9.

**Diagnostic Evaluation**

Because sepsis is easy to confuse with other neonatal disorders, the definitive diagnosis is established by laboratory and radio-
graphic examination. Isolation of the specific organism is always attempted through cultures of blood, urine, and CSF. Blood studies may show signs of anemia, leukocytosis, or leukopenia. Leukopenia is usually an ominous sign because of its frequent association with high mortality. An elevated number of immature neutrophils (left-shift), decreased or increased total neutrophils, and changes in neutrophil morphologic characteristics also suggest an infectious process in the neonate. Other diagnostic data that are helpful in the determination of neonatal sepsis include C-reactive protein and interleukins, specifically interleukin-6 (Volante, Moretti, Pisani, et al, 2004; Laborada, Rego, Jain, et al, 2003).

**Therapeutic Management**

In addition to the institution of rigorous preventive measures such as good hand washing, early recognition and diagnosis are essential to increase the infant’s chance for survival and reduce the likelihood of permanent neurologic damage. Diagnosis of sepsis is often based on suspicion of initial clinical signs and symptoms, and antibiotic therapy is initiated before laboratory results are available for confirmation and identification of the exact organism. Treatment consists of circulatory support, respiratory support, and aggressive administration of antibiotics.

Supportive therapy usually involves administration of oxygen (if respiratory distress or hypoxia is evident), careful regulation of fluids, correction of electrolyte or acid-base imbalance, and temporary discontinuation of oral feedings. Blood transfusion may be needed to correct anemia; IV fluids for shock, electronic monitoring of vital signs, and regulation of the thermal environment are mandatory.

Antibiotic therapy is continued for 7 to 10 days if cultures are positive, discontinued in 36 to 72 hours if cultures are negative and the infant is asymptomatic, and most often administered via IV infusion. Antifungal and antiviral therapies are implemented as appropriate, depending on causative agents.

**Prognosis**

The prognosis for neonatal sepsis is variable. Severe neurologic and respiratory sequelae may occur in ELBW and VLBW infants with early-onset sepsis. Late-onset sepsis and meningitis may also result in poor outcomes for immunocompromised neonates.

The introduction of new markers for neonatal sepsis such as acute phase proteins, cytokines, cell surface antigens, and bacterial genomes may prove to be particularly helpful in early differentiation of true sepsis from RDS and in guidance for antibiotic therapy (Arnon and Litmanovitz, 2008). Future experimental methods being explored to combat infection in neonates include monoclonal antibody therapy, fibronectin infusion, and lymphokine enhancement.

**Nursing Care Management**

Nursing care of the infant with sepsis involves observation and assessment as outlined for any high-risk infant. Recognition of the existing problem is of paramount importance. It is usually the nurse who observes and assesses infants and identifies that “something is wrong” with them. Awareness of the potential modes of infection transmission also helps the nurse identify those at risk for developing sepsis. Much of the care of infants with sepsis involves the medical treatment of the illness. Knowledge of the side effects of the specific antibiotic and proper regulation and administration of the drug are vital. Antibiotics are usually administered via a special injection port near the infusion site. The appropriately diluted medication is administered slowly by mechanical pump.

Prolonged antibiotic therapy poses additional hazards for affected infants. Oral antibiotics, if administered, destroy intestinal flora responsible for the synthesis of vitamin K, which can reduce blood coagulability. In addition, antibiotics predispose the infant to growth of resistant organisms and superinfection from fungal or mycotic agents, such as *C. albicans*. Nurses must be alert for evidence of such complications. Nystatin oral suspension may be administered for prophylaxis against oral candidiasis.

Part of the total care of infants with sepsis is to decrease any additional physiologic or environmental stress. This includes providing an optimum thermoregulated environment and anticipating potential problems such as dehydration or hypoxia. Precautions are implemented to prevent the spread of infection to other newborns, but to be effective, activities must be carried out by all caregivers. Proper hand washing, the use of disposable...
equipment (e.g., linens, catheters, feeding supplies, and IV equipment), disposal of secretions (e.g., vomitus and stool), and adequate housekeeping of the environment and equipment are essential. Because nurses are the most consistent caregivers involved with sick infants, it is usually their responsibility to ensure that everyone maintains all phases of contact isolation or Standard Precautions.

Another aspect of caring for infants with sepsis involves observation for signs of complications, including meningitis and septic shock, a severe complication caused by toxins in the bloodstream.

A number of viral agents—namely, cytomegalovirus, herpes, hepatitis, and human immunodeficiency virus (HIV)—may also be transmitted to the fetus from the mother. When acquired prenatally (congenital), these viruses represent a serious threat to the infant’s life. (See Table 10–11 for viral infections.)

**NECROTIZING ENTEROCOLITIS**

NEC is an acute inflammatory disease of the bowel with increased incidence in preterm and other high-risk infants; it is most common in preterm infants. Because the signs are similar to those observed in many other disorders of the newborn, nurses must constantly be aware of the possibility of this disease.

**Pathophysiology**

The precise cause of NEC is still uncertain, but it appears to occur in infants whose GI tract has suffered vascular compromise. Intestinal ischemia of unknown etiology, immature GI host defenses, bacterial proliferation, and feeding substrate are now believed to have a multifactorial role in the etiology of NEC. Prematurity remains the most prominent risk factor in this disease (Schurr and Perkins, 2008).

The damage to mucosal cells lining the bowel wall is great. Diminished blood supply to these cells causes their death in large numbers; they stop secreting protective, lubricating mucus; and the thin, unprotected bowel wall is attacked by proteolytic enzymes. Thus the bowel wall continues to swell and break down; it is unable to synthesize protective IgM, and the mucosa is permeable to macromolecules (e.g., toxins), which further hamper intestinal defenses. Gas-forming bacteria invade the damaged areas to produce intestinal **pneumatosis**, the presence of air in the submucosal or subserosal surfaces of the bowel.

**Clinical Manifestations**

The prominent clinical signs of NEC are a distended abdomen, gastric residuals, and blood in the stools. Because NEC closely resembles septicemia, the infant may “not look well.” Nonspecific signs include lethargy, poor feeding, hypotension, apnea, vomiting (often bile stained), decreased urinary output, and hypothermia. The onset is usually between 4 and 10 days after the initiation of feedings, but signs may be evident as early as 4 hours of age and as late as 30 days. NEC in full-term infants almost always occurs in the first 10 days of life; late-onset NEC is confined primarily to preterm infants and coincides with the onset of feedings after they have passed through the acute phase of an illness such as RDS.

**Diagnostic Evaluation**

Radiographic studies show a sausage-shaped dilatation of the intestine that progresses to marked distention and the characteristic intestinal pneumatosis—“soapsuds,” or the bubbly appearance of thickened bowel wall and ultralumina. Air may be present in the portal circulation or free air observed in the abdomen, indicating perforation. Laboratory findings may include anemia, leukopenia, leukocytosis, metabolic acidosis, and electrolyte imbalance. In severe cases coagulopathy (disseminated intravascular coagulation) or thrombocytopenia may be evident. Organisms may be cultured from blood, although bacteremia or septicemia may not be prominent early in the course of the disease.

**Therapeutic Management**

Treatment of NEC begins with prevention. Oral feedings may be withheld for at least 24 to 48 hours from infants who are believed to have suffered birth asphyxia. Breast milk is the preferred enteral nutrient because it confers some passive immunity (IgA), macrophages, and lysozymes.

Minimum enteral feedings (trophic feeding, GI priming) in VLBW infants have gained acceptance. However, the question as to whether or not trophic feeding increases the incidence of NEC remains unanswered. Some studies have shown that such feedings may be protective against NEC in nonasphyxiated preterm infants (Schanler, Shulman, Lau, et al, 1999; Newell, 2000; Hay, 2008). Some researchers, however, suggest there is insufficient evidence to completely advocate for trophic feedings to prevent NEC (Tyson and Kennedy, 2005; Tyson, Kennedy, Lucke, et al, 2007). A study by Kamitsuka, Horton, and Williams (2000) demonstrated a reduction in NEC by 84% after implementation of a standardized feeding protocol in infants weighing 1250 to 2500 g (2.7 to 5.5 lb) who were less than 35 weeks of gestation.

The role of probiotics such as *Lactobacillus acidophilus* and *Bifidobacterium infantis* administered with enteral feedings for the prevention of NEC has yet to be explored fully enough to advocate widespread use in all VLBW infants. In some studies probiotics decreased the incidence of NEC (Alfaleh, Anabrees, and Bassler, 2009; Bin-Nun, Bromiker, Wilschanski, et al, 2005). There is evidence that human milk may have a protective effect against the development of NEC (Sisk, Lovelady, Gruber, et al, 2007). The administration of maternal antenatal steroids may prevent NEC in some infants by promoting early gut closure and maturation of the gut barrier mucosa (Thompson and Bizzarro, 2008).

Medical treatment of confirmed NEC consists of discontinuation of all oral feedings; institution of abdominal decompression via nasogastric suction; administration of IV antibiotics; and correction of extravascular volume depletion, electrolyte abnormalities, acid-base imbalances, and hypoxia. Replacing oral feedings with parenteral fluids decreases the need for oxygen and circulation to the bowel. Serial abdominal radiograph films (every 4 to 6 hours in the acute phase) are taken to monitor for possible progression of the disease to intestinal perforation.

With early recognition and treatment, medical management is increasingly successful. If there is progressive deterioration...
under medical management or evidence of perforation, surgical resection and anastomosis are performed. Extensive involvement may necessitate surgical intervention and establishment of an ileostomy, jejunalostomy, or colostomy. Sequela of surviving infants include short-bowel syndrome, colonic stricture with obstruction, fat malabsorption, and failure to thrive secondary to intestinal dysfunction. Various surgical interventions for NEC are available and depend on the extent of bowel necrosis, associated illness factors, and infant stability. Intestinal transplantation has been successful in some former preterm infants with NEC-associated short-bowel syndrome who had already developed life-threatening complications related to total parenteral nutrition. More than 50% of these patients survived with improved quality of life. Bowel lengthening procedures and intestinal transplantation may be lifesaving options for infants who previously faced high morbidity and mortality (Nucci, Burns, Armah, et al, 2008). Animal research is now under way using tissue-engineered small intestine as a possible lifesaving treatment for short-bowel syndrome (Guner, Chokshi, Petrosyan, et al, 2008).

**Nursing Care Management**

The nurse is a key factor in the prompt recognition of the early warning signs of NEC. When the disease is suspected, the nurse assists with diagnostic procedures and implements the therapeutic regimen. Vital signs, including blood pressure, are monitored for changes that might indicate bowel perforation, sepsis, or cardiovascular shock, and measures are instituted to prevent possible transmission to other infants. It is especially important to avoid rectal temperatures because of the increased danger of perforation. To avoid pressure on the distended abdomen and to facilitate continuous observation, infants are often left undiapered and positioned supine or on the side.

**HIGH RISK RELATED TO CARDIOVASCULAR AND HEMATOLOGIC COMPLICATIONS**

**PATENT DUCTUS ARTERIOSUS**

PDA is a common complication of severe respiratory disease in preterm infants. It occurs in the majority of preterm infants under 1200 g (2.6 lb), and the incidence diminishes in direct relationship to increasing birth weight. During fetal life the ductus remains patent through the vasodilatory action of prostaglandin, which is produced by the placenta and circulated to the fetus. Postnatally the increase in oxygen tension has a constricting effect on the ductus, but it may reopen in preterm infants in response to the lowered oxygen tension associated with respiratory impairment.

Lack of ductal smooth muscle in preterm infants also prolongs patency of the ductus arteriosus. Functional closure occurs usually within 3 to 4 days, but complete anatomic closure with fibrosis and permanent sealing of the lumen may take up to 2 to 3 weeks.

**Clinical Manifestations**

Signs of PDA may appear within the first week of life. Early signs are increased PaCO$_2$, decreased PaO$_2$, increased FiO$_2$, increased work of breathing, and recurrent apnea. Other signs include bounding peripheral pulses; wide pulse pressure with decreased diastolic blood pressure; pericardial hyperactivity; cardiomegaly; and a systolic or continuous murmur usually referred to as a “machinery-type” murmur, heard loudest in systole. If the PDA is wide open, a murmur may not be heard. Spontaneous closure usually occurs within 12 weeks, but in infants with severe lung involvement, the left-to-right shunting of blood leads to pulmonary edema and may prevent timely weaning from mechanical ventilation. The diagnosis is confirmed by echocardiography.

**Therapeutic Management**

Therapy consists of careful fluid regulation; respiratory support; and administration of indomethacin or ibuprofen, which inhibit prostaglandin synthetase inhibitor. However, indomethacin inhibits platelet function and affects renal function in neonates, so close monitoring for bleeding and renal dysfunction is necessary if this drug is used. If a ductus reopens after cessation of therapy, readministration of the medication may produce a favorable response; as many as four doses may be used to accomplish ductal closure. Surgical ligation may be necessary if medical therapy is unsuccessful, since ductal shunting is perceived as an important contributor to respiratory distress and BPD.

**Nursing Care Management**

Nursing observations are important in the recognition and management of PDA. Assisting in early detection, carefully
assessing cardiovascular status, and monitoring for complications after implementation of therapy are nursing responsibilities. Activities related to therapy include collection of specimens for laboratory examination, continued assessment of renal function (adequate urinary output, any abnormal laboratory findings such as blood urea nitrogen and creatinine levels), and observation for any bleeding tendencies (Hematest-positive stools or gastric aspirate, oozing from heel sticks or venipuncture sites, and laboratory evidence of clotting abnormalities).

Postoperative care includes monitoring for pneumothorax or atelectasis on the affected side, assessment for bleeding and signs or symptoms of infection, supportive respiratory care, and pain management. Other nursing observations and management are the same as for the high-risk infant and the infant with congenital heart disease. (See Chapter 34.)

ANEMIA

Preterm infants tend to develop anemia that is more severe and appears earlier than in more mature infants. It may be a result of hemorrhage during pregnancy or labor and delivery (loss of placental integrity, anomalies of the umbilical cord, fetomaternal hemorrhage), hemorrhage during the neonatal period (ICH, visceral trauma), or blood disorders (hemolytic disease, thrombocytopenia). Anemia may also be iatrogenic from blood withdrawn in the NICU for laboratory tests. Physiologic characteristics of prematurity tend to contribute to the development of anemia (i.e., a decreased red blood cell mass at birth, a drop in the production of hemoglobin, and shortened survival time of red blood cells). This lag in hematopoiesis during continued growth results in physiologic anemia, probably as a consequence of diminished erythropoietin values.

Fortunately, even VLBW infants are able to accommodate the GI absorption of iron required for their high needs. Iron is supplied in iron-fortified formulas or iron supplements as both a preventive and therapeutic measure. Transfusions with packed red blood cells are often required for severe anemia, usually for replacement of blood loss from iatrogenic measures. At 4 to 12 weeks of age, “physiologic anemia” reaches a peak, at which time infants sometimes display signs that suggest true anemia.

Nursing Care Management

One of the most common causes of anemia in acutely ill preterm infants is blood loss associated with frequent sampling for blood gas and metabolic analyses. Therefore an important nursing responsibility is careful monitoring and recording of all blood drawn for tests. It is surprising how easily and rapidly the small total blood volume of preterm infants is depleted by repeated withdrawals. In light of hepatitis and HIV transmission and the potential for other blood-borne pathogens, measures to reduce iatrogenic blood loss and to minimize the need for transfusions of blood products is an important consideration.

Observation for signs of anemia is a vital nursing function. The signs of anemia in the preterm infant are poor feeding, decreased oxygen saturation, systolic murmur, dyspnea, tachycardia, tachypnea, diminished activity, and pallor. However, some infants may not display all these signs. Poor weight gain may be an indication of a lowered hemoglobin level. (Chapter 35 discusses nursing precautions and observations during blood transfusion.)

POLYCYTHEMIA

The current definition of polycythemia is a venous hematocrit of 65% or more (Sarkar and Rosenkrantz, 2008). With a hematocrit above 65%, blood flow becomes increasingly sluggish and hyperviscous, resulting in hypoperfusion of organs. Polycythemia may result from in utero twin-to-twin transfusion and maternal-fetal transfusion, delayed cord clamping or stripping of the umbilical cord, maternal diabetes, or intrapartum asphyxia. The small-for-gestational-age infant is the most at risk for polycythemia; increased red blood cell consumption of glucose further predisposes the infant to hypoglycemia. Infants with polycythemia have a high incidence of cardiopulmonary distress symptoms (PPHN, cyanosis, and apnea), seizures, hyperbilirubinemia, and GI abnormalities.

Appropriate therapy for correcting metabolic disturbances (e.g., hypoxia, hypoglycemia, and hyperbilirubinemia) is implemented. Lowering blood viscosity by partial plasma exchange transfusion may be considered in symptomatic cases.

Nursing Care Management

Nursing care involves watching for signs of polycythemia (e.g., plethora, peripheral cyanosis, respiratory distress, lethargy, jitteriness or seizure activity, hypoglycemia, hyperbilirubinemia) and assisting with diagnostic tests and therapeutic procedures. (Care of the infant with hyperbilirubinemia is discussed in Chapter 9.)

RETINOPATHY OF PREMATURENESS

Although often discussed in relation to respiratory dysfunction, retinopathy of prematurity (ROP) is a disorder involving immature retinal vasculature. Formerly known as retrolental fibroplasia, ROP is a term used to describe retinal changes observed in preterm infants. The incidence and severity of the disease correlates with the degree of the infant’s maturity—the younger the gestational age, the greater the likelihood of the development of ROP, with extremely preterm infants being the group most at risk. However, cases have been documented of ROP in full-term infants who received no oxygen therapy (Korones, 2003).

In addition to immaturity, numerous factors have been implicated in the etiology of ROP, including hyperoxemia and hypoxemia, hypercarbia and hypocarbia, PDA, apnea, intralipid administration, IVH, infection, vitamin E and A deficiency, prenatal infection, exposure to light, and genetic factors (Askin and Diehl-Jones, 2009b). Previously considered an iatrogenic disease related to hyperoxia, ROP is now believed to be a complex disease of prematurity with multiple causes and therefore difficult to completely prevent.

Pathophysiology

Severe vascular constriction in the immature retinal vasculature, followed by hypoxia in those areas, is characteristic of ROP. This appears to stimulate vascular proliferation of retinal capillaries into the hypoxic areas, where veins become
Numerous and dilate. As new vessels multiply toward the lens, the aqueous humor and vitreous humor become turbid. The retina becomes edematous, and hemorrhages and scarring occur, which separates the retina from its attachment. This extensive retinal detachment and scarring result in irreversible blindness.

**Diagnostic Evaluation**

A system of classification has been established to describe the location and extent of the developing vasculature involved (International Committee for the Classification of Retinopathy of Prematurity, 2005). Normal vascular growth proceeds in an orderly fashion from the optic disc toward the ora serrata, the irregular anterior margin of the retina. Box 10-10 outlines the stages of ROP. ROP is further classified by location of damage in the retina and by the extent of abnormally developing vascularization. U.S. guidelines recommend that all infants with a birth weight of less than 1500 g (3.3 lb) or a gestational age of less than 32 weeks, and selected infants who are believed to be at high risk, undergo ROP screening (American Academy of Pediatrics, American Association for Pediatric Ophthalmology and Strabismus, and American Academy of Ophthalmology, 2006). The frequency of follow-up examination is determined by the ophthalmologist and is outlined in the 2006 recommendations. With increased survival of extremely preterm infants, most authorities agree that the incidence of ROP is not likely to decrease until definitive causative factors are identified.

**Therapeutic Management**

Studies have demonstrated an association between the development of ROP and high arterial oxygen saturations in ELBW and VLBW infants. Fluctuations in arterial oxygen saturation in the first few weeks of life have also been implicated in the development of ROP. Although there is no consensus on the ideal arterial oxygen saturation in preterm infants—to prevent either hypoxemia or hyperoxemia—evidence is mounting that oxygen saturations of 100% are undesirable and may have a significant role in the development of ROP in preterm infants. Further studies are needed to clarify optimal arterial oxygen saturation (Pollan, 2009). Therefore the management and treatment of ROP are primarily aimed at preventing fluctuations in arterial concentrations of oxygen in preterm neonates. Studies also indicate that decreasing ambient light exposure in preterm infants did not decrease the incidence of ROP (Phelps and Watts, 2001).

The early recognition of ROP, treatment, and follow-up care are essential components of disease management. Although prevention is the primary goal of therapeutic management, treatment of retinal pathologic conditions is directed toward arresting the proliferation process. Early treatment of high-risk prethreshold ROP significantly reduced unfavorable outcomes when evaluated at a corrected age of 9 months (Jones, MacKinnon, Good, et al, 2005). Cryotherapy ablation of the avascular retina and laser photocoagulation therapy are the most effective treatments for ROP. Laser photocoagulation is reported to be more effective than cryotherapy, and some studies indicate that early laser treatment produces better outcomes (Drenser and Capone, 2008).

Recently there has been increased interest in the administration of an antivascular endothelial growth factor (anti-VEGF) drug bevacizumab, which arrests the proliferation of vessels and prevents retinal detachment commonly seen in ROP. If successful this therapy may preclude the use of laser therapy (Mintz-Hittner and Best, 2009).

**Nursing Care Management**

The nursing care of extremely preterm infants and those at risk for development of ROP should focus on decreasing or avoiding events known to cause fluctuations in systemic blood pressure and oxygenation. The infant’s oxygenation status should be carefully monitored and targeted SpO2 ranges maintained for each infant. Individualized care of the preterm infant is essential to aid in further decreasing the incidence of ROP.

Intraoperative nursing care for the infant undergoing either cryotherapy or laser surgery involves proper infant identification, stabilization and monitoring of vital signs as required, monitoring of IV therapy, and administration of the necessary medications. Postoperative nursing care also includes monitoring the infant for signs of pain and appropriate pain management as needed. After surgery the infant’s eyelids will be edematous and closed; the nurse informs the parents of this preoperatively. Eye medications are administered as needed, and the infant’s tolerance of these medications is monitored closely. Most infants are able to bottle- or breast-feed once awake and alert in the postoperative period. When the infant suffers partial or complete visual impairment, the parents need a considerable amount of support and assistance in meeting his or her special developmental needs. (See Chapter 24.)

**HIGH RISK RELATED TO Neurologic Disturbance**

Neurologic complications are observed with increased frequency in preterm infants and in infants born after a difficult labor and delivery. A disproportionately high incidence of perinatal encephalopathy and psychomotor delay occurs in the high-risk infant population, especially ELBW and VLBW infants. Preterm infants are also more vulnerable to cerebral insults (e.g., hypoxia) and chemical alterations (e.g., decreased blood glucose). In addition, fragility and increased permeability of capillaries and prolonged prothrombin time predispose the...
Preterm infant’s brain to trauma when delicate structures are subjected to increased pressure, such as the forces of labor, high ventilatory pressures, fluid and electrolyte imbalances, sepsis, acidosis, and seizure activity. All these factors contribute to intracranial insults, including traumatic bleeding in the newborn, which consists of four major types: intraventricular, subdural, primary subarachnoid, and intracerebellar.

**PERINATAL HYPOXIC-ISCHEMIC BRAIN INJURY**

Hypoxic-ischemic brain injury, or hypoxic-ischemic reperfusion injury, is the most common cause of neurologic impairment observed in term and preterm infants. The brain damage usually results from asphyxia before, during, or after delivery. Ischemia and hypoxemia may occur simultaneously, or one may precede the other. The fetal brain is somewhat protected against mild hypoxic events but may be damaged when there is a decrease in cerebral blood flow, systemic blood pressure, and oxygen and nutrients such as glucose. Subsequent reperfusion after the event may further result in bleeding of the fragile capillaries and tissue ischemia.

**Hypoxic-ischemic encephalopathy (HIE)** is the resultant cellular damage from hypoxic-ischemic injury that causes the clinical manifestations observed in each case. Such clinical manifestations are variable and may be mild, moderate, or severe. In some infants little or no residual damage may be observed. In general, hypoxia that is severe enough to cause HIE will also damage other organs such as the liver, kidneys, myocardium, and GI tract (Verklan, 2009; Hanks, Koen, Gei, et al, 2002). In the preterm infant HIE may occur in conjunction with IVH. As a consequence of prematurity and general organ and system immaturity, the preterm infant may also suffer hypoxic-ischemic brain damage in the neonatal period as a result of altered cerebral blood flow, systemic hypotension, and decreased cellular nutrients (blood glucose and oxygen).

The site of the hypoxic-ischemic injury varies according to the infant’s gestational age. In the full-term infant the primary ischemic damage is parasagittal cerebral injury with cortical necrosis (deeper region of the brain). In the preterm infant the primary ischemic lesion is in the white matter near the ventricles, or periventricular, with resultant periventricular leukomalacia (Volpe, 2008).

**Clinical Manifestations**

The neurologic signs of encephalopathy appear within the first hours after the hypoxic episode, with manifestations of bilateral cerebral dysfunction. The infant may be stuporous or comatose. Seizures begin after 6 to 12 hours in approximately 50% of the infants, and they become more frequent and severe by 12 to 24 hours. Between 24 and 72 hours the level of consciousness may deteriorate, and after 72 hours persistent stupor, abnormal tone (usually hypotonia), and evidence of disturbances of sucking and swallowing may occur. Muscular weakness of the hips and shoulders occurs in full-term infants, and lower limb weakness occurs in preterm infants. Apneic episodes happen in approximately 50% of the affected infants.

Improvement in the neurologic deficiencies is highly variable and difficult to predict. Infants who demonstrate the most rapid initial improvement appear to have the best prognosis. Myocardial failure and acute tubular necrosis are frequent complications. The major long-term sequelae of hypoxic-ischemic injury are cognitive impairment, seizures, and CP.

**Therapeutic Management**

Treatment involves aggressive resuscitation at birth, supportive care to provide adequate ventilation and avoid aggravating the existing hypoxia, and measures to maintain cerebral perfusion and prevent cerebral edema. Recent research has shown that therapeutic hypothermia provided by either cooling the infant’s head or the whole body reduces the severity of the neurologic damage when it is applied in the early stages of injury (first 6 hours after delivery) (Azzopardi, Strohm, Edwards, et al, 2009; Edwards and Azzopardi, 2006; Jacobs, Hunt, Tarnow-Mordi, et al, 2007; Laptook, 2009). Seizures are managed as described on p. 371. However, prevention is the most important therapy, and every effort should be made to recognize high-risk pregnancies, monitor the fetus, and initiate appropriate therapy early.

**Nursing Care Management**

Nursing care is primarily the same as for any high-risk infant: careful assessment and observation for signs that might indicate cerebral hypoxia or ischemia; monitoring of ventilatory and IV therapy; observation and management of seizures; and general supportive care to infants and parents, including guidelines for management in the event of cognitive impairment. During therapeutic hypothermia, the nurse directs care toward careful regulation of the infant’s body temperature according to the parameters in the cooling protocol being used. The protocol also directs the frequency of blood work, vital signs, and other parameters to be monitored such as a continuous brain wave recording. (See Chapter 24.)

**INTRAVENTRICULAR HEMORRHAGE**

Germinal matrix–intraventricular hemorrhage is known by a variety of terms according to the locus of bleeding: intraventricular hemorrhage, periventricular hemorrhage, and subependymal-intraventricular hemorrhage. Most authorities use the term intraventricular hemorrhage (IVH) to describe this disorder, which is responsible for a significant percentage of seriously ill infants and neonatal mortality. The incidence of IVH ranges from 20% to 25% of VLBW infants (McCrea and Ment, 2008). IVH is extremely common in preterm infants, especially ELBW and VLBW infants less than 32 weeks of gestation; the degree of neonatal immaturity correlates with the incidence of hemorrhage, and subsequent neurologic handicap is not uncommon.

**Pathophysiology**

During the early months of prenatal development an extensive but fragile vascular network in the region of the ventricles receives a disproportionately large amount of cerebral blood flow. Blood is directed to the germinal matrix located in the periventricular region near the caudate nuclei of the cerebrum. Therefore preterm infants are subject to bleeding in this heavily vascularized region, especially during events that are likely to cause fluctuations in cerebral blood flow, such as hypoxic epi-
sodes and the associated increased venous pressure. In IVH the bleeding originates in these capillaries. The blood may rupture through the ependymal lining of the ventricles and fill all or part of the ventricular system. In severe cases the hemorrhage extends into the cerebral parenchyma. Bleeding in the cerebral parenchyma may lead to the development of cystic lesions referred to as periventricular leukomalacia, which is a significant risk factor for CP. Table 10-9 lists the classification of degrees of IVH.

Following bleeding in the ventricle, clots and other debris can obstruct the passages between the ventricles, causing the ventricles to dilate and resulting in the development of hydrocephalus. Several clinical features are associated with IVH, such as birth asphyxia, early gestational age, LBW, respiratory distress, asynchronous breathing on ventilatory therapy, pneumothorax, low blood glucose, noxious stimulation, hypercarbia, coagulation and platelet disorders, and hypotension. Posthemorrhagic hydrocephalus and damage to the periventricular white matter of the brain (such as in grade III+) are major determinants of associated chronic problems and prognosis.

**Clinical Manifestations**

Volpe (2008) classifies clinical manifestations of IVH into three categories:

1. **Catastrophic deterioration**—Begins within minutes to hours of the insult with a coma or deep stupor, respiratory abnormalities such as apnea and hypoventilation, fixed pupils, decerebrate posturing, generalized tonic seizures, flaccid quadriplegia, and cardiac arrhythmias
2. **Saltatory deterioration**—More subtle; signs appear over several hours, may stop altogether, then reappear; signs consist of altered level of consciousness, hypotonia, subtle abnormal eye position and movements, decreased spontaneous or abnormal movements and an abnormally tight popliteal angle; respiratory abnormalities observed in some cases
3. **Clinically silent deterioration**—Often overlooked clinically, but a sudden unexplained decrease in hematocrit may be the only clinical sign of IVH

Approximately 50% of all IVHs occur on the first postnatal day of life, 25% on the second, 15% on the third, and 10% on or after the fourth day of life (Volpe, 2008).

**Diagnostic Evaluation**

When IVH is suspected or the infant is at risk, studies of intracranial structures are performed by ultrasonography, computed tomography (CT), or magnetic resonance imaging (MRI). In many NICUs screening with cranial ultrasonography is performed at the bedside (via the anterior fontanel) within hours of birth if there is suspicion of IVH or within 4 to 7 days for high-risk infants (<32 weeks of gestation). A positron emission tomography scan may also be helpful in identifying cerebral blood flow in and around the site of the hemorrhage.

**Therapeutic Management**

The treatment of IVH is aimed at prevention, particularly of prematurity and any events that may lead to IVH. The maintenance of adequate oxygenation by decreasing iatrogenic events is the key to keeping ELBW and VLBW infants neurologically intact. A number of factors associated with prematurity and RDS may predispose the preterm infant to IVH; these factors include acidosis, electrolyte imbalances and rapid fluid shifts (extracellular to intracellular), administration of hyperosmolar solutions (such as sodium bicarbonate), and hypotension followed by rapid volume expansion. Medical treatment aimed at preventing IVH with vitamin E, maternal vitamin K, pancuronium (to decrease blood pressure fluctuations), ibuprofen, phenobarbital, ethamsylate, magnesium sulfate, indomethacin, and surfactant (for RDS) has met with varying degrees of success. Antenatal betamethasone administration has played a significant role in the reduction of IVH in preterm infants (Volpe, 2008).

In the event of IVH, treatment is both preventive and supportive; prompt detection by clinical signs or periodic ultrasonography is a key element in implementing strategies to prevent further damage. Posthemorrhagic hydrocephalus is a common occurrence within 1 month of the event. Serial lumbar punctures may be used to decrease the amount of CSF and thus decrease ventricular size. A closed reservoir may be attached to an intraventricular shunt, with the reservoir tapped or drained intermittently to relieve pressure on the ventricles. Ventricular dilatation (grade III to grade III+) may be managed with shunting (ventriculoperitoneal or subgaleal) or a temporary external ventricular drainage.

The long-term outcome of IVH is variable and unpredictable and is influenced by the size of the hemorrhage and the extent of parenchymal involvement. Infants with small lesions have an excellent prognosis for neurologic outcome (Hill, 2005).

**Nursing Care Management**

In addition to routine observations and management, the nurse also directs care toward prevention of fluctuations in cerebral blood flow. It has been observed that some nursing procedures increase intracranial pressure. For example, blood pressure increases significantly during ET succioning in preterm infants, and head positioning produces measurable changes in intracranial pressure. Researchers have found that intracranial pressure is highest when infants are in the dependent position and decreases when the head is in a midline position and elevated 30 degrees.
Cerebral pressure is lower when infants are in a midline position as opposed to a right side-lying position. When the head is turned to the right without body alignment, the resulting venous congestion creates hydrostatic pressure fluctuations that increase intracranial pressure. Infants encumbered with tubes and monitoring equipment are more difficult to turn while maintaining head-body alignment.

Other interventions that may reduce the risk of increased intracranial pressure include avoiding interventions that cause crying (such as painful procedures). Crying (which essentially creates a Valsalva effect) can impede venous return, increase cerebral blood volume, and compromise cerebral oxygenation in LBW infants. Avoid rapid volume expansion following hypotension (primarily in preterms) and administration of hyperosmolar solutions such as sodium bicarbonate. Because air leaks such as pneumothorax produce variable cerebral blood flow, rapid detection and intervention are a key component of nursing care of the high-risk infant. Monitoring serum blood glucose levels and preventing hypoglycemia are also important factors in keeping the infant neurologically intact. Many units practice minimum handling of infants at high risk to avoid fluctuations in cerebral blood flow. In addition, research has implicated noxious external stimuli (e.g., pain and noise) as having a potential role in stimulation that may lead to IVH. Care includes evaluating manipulations and handling and administering analgesics to reduce discomfort.

**INTRACRANIAL HEMORRHAGE**

ICH in neonates, although manifested in the same ways as those described in older children, occurs with different frequencies and different degrees of severity.

**Subdural Hemorrhage**

A subdural hematoma is a life-threatening collection of blood in the subdural space. The stretching and tearing of the large veins in the tentorium cerebelli, the dural membrane that separates the cerebrum from the cerebellum, is the most common cause. With improved obstetric care this condition has become relatively uncommon; however, it is especially serious because of the inaccessibility of the hematoma to aspiration by subdural tap. Less commonly, hemorrhage occurs when veins in the subdural space over the surface of the brain are torn. (See Head Injury, Chapter 37.)

**Subarachnoid Hemorrhage**

Subarachnoid hemorrhage, the most common type of ICH, occurs in full-term infants as a result of trauma and in preterm infants as a result of the same types of events that cause IVH. Small hemorrhages are the most common. Bleeding is of venous origin, and underlying contusion may also occur.

**Intracerebellar Hemorrhage**

Intracerebellar hemorrhage is a common finding on postmortem examination of the preterm infant and can be a primary hemorrhage in the cerebellum associated with skull compression during abrupt, precipitous delivery, or it may occur secondary to extravasation of blood into the cerebellum from a ventricular hemorrhage. In the full-term infant the bleeding may follow a difficult delivery.

**Nursing Care Management**

Nursing care is the same as care of the infant with IVH or with perinatal hypoxic-ischemic brain injury.

**NEONATAL/PERINATAL STROKE**

Neonatal stroke is reported to occur in 1 in 4000 live term births (Nelson and Lynch, 2004). Neonatal stroke has been defined to encompass all ischemic and hemorrhagic events that affect the venous and arterial distribution of blood supply from early gestation to the first 28 days of life (Golomb, Cvijanovich, and Ferriero, 2006). Perinatal stroke refers to strokes that occur between 28 weeks of gestation and the first 7 days of life, primarily as a result of altered arterial blood flow and ischemia (Nelson and Lynch, 2004). Fetal stroke may occur as early as 8 weeks’ gestation (Kirtton and deVeber, 2009).

Neonatal stroke is the second leading cause of seizures in term neonates and may be caused by arterial, thrombotic, or ischemic events that result in altered brain blood flow and infarction. Neonatal stroke is more predominant in males, and there is an increased tendency toward left-sided involvement. Known risk factors for neonatal and perinatal stroke include the presence of maternal and/or fetal factor V Leiden, antiphospholipid, and prothrombin factors (Curry, Bhullar, Holmes, et al, 2007; Simchen, Goldstein, Lubetsky, et al, 2009). Cerebral palsy, motor deficits, epilepsy, and language deficits, and visual deficits may occur as a result of neonatal stroke (Kirtton and deVeber, 2009).

Diagnosis with MRI and venography is most accurate because head ultrasonography may be negative with an ischemic event; the electroencephalogram may be normal (Golomb, Cvijanovich, and Ferriero, 2006).

Because neonatal stroke can only be diagnosed retrospectively, it is important for the nurse to be vigilant for apnea or seizure activity in the first year of life, the time when clinical manifestations will appear.

**NEONATAL SEIZURES**

Seizures in the neonatal period are usually the clinical manifestation of a serious underlying disease. The most common cause of seizures in the neonatal period (for term and preterm infants) is HIE secondary to perinatal asphyxia (Volpe, 2008). Although not life threatening as an isolated entity, seizures constitute a medical emergency because they signal a disease process that may produce irreversible brain damage. Consequently, it is imperative to recognize a seizure and its significance so that the cause, as well as the seizure, can be treated (Box 10-11).

**Pathophysiology**

The features of neonatal seizures are different from those observed in the older infant or child. For example, the well-organized, generalized tonic-clonic seizures seen in older children are rare in infants, especially preterm infants. The newborn brain, with its immature anatomic and physiologic status and reduced cortical organization, is developmentally insufficient to
allow ready development and maintenance of a generalized seizure. The advanced degree of development of limbic structures with connections to the diencephalon and brainstem probably accounts for the higher frequency of seizure manifestations (such as oral movements, oculomotor deviations, and apnea) that originate in these structures.

**Clinical Manifestations**

Seizures in newborns may be subtle and barely discernible or grossly apparent. Because most neonatal seizures are subcortical, they do not have the etiologic and prognostic significance of seizures in children. The type of seizure is seldom important because one may produce any of a variety of manifestations. Neonatal seizures can be divided into four major types: clonic, tonic, myoclonic, and subtle seizures. Table 10-10 lists these classifications in order of frequency (Volpe, 2008). Clonic, multifocal clonic, and migratory clonic seizures are more common in full-term infants.

**BOX 10-11 CAUSES OF NEONATAL SEIZURES**

<table>
<thead>
<tr>
<th>Metabolic</th>
<th>Toxic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoglycemia, hyperglycemia</td>
<td>Uremia</td>
</tr>
<tr>
<td>Hypernatremia, hypotension</td>
<td>Bilirubin encephalopathy (kernicterus)</td>
</tr>
<tr>
<td>Hypocalcemia, hypomagnesemia</td>
<td>Prenatal Infections</td>
</tr>
<tr>
<td>Pyridoxine deficiency</td>
<td>Toxoplasmosis</td>
</tr>
<tr>
<td>Aminoacidurias (e.g., phenylketonuria, maple syrup urine disease)</td>
<td>Syphilis</td>
</tr>
<tr>
<td>Hyperammonemia</td>
<td>Cytomegalovirus</td>
</tr>
<tr>
<td></td>
<td>Herpes simplex</td>
</tr>
<tr>
<td></td>
<td>Hepatitis</td>
</tr>
<tr>
<td>Postnatal Infections</td>
<td>Trauma at Birth</td>
</tr>
<tr>
<td>Bacterial meningitis</td>
<td>Hypoxic brain injury</td>
</tr>
<tr>
<td>Viral meningoencephalitis</td>
<td>Intracranial hemorrhage</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Subarachnoid, subdural hemorrhage</td>
</tr>
<tr>
<td>Brain abscess</td>
<td>Intraventricular hemorrhage</td>
</tr>
<tr>
<td>Malformations</td>
<td>Myoclonic</td>
</tr>
<tr>
<td>Central nervous system agenesis</td>
<td>Rapid jerks that involve flexor muscle groups</td>
</tr>
<tr>
<td>Hydranencephaly</td>
<td>Focal</td>
</tr>
<tr>
<td>Panencephaly</td>
<td>Involves upper extremity flexor muscle group</td>
</tr>
<tr>
<td>Tuberous sclerosis</td>
<td>Multifocal</td>
</tr>
<tr>
<td></td>
<td>Asynchronous twitching of several parts of the body</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Generalized</td>
</tr>
<tr>
<td>Degenerative disease</td>
<td>Bilateral jerks of upper and lower limbs</td>
</tr>
<tr>
<td>Benign familial neonatal seizures</td>
<td>Associated with EEG discharges observed</td>
</tr>
<tr>
<td>Narcotic withdrawal</td>
<td>Stroke (fetal, perinatal, or neonatal)</td>
</tr>
</tbody>
</table>

**TABLE 10-10 CLASSIFICATIONS OF NEONATAL SEIZURES**

<table>
<thead>
<tr>
<th>TYPE</th>
<th>CHARACTERISTICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clonic</td>
<td>Slow, rhythmic jerking movements</td>
</tr>
<tr>
<td></td>
<td>Approximately 1-3/sec</td>
</tr>
<tr>
<td>Focal</td>
<td>Involves face, upper or lower extremities on one side of body</td>
</tr>
<tr>
<td></td>
<td>May involve neck or trunk</td>
</tr>
<tr>
<td></td>
<td>Infant is conscious during event</td>
</tr>
<tr>
<td>Multifocal</td>
<td>May migrate randomly from one part of the body to another</td>
</tr>
<tr>
<td></td>
<td>Movements may start at different times</td>
</tr>
<tr>
<td>Tonic</td>
<td>Extension, stiffening movements</td>
</tr>
<tr>
<td>Generalized</td>
<td>Extension of all four limbs (similar to decerebrate rigidity)</td>
</tr>
<tr>
<td></td>
<td>Upper limbs maintained in a stiffly flexed position</td>
</tr>
<tr>
<td></td>
<td>(resembles decorticate rigidity)</td>
</tr>
<tr>
<td>Focal</td>
<td>Sustained posturing of a limb</td>
</tr>
<tr>
<td></td>
<td>Asymmetric posturing of trunk or neck</td>
</tr>
<tr>
<td>Subtle</td>
<td>May develop in either full-term or preterm infants but more common in preterm</td>
</tr>
<tr>
<td></td>
<td>Often overlooked by inexperienced observers</td>
</tr>
<tr>
<td></td>
<td>Signs:</td>
</tr>
<tr>
<td></td>
<td>• Horizontal eye deviation</td>
</tr>
<tr>
<td></td>
<td>• Repetitive blinking or fluttering of the eyelids, staring</td>
</tr>
<tr>
<td></td>
<td>• Sucking or other oral-buccal-lingual movements</td>
</tr>
<tr>
<td></td>
<td>• Arm movements that resemble rowing or swimming</td>
</tr>
<tr>
<td></td>
<td>• Leg movements described as pedaling or bicycling</td>
</tr>
<tr>
<td></td>
<td>• Apnea (common)</td>
</tr>
<tr>
<td></td>
<td>Signs may appear alone or in combination</td>
</tr>
<tr>
<td>Myoclonic</td>
<td>Rapid jerks that involve flexor muscle groups</td>
</tr>
<tr>
<td>Focal</td>
<td>Involves upper extremity flexor muscle group</td>
</tr>
<tr>
<td></td>
<td>No electroencephalogram (EEG) discharges observed</td>
</tr>
<tr>
<td>Multifocal</td>
<td>Asynchronous twitching of several parts of the body</td>
</tr>
<tr>
<td></td>
<td>No associated EEG discharges observed</td>
</tr>
<tr>
<td>Generalized</td>
<td>Bilateral jerks of upper and lower limbs</td>
</tr>
<tr>
<td></td>
<td>Associated with EEG discharges</td>
</tr>
</tbody>
</table>


**Jitteriness** or tremulousness in the newborn is a repetitive shaking of an extremity or extremities that may be observed with crying, may occur with changes in sleeping state, or may be elicited with stimulation. Jitteriness is relatively common in newborns, and in a mild degree may be considered normal during the first 4 days of life. Jitteriness can be distinguished from seizures by several characteristics; jitteriness is not accompanied by ocular movement as are seizures; the dominant movement in jitteriness is tremor, whereas seizure movement is clonic jerking that cannot be stopped by flexion of the affected limb; and jitteriness is highly sensitive to stimulation, whereas seizures are not. Further evaluation is indicated if jittery movements persist beyond the fourth day, if the movements are persistent and prolonged after a stimulus, or if they are easily elicited with minimum stimulus.

A **tremor** is repetitive movements of both hands (with or without movement of legs or jaws) at a frequency of two to five per second and lasting more than 10 minutes. It is common in newborn infants and has a variety of causes, including neurologic damage, hypoglycemia, and hypocalcemia. In most instances tremors are of no pathologic significance.
Diagnostic Evaluation
Early evaluation and diagnosis of seizures are urgent. In addition to a careful physical examination, the pregnancy and family histories are investigated for familial and prenatal causes. Blood is drawn for glucose and electrolyte examination, and CSF is obtained for examination for gross blood, cell count, protein, glucose, and culture. Electroencephalography may help identify subtle seizures but is less helpful in establishing a diagnosis. Other diagnostic procedures, such as CT, ultrasonography, and echoencephalography, may be indicated.

Therapeutic Management
Direct treatment toward the prevention of cerebral damage, correction of metabolic derangements, respiratory and cardiovascular support, and suppression of the seizure activity. The underlying cause is treated (e.g., glucose infusion for hypoglycemia, calcium for hypocalcemia, and antibiotics for infection). If needed, respiratory support is provided for hypoxia, and anticonvulsants may be administered, especially when the other measures fail to control the seizures. Phenytoin is the drug of choice given intravenously or orally and is used if seizures are severe and persistent. Other drugs that may be used are fosphenytoin sodium, phenytoin (Dilantin), and lorazepam.

Nursing Care Management
The major nursing responsibilities in the care of infants with seizures are to recognize when the infant is having a seizure so that therapy can be instituted, to carry out the therapeutic regimen, and to observe the response to the therapy and any further evidence of seizures or other symptomatology. Assessment and other aspects of care are the same as for all high-risk infants. Parents need to be informed of their infant’s status, and the nurse should reinforce and clarify the practitioner’s explanations. The infant’s behaviors need to be interpreted for the parents, and the infant’s responses to the treatment must be anticipated and their significance explained. Encourage parents to visit their infant and perform the parenting activities consistent with the care plan. Seizures are frightening phenomena and generate a great deal of anxiety and fear, and the staff’s concern, which is justifiable, can heighten that anxiety. Providing support and guidance is an important nursing function.

Effects of Diabetes on the Fetus
Hypoglycemia may appear a short time after birth and in IDMs is associated with increased insulin activity in the blood. A standardized definition for neonatal hypoglycemia remains elusive and controversial. At best, authorities agree that reliance on a single numeric value for every clinical situation is inadequate (see Therapeutic Management section). Hypoglycemia in the IDM is related to hyperthyroid and hyperplasia of the pancreatic islet cells, causing a transient state of hyperinsulinism.

High maternal blood glucose levels during fetal life provide a continuous stimulus to the fetal islet cells for insulin production. This sustained hyperglycemia promotes fetal insulin secretion that ultimately leads to excessive growth and deposition of fat, which probably accounts for the infants who are large for gestational age, or macrosomic. When the neonate’s glucose supply is removed abruptly at the time of birth, the continued production of insulin soon depletes the blood of circulating glucose, creating a state of hyperinsulinism and hypoglycemia within 1½ to 4 hours, especially in infants of mothers with poorly controlled diabetes. Precipitous drops in blood glucose levels can cause serious neurologic damage or death. The birth defects observed in IDMs are thought to occur as a result of multifactorial teratogenic factors, rather than hyperglycemia alone (Leguizamon, Igarzabal and Reece, 2007).

Clinical Manifestations
IDMs have a characteristic appearance. They are usually macrosomic for their gestational age, very plump and full faced, liberally coated with vernix caseosa, and plethoric. The placenta and umbilical cord are also larger than average. However, infants of mothers with advanced diabetes may be small for gestational age, have IUGR, or be appropriate for gestational age because of the maternal vascular (placental) involvement. IDMs have an increased incidence of hypoglycemia, hypocalcemia, hyperbilirubinemia, hypomagnesemia, and RDS. Hyperglycemia in the diabetic mother and subsequent fetal hyperinsulinemia may be a factor in reducing fetal surfactant synthesis, thus contributing to the development of RDS.
Morbidities in IDMs are the result of exposure to elevated glucose and ketone levels, placental insufficiency, and prematurity. Although large, these infants may be delivered before term because of maternal complications or increased fetal size.

**Therapeutic Management**

The most effective management of IDMs is careful monitoring of serum glucose levels and observation for accompanying complications such as RDS. Examine these infants for any anomalies or birth injuries, and regularly obtain blood studies for determinations of glucose, calcium, hematocrit, and bilirubin. A common definition of hypoglycemia has not been established. Several authors have suggested the use of operational thresholds at which hypoglycemia should be closely monitored and treated. The researchers recommend close observation in infants with known risk factors such as maternal diabetes and close observation if plasma glucose values are below 45 mg/dl (2.5 mmol/L) (Cornblath, Hawdon, Williams, et al, 2000; Canadian Paediatric Society, 2004; Deshpande and Ward Platt, 2005). If a feeding fails to increase the glucose levels in such cases or if abnormal signs develop, IV glucose should be administered to maintain glucose levels above 45 mg/dl (2.5 mmol/L). A newborn with levels at or below 30 mg/dl should receive IV glucose.

Because the hypertrophied pancreas is so sensitive to blood glucose concentrations, the administration of oral glucose may trigger a massive insulin release, resulting in rebound hypoglycemia. Therefore feedings of breast milk or formula begin within the first hour after birth provided that the infant’s cardiorespiratory condition is stable. Approximately half of IDMs do well and adjust without complications. Infants born to mothers with uncontrolled diabetes may require IV infusion of dextrose. Oral and IV intake may be titrated to maintain adequate blood glucose levels. Frequent blood glucose determinations are needed for the first 2 days of life to assess the degree of hypoglycemia present at any given time. Testing blood taken from the heel with point-of-care portable reflectance meters (glucometer) is a simple and effective screening evaluation that can then be confirmed by laboratory examination.

**Nursing Care Management**

The nursing care of IDMs involves early examination for congenital anomalies and signs of possible respiratory or cardiac problems, maintenance of adequate thermoregulation, early introduction of carbohydrate feedings as appropriate, and monitoring of serum blood glucose levels. The latter is of particular importance because many hypoglycemic infants may remain asymptomatic. IV glucose infusion requires careful monitoring of the site and the neonate’s reaction to therapy; high glucose concentrations (>12.5%) should be infused via a central line instead of a peripheral one. Because macrosomic infants are at risk for problems associated with a difficult delivery, they are monitored for birth injuries such as brachial plexus injury and palsy, fractured clavicle, and phrenic nerve palsy. Additional monitoring of the infant for associated problems (RDS, polycythemia, hypocalcemia, poor feeding, and hyperbilirubinemia) is also a vital nursing function.

There is evidence that IDMs have an increased risk of acquiring metabolic syndrome (obesity, hypertension, dyslipidemia, and glucose intolerance) in childhood or early adulthood; therefore nursing care of IDMs should also focus on healthy lifestyle and prevention later in life (Boney, Verma, Tucker, et al, 2005).

**DRUG-EXPOSED INFANTS***

**Overview**

In the 2002 to 2003 National Survey on Drug Use and Health, 4.3% of pregnant women ages 15 to 44 years reported illicit drug use within the past month (Substance Abuse and Mental Health Services Administration, 2005). Given the self-reporting nature of survey data, it is likely that this number is considerably lower than the actual number of substance-using pregnant women. Determining the effects of intrauterine drug and alcohol exposure is difficult for a variety of reasons. Many substance-using women ingest multiple drugs or a combination of drugs and alcohol, and some women who use drugs or alcohol may be undernourished or suffer from chronic medical conditions. Some may not seek prenatal care; for others, the drugs used may be cut with a variety of materials, and the strength, dose, and duration of exposure are likely to be unknown (Schempf, 2007).

**Clinical Manifestations**

Most infants of drug-dependent mothers appear normal at birth but may begin to exhibit signs of drug withdrawal within 12 to 24 hours, depending on the substance and the mother’s pattern of use. If mothers have been taking methadone, the signs appear somewhat later—anywhere from 1 or 2 days to 2 to 3 weeks or more after birth. The clinical manifestations of withdrawal may fall into one or all of the following categories: CNS, GI, respiratory, and autonomic nervous system signs (Kuschel, 2007). The manifestations become most pronounced between 48 and 72 hours of age and may last from 6 days to 8 weeks (Box 10-12).

In a study of polydrug use during pregnancy the most prominent signs of withdrawal were increased tone, increased respiratory rate, disturbed sleep, fever, excessive sucking, and loose watery stools. Other signs observed included projectile vomiting, mottling, crying, nasal stuffiness, hyperactive Moro reflex,

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*It is important to note that the term addiction is often associated with behaviors whereby the person seeks the drug to experience a high or euphoria, escape from reality, or satisfy a personal need. Newborns are not addicted in a behavioral sense, yet they may experience mild to strong physiologic signs as a result of the mother’s drug use. Therefore to say that an infant born to a mother who uses substances is addicted is incorrect; drug-exposed newborn is a better term, which implies intrauterine drug exposure.
and tremors (D’Apolito and Hepworth, 2001). Although these infants suck avidly on fists and display an exaggerated rooting reflex, they are poor feeders with uncoordinated and ineffectual sucking and swallowing reflexes.

One observation in a large percentage of these infants is generalized perspiring, which is unusual in newborn infants. It is significant that, although drug-exposed infants may have some tachypnea, cyanosis, or apnea, they rarely develop RDS when born near term. Apparently, narcotics or stress factors in the intrauterine environment cause accelerated lung maturation even with a high incidence of prematurity.

Not all infants of narcotic-addicted mothers show signs of withdrawal. Because of irregular and varying degrees of drug use, quality of drug, and mixed drug usage by the mother, some infants display mild or variable manifestations. Most manifestations are the vague, nonspecific signs characteristic of infants in general; therefore it is important to differentiate between drug withdrawal and other disorders before instituting specific therapy. Other states (e.g., hypocalcemia, hypoglycemia, or sepsis) often coexist with the drug withdrawal.

A concern regarding substance abuse is that many of the mothers often use several drugs, such as tranquilizers, nicotine, sedatives, narcotics, amphetamines, phencyclidine (PCP), marijuana, and other psychotropic agents. Of increasing concern in the United States is the number of newborns who are exposed to methamphetamines and selective serotonin reuptake inhibitors in utero.

Therapeutic Management
The treatment of the drug-exposed infant initially consists of modulating the environment to decrease external stimuli. Drug therapies to decrease withdrawal side effects are implemented once neonatal abstinence syndrome (NAS) is identified.

BOX 10-12 SIGNS OF WITHDRAWAL IN THE NEONATE

- I irritability
- Tachypnea (>60 beats/min)
- Tremors
- Excoriations (knees, face)
- Shril cry
- Mottling (skin)
- Hypertonicity of muscles
- Sneezing
- Frantic sucking of hands
- Yawning
- Poor feeding
- Vomiting, often projectile
- Hyperactivity
- Temperature instability
- Perspiring
- Loose diarrhea stools
- Fever
- Seizures
- Nasal stuffiness
- Sleep disturbances

Nursing Care Management
When possible, alert the nursery personnel to the likelihood of a drug-exposed infant requiring admittance. If the mother has had good prenatal care, the practitioner is aware of the problem and substance abuse treatment may have been instituted before delivery. However, a number of mothers deliver their infants without the benefit of adequate care, and the addiction is unknown to health care personnel at the time of delivery. The degree of narcosis or withdrawal is closely related to the amount of drug the mother has habitually taken, the length of time she has been taking the drug, and her drug level at the time of delivery. The most severe symptoms occur in the infants of mothers who have taken large amounts of drugs over a long period. In addition, the nearer to the time of delivery that the mother takes the drug, the longer it takes the child to develop withdrawal, and the more severe the manifestations. The infant may not exhibit withdrawal symptoms until 7 to 10 days after delivery.

Once the presence of NAS is identified in an infant, direct nursing care toward reducing external stimuli that might trigger hyperactivity and irritability (e.g., dimming the lights and decreasing noise levels), providing adequate nutrition and hydration, and promoting positive and nurturing maternal-infant relationships. Providing care on demand rather than on a fixed schedule may help reduce irritability for infants. Appropriate individualized developmental care is implemented, such as care with preterm infants to facilitate self-consoling and self-regulating behaviors (see Table 10-5). Some irritable and hyperactive infants respond to comforting, movement, containment, and close contact. Wrapping infants snugly and rocking and holding them tightly limit their ability to self-stimulate. The infant’s arms should remain flexed with hands close to the mouth for sucking as appropriate; sucking on fingers or hands is a form of self-control and comfort. Arranging nursing activities to reduce disturbances helps decrease exogenous stimulation.

The Neonatal Abstinence Scoring System has been developed to monitor infants in an objective manner and evaluate the infant’s response to clinical and pharmacologic interventions (Finnegan, 1985). This system also assists nurses and other health care workers in evaluating the severity of the infant’s withdrawal symptoms.

Another scoring tool has been recently developed specifically aimed at measuring neurologic behavior and resultant effects on the neonate when substances are used during pregnancy. The NICU Network Neurobehavioral Scale, developed by the National Institutes of Health, provides an assessment of neurologic, behavioral, and stress-abstinence function in the neonate. The test combines items from other tests such as the Neonatal Behavioral Assessment Scale (NBAS); stress-abstinence items developed by Finnegan (1985); and a complete neurologic examination, which includes primitive reflexes and active and passive tone (Law, Stroud, LaGasse, et al, 2003).

Loose stools and poor intake and regurgitation after feeding predispose the infants to malnutrition, dehydration, and electrolyte imbalance. An oral opioid such as morphine may be administered to control loose watery stools (D’Apolito and Hepworth, 2001). It takes considerable time and patience to
ensure that these infants receive a sufficient caloric and fluid intake.

Monitoring and recording the activity level and its relationship to other activities, such as feeding and preventing complications, are important nursing functions.

A valuable aid to anticipating problems in the newborn is recognizing drug abuse in the mother. Unless the mother is enrolled in a methadone rehabilitation program, she seldom risks calling attention to her habit by seeking prenatal care. Consequently, infants and mothers are exposed to the additional hazards of obstetric and medical complications resulting from the lack of adequate prenatal care. Moreover, the nature of heroin addiction makes the user susceptible to disorders such as infection (hepatitis B and HIV related to IV needle use), foreign body reaction, and the hazards of inadequate nutrition and preterm birth. Methadone treatment does not prevent withdrawal reaction in neonates, but the clinical course may be modified. Also, intensive psychologic support of mothers is a factor in the treatment and reduction of perinatal mortality. Experience has indicated that mothers are usually anxious and depressed, lack confidence, have poor self-image, and have difficulty with interpersonal relationships. They may have a psychologic need for the pregnancy and an infant.

Initial symptoms or the recurrence of withdrawal symptoms may develop after discharge from the hospital. Therefore it is important to establish rapport and maintain contact with the family so that they return for treatment if this occurs. The demands of the drug-exposed infant on the caregiver are enormous and unrewarding in terms of positive feedback. The infants are difficult to comfort, and they cry for long periods, which can be especially trying for the caregiver after the infant’s discharge from the hospital. Long-term follow-up to evaluate the status of the infant and family is important.

An important aspect of nursing care is identification of an infant who was exposed to drugs in utero. Observation of signs mentioned previously may warrant further investigation so prompt treatment can be implemented. Newborn urine, rarely hair, or meconium sampling may be required to identify drug exposure and implement appropriate early interventional therapies aimed at minimizing the consequences of intrauterine drug exposure. Meconium sampling for fetal drug exposure provides more screening accuracy than urine, since drug metabolites accumulate in meconium (Kuschel, 2007). Urine toxicology screening has less accuracy because it only reflects recent substance intake by the mother (Huestis and Choo, 2002). Meconium testing for drug metabolites has the advantage of being easy to collect, noninvasive, and more accurate.

Pharmacologic treatment is usually based on the severity of withdrawal symptoms, as determined by an assessment tool. Drug therapies to decrease withdrawal side effects include administration of phenobarbital, morphine, dilute tincture of opium, methadone (Coyle, Ferguson, Lagasse, et al, 2002; Johnson, Gerada, and Greenough, 2003) buprenorphine (Kraft, Gibson, Dysart, et al, 2008), or clonidine (Agthe, Kim, Mathias, et al, 2009). A combination of these drugs may be necessary to treat infants exposed to multiple drugs in utero, and careful attention should be given to possible adverse effects of the treatment drugs (Johnson, Gerada, and Greenough, 2003).

Many problems relate to the disposition of infants of drug-dependent mothers. Those who advocate separation of mothers and children argue that the mothers are not capable of assuming responsibility for their infant’s care, that child care is frustrating to them, and that their existence is too disorganized and chaotic. Others encourage the maternal-infant bond and recommend a protected environment such as a therapeutic community; a halfway house; or continuous ongoing, supportive services in the home after discharge. Careful evaluation and the cooperative efforts of a variety of health professionals are required, whether the choice is foster home placement or supportive follow-up care of mothers who keep their infants.

**Opiate Exposure**

Narcotics, which have a low molecular weight, readily cross the placental membrane and enter the fetal system. When the mother is a habitual user of narcotics, especially heroin or methadone, the unborn child may also become passively physiologically addicted to the drug, which places the infant at risk during the early neonatal period. NAS is the term used by many to describe the set of behaviors exhibited by the infant exposed to chemical substances in utero.

Prescription opioids such as oxycodone (Percodan) have been identified as increasingly popular drugs of abuse, which may cause withdrawal symptoms in neonates (Rao and Desai, 2002). Other chemical substances that may cause neonatal withdrawal include methadone, caffeine, and PCP.

**Methadone Exposure**

Methadone, a synthetic opiate, has been the therapy of choice for heroin addiction since 1965. Methadone crosses the placenta. An increasing number of infants have been born to methadone-maintained mothers, who seem to have better prenatal care and a somewhat better lifestyle than those taking heroin.

Some question exists concerning the benefits of methadone therapy during pregnancy because of its effect on the fetus. Methadone withdrawal resembles heroin withdrawal but tends to be more severe and prolonged. Signs of methadone withdrawal include tremors, irritability, state lability, hypertonicity, hypersensitivity, vomiting, mottling, and nasal stuffiness (Jansson, Velez, and Harrow, 2004). These infants exhibit a disturbed sleep pattern similar to that seen in heroin withdrawal. They have a higher birth weight than those infants in heroin withdrawal, usually appropriate for gestational age. No increased incidence of congenital anomalies is seen. The American Academy of Pediatrics, Committee on Drugs (2001), has revised its statement regarding breast-feeding for mothers who are in a methadone treatment program, suggesting such mothers be allowed to breast-feed regardless of the methadone dosage; follow-up counseling and monitoring of the mother and infant are recommended.

Late-onset withdrawal occurs at age 2 to 4 weeks and may continue for weeks or months. A higher incidence of SIDS also has been reported in these infants (Wagner, Katikaneni, Cox, et al, 1998). This factor is important for perinatal nurses who coordinate follow-up care for the infant and education for the mother or other caregiver. Community health nurses must know about the potential for withdrawal symptoms to occur.
Therapy for methadone withdrawal is similar to that for heroin withdrawal. The few available follow-up studies of these infants reveal a high incidence of hyperactivity, learning and behavior disorders, and poor social adjustment.

**Cocaine Exposure**

Cocaine, a commonly used illicit drug in the United States, has multiple modes of use. However, use of the relatively inexpensive and easily administered “crack” form increased significantly among pregnant women and women of childbearing age in the 1990s (Askin and Diehl-Jones, 2001; Eyler, Behnke, and Conlon, 1998). Because crack vaporizes at relatively low temperatures, it is smoked and absorbed in large quantities through pulmonary vasculature. The drug readily enters the placenta, placing the fetus at risk (Malanga and Kosofsky, 1999).

Cocaine is a CNS stimulant and sympathetic mimetic, and the effects on the fetus may be direct or indirect. Indirect effects include fetal hypoxemia secondary to impaired uterine blood flow. Cocaine also appears to affect fetal cardiac function and suppress the fetal immune system. The difficulties encountered by cocaine-exposed infants are compounded when the mother takes the drug in conjunction with other illicit drugs (Askin and Diehl-Jones, 2001). Studies have found that women who use cocaine in pregnancy are less likely to have adequate prenatal care, are more likely to smoke tobacco and consume alcohol, are more likely to be malnourished, and are more likely to have sexually transmitted infections than nonusers (Tronick and Beeghly, 1999). These variables compound the problem of drug exposure and effects on the fetus.

**Clinical Manifestations**

Infants who are exposed to cocaine in utero may demonstrate no immediate untoward effects. Previous reports of catastrophic neurologic effects have been published, yet the findings have considerable variability because of poor reliability of maternal history, maternal polydrug use, prematurity, poor social environment, and poor specificity in detecting cocaine exposure. A large meta-analysis of 15,208 pregnancies did not find an association between illicit drug use and congenital anomalies (van Gelder, Reefhuis, Caton, et al, 2009). It may be, however, that habitual cocaine use in pregnancy has negative effects that are too subtle to notice in the newborn and infancy period (Askin and Diehl-Jones, 2001).

Clinical manifestations of intrauterine cocaine exposure include IUGR, decreased head circumference, association with preterm delivery, NEC, cerebral infarcts, respiratory disturbances such as apnea, cardiac arrhythmias, transient electroencephalogram abnormalities, and IVH (Askin and Diehl-Jones, 2001; Chiriboga, Brust, Bateman, et al, 1999). Other findings related to neurobehavioral effects include sleep disturbances; increased tone; jitteriness; delayed language acquisition; behavior problems in school; poor impulse control; hypertonia; abnormal reflexes; poor NBAS scores; significant cognitive delays in the first 2 years; and poor responses to auditory, arousal, and visual stimuli (Chiriboga, Kuhn, and Wasserman, 2007; Chiriboga, Brust, Bateman, et al, 1999; Delaney-Black, Covington, Templin, et al, 1998; Eyler, Behnke, and Conlon, 1998; Schuler and Nair, 1999; Singer, Minnes, Short, et al, 2004; Levine, Liu, Das, et al, 2008). Environmental and sociodemographic factors likely play an important role in the outcome of children exposed to cocaine in utero.

**Therapeutic Management**

Infants exposed to cocaine alone are less likely than other drug-exposed infants to demonstrate signs of withdrawal. Regardless of the type of drug or substance to which the newborn was exposed, treatment begins with prompt identification of a potential problem by obtaining a comprehensive maternal history, identifying potential risks associated with exposure, and maintaining a safe environment. Newborn urine, hair (rarely), or meconium sampling may be required to identify intrauterine drug exposure and implement appropriate early interventional therapies aimed at minimizing the consequences.

**Nursing Care Management**

Nursing care of cocaine-exposed infants is similar to that of infants exposed to other drugs. Individualized assessment help determine appropriate intervention strategies. If the nurse identifies hypertonicity and sleep disturbance, the environment is modified accordingly to decrease noxious stimuli. The use of swaddling, containment, gentle rocking, NNS, and undisturbed periods of rest may help promote self-containment and state regulation. As previously noted, tissue samples may be required for identification of drug exposure. Because cocaine is easily passed in breast milk (Winecker, Goldberger, Tebbett, et al, 2001), mothers should be counseled regarding avoidance of breast-feeding. A fussy newborn may be interpreted by caretakers to be consistently hungry, and thus overfeeding and vomiting may be problematic. Provision of a safe environment in which the mother and newborn may interact is imperative. Opportunities for appropriate family bonding and attachment should be provided as with any other newborn. Because a large percentage of women who use cocaine during pregnancy have sexually transmitted infections, consider viral titers and hepatitis screening for the newborn (Askin and Diehl-Jones, 2001).

Referral to early intervention programs, including child health care, parental drug treatment, individualized developmental care, and parenting education, is essential in promoting optimum outcomes for these children. Children exposed to maternal cocaine use often live in impoverished conditions, putting them at high risk for cognitive delays, poor child health care, and inadequate nutrition; they would benefit from an early intervention program (Tronick and Beeghly, 1999, Singer, Minnes, Short, et al, 2004). Comprehensive health care services for both mother and child may be provided at one location in the “one-stop shopping” model (Tanney and Lowenstein, 1997). It is essential that nurses caring for these infants and their mothers understand the depth of the problem of prenatal drug exposure, have a positive attitude toward cocaine-using mothers and their children, be aware of community resources, and encourage positive parenting (Pokorni and Stanga, 1996).

**Methamphetamine Exposure**

The fetal and neonatal effects of maternal use of methamphetamine in pregnancy are not well known but appear to be dose related (Smith, Yonekura, Wallace, et al, 2003). LBW, preterm birth, and perinatal mortality may be consequences of higher doses used throughout pregnancy. In addition, a higher inci-
dence of cleft lip and palate and cardiac defects has been reported in infants exposed to methamphetamines in utero (Plessinger, 1998). Behavioral changes in infants exposed prenatally to methamphetamines include decreased arousal, increased stress, and alterations in movement (Smith, Lagasse, Derauf, et al, 2008).

Methamphetamine use has increased significantly in the past 10 years in certain regions of the United States. In a 2003 study by Smith, Yonekura, Wallace, and colleagues, 63% of pregnant women using methamphetamines reported using the drug throughout the pregnancy. A higher incidence of preterm delivery and placental abruption was associated with methamphetamine use. In addition, fetal growth restriction (small for gestational age) was slightly higher in methamphetamine-exposed offspring; however, 80% of these neonates’ mothers also had significant alcohol and tobacco use.

Study reports vary in the time of clinical manifestations of withdrawal from this drug: one study did not identify any signs of withdrawal in the first 3 days after birth, but long-term data were not collected (Smith, Yonekura, Wallace, et al, 2003). A study of infants exposed to methamphetamine in utero showed that such infants had significantly smaller head circumferences and birth weights than those not exposed. In addition, the exposed infants exhibited withdrawal signs of agitation, vomiting, and tachypnea, which were not observed in the unexposed infants (Chomchai, Na Manorom, Watanarungsan, et al, 2004). After birth, infants may experience bradycardia or tachycardia that resolves as the drug is cleared from the system. Lethargy may continue for several months, along with frequent infections and poor weight gain. Emotional disturbances and delays in gross and fine motor coordination may occur during early childhood.

The long-term effects of methamphetamine exposure on children living in households where the product is manufactured are not known, but there are early reports of burns in exposed children and concerns regarding the effects of the toxic by-products of methamphetamine production on small children. Skin rashes and respiratory tract illnesses are common problems seen in methamphetamine-exposed children; physical neglect and speech and language developmental delays are of significant concern as well (Crocker, 2005).

**Marijuana Exposure**

Marijuana has replaced cocaine as the most common illicit drug used by women ages 18 to 44 years (nonpregnant and pregnant) in the United States (Ebrahirn and Grofroer, 2003). Marijuana crosses the placenta. Some studies have shown that its use during pregnancy may result in a shortened gestation and a higher incidence of IUGR (Wagner, Katikaneni, Cox, et al, 1998). A review of studies examining the effects of marijuana during pregnancy found inconsistent results regarding the drug’s effect on birth weight and gestational age (Schempf, 2007), but another study reported a strong association between the use of marijuana and a decrease in fetal growth and infant birth weight and length (Hurd, Wang, Anderson, et al, 2005). Other investigators have found a higher incidence of meconium staining (Bandstra and Accornero, 2006). Compounding the issue of the effects of marijuana, especially among women ages 18 to 30 years (Ebrahirn and Grofroer, 2003), is multidrug use, which combines the harmful effects of marijuana, tobacco, alcohol, opiates, and cocaine. Long-term follow-up studies on exposed infants are needed.

**Fetal Alcohol Spectrum Disorder**

Infants and children exposed to alcohol in utero were previously reported to have characteristic facial features, prenatal and postnatal growth failure, and neurodevelopmental deficits. This triad of findings, termed fetal alcohol syndrome (FAS), was attributed to excessive ingestion of alcohol by the mother during pregnancy. It has since been shown that infants may not initially display the dysmorphic facial features. These are believed to be more well defined with increasing age during childhood. A number of terms (including alcohol-related neurodevelopmental birth defects and fetal alcohol spectrum disorders) have been proposed to describe the combination of findings. The umbrella term fetal alcohol spectrum disorder is now recommended to describe the continuum of defects seen in children affected by maternal alcohol intake, including classic FAS at the most severe end of the spectrum.

The three Centers for Disease Control and Prevention (2005) categories for diagnosis of FAS are (1) growth restriction, both prenatal and postnatal; (2) midfacial dysmorphic facial features; and (3) CNS involvement (structural, neurologic, or functional abnormality). Any single or multiple combination of these may be present in addition to confirmed or unknown history of maternal alcohol consumption. The Institute of Medicine has established diagnostic criteria (Welch-Carre, 2005), which include alcohol-related neurodevelopmental disorder (ARND) and alcohol-related birth defect (ARBD) criteria, but the Centers for Disease Control and Prevention criteria relate exclusively to FAS. The diagnosis of FAS is complicated by the absence of a specific single biologic marker and by manifestations that are often seen in other childhood conditions.

When possible, long-term disabilities are prevented by early evaluation and implementation of therapy. The family should learn any special handling techniques needed for the care of their infant and signs of complications or possible sequelae. When sequelae are inevitable, the family needs assistance in determining how to best cope with the problems, such as with home care assistance, referral to appropriate agencies, or placement in an institution for care.

The major goal of nursing care is prevention of these disorders through provision of adequate prenatal care for the expectant mother and precautions regarding exposure to potentially harmful infections.

FAS is recognized as the leading cause of cognitive impairment (American Academy of Pediatrics, 2000). The incidence of FAS is on the rise in the United States despite public warnings, including the U.S. surgeon general’s warning that consumption of alcohol during pregnancy may cause cognitive impairment and other defects. The incidence of FAS (ARBD) in the United States is about 0.2 to 1.5 per 1000 live births (Centers for Disease Control and Prevention, 2005). The reported incidence of maternal alcohol consumption during pregnancy did not change substantially during the 1991 to 2005 period despite widespread education and information regarding periconceptional and gestational effects of drinking (Centers for Disease Control and Prevention, 2009). Among pregnant
women ages 18 to 44 years, the average annual rates were 12.2% for alcohol use and 1.9% for binge drinking. In addition, among nonpregnant women any alcohol consumption was 53.7% while binge drinking was reported to be 12.1%. The Centers for Disease Control and Prevention (2009) found that pregnant women who were older, unmarried, more educated, and employed were more likely to use alcohol.

Alcohol (ethanol and ethyl alcohol) interferes with normal fetal development. The effects on the fetal brain are permanent, and even moderate use of alcohol during pregnancy may cause long-term postnatal difficulties, including impaired maternal-infant attachment. Because there is no known safe level of alcohol consumption in pregnancy, women should stop consuming alcohol at least 3 months before they plan to conceive.

Fetal abnormalities are not related to the amount of the mother’s alcohol intake per se, but to the amount consumed in excess of the liver’s ability to detoxify it. The liver’s capacity to detoxify alcohol is limited and inflexible; when the liver receives more alcohol than it is able to handle, the excess is continually recirculated until the organ is able to reduce it to carbon dioxide and water. This circulating alcohol has a special affinity for brain tissue. There is no specific critical period at which alcohol toxicity may occur, although early gestation is considered the most vulnerable period; however, exposure at any period may cause subtle damage to the developing fetus (Brust, 2009). Other factors that contribute to the teratogenic effects include toxic acetyl aldehyde (a degradation byproduct of ethanol) and other substances that may be added to the alcohol. Poor nutritional state, smoking, polydrug intake, and infrequent or lack of prenatal care may compound the problem of alcohol abuse (Jones and Bass, 2003).

The effects on the fetal brain are reflected in CNS manifestations of FAS (Box 10-13). Cognitive and motor delays, hearing disorders, and a variety of defects in craniofacial development are prominent features (Fig. 10-19). MRI studies of children with diagnosed FAS revealed a high incidence of midbrain anomalies, including displacements in the corpus callosum, and changes in symmetry in the temporal lobes. Alcohol-exposed infants also demonstrate narrowing in the temporal region and reduced brain growth in portions of the frontal lobe (Riley, McGee, and Sowell, 2004). Some affected infants display physical features of the syndrome; behaviors, however, are nonspecific in newborns and may therefore pass undetected. These include difficulty in establishing respiration, irritability, lethargy, poor suck reflex, and abdominal distention.

**Nursing Care Management**

Nursing care of affected infants involves the same assessment and observations that are employed for any high-risk infant. Poor feeding is characteristic of infants with FAS and is a significant problem throughout infancy. Strategies to provide individualized developmental care are aimed at reducing noxious environmental stimuli and helping the infant achieve self-regulation (see Developmental Outcome, p. 332). Monitoring weight gain, analyzing feeding behaviors, and devising strategies to promote nutritional intake are especially important.

The effects of FAS have been identified in adolescents and young adults, primarily in relation to growth deficiencies, delayed motor development, and cognitive impairment. In one study children who were exposed to only small amounts of alcohol prenatally showed more aggressiveness, delinquent behavior, and attention problems at 6 to 7 years of age compared with unexposed controls (Sood, Delaney-Black, Covington,
et al, 2001). Another study found that young adult offspring prenatally exposed to alcohol had significant alcohol-related problems by age 21 (Baer, Sampson, Barr, et al, 2003). Facial characteristics in adults tend to be more subtle than in infants and children.

Early diagnosis and intervention are reported to be beneficial for reducing the effects of alcohol exposure on the growing child (Stoler and Holmes, 2004); therefore nurses should be actively involved in identifying and referring children exposed to alcohol prenatally.

The dangers of heavy drinking are known, and all women should be counseled regarding the risks to the fetus. The nurse should emphasize to women of all ages that there is no known “safe” amount of alcohol intake during pregnancy that will preclude FASD. Furthermore, FASD is a totally preventable birth defect. A change in drinking habits even as late as the third trimester (when brain growth in the fetus is greatest) is associated with improved fetal outcome.*

### Infants of Mothers Who Smoke

Cigarette smoking during pregnancy is clearly associated with significant birth weight deficits—up to 440 g (about 1 lb) in full-term newborns—and there is a definitive dose-response relationship between the number of cigarettes smoked by the mother and these deficits (Law, Stroud, LaGasse, et al, 2003). This dose-related response also affects the Apgar scores. The number of infants with low Apgar scores whose mothers smoked three packs per day is nearly four times higher than for infants whose mothers smoked none or only one pack per day. Large studies indicate that 21% to 39% of the incidence of LBW infants whose mothers smoked none or only one pack per day. The rate of preterm births is increased in mothers who smoke, but the infants are smaller at all stages of gestation. They show fetal growth restriction in length, weight, and chest and head circumference; these deficits are not related to maternal appetite or weight gain. The concentration of a pharmacologically active substance found in tobacco—nicotine—has been found to be higher in newborns of mothers who smoke than in the mothers themselves. Nicotine is metabolized to cotinine and secreted in breast milk and has a half-life of 70 to 80 minutes. In addition, it is now recognized that neonates may experience withdrawal symptoms after exposure to nicotine, whether in tobacco smoke or chewable form. It has also been shown that cigarette smoking has detrimental effects beyond the neonatal period, with deficits in growth, intellectual and emotional development, and behavior. Maternal smoking and passive smoking by household members has been correlated with an increased incidence of SIDS (Hunt and Hauck, 2006), respiratory tract illnesses (Jorgensen, 1999), spontaneous abortion, premature rupture of membranes, preterm delivery, and deficits in learning and behavior (Shea and Steiner, 2008). (See Environmental Tobacco Smoke Exposure, Chapter 32.)

### Nursing Care Management

Nurses are prime candidates for disseminating information to expectant mothers regarding smoking-related risks. Mothers who stop or substantially reduce smoking during pregnancy improve the quality of life for their unborn infants. In one study, infants of expectant mothers who were given information, support, encouragement, practical guidance, and behavior modification during pregnancy delivered infants with significantly higher birth weights than did controls. If mothers continue to smoke while breast-feeding, encourage them to do so immediately after breast-feeding to reduce the amount of nicotine and cotinine in the breast milk. Smoking decreases milk production in the breast-feeding mother (Lawrence and Lawrence, 2005). Parents should make all efforts to avoid second-hand smoke around all infants, but especially around those born with respiratory or cardiac problems and those born prematurely.

### MATERNAL INFECTIONS

The range of pathologic conditions produced by infectious agents is large, and the difference between the maternal and fetal effects caused by any one agent is also great. Some maternal infections, especially during early gestation, can result in fetal loss or malformations because the fetus’s ability to handle infectious organisms is limited and the fetal immunologic system is unable to prevent the dissemination of infectious organisms to the various tissues. Not all prenatal infections produce teratogenic effects. Furthermore, the clinical picture of disorders caused by transplacental transfer of infectious agents is not always well defined. Some microbial agents can cause remarkably similar manifestations, and it is not uncommon to test for all when a prenatal infection is suspected. This is the so-called TORCHS complex, an acronym for:

- T—Toxoplasmosis
- O—Other (e.g., hepatitis B, parvovirus, HIV)
- R—Rubella
- C—Cytomegalovirus infection
- H—Herpes simplex
- S—Syphilis

To determine the causative agent in a symptomatic infant, perform tests to rule out each of these infections. The O category may involve testing for several viral infections (e.g., hepatitis B, varicella zoster, measles, mumps, HIV, human papillomavirus, and human parvovirus). Although this acronym has received substantial criticism because it does not cover the entire spectrum of congenital infections (Klein, Baker, Remington, et al, 2006), it is still used in clinical settings. Bacterial infections are not included in the TORCHS workup, since they are usually identified by clinical manifestations and readily available laboratory tests. Gonococcal conjunctivitis (ophthalmia neonatorum) and chlamydial conjunctivitis have been significantly reduced by prophylactic measures at birth. (See Chapter 8.) HIV infection is discussed in Chapter 35. The major maternal infections, their possible effects, and specific nursing considerations are outlined in Table 10-11.

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*Further information is available from National Organization on Fetal Alcohol Syndrome, 900 17th St. NW, Washington, DC 20006; 202-785-4858; www.nofas.org; and Fetal Alcohol Syndrome Branch, National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, www.cdc.gov/ncbddd/fas.
### TABLE 10-11  INFECTIOnS ACQUIRED FROM MOTHER BEFORE, DURING, OR AFTER BIRTH*

<table>
<thead>
<tr>
<th>FETAL OR NEWBORn EFFECT</th>
<th>TRANSMISSION</th>
<th>NURSING CONSIDERATIONS†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Human Immunodeficiency Virus (HIV)</strong></td>
<td>Transplacental; during vaginal delivery; potentially in breast milk</td>
<td>Administer antiviral prophylaxis to the mother beginning at 14 wk of pregnancy. The choice of regimens is determined by examining a number of factors, including the mother’s current treatment. Detailed recommendations can be obtained from Perinatal HIV Guidelines Working Group (2009). During labor ZDV is recommended for all HIV-infected pregnant women, regardless of the antenatal treatment regimen. HIV-exposed neonates should receive a 6-wk course of ZDV (consider addition of another antiretroviral drug based on maternal treatment and exposure). Cesarean section in HIV-positive mother is recommended to reduce transmission. For chemoprophylaxis against <em>Pneumocystis carinii</em> pneumonia in HIV-exposed infants, drug of choice is trimethoprim-sulfamethoxazole (Bactrim, Septra). Documented routine HIV education and routine testing with consent for all pregnant women in United States are recommended.</td>
</tr>
<tr>
<td><strong>Chickenpox (Varicella-Zoster Virus [VZV])</strong></td>
<td>1st trimester (fetal varicella syndrome); perinatal period (infection)</td>
<td>Use varicella zoster immunoglobulin (VariZIG) or IV immunoglobulin (IVIG) to treat infants born to mothers with onset of disease within 5 days before or 2 days after delivery. Institute Isolation Precautions in newborn born to mother with varicella up to 21-28 days (latter time if newborn received VariZIG or IVIG) after birth (if hospitalized). Prevention—universal immunization of children with varicella vaccine according to recommended schedule.</td>
</tr>
<tr>
<td><strong>Chlamydia Infection (Chlamydia trachomatis)</strong></td>
<td>Last trimester or perinatal period</td>
<td>Standard ophthalmic prophylaxis for gonococcal ophthalmia neonatorum (topical antibiotics, silver nitrate, or povidone-iodine) is not effective in treatment or prevention of chlamydial ophthalmia. Treat with oral erythromycin for 14 days.</td>
</tr>
<tr>
<td><strong>Coxsackievirus (Group B Enterovirus–Nonpolio)</strong></td>
<td>Peripartum</td>
<td>Treatment is supportive. Provide IVG in neonatal infections.</td>
</tr>
<tr>
<td><strong>Cytomegalovirus (CMV)</strong></td>
<td>Throughout pregnancy</td>
<td>Infection acquired at birth, shortly thereafter, or via human milk is not associated with clinical illness. Affected individuals excrete virus. Virus is detected in urine or tissue by electron microscopy. Avoid kissing affected child. Pregnant women should avoid close contact with known cases. To treat infection, administer IV antivirals such as ganciclovir to newborn.</td>
</tr>
<tr>
<td><strong>Erythema Infectiosum (Parvovirus B19)</strong></td>
<td>Transplacental</td>
<td>First trimester infection has most serious effects. Pregnant health care workers should not care for patients who might be highly contagious (e.g., child with aplastic crisis). Routine exclusion of pregnant women from workplace where disease is occurring is not recommended.</td>
</tr>
</tbody>
</table>

*This table is not an exhaustive representation of all perinatally transmitted infections. For further information regarding specific diseases or treatment not listed here, refer to American Academy of Pediatrics, Committee on Infectious Diseases, Pickering L, editor: 2009 Red book: report of the Committee on Infectious Diseases, ed 28, Elk Grove Village, Ill, 2009, The Academy. Isolation Precautions depend on institutional policy. (See Infection Control, Chapter 27.)

**Continued**
**TABLE 10-11  INFECTIONS ACQUIRED FROM MOTHER BEFORE, DURING, OR AFTER BIRTH**—cont’d

<table>
<thead>
<tr>
<th>Fetal or Newborn Effect</th>
<th>Transmission</th>
<th>Nursing Considerations†</th>
</tr>
</thead>
<tbody>
<tr>
<td>** Gonococcal Disease (Neisseria gonorrhoeae)**</td>
<td>Last trimester or perinatal period</td>
<td>Apply prophylactic medication to eyes at time of birth. Obtain smears for culture. To treat infection, administer penicillin.</td>
</tr>
<tr>
<td>Ophthalmitis</td>
<td></td>
<td></td>
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<tr>
<td>Neonatal gonococcal arthritis, septicemia, meningitis</td>
<td></td>
<td></td>
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<tr>
<td>** Hepatitis B Virus (HBV)**</td>
<td>Transplantal;</td>
<td>Administer hepatitis B immunoglobulin (HBIG) to all infants of HBsAg-positive mothers within 12 hr of birth; in addition, administer hepatitis B vaccine at separate site. Prevention—universal immunization of all infants with Hep B vaccine. (See Immunizations, Chapter 12.)</td>
</tr>
<tr>
<td>May be asymptomatic at birth</td>
<td>contaminated maternal fluids or secretions during delivery</td>
<td></td>
</tr>
<tr>
<td>Acute hepatitis, changes in liver function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>** Herpes, Neonatal (Herpes Simplex Virus)**</td>
<td>History of genital infection in mother or partner in 50% of cases</td>
<td>Cesarean sections sometimes are preventive measure for mothers with active lesions. Vaginal delivery is recommended for infants of mothers with recurrent infection thought to be at lower risk. Infants should room-in with mother in private room.</td>
</tr>
<tr>
<td>Cutaneous lesions—vesicles at 5-10 days of age; may be no lesions</td>
<td>Transmitted intrapartum, either by ascending infection or direct contact, especially primary infection</td>
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</tr>
<tr>
<td>Disseminated disease resembling sepsis—encephalitis in 60%-70%</td>
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<tr>
<td>Visceral involvement—granulomas</td>
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</tr>
<tr>
<td>Early non specific signs—fever, lethargy, poor feeding, irritability, vomiting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early nonspecific signs—crying, irritability, poor feeding</td>
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<td></td>
</tr>
<tr>
<td>May include hyperbilirubinemia, seizures, flaccid or spastic paralysis, apneic episodes, respiratory distress, lethargy, or coma</td>
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<td></td>
</tr>
<tr>
<td>Rubella, Congenital (Rubella Virus)</td>
<td>1st trimester; early 2nd trimester</td>
<td>Pregnant women should avoid contact with all affected persons, including infants with rubella syndrome. Emphasize vaccination of all unimmunized prepubertal children, susceptible adolescents, and women of childbearing age (nonpregnant). Caution women against pregnancy for at least 3 mo after vaccination.</td>
</tr>
<tr>
<td>Eye defects—cataracts (unilateral or bilateral), microphthalmos, retinitis, glaucoma</td>
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<td></td>
</tr>
<tr>
<td>CNS signs—microcephaly, seizures, severe cognitive impairment</td>
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<tr>
<td>Congenital heart defects—patent ductus arteriosus</td>
<td></td>
<td></td>
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<tr>
<td>Auditory—high incidence of delayed hearing loss</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intrauterine growth restriction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperbilirubinemia, meningoencephalitis, thromboembolic, hepatomegaly</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Listeriosis (Listeria monocytogenes)</td>
<td>Transplantal, by ascending infection or exposure at delivery</td>
<td>Hand washing is essential to prevent nosocomial spread. Treat infected newborn with antibiotics—ampicillin and gentamicin.</td>
</tr>
<tr>
<td>Maternal infection associated with abortion, preterm delivery, and fetal death</td>
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</tr>
<tr>
<td>Preterm birth, sepsis, and pneumonia seen in early-onset disease; late-onset disease usually manifests as meningitis</td>
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<tr>
<td>Syphilis, Congenital (Treponema pallidum)</td>
<td>Transplantal; can be anytime during pregnancy or at birth</td>
<td>This is most severe form of syphilis. Treatment consists of IV penicillin. Diagnostic evaluation depends on maternal serology testing and infant symptoms (American Academy of Pediatrics, 2009b).</td>
</tr>
<tr>
<td>Stillbirth, prematurity, hydramnios fatalis</td>
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<td></td>
</tr>
<tr>
<td>May be asymptomatic at birth and in 1st few weeks of life or may have multisystem manifestations: hepatospleno megaly, lymphadenopathy, hemolytic anemia, and thrombocytopenia</td>
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<td></td>
</tr>
<tr>
<td>Copper-colored maculopapular cutaneous lesions (usually after 1st few weeks of life), mucous membrane patches, hair loss, nail exfoliation, snuffles (syphilitic rhinitis), profound anemia, poor feeding, pseudoparalysis of one or more limbs, dysmorphic teeth (older child)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toxoplasmosis (Toxoplasma gondii)</td>
<td>Throughout pregnancy</td>
<td>Caution pregnant women to avoid contact with cat feces (e.g., emptying cat litter boxes). Administer sulfa nafonides (trimethoprim-sulfamethoxazole) or pyrimethamine (Daraprim).</td>
</tr>
<tr>
<td>Predominant host for organism is cats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>May be transmitted through cat feces or poorly cooked or raw infected meats</td>
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</tbody>
</table>

CNS, Central nervous system; HBsAg, hepatitis B surface antigen; IV, intravenous.

*This table is not an exhaustive representation of all perinatally transmitted infections. For further information regarding specific diseases or treatment not listed here, refer to American Academy of Pediatrics, Committee on Infectious Diseases, Pickering L, editor: 2009 Red book: report of the Committee on Infectious Diseases, ed 28, Elk Grove Village, Ill, 2009, The Academy.

†Isolation Precautions depend on institutional policy. (See Infection Control, Chapter 27.)
Nursing Care Management

One of the major goals in care of infants suspected of having an infectious disease is identification of the causative organism. Until the diagnosis is established, implement Standard Precautions according to institutional policy. In suspected cytomegalovirus and rubella infections, pregnant personnel are cautioned to avoid contact with the infant. Herpes simplex is easily transmitted from one infant to another; therefore risk of cross-contamination is reduced or eliminated by wearing gloves for patient contact. The American Academy of Pediatrics’ 2009 Red Book: Report of the Committee on Infectious Diseases (2009b) provides guidelines for the type and duration of precautions for most bacterial and viral exposures. Careful hand washing is the most important nursing intervention in reducing the spread of any infection.

**KEY POINTS**

- High-risk neonates may be defined as newborns, regardless of gestational age or birth weight, who have a greater than average chance of morbidity or mortality because of conditions or circumstances superimposed on the normal course of events associated with birth and adjustment to extrauterine existence.
- Identification of high-risk newborns may occur during any of the following stages: prenatal, natal, or postnatal.
- High-risk infants may be classified according to birth weight, gestational age, and morbidity factors.
- Late-preterm infants, by nature of their limited gestation, remain at risk for problems related to thermoregulation, hypoglycemia, hyperbilirubinemia, sepsis, and respiratory function.
- General management of the newborn entails immediate care, protection from infection, monitoring of physiologic data (including heart rate, respiratory activity, temperature, and blood pressure), laboratory data, and systematic assessment of the high-risk infant.
- Assessment of the high-risk newborn includes general, respiratory, cardiovascular, GI, genitourinary, neurologic-musculoskeletal, skin, and temperature assessments.
- Because many of their metabolic processes are immature, high-risk newborns are placed in a heated environment to help maintain thermal stability.
- Because of the immature, fragile skin of preterm infants, the nurse should use caution when applying topical preparations and, when possible, avoid adhesives.
- Meeting the high-risk infant’s nutritional needs requires specific knowledge of physiologic characteristics, the infant’s particular needs, and methods of feeding.
- Delayed development in high-risk neonates is a concern; developmental interventions are individualized to ameliorate the effects and increase infant well-being.
- Parental involvement in the care of high-risk infants is important, and nurses should encourage parent-infant relationships from birth to discharge.
- Prematurity accounts for the largest number of admissions to an NICU.
- Several severe respiratory conditions place the infant at high risk: AOP, RDS, MAS, air leak syndromes, and BPD.
- Therapeutic management of RDS includes oxygen therapy and assisted ventilation.
- Newborns are highly susceptible to infection, particularly septicemia.
- Cardiovascular complications in the high-risk infant may include PDA and PPHN.
- Neurologic disturbances in the high-risk newborn may include perinatal hypoxic-ischemic brain injury, IVH, ICH, neonatal seizures, and stroke.
- Nurses play an important role in end-of-life care of the family of the dying infant.
- Maternal conditions that pose a threat to the newborn include diabetes and substance abuse during pregnancy.
- Prenatal environmental conditions, especially selected maternal viral and bacterial infections and maternal alcohol ingestion, are responsible for high-risk problems in some newborns.

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CHAPTER 10
The High-Risk Newborn and Family


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