LEARNING OBJECTIVES

- Summarize assessment and care of the newborn with soft-tissue, skeletal, and nervous system injuries caused by birth trauma.
- Identify maternal conditions that place the newborn at risk for infection.
- Describe methods used to identify infection in the newborn.
- Identify clinical signs of infection in the newborn.
- Identify the effects of maternal use of alcohol, heroin, methadone, marijuana, methamphetamine, mine, cocaine, and smoking tobacco on the fetus and newborn.
- Outline the assessment of a newborn exposed to recreational drugs in utero.
- Compare neonatal Rh and ABO incompatibility.
- Describe preoperative and postoperative nursing care of the newborn.
- Explain congenital disorders presented in this chapter and identify the priority of nursing care for each.

KEY TERMS AND DEFINITIONS

**ABO incompatibility** Hemolytic disease that occurs when the mother’s blood type is O and the newborn’s is A, B, or AB.

**alcohol-related birth defects (ARBD)** Congenital abnormality or anomaly resulting from excessive maternal alcohol intake during pregnancy; characterized by typical craniofacial and limb defects, cardiovascular defects, intrauterine growth restriction, and developmental delay; newer terminology for fetal alcohol syndrome (FAS).

**alcohol-related neurodevelopmental disorder (ARND)** Disorder in infants affected by prenatal exposure to alcohol but who do not meet the criteria for FAS; previously referred to as fetal alcohol effects (FAE)

**anencephaly** Congenital deformity characterized by the absence of cerebrum, cerebellum, and flat bones of the skull.

**cleft lip** Incomplete closure of the lip; lay term is harelip.

**cleft palate** Incomplete closure of the palate or roof of the mouth; a congenital fissure.

**Coombs’ test** Indirect: Determination of Rh-positive antibodies in maternal blood; direct: determination of maternal Rh-positive antibodies in fetal cord blood; positive test result indicates the presence of antibodies or titers.

**developmental dysplasia of the hip** Abnormal development of the hip joint, resulting in instability of the hip causing one or both of the femoral heads to be displaced from the acetabulum (hip socket).

**erythroblastosis fetalis** Hemolytic disease of the newborn usually caused by isoimmunization resulting from Rh incompatibility or ABO incompatibility.

**exchange transfusion** Replacement of 75% to 85% of circulating blood by withdrawal of the recipient’s blood and injection of a donor’s blood in equal amounts, the purposes of which are to prevent an accumulation of bilirubin in the blood above a dangerous level, to prevent the accumulation of other by-products of hemolysis in hemolytic disease, and to correct anemia and acidosis.

**gastrochisis** Abdominal wall defect at the base of the umbilical stalk.

**hydrops fetalis** Most severe expression of fetal hemolytic disorder, a possible sequela to maternal Rh isoimmunization; infants exhibit gross edema (anasarca), cardiac decompensation, and profound pallor from anemia and seldom survive.

**inborn error of metabolism** Group of recessive disorders caused by a metabolic defect that results from the absence of or change in a protein, usually an enzyme, and mediated by the action of a certain gene.

**microcephaly** Abnormal smallness of the head in relation to the rest of the body and underdevelopment of the brain, resulting in some degree of mental retardation.

**myelomeningocele** External sac containing meninges, spinal fluid, and nerves that protrudes through defect in vertebal column.

**Chapter 27**

The Newborn at Risk: Acquired and Congenital Problems

SHANNON E. PERRY
A challenge for the nurse is the birth of an infant at risk because of conditions or circumstances that are superimposed on the normal course of events associated with birth and the adjustment to extrauterine existence. The infant may be considered high risk because of birth trauma, maternal substance abuse, infection, or congenital anomalies. Birth trauma includes physical injuries a neonate sustains during labor and birth. Congenital anomalies include such conditions as gastrointestinal (GI) malformations, neural tube defects (NTDs), abdominal wall defects, and cardiac defects.

At times the nurse is able to anticipate problems, such as when a woman is admitted in premature labor or a congenital anomaly is diagnosed by ultrasound before birth. At other times the birth of a high risk infant is unanticipated. In either case the personnel and equipment necessary for immediate care of the infant must be available.

Birth trauma (injury) is physical injury sustained by a neonate during labor and birth. It remains an important source of neonatal morbidity. In theory, most birth injuries may be avoidable, especially if careful assessment of risk factors and appropriate planning of birth occur. The use of fetal ultrasonography allows antepartum diagnosis of many conditions that may be treated in utero or shortly after birth. Elective cesarean birth can be chosen for some pregnancies to prevent significant birth or vacuum extraction or from pressure of the fetal skull against the maternal pelvis.

Many injuries are minor and resolve readily in the neonatal period without treatment. Other trauma requires some degree of intervention; few are serious enough to be fatal. The nurse’s contributions to the welfare of the newborn begin with early observation of the newborn’s transition. The prompt reporting of signs that indicate deviations from normal permits early initiation of appropriate therapy. Table 27-1 provides an overview of neurologic birth injuries and the sites in which they occur.

When the newborn is born the nurse makes a rapid inspection and physical assessment to determine if there are any life-threatening conditions requiring immediate medical or surgical attention. A comprehensive physical assessment of the newborn is performed after the parents have had the opportunity to interact with the newborn. Because evidence of some birth injuries may not be apparent at the initial examination, assessment continues during each contact with the neonate.

Soft-tissue injuries that commonly occur at birth including caput succedaneum and cephalohematoma are discussed in Chapter 19.

Skeletal Injuries

The newborn’s immature, flexible skull can withstand a greater degree of deformation (molding) before fracture results. Considerable force is required to fracture the newborn’s skull.
Two types of skull fractures typically are identified in the newborn: linear fractures and depressed fractures. The location of the fracture and involvement of underlying structures determine its significance.

If an artery lying in a groove on the undersurface of the skull is torn as a result of the fracture, increased intracranial pressure (ICP) will follow. Unless a blood vessel is involved, linear fractures, which account for 70% of all fractures for this age group, heal without special treatment. The soft skull may become indented without laceration of either the skin or the dural membrane. These depressed fractures, or ping-pong ball indentations, may occur during difficult births from pressure of the head on the bony pelvis. They also can occur as a result of injudicious application of forceps. Spontaneous or nonsurgical elevation of the indentation by using a hand breast pump or vacuum extractor has been reported (Mangurten, 2002).

The clavicle is the bone most often fractured during birth. Generally the break is in the middle third of the bone (Fig. 27-1). Dystocia, particularly shoulder impaction, may be the predisposing problem. Limitation of motion of the arm, crepitus over the bone, and the absence of the Moro reflex on the affected side are diagnostic. Except for use of gentle rather than vigorous handling, no accepted treatment for fractured clavicle in the newborn exists, and the prognosis is good. The humerus and femur are other bones that may be fractured during a difficult birth. Fractures in newborns generally heal rapidly. Immobilization is accomplished with slings, splints, swaddling, and other immobilization devices.

The parents need support in handling these infants because they often are fearful of hurting them. Parents are encouraged to practice handling, changing diapers, and feeding the affected neonate under the guidance of nursery personnel. This increases their confidence and knowledge and facilitates attachment. A plan for follow-up therapy is developed with the parents so that the times and arrangements for therapy are acceptable to them.

### Peripheral Nervous System Injuries

Plexus injury results from forces that alter the normal position and relationship of the arm, shoulder, and neck. Erb palsy (Erb-Duchenne paralysis) is caused by damage to the upper plexus and usually results from a stretching or pulling away of the shoulder from the head such as might occur with shoulder dystocia or with a difficult vertex or breech birth. The less common lower plexus palsy, or Klumpke palsy, results from severe stretching of the upper extremity while the trunk is relatively less mobile.

The clinical manifestations of Erb palsy are related to the paralysis of the affected extremity and muscles. The arm hangs limp alongside the body. The shoulder and arm are adducted and internally rotated. The elbow is extended, and the forearm is pronated, with the wrist and fingers flexed; a grasp reflex may be present because finger and wrist movement remain normal (Tappero, 2003) (Fig. 27-2). In lower plexus palsy, the muscles of the hand are paralyzed, with consequent wrist drop and relaxed fingers. In a third and

<table>
<thead>
<tr>
<th>SITE OF INJURY</th>
<th>TYPE OF INJURY</th>
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<tbody>
<tr>
<td>Scalp</td>
<td>Caput succedaneum</td>
</tr>
<tr>
<td></td>
<td>Subgaleal hemorrhage</td>
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<tr>
<td></td>
<td>Cephalhematoma</td>
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<tr>
<td>Skull</td>
<td>Linear fracture</td>
</tr>
<tr>
<td></td>
<td>Depressed fracture</td>
</tr>
<tr>
<td>Intracranial</td>
<td>Epidural hematoma</td>
</tr>
<tr>
<td></td>
<td>Subdural hematoma (faceration of falx, tentorium, or superficial veins)</td>
</tr>
<tr>
<td></td>
<td>Subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Spinal cord (cervical)</td>
<td>Vertebral artery injury</td>
</tr>
<tr>
<td>Plexus</td>
<td>Intraspinal hemorrhage</td>
</tr>
<tr>
<td></td>
<td>Spinal cord transection or injury</td>
</tr>
<tr>
<td></td>
<td>Erb palsy</td>
</tr>
<tr>
<td></td>
<td>Klumpke palsy</td>
</tr>
<tr>
<td></td>
<td>Total (mixed) brachial plexus injury</td>
</tr>
<tr>
<td></td>
<td>Horner syndrome</td>
</tr>
<tr>
<td></td>
<td>Diaphragmatic paralysis</td>
</tr>
<tr>
<td>Cranial and peripheral nerve</td>
<td>Lumbosacral plexus injury</td>
</tr>
<tr>
<td></td>
<td>Radial nerve palsy</td>
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<tr>
<td></td>
<td>Median nerve palsy</td>
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<tr>
<td></td>
<td>Sciatic nerve palsy</td>
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<tr>
<td></td>
<td>Laryngeal nerve palsy</td>
</tr>
<tr>
<td></td>
<td>Diaphragmatic paralysis</td>
</tr>
<tr>
<td></td>
<td>Facial nerve palsy</td>
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</tbody>
</table>

more severe form of brachial palsy the entire arm is paralyzed and hangs limp and motionless at the side. The Moro reflex is absent on the affected side for all of the forms of brachial palsy (Dunham, 2003).

Treatment of the affected arm is aimed at preventing contractures of the paralyzed muscles and maintaining correct placement of the humeral head within the glenoid fossa of the scapula. Complete recovery from stretched nerves usually takes 3 to 6 months. However, avulsion of the nerves (complete disconnection of the ganglia from the spinal cord that involves both anterior and posterior roots) results in permanent damage. For those injuries that do not improve spontaneously by 3 months, surgical intervention may be needed to relieve pressure on the nerves or to repair the nerves with grafting (Volpe, 2001). In some cases, injection of botulinum toxin A into the triceps muscle may be effective in reducing muscle contractures after birth-related brachial plexus injuries (Rollnik et al., 2000).

Nursing care of the newborn with brachial palsy is concerned primarily with proper positioning of the affected arm. The affected arm should be gently immobilized on the upper abdomen; passive range-of-motion exercises of the shoulder, wrist, elbow, and fingers are initiated in the latter part of the first week (Volpe, 2001). Wrist flexion contractures may be prevented with the use of supportive splints. In dressing the infant, preference is given to the affected side. Un-dressing begins with the unaffected arm, and redressing begins with the affected arm to prevent unnecessary manipulation and stress on the paralyzed muscles. Parents are taught to use the “football” position when holding the infant and to avoid picking the child up from under the axillae or by pulling on the arms.

Pressure on the facial nerve during birth may result in injury to cranial nerve VII. The primary clinical manifestations are loss of movement on the affected side, such as an inability to completely close the eye, drooping of the corner of the mouth, and absence of wrinkling of the forehead and nasolabial fold (Fig. 27-3). Facial palsy or paralysis is most noticeable when the infant cries. The mouth is drawn to the unaffected side, the wrinkles are deeper on the normal side, and the eye on the involved side remains open. Often the condition is temporary, resolving within hours or days of birth. Permanent paralysis is rare.

Nursing care of the infant with facial nerve paralysis involves aiding the infant in sucking and helping the mother with feeding techniques. The infant may require gavage feeding to prevent aspiration. Breastfeeding is not contraindicated, but the mother will need additional assistance in helping the infant grasp and compress the areolar area.

If the lid of the eye on the affected side does not close completely, artificial tears can be instilled daily to prevent drying of the conjunctiva, sclera, and cornea. The lid is often taped shut to prevent accidental injury. If eye care is needed at home, the parents are taught the procedure for administering eye drops before the infant is discharged from the nursery.

Phrenic nerve paralysis results in diaphragmatic paralysis as demonstrated by ultrasonography, which shows paradoxical chest movement and an elevated diaphragm. Initially, radiography may not demonstrate an elevated diaphragm if the
neonate is receiving positive pressure ventilation (Volpe, 2001). The injury sometimes occurs in conjunction with brachial palsy. Respiratory distress is the most common and important sign of injury. Because injury to the phrenic nerve is usually unilateral, the lung on the affected side does not expand, and respiratory efforts are ineffectual. The infant is positioned on the affected side to facilitate maximum expansion of the uninvolved lung. Breathing is primarily thoracic, and cyanosis, tachypnea, or complete respiratory failure may be seen. Pneumonia and atelectasis on the affected side may also occur.

The infant with phrenic nerve paralysis requires the same nursing care as any infant with respiratory distress. As with other birth injuries, the emotional needs of the family are similar to those discussed for soft-tissue injury (see Chapter 19). Follow-up is also essential because of the extended length of recovery.

**Central Nervous System Injuries**

All types of intracranial hemorrhage (ICH) occur in newborns. ICH as a result of birth trauma is more likely to occur in the full-term, large infant. The frequency and degree of severity of ICH are different in the newborn than in older children or adults. In the newborn, more than one type of hemorrhage can and does commonly occur.

A subdural hematoma, or life-threatening collection of blood in the subdural space, most often is produced by the stretching and tearing of the large veins in the tentorium of the cerebellum, the dural membrane that separates the cerebrum from the cerebellum. When this type of bleeding occurs, the typical history includes a primiparous mother, with the total labor and birth occurring in less than 2 or 3 hours; a difficult birth; or a large-for-gestational-age infant. Subdural hemorrhage occurs infrequently because of improvements in obstetric care. However, it is especially serious because of its inaccessibility to aspiration by subdural tap.

Subarachnoid hemorrhage, the most common type of ICH, occurs in term infants as a result of trauma and in preterm infants as a result of hypoxia. Small hemorrhages are the most common. Bleeding is of venous origin, and underlying contusion also may occur.

The clinical presentation of hemorrhage in the full-term infant can vary considerably. In many infants, signs are absent, and hemorrhaging is diagnosed only because of abnormal findings on lumbar puncture—for example, red blood cells (RBCs) in the cerebrospinal fluid (CSF) or a hemorrhage is seen on a computed tomography (CT) scan. The initial clinical manifestations of neonatal subarachnoid hemorrhage may be the early onset of alternating central nervous system (CNS) depression and irritability, with refractory seizure. Poor feeding, apnea, and unequal pupils may suggest an intracranial insult. Occasionally the infant appears normal initially then has seizures on the second or third day of life, followed by no apparent sequelae.

In general, nursing care of an infant with ICH is supportive and includes monitoring neurologic signs, intra-venous (IV) therapy, observation and management of seizures, and prevention of increased ICP.

Spinal cord injuries almost always result from breech births, especially difficult ones in which version and extraction are used. This type of injury is rarely seen today because cesarean birth is often used for breech presentation (Page & Carney, 2002).

**NEONATAL INFECTIONS**

**Sepsis**

Sepsis (the presence of microorganisms or their toxins in the blood or other tissues) continues to be one of the most significant causes of neonatal morbidity and mortality. Maternal immunoglobulin M (IgM) does not cross the placenta. Immunoglobulin A (IgA) and IgM require time to reach optimum levels after birth. Phagocytosis (process by which cells engulf and destroy microorganisms and cellular debris) is less efficient. Serum complement levels are inadequate; serum complement (C1 through C6) is involved in immunologic reactions, some of which kill or lyse bacteria and enhance phagocytosis. Dysmaturity seen with intrauterine growth restriction (IUGR) and preterm and postdate birth further compromises the neonate’s immune system.

Table 27-2 outlines risk factors for neonatal sepsis. Special precautions for preventing infection, as well as prompt recognition when it occurs, are necessary for optimum newborn care. Neonatal infections may be acquired in utero, during birth or resuscitation, and nosocomially.

**TABLE 27-2**

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>RISK FACTORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal</td>
<td>Low socioeconomic status, Poor prenatal care, Poor nutrition, Substance abuse, Maternal fever, Chorioamnionitis, Prolonged labor, Rupture of membranes &gt;12 to 18 hr, Premature labor, Maternal urinary tract infection, Twin or multiple gestation, Male, Birth asphyxia, Meconium aspiration, Congenital anomalies of skin or mucous membranes, Galactosemia, Absence of spleen, Low birth weight or prematurity, Malnutrition, Prolonged hospitalization</td>
</tr>
</tbody>
</table>
Neonatal bacterial infection is classified into two patterns according to the time of presentation. Early-onset or congenital sepsis usually manifests within 24 to 48 hours of birth, progresses more rapidly than later-onset infection, and carries a mortality rate as high as 50%. Early-onset infection is usually caused by microorganisms from the normal flora of the maternal vaginal tract, including group B streptococci (GBS), Haemophilus influenzae, Listeria monocytogenes, Escherichia coli, and Streptococcus pneumoniae (Merenstein, Adams, & Weisman, 2002). With the widespread use of intrapartum penicillin for prevention of GBS infection, E. coli has been reported to be the most common offending pathogen in early-onset sepsis (Stoll et al., 2002). Coagulase-negative staphylococci has also been reported in some centers as the most common pathogen (Edwards et al., 2003), but this is debatable because some consider it to be a contaminant (Polak, Ringler, & Daugherty, 2004). Early-onset sepsis is associated with a history of obstetric events such as preterm labor, prolonged rupture of membranes (>12–18 hours), maternal fever during labor, and chorioamnionitis (Merenstein, Adams, & Weisman, 2002).

Nosocomial infection (late-onset) is most commonly seen after 2 weeks of age and is slower in progression. Bacteria responsible for late-onset sepsis are varied, may be acquired from the birth canal or from the external environment, and include Staphylococcus aureus, Staphylococcus epidermidis, Pseudomonas organisms, and GBS. Viral infections may cause miscarriage, stillbirth, intrauterine infection, congenital malformations, and acute neonatal disease. These pathogens also may cause chronic infection, with subtle manifestations that may be recognized only after a prolonged period. It is important to recognize these manifestations in the neonatal period to be able to treat the acute infection, to prevent nosocomial infections in other infants, and to anticipate effects on the infant’s subsequent growth and development. Fungal infections are of greatest concern in the immunocompromised or premature infant. Occasionally, fungal infections such as thrush are found in otherwise healthy term infants. The term septicemia refers to a generalized infection in the bloodstream. Pneumonia, the most common form of neonatal infection, is one of the leading causes of perinatal death. Bacterial meningitis affects 1 in 2500 live-born infants. Gastroenteritis is sporadic, depending on epidemic outbreaks. Local infections such as conjunctivitis and omphalitis occur commonly. Infection continues to be a significant factor in fetal and neonatal morbidity and mortality.

CARE MANAGEMENT

Assessment and Nursing Diagnoses

The prenatal record is reviewed for risk factors associated with infection and the signs and symptoms suggestive of infection. Maternal vaginal or perineal infection may be transmitted directly to the infant during passage through the birth canal. Psychosocial history and history of sexually transmitted infections (STIs) may indicate possible human immunodeficiency virus (HIV), hepatitis B virus (HBV), herpes (HSV-2), or CMV infection.

Perinatal events also are reviewed. Premature rupture of membranes (PROM) may be caused by maternal or intrauterine infection. Ascending infection may occur after prolonged PROM, prolonged labor, or intrauterine fetal monitoring. In some cases infection may occur with intact membranes or contribute to early rupture. A maternal history of fever during labor or the presence of foul-smelling amniotic fluid may also indicate the presence of infection. Antibiotic therapy initiated during labor should be noted. The neonate’s gestational age, maturity, birth weight, and sex all affect the incidence of infection. Sepsis occurs about twice as often and results in a higher mortality in male than in female infants. The neonate is assessed for respiratory distress, skin abscesses, rashes, and other indications of infection.

During the postnatal period, the time of onset of suspicious signs is noted. Onset within the first 48 hours of life is more often associated with prenatal or perinatal predisposing factors. Onset after 2 or 3 days more often reflects disease acquired at or subsequent to birth.

The earliest clinical signs of neonatal sepsis are characterized by a lack of specificity. The nonspecific signs include lethargy, poor feeding, poor weight gain, and irritability. The nurse or parent may simply note that the infant is just not doing as well as before. Differential diagnosis may be difficult because signs of sepsis are similar to signs of noninfectious neonatal problems such as hypoglycemia and stress. Additional clinical and laboratory information and appropriate cultures supplement the findings described. Table 27-3 outlines the clinical signs associated with neonatal sepsis.

Laboratory studies are important. Specimens for cultures include blood, CSF, stool, and urine. Fluids such as urine and CSF may be evaluated by counterimmuneelectrophoresis (CIE) or latex agglutination (LA) to assist in the identification of the bacteria. A complete blood cell count with differential is performed to determine the presence of bacterial infection or increased or decreased white blood cell (WBC) count (the latter is an ominous sign). The total neutrophil count, immature to total neutrophil (I:T) ratio, absolute neutrophil count (ANC), and C-reactive protein level may be used to determine the presence of sepsis (Table 27-4). Newer technology includes detection of viral DNA or antibodies by polymerase chain reaction (PCR) amplification in fluids (Frenkel, 2005). Detection of antepartal infection can now be successfully treated with a number of antiviral medications to decrease viral replication and fetal transmission of disease; neonates may also be treated with antiviral medications such as ganciclovir. Treatment with antibiotics is initiated after cultures are obtained in neonates. In high risk infants with significant illness, antiviral or antibiotic treatment may begin once cultures are obtained;
when the pathogen is identified, antibiotic therapy may be modified. Vigilant assessment continues during and after treatment. The newborn continues to be assessed for sequelae to septicemia, which include meningitis, disseminated intravascular coagulation (DIC), necrotizing enterocolitis, pneumonia, and septic shock. Septic shock results from the toxins released into the bloodstream. The most common signs include decreasing oxygen saturations, poor perfusion, tachycardia, respiratory distress, and hypotension.

Various nursing diagnoses are possible, depending on the infant’s gestational age and birth weight, the organ systems involved, and the nature of the infection. Examples of nursing diagnoses related to neonatal infections include the following:

**Newborn**

- Risk for infection related to
  - maternal vaginal (or other) infection
  - indwelling umbilical catheters, parenteral fluids (invasive procedures)
  - intraventricular electronic fetal monitoring
  - dysmaturity, IUGR, gestational age
- Ineffective thermoregulation related to
  - systemic infection

**TABLE 27-3**

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>SIGNS</th>
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<tbody>
<tr>
<td>Respiratory</td>
<td>Apnea, bradycardia</td>
</tr>
<tr>
<td></td>
<td>Tachypnea</td>
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<tr>
<td></td>
<td>Grunting, nasal flaring</td>
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<tr>
<td></td>
<td>Retractions</td>
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<td></td>
<td>Decreased oxygen saturation</td>
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<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Decreased cardiac output</td>
</tr>
<tr>
<td></td>
<td>Tachycardia</td>
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<tr>
<td></td>
<td>Hypotension</td>
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<tr>
<td></td>
<td>Decreased perfusion</td>
</tr>
<tr>
<td></td>
<td>Temperature instability</td>
</tr>
<tr>
<td></td>
<td>Lethargy</td>
</tr>
<tr>
<td></td>
<td>Hypotonia</td>
</tr>
<tr>
<td></td>
<td>Irritability, seizures</td>
</tr>
<tr>
<td>Central nervous</td>
<td>Feeding intolerance (decreased suck strength and intake; increasing residuals)</td>
</tr>
<tr>
<td></td>
<td>Abdominal distention</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Vomiting, diarrhea</td>
</tr>
<tr>
<td></td>
<td>Jaundice</td>
</tr>
<tr>
<td></td>
<td>Pallor</td>
</tr>
<tr>
<td></td>
<td>Petechiae</td>
</tr>
<tr>
<td>Integumentary</td>
<td>Mottling</td>
</tr>
</tbody>
</table>


*Laboratory findings include neutropenia, increased bands, hypoglycemia or hyperglycemia, metabolic acidosis, and thrombocytopenia.

- Impaired skin integrity related to
  - use of multiple supportive invasive measures (e.g., physiologic monitoring, parenteral fluid therapy, inhalation therapy)
- Acute pain related to
  - multiple supportive invasive measures

**Expected Outcomes**

Expected outcomes include the following:

- The newborn will remain free of infection.
- The newborn’s early signs of sepsis will be recognized, and appropriate therapy will be instituted.
- If therapy is necessary, the newborn will suffer no harmful sequelae.
- Parents will begin interacting and caring for newborn and be involved in his or her care.
- Parents will maintain self-esteem by understanding that their role as parents is important to the infant’s well-being.

**Plan of Care and Interventions**

**Prevention**

Virtually all controlled clinical trials have demonstrated that effective handwashing is responsible for the prevention of nosocomial infection in nursery units. Nursing is directly or indirectly responsible for minimizing or eliminating environmental sources of infectious agents in the nursery. Measures to be taken include Standard Precautions, careful and thorough cleaning of contaminated equipment, frequent replacement of used equipment (e.g., changing IV and nasogastric tubing per hospital protocol, and cleaning resuscitation and ventilation equipment, IV pumps, and incubators), and disposal of contaminated linens and diapers in an appropriate manner. Overcrowding must be avoided in nurseries. Guidelines for space, visitation, and general infection control in areas where newborns receive care have been established and published (American Academy of Pediatrics [AAP] & American College of Obstetricians and Gynecologists [ACOG], 2002).

Infants cared for in neonatal intensive care units (NICU) are at high risk for infection. There is center-to-center variability, with infection rates from 11.5% to 34% (Buus-Frank, 2004). Handwashing is the single most effective measure to reduce nosocomial infection. However, the rate of compliance with standards for hand hygiene is only...
The combined use of alcohol, hand hygiene, and gloves is effective in reducing the incidence of systemic infection (Buus-Frank, 2004). It is incumbent on caregivers to strictly adhere to recommended guidelines for hand hygiene. The skin, its secretions, and normal flora are natural defenses that protect against invading pathogens. Warm water may be used to remove blood and meconium from the neonate’s face, head, and body. A mild nonmedicated soap (in single-use container or in the form of a small bar reserved for a single newborn) can be used with careful water rinsing.

Artificial and long natural fingernails worn by nurses and other caregivers have been associated with serious neonatal infection and morbidity from Pseudomonas aeruginosa in the NICU (Moolenaar et al., 2000).

**CARE MANAGEMENT**

Breastfeeding or feeding the newborn breast milk from the mother is encouraged. Breast milk provides protective mechanisms. Colostrum contains IgA, which offers protection against infection in the GI tract. Human milk contains iron-binding protein that exerts a bacteriostatic effect on *E. coli*. Human milk also contains macrophages and lymphocytes. The vulnerability of infants to common mucosal pathogens such as respiratory syncytial virus (RSV) may be reduced by passive transfer of maternal immunity in the colostrum and breast milk. Some evidence indicates that early enteral feedings (trophic or minimal enteral feedings) may be beneficial in establishing a natural barrier to infection in extremely low-birth-weight (ELBW) and very low-birth-weight (VLBW) infants; further studies are needed to make general recommendations and establish protocols (Strodtbeck, 2003).

**TABLE 27-4**

<table>
<thead>
<tr>
<th>Suspected Neonatal Sepsis</th>
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<tr>
<td><strong>ASSESSMENTS</strong></td>
</tr>
<tr>
<td>1. Potential maternal risk factors and unstable vital signs, especially temperature instability</td>
</tr>
<tr>
<td>2. Sepsis screen in first hour (CBC with differential, platelets, and CRP level) if there are significant maternal risk factors (prolonged rupture of membranes, maternal temperature) or if infant demonstrates physiologic signs of sepsis</td>
</tr>
<tr>
<td><strong>TREATMENT</strong></td>
</tr>
<tr>
<td>1. Start IV administration of antibiotics by peripheral IV</td>
</tr>
<tr>
<td>2. Provide other treatments as needed for additional physiologic problems (supplemental oxygen or ventilator for respiratory distress, incubator for temperature instability)</td>
</tr>
<tr>
<td><strong>POSSIBLE CONSULTATIONS</strong></td>
</tr>
<tr>
<td>1. Neonatologists and advanced practice nurses for care of unstable infants</td>
</tr>
<tr>
<td>2. Medical specialists for care of infants with additional problems (congenital deformities)</td>
</tr>
<tr>
<td>3. Lactation consultant, interpreter, social worker, and chaplain as needed or requested</td>
</tr>
<tr>
<td><strong>ADDITIONAL ASSESSMENTS</strong></td>
</tr>
<tr>
<td>1. Weight and measurements</td>
</tr>
<tr>
<td>2. Blood culture, chest x-ray examination, urinalysis, and lumbar puncture, if infant is symptomatic or CRP level is positive</td>
</tr>
<tr>
<td>3. Repeat determination of CRP level in the morning for 2 days; if negative and infant not symptomatic, stop antibiotic treatment</td>
</tr>
<tr>
<td>4. Continuous cardiac and oxygen saturation monitor assessment if infant's condition is unstable</td>
</tr>
<tr>
<td><strong>DIRECT INFANT CARE</strong></td>
</tr>
<tr>
<td>1. Vital signs every 1 to 2 hr for the first 4 hr, then every 4 hr</td>
</tr>
<tr>
<td>2. Advance oral feedings as tolerated (infant on NPO status only if condition is physiologically unstable)</td>
</tr>
<tr>
<td>3. Bath and cord care done per unit protocols</td>
</tr>
<tr>
<td><strong>TEACHING AND DISCHARGE PLANNING</strong></td>
</tr>
<tr>
<td>1. Initiate on admission. Provide parents with written and oral information on suspected sepsis</td>
</tr>
<tr>
<td>2. Reinforce information and determine parents’ understanding of information before discharge. Include information on well-baby care and community follow-up with the family’s primary health care provider</td>
</tr>
</tbody>
</table>
TORCH Infections

The occurrence of certain maternal infections during early pregnancy is known to be associated with various congenital malformations and disorders. The most common and best understood infections are represented by the acronym TORCH (Box 27-1). One of the problems with these viral infections—TORCH infections—is the lack of maternal symptomatology, resulting in lack of treatment and thus often producing an affected newborn at birth. With the advent of newer diagnostic methods these viral infections may be diagnosed in utero and interventions planned based on the outcomes established for care are met.

Evaluation

The nurse can be reasonably assured that care was effective if the outcomes established for care are met.

TORCH Infections

The incidence of gonococcal infection in pregnant women ranges from 2.5% to 7.3%. With this high incidence, it is not surprising that neonatal infection with Neisseria gonorrhoeae occurs. After rupture of membranes, ascending infection can result in orogastric contamination of the fetus. The organism also may invade mucosal surfaces such as the conjunctiva (ophthalmitis neonatorum), rectal mucosa, and pharynx. Contamination may occur as the infant passes through the birth canal, or it may occur postnatally from an infected adult. Neonatal gonococcal arthritis, septicemia, meningitis, vaginitis, and scalp abscesses can also develop.

Eye prophylaxis (e.g., with 0.5% erythromycin ointment) is administered at or shortly after birth to prevent opth-
should be investigated for congenital syphilis. Infants born to women treated after 20 weeks of gestation when treatment is given in the third trimester; therefore, in- 

genital syphilis. Treatment failure can occur, particularly when treatment is given in the third trimester; therefore, infants born to women treated after 20 weeks of gestation should be investigated for congenital syphilis.

Fetal infection with the spirochete *Treponema pallidum* is blocked by Langhans’ layer in the chorion until this layer begins to atrophy at between 16 and 18 weeks of gestation. If spirochetemia is untreated, it will result in fetal death by midtrimester, miscarriage, or stillbirth (in one in four cases). All neonates in whom the infection occurs before 7 months of gestation are affected. Only 60% are affected if the in- 
fection occurs late in pregnancy. If maternal infection is treated adequately before the eighteenth week, neonates sel- 
dom demonstrate signs of the disease. Although treatment after the eighteenth week may cure fetal spirochetemia, pathologic changes may not be prevented completely.

Because the fetus becomes infected after the period of organogenesis (first trimester), organs develop normally. Congenital syphilis may stimulate preterm labor, but no ev- 

dence indicates that it causes IUGR. Organs affected later by congenital syphilis may include the liver, spleen, kidneys, adrenal glands, and bone covering and marrow. Disorders of the CNS, teeth, and cornea may not become evident un- 
til several months after birth.

The most severely affected infants are born to untreated mothers, and the newborn may be hydrotic (edematous) and anemic, with enlarged liver and spleen. Hepatosplenomegaly probably results from extramedullary hematopoietic activity stimulated by the severe anemia. In some infants, signs of congenital syphilis do not appear until late in the neonatal period. In these newborns, early signs such as poor feeding, slight hyperthermia, and snuffles may be nonspe- 
cific. The term *snuffles* refers to the copious, clear, serosan- 

guinous mucous discharge from the neonate’s nose. A mu- 
copurulent discharge indicates secondary infection, usually by streptococci or staphylococci.

By the end of the first week of life, a copper-colored maculopapular dermal rash appears in untreated newborns. The rash is characteristically first noticeable on the palms of the hands, soles of the feet (Fig. 27-4), and the diaper area and around the mouth and anus. The maculopapular lesions may become vesicular and confluent and extend over the trunk and extremities. Condylomata (i.e., elevated, wartlike lesions) may be seen around the anus. Rough, cracked, mucocutaneous lesions of the lips heal to form circumanal ra- 
diating scars known as *rhagades*.

If the mother was adequately treated before giving birth, and serologic testing of the infant does not show syphilis, generally the infant is not treated with antibiotics. The in- 
fant is checked for antibody titer (received from the mother through the placenta) every 2 weeks for 3 months, at which time the test result should be negative. Some physicians rec- 
ommend antibiotic therapy for asymptomatic or inconclu- 
sive cases.

A 10-day course of aqueous penicillin G or procaine peni- 
cillin G (consult drug references for dosage and adminis- 
tration route for each) is the usual treatment for congenital 

syphilis (Boyer & Boyer, 2004). Erythromycin is the substi- 
tute antibiotic of choice for infants sensitive to penicillin.

**NURSE ALERT** The infant born of a mother who has un- 
treated syphilis at the time of labor and birth will be highly contagious until after the first bath. It is therefore imperative that caregivers use Standard Precautions with all newborns.

In general, treatment of syphilis is more effective if it is begun early rather than later in the course of the disease. However, a recurrence rate of 5% can be expected. Even ade- 
quate treatment of congenital syphilis after birth does not always prevent late complication (e.g., 5 to 15 years after ini- 
tial infection). Potential complications include neurosyphilis, deafness, Hutchinson’s teeth (notched incisors), saber shins, joint involvement, saddle nose (depressed bridge), gammas (soft, gummy tumors) over the skin and other organs, and intussusception (inflammation of the cornea).

**Varicella-zoster**

The varicella-zoster virus, responsible for chickenpox and shingles, is a member of the herpes family. About 90% of
women in their childbearing years are immune, therefore the risk of infection in pregnancy is low—5 per 10,000 births (Cowles & Gonik, 2002).

Varicella transmission to the fetus may occur across the placenta when the disease is contracted in the first half of pregnancy, but this is relatively infrequent. When transmission to the fetus does occur in the early part of pregnancy, especially between weeks 13 and 20, the effects on the fetus include limb atrophy, neurologic abnormalities, eye abnormalities, and IUGR.

When maternal infection occurs in the last few days of pregnancy, 20% of infants born to these mothers will develop clinical varicella (Boyer & Boyer, 2004). The severity of the infant’s illness increases greatly if maternal infection occurred within 5 days before or 2 days after birth. The mortality in severe illness is 30% (Gibbs, Sweet, & Duff, 2004). Infants born to mothers who develop chickenpox between 5 days before birth and 48 hours after birth should be given varicella-zoster immune globulin (VZIG) at birth because of the risk of severe disease. Acyclovir can be used to treat infants with generalized involvement and pneumonia (Myers, Stanberry, & Seward, 2004). Infants exposed to chickenpox after birth will have either a mild infection or no infection if they are born to immune mothers. Those born to nonimmune mothers may develop chickenpox, but the course is not usually severe. Experts are divided as to whether this group of infants should receive VZIG. Infants younger than 28 weeks of age are at risk regardless of their mother’s status and probably benefit from VZIG if exposed to chickenpox.

Hepatitis B virus

HBV infection during pregnancy is not associated with an increase in maternal infections, stillbirths, or IUGR; however, about a 32% increase in risk exists for preterm birth. The transmission rate of HBV to the newborn is as high as 90% when the mother is seropositive for both hepatitis B surface antigen (HBsAg) and hepatitis B e antigen (HBeAg) (Duff, 1998). Transmission occurs transplacentally; serum to serum; and by contact with contaminated blood, urine, feces, saliva, semen, or vaginal secretions during birth. Infants are most commonly infected during birth or in the first few days of life. The rate of transmission is highest when the mother contracts the virus immediately before birth. These mothers will be positive for HBsAg. Transmission may occur through breast milk, but antibodies also develop in formula-fed infants at the same or a higher rate. Diagnosis is made by viral culture of amniotic fluid as well as the presence of HBsAg and IgM in the cord blood or newborn’s serum.

Neonatal and fetal effects are serious. Preterm birth exposes the neonate to the problems of prematurity. Infants may be symptom free at birth or may show evidence of acute hepatitis with changes in liver function. The mortality for full-blown hepatitis is 75%. Infants who become carriers are at high risk for chronic hepatitis, cirrhosis of the liver, or liver cancer even years later (Cowles & Gonik, 2002).

Infants whose mothers have antibodies for HBsAg or who have developed hepatitis during pregnancy or the postpartum period should be treated with hepatitis B immunoglobulin (HBIG), 0.5 milliliter IM, as soon as possible after birth or within the first 12 hours of life. The hepatitis B vaccine should also be given concurrently, but at a different site (AAP Committee on Infectious Diseases, 2003). The second dose of vaccine is given at 1 month, and the third dose at 6 months. The vaccine should protect the child for up to 9 years. After the infant has been cleansed thoroughly and has received the vaccine, breastfeeding may be initiated. Vaccination for infants not exposed to maternal HBV is recommended before discharge from the birth hospital; breastfeeding for these infants may begin before the vaccine is given.

Human immunodeficiency virus (HIV)

There were 790,000 children newly affected with HIV in 2003, mostly through mother-to-child transmission of HIV. Most (90%) of these infections occurred in sub-Saharan Africa. Fewer than 1000 children were estimated to become infected in North America and Western Europe during the same time. Globally, about 2.5 million children are living with the virus (World Health Organization, 2004). Universal counseling and screening of pregnant women is recommended in the United States and Canada.

Transmission of HIV from the mother to the infant may occur transplacentally at various gestational ages. The risk of infection in an infant born to an HIV-positive mother (not treated) is approximately 13% to 39% (AAP Committee on Infectious Diseases, 2003). Globally the rate of maternal transmission of the virus is estimated to be 25%. With antepartum, intrapartum, and neonatal zidovudine (ZDV) treatment the incidence of neonatal HIV infection is decreased to 5% to 8%, and compliance with highly active antiretroviral therapy (HAART) is said to further reduce newborn infection rates to 1% to 2% (Cooper et al., 2002; Knehs, 2002). A critical factor in perinatal transmission is the maternal viral load; a high viral load creates a greater chance for perinatal transmission of the virus. Postpartum transmission may also occur, with an additional risk of 14% attributed to breast milk contact (Weinberg, 2000).

Diagnosis of HIV infection in the neonate is complicated by the presence of maternal IgG antibodies, which cross the placenta after 32 weeks of gestation. The most accurate test for newborns and infants younger than 18 months is the HIV DNA PCR (deoxyribonucleic acid polymerase chain reaction) assay, which is performed on neonatal blood, not cord blood; results may be obtained by 24 hours (AAP Committee on Infectious Diseases, 2003). Follow-up testing for infants born to HIV-positive mothers is recommended at several intervals within the first year of life.

Typically the HIV-infected neonate is asymptomatic at birth. Early-onset illness (i.e., virus detected within 48 hours of birth) is attributed to prenatal infection and occurs in 10%
to 15% of infected infants. These infants develop opportunistic infections (Candida and Pneumocystis carinii pneumonia [PCP]) and rapid progression of immunodeficiency, which progresses to death in the first 1 to 2 years of life.

The remainder of infants seroconvert over a period of months to years. By 1 year of life, 80% to 90% of perinatally infected infants show signs of infection. Some children infected at birth show no signs of disease 8 to 10 years later. The age of onset of symptoms predicts the length of survival.

The presenting signs and symptoms of HIV infection vary from severe immunodeficiency to nonspecific findings such as failure to thrive, parotitis, and recurrent or persistent upper respiratory infections. In the first year of life, lymphadenopathy and hepatosplenomegaly are common. The infant may have fever, chronic diarrhea, chronic dermatitis, interstitial pneumonitis, persistent thrush, and AIDS-defining secondary infections. Common secondary infections include PCP, candidiasis, CMV infection, cryptosporidiosis, herpes simplex or herpes zoster, and disseminated varicella.

Although it is rare for an infant to be born with symptoms of HIV infection, all infants born to seropositive mothers should be presumed to be HIV positive until proven otherwise. Management begins by implementing Standard Precautions. Measures should also be undertaken to protect the infant from further exposure to maternal blood and body fluids. Regimens for the prevention of HIV transmission include antepartum, intrapartum, and neonatal treatment with HAART. Neonates may be treated with a combination of ZDV, didanosine, and nevirapine. In some cases lamivudine or stavudine may be used instead of didanosine for neonatal treatment (Bell, 2004). If the infant is diagnosed with HIV infection, the family should be counseled about conventional and investigational treatment options.

Counseling regarding the care of the mothers themselves, the family’s care of the infant, and future pregnancies should be provided. Social services are required in these cases. If the parent chooses to keep the infant, home health care may be arranged. The risk for transmission among members of the same household is minimal. For more information and updated information, parents are offered the following resources: the National AIDS Hotline, 1-888-342-AIDS.

In the United States, breastfeeding in the HIV-positive mother is contraindicated; however, in developing countries, the issue of risks versus benefits in relation to number of infant deaths attributed to poor sanitary conditions and availability of an appropriate food supply for infants and the theoretic risk of HIV transmission via breast milk is less clear (Krebs, 2002). HIV-infected women should avoid breastfeeding when replacement feeding is available, affordable, and safe. Otherwise, the recommendation is for exclusive breastfeeding during the first month of life (WHO, 2005).

The family must be counseled about vaccinations. Children with symptomatic or asymptomatic HIV infection should receive all routine vaccines. Although data regarding children with HIV and varicella vaccine are limited, the AAP Committee on Infectious Diseases (2003) recommends that children with no or mild symptoms be immunized for varicella.

**Rubella infection**

Since rubella vaccination was begun in 1969, cases of congenital rubella have been reduced dramatically; however, it is still seen occasionally in the newborn. Vaccination failures, lack of compliance, and the immigration of nonimmunized persons result in periodic outbreaks of rubella, also known as German measles or 3-day measles.

The risk for congenital anomalies varies with the gestational age of the fetus at the time maternal infection occurs. Abnormalities are most severe if the mother contracts the virus during the first trimester.

More than two-thirds of infected infants have no symptoms apparent at birth, but sequelae may develop years later. Hearing loss, the most common result, appears to be progressive after birth. Initially the newborn may present with hepatosplenomegaly, lymphedema, IUGR, jaundice, hepatitis, thrombocytopenic purpura with petechiae, and the characteristic blueberry muffin lesions. Congenital rubella syndrome often includes chronic problems such as cataracts or glaucoma, sensorineural hearing impairment, hypogammaglobulinemia, peripheral pulmonary stenosis, and diabetes mellitus type 1 (Boyer & Boyer, 2004). The rubella virus has been cultured in infants for up to 18 months after their birth. These infants are a serious source of infection to susceptible individuals, particularly women in the childbearing years. Extended pediatric isolation is mandatory until the noncontagious stage of rubella has been reached (i.e., the infant should be isolated until pharyngeal mucus and the urine are free of virus).

**Cytomegalovirus infection**

Cytomegalovirus (CMV) infection during pregnancy may result in miscarriage, stillbirth, or congenital illness. It is the most common cause of congenital viral infections in the United States (Boyer & Boyer, 2004). Most (90% to 95%) of the affected infants are asymptomatic at birth; however, sensorineural hearing impairment and learning disabilities have been reported in previously asymptomatic infants.

The neonate with classic, full-blown CMV displays IUGR and has microcephaly. The neonate may also have a rash, jaundice, and hepatosplenomegaly (Fig. 27-5). Anemia, thrombocytopenia, and hyperbilirubinemia are common in the early stages of the illness. Intracranial, periventricular calcification often is noted on radiography. Inclusion bodies (“owl’s eye” figures) in cells sedimented from freshly voided urine or in liver biopsy specimens are typical.

The virus may be isolated from urine or saliva of the newborn using the PCR assay. Differential diagnosis includes other causes of jaundice, pyelitis (positive Veneral Disease Research Laboratories [VDRL] findings), toxoplasmosis (positive Sabin-Feldman dye test result), hemolytic disease of the newborn (positive Coombs’ test reaction), or conchosarcoma virus infection (positive culture).
Congenital infection is rare and is characterized by intrauterine destruction of normally formed organs. Affected infants are growth restricted. They have severe psychomotor restriction, with intracranial calcifications, microcephaly, hypertonicity, and seizures. They suffer eye involvement, including microphthalmus, cataracts, chorioretinitis, blindness, and retinal dysplasia. Some infants have patent ductus arteriosus, limb anomalies, and recurrent skin vesicles, with a short life expectancy.

Most infants are infected directly during passage through the birth canal. The risk of infection during vaginal birth in the presence of genital herpes has not been clearly delineated. It may be as high as 33% to 50%, with active primary infection at term. Primary maternal infections after 32 weeks of gestation carry a higher risk for the fetus and newborn than do recurrent infections (Baley & Toltzis, 2002). The transmission rate of chronic vaginal herpes from the pregnant woman to her newborn is low. Passive intrauterine immunity to herpes may be responsible.

Postnatal acquisition of the virus and spread within a nursery have been documented by DNA analysis. Both mother and father, as well as maternal breast lesions, have been implicated in neonatal infections. There also is concern regarding symptomatic and asymptomatic shedding among hospital personnel. Nursery personnel with cold sores should practice strict handwashing and wear a mask, but no evidence indicates they should be removed from the nursery unless they have a herpetic whitlow (primary HSV infection of the terminal segment of a finger).

Clinically, neonatal HSV infections are classified as disseminated infection; localized CNS disease; or localized infection of the skin, eye, or mouth. Disseminated infections may involve virtually every organ system, but those primarily involved are the liver, adrenal glands, and lungs. Affected infants exhibit initial symptoms usually in the first week of life but sometimes in the second week, with signs of bacterial sepsis or shock. Clinical manifestations include skin vesicles in about 33% of infants (Fig. 27-6). Death results from progression of CNS involvement, respiratory distress and pneumonitis, shock, DIC, and bleeding. The risk of serious sequelae or death in disseminated infections is approximately 50% (Gibbs, Sweet, & Duff, 2004).

Standard Precautions should be observed when caregivers have contact with these infants. The neonate’s eyes, oral cavity, and skin are inspected carefully for the presence of any lesions (Fig. 27-7). Cultures are obtained from the mouth, eyes, and lesions. Circumcision, if performed, is delayed until the infant is ready to be discharged. The infant may be discharged with the mother if the infant’s cultures are negative for the virus. As long as no suspicious lesions are on the mother’s breasts, breastfeeding is allowed. For the infant at risk, a prophylactic topical eye ointment (vidarabine or trifluridine) is administered for 5 days to prevent keratoconjunctivitis. Acyclovir should also be given to infants with ocular manifestation. No current recommendations exist for prophylactic systemic therapy; each case should be consid-
Blood, urine, and CSF specimens should be cultured when indicated clinically. If herpetic lesions first occur after 6 weeks of life, the risk of dissemination and severe illness is very low (Baley & Tolzis, 2002). Therapy includes general supportive measures, as well as treatment with IV acyclovir. Acyclovir is the most commonly used and recommended drug for treatment of HSV. It is considered safe because only viral replication is inhibited, although long-term sequelae are not yet known. Although acyclovir is easier to administer than vidarabine, there is no difference between the two drugs regarding treatment of HSV. Continuing therapy may be required in recurrences. Ophthalmic ointment should be administered simultaneously.

**Parvovirus B19**

Parvovirus B19 is well-known in older children as fifth disease or “slapped-cheek illness” because of the characteristic facial appearance of the affected child. During pregnancy infection may result in fetal miscarriage or the development of non-immune fetal hydrops. The estimated risk of transplacental transmission is approximately 30%, and fetal death may occur in about 9% of those affected (Boyer & Boyer, 2004). Protocols for intrauterine management have not been well developed, but intrauterine transfusion has offered limited success. Serial ultrasounds to detect fetal hydrops are possible. The virus may be isolated from amniotic fluid, fetal blood, or tissues using DNA PCR assay (Boyer & Boyer, 2004). Pericardial, pleural, and peritoneal effusions are common and fatal if not treated immediately, with cardiac failure from anemia being the most common cause of death.

**Bacterial Infections**

**Group B streptococcus**

Until recently GBS has been the most common cause of neonatal sepsis and meningitis in the United States; however, antepartum maternal screening and administration of penicillin has significantly decreased the incidence of GBS. Early-onset GBS infection in the neonate occurs in the first 7 days of life but most commonly manifests in the first 24 hours following birth. Risk factors for the development of early-onset GBS infection include low birth weight, preterm birth, rupture of membranes of more than 18 hours, maternal fever, previous GBS-infected infant, maternal GBS bacteriuria, and multiple gestation. Usually resulting from vertical transmission from the birth canal, early-onset disease results in a respiratory illness that mimics the symptoms of severe respiratory distress syndrome. The infant may rapidly develop septic shock, which has a significant mortality rate. Late-onset GBS infection manifests between 1 week and 3 months of age, with an average age of onset of 24 days. Of infants with late-onset GBS, 85% have meningitis; this population has a mortality rate of 0% to 23%. Fifty percent of the survivors develop neurologic damage.

**Escherichia coli**

E. coli is the second most common cause of neonatal sepsis and meningitis in the United States, although some preliminary reports suggest that this organism has increased in some NICUs (Stoll et al., 2002). E. coli is found in the GI tract soon after birth and makes up the bulk of human fecal flora. In addition to meningitis, E. coli can also cause infections in other body systems, including the urinary tract. There is...
affected area is intensely erythematous, with a sharply demarcated, scalloped edge, often with numerous satellite lesions that extend beyond the larger lesion. The source of the infection can be through the GI tract or caretakers’ hands. Oral candidiasis (thrush or mycotic stomatitis) is characterized by the appearance of white plaques on the oral mucosa, gums, and tongue. The white patches are easily differentiated from milk curds; the patches cannot be removed and tend to bleed when touched. In most cases the infant does not seem to be in discomfort from the infection; however, some will pull away from the breast or bottle and cry. The child may be brought to the health care provider with a complaint of poor oral intake.

Infants who are sick, debilitated, or receiving prolonged antibiotic therapy are more susceptible to thrush. Those with conditions such as cleft lip or palate, neoplasms, and hyperparathyroidism seem to be more vulnerable to mycotic infection.

The objectives of management are to eradicate the causative organism and to control exposure to C. albicans. Interventions include maintenance of scrupulous cleanliness (by nursing personnel, parents, and others) to prevent reinfection. Good handwashing technique is imperative. Clean surfaces should be provided for neonates. Proper cleanliness of the equipment and environment is critical. Diaper dermatitis is treated with a topical fungicide at each diaper change. When possible, exposing the perineal area to dry air is recommended because yeast prefers a moist environment.

Topical application of 1 ml of nystatin (Mycostatin) over the surfaces of the oral cavity four times a day, or every 6 hours, is usually sufficient to prevent spread of the disease or prolongation of its course. Several other drugs may be used, including amphotericin B (Fungizone), clotrimazole (Lotrimin, Mycelex), or miconazole (Monistat, Micatin). Gentian violet solution may be used in addition to one of the antifungal drugs in chronic cases of oral thrush; however, the former does not treat GI Candida and may be irritating to the oral mucosa.

**Nurse Alert** Nystatin is best absorbed when given either 1 hour before feeding or after a feeding. Using a needleless syringe or medicine dropper, apply the medication to each side of the infant’s mouth for optimal absorption.

Infants who are breastfed may acquire thrush from the mother; in the event that the mother is colonized, treatment for mother and infant is recommended. There is no need to stop breastfeeding even if the mother is receiving systemic antifungal medications (Lawrence & Lawrence, 2005).

**Substance Abuse**

Certain maternal behaviors result in perinatal risk. Maternal habits hazardous to the fetus and neonate include rece-
ational drug abuse, smoking, and alcohol abuse. Physiologic signs of withdrawal have been reported in neonates of mothers who use to excess such drugs as barbiturates, alcohol, or amphetamines. Prescription opioids such as oxycodone (Perco
codan) have been identified as increasingly popular drugs of abuse that may cause withdrawal symptoms in neonates (Rao & Desai, 2002). Serious withdrawal reactions are seen in neonates whose mothers abuse psychoactive drugs. Mothers receiving methadone in substance abuse treatment may give birth to an infant who exhibits withdrawal symptoms requiring treatment. Almost 50% of pregnancies of women addicted to opioids result in low-birth-weight (LBW) infants who are not necessarily preterm. Alcohol is a teratogen that produces CNS effects that may not be evident for years.

It is important to note that the term addiction is often associated with behaviors whereby the person seeks the drug(s) to experience a high or euphoria, to escape from reality, or to satisfy a personal need. Newborns who have been exposed to drugs in utero are not addicted in a behavioral sense, yet they may experience mild to strong physiologic signs as a result of the exposure. Therefore, to say that an infant born to a mother who uses substances is addicted is incorrect; drug-exposed newborn, which implies intrauterine drug exposure, is a better term.

The adverse effects of exposure of the fetus to drugs are varied. They include transient behavioral changes such as fetal breathing movements and irreversible effects such as fetal death, IUGR, structural malformations, cognitive and motor delay, and behavioral problems. Critical determinants of the effect of the drug on the fetus include the specific drug, the dosage, the route of administration, the genotype of the mother or fetus, and the timing of the drug exposure.

Fig. 27-8 shows critical periods in human embryogenesis and the teratogenic effects of drugs. Table 27-5 summarizes the effects of commonly abused substances on the fetus and neonate.

Alcohol

Maternal ethanol abuse during gestation can lead to a readily identifiable alcohol-related birth defects (ARBD), fetal alcohol syndrome (FAS) or a constellation of neurobehavioral and cognitive problems which may only be identified by maternal history and behavioral characteristics.

The incidence of ARBD in the United States is about 0.2 to 1.5 per 1000 live births (CDC, 2004). The incidence of ARBD in the United States is about 0.2 to 1.5 per 1000 live births (CDC, 2004). ARBD is based on minimum criteria of signs in each of three categories: prenatal and postnatal growth restriction; CNS malfunctions, including mental retardation; and craniofacial features such as microcephaly, small eyes or short palpebral fissures, thin upper lip, flat midface, and an indistinct philtrum (Fig. 27-9) (Dunbar, 2003). Neurologic problems in ARBD children include some degree of intelligence quotient (IQ) deficit, attention deficit disorder, diminished fine motor skills, and poor speech (Jones & Bass, 2003). Infants exposed prenatally to alcohol who are affected but do not meet the criteria for ARBD may be said to have alcohol-related neurodevelopmental disorder (ARND), formerly referred to as fetal alcohol effects (FAE) (CDC, 2004). These effects range from learning disabilities and behavioral problems to speech or language problems and hyperactivity. Often these problems are not detected until the child goes to school and learning problems become evident. Predictable abnormal patterns of fetal and neonatal morphogenesis are often attributed to severe, chronic alcoholism in women who continue to drink heavily.
during pregnancy; however, the amount of alcohol consumption does not always correlate with identifiable features. Rather, it is the amount of alcohol consumed in excess of the maternal liver’s ability to detoxify alcohol that defines what manifestations or effects will be evidenced from one child to another. The pattern of growth restriction begun in prenatal life persists after birth, especially in the linear growth rate, rate of weight gain, and growth of head circumference.

Ocular structural anomalies are common findings. Limb anomalies and various cardiocirculatory anomalies, especially ventricular septal defects, pose problems for the child. Table 27-6 outlines physical findings in ARBD. Mental retardation (e.g., IQ of 79 or below at 7 years of age), hyperactivity, and fine motor dysfunction (e.g., poor hand-to-mouth coordination, weak grasp) add to the handicapping problems that maternal alcoholism can impose. Genital abnormalities are seen in daughters of alcohol-addicted mothers. Two thirds of newborns with ARBD are girls; the cause of this altered fetal sex ratio is unknown. Severe and chronic alcoholism (ethanol toxicity), not maternal malnutrition, is responsible for the severity and consistency of postnatal performance problems. Other disorders include recurrent otitis media and hearing loss. Craniofacial features may be important in diagnosing craniofacial and oral anomalies, dental development abnormalities, and long-term body growth patterns. Feeding difficulties are related to preterm birth, poor sucking ability, and possible

during pregnancy; however, the amount of alcohol consumption does not always correlate with identifiable features. Rather, it is the amount of alcohol consumed in excess of the maternal liver’s ability to detoxify alcohol that defines what manifestations or effects will be evidenced from one child to another. The pattern of growth restriction begun in prenatal life persists after birth, especially in the linear growth rate, rate of weight gain, and growth of head circumference.

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### Table 27-5

<table>
<thead>
<tr>
<th>Substance</th>
<th>Neonatal Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>Alcohol-related birth defects (ARBD) fetal alcohol syndrome (FAS): craniofacial features vary, may include short eyelid opening, flat midface, flat upper lip groove, thin upper lip; microcephaly; hyperactivity; developmental delays; attention deficits Alcohol-related neurodevelopmental disorder (ARND): varying forms of ARBD, cognitive, behavioral, and psychosocial problems without typical physical features</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Prematurity, small size for gestational age, microcephaly, poor feeding, irregular sleep patterns, diarrhea, visual attention problems, hyperactivity, difficulty in consoling, hypersensitivity to noise and external stimuli, irritability, developmental delays, congenital anomalies such as prune belly syndrome (i.e., distended, flabby, wrinkled abdomen caused by lack of abdominal muscles)</td>
</tr>
<tr>
<td>Heroin</td>
<td>Low birth weight, small size for gestational age, irritability, tachypnea, feeding difficulties, vomiting, high-pitched cry, seizures</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>Small size for gestational age, prematurity, poor weight gain, lethargy, behavioral problems later in childhood</td>
</tr>
<tr>
<td>Tobacco</td>
<td>Prematurity; low birth weight; increased risk for sudden infant death syndrome; increased risk for bronchitis, pneumonia, developmental delays</td>
</tr>
<tr>
<td>Marijuana</td>
<td>Possible neonatal tremors, low birth weight, growth restriction</td>
</tr>
</tbody>
</table>

The infant of a mother who abuses alcohol is faced with many clinical problems. Identification of the problems leads to the medical diagnosis of ARBD. The infant may suffer respiratory distress related to preterm birth, neurologic damage, and a “floppy” epiglottis and small trachea. Tracheoepiglottal anomalies may cause cardiopulmonary arrest. Other disorders include recurrent otitis media and hearing loss. Craniofacial features may be important in diagnosing craniofacial and oral anomalies, dental development abnormalities, and long-term body growth patterns. Feeding difficulties are related to preterm birth, poor sucking ability, and possible

![Fig. 27-9](https://via.placeholder.com/150)
Features of Alcohol-Related Birth Defects

<table>
<thead>
<tr>
<th>Affected Part</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eyes</td>
<td>Epicanthal folds, strabismus, ptosis, hypoplastic retinal vessels</td>
</tr>
<tr>
<td>Mouth</td>
<td>Poor suck, cleft lip, cleft palate, small teeth</td>
</tr>
<tr>
<td>Ears</td>
<td>Sensorineural hearing deficits</td>
</tr>
<tr>
<td>Skeleton</td>
<td>Radioulnar synostosis, fusion of cervical vertebral, restricted bone growth</td>
</tr>
<tr>
<td>Heart</td>
<td>Atrial and ventricular septal defects, tetralogy of Fallot, patent ductus arteriosus</td>
</tr>
<tr>
<td>Kidney</td>
<td>Renal hypoplasia, hydronephrosis, urogenital sinus</td>
</tr>
<tr>
<td>Liver</td>
<td>Extrarenal biliary atresia, hepatic fibrosis</td>
</tr>
<tr>
<td>Immune system</td>
<td>Increased infections: otitis media, upper respiratory infections, immune deficiencies</td>
</tr>
<tr>
<td>Tumors</td>
<td>Nonspecific neoplasms</td>
</tr>
<tr>
<td>Skin</td>
<td>Abnormal palmar creases, irregular hair, whorls</td>
</tr>
</tbody>
</table>


The Newborn at Risk: Acquired and Congenital Problems

Marijuana

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 (Ebrahim & Gfoerer, 2004). Marijuana crosses the placenta. Its use during pregnancy may result in a shortened gestation and a higher incidence of IUGR. A strong association has been reported between the use of marijuana and a decrease in fetal growth and infant birth weight and length (Hurd et al., 2005). Other investigators have found a higher incidence of meconium staining (Rosen & Bateman, 2002). Compounding the issue of the effects of marijuana, especially among women ages 18 to 30 years (Ebrahim & Gfoerer, 2004), is polydrug use, which combines the harmful effects of marijuana, tobacco, alcohol, opiates, and cocaine. Long-term follow-up studies on exposed infants are needed.

Cocaine

Cocaine, a common illicit drug used in the United States, has multiple modes of use. However, use of the relatively inexpensive and easily administered “crack” form is increasingly common, especially among women of childbearing age. Because crack vaporizes at relatively low temperatures, it is

Tobacco

Cigarette smoking in pregnancy is associated with birth weight deficits of up to 250 g for a full-term neonate (Azanda, Edwards, Hales, & Riedel, 2002). Maternal cigarette smoking is implicated in 21% to 39% of LBW infants. Passive exposure to secondhand smoke by a pregnant woman may also result in the birth of an LBW infant. The rate of miscarriage and preterm birth is increased in the smoking population. Nicotine and cotinine, the two pharmacologically active substances in tobacco, are found in higher concentrations in infants whose mothers smoke. These substances can be sequestered in breast milk for up to 2 hours after the mother has smoked. Cigarette smoke contains more than 2000 compounds, including carbon monoxide, dioxin, cyanide, and cadmium. Deficits in growth and intellectual and emotional development, poor auditory responsiveness, increased fine motor tremors, hypertonicity, and decreased verbal comprehension have been observed in infants exposed to smoke. There is also a positive dose-response relationship between the amount of tobacco exposure and newborn neurobehavior; increased tobacco exposure in utero is related to increasing negative neurobehavioral effects (Law et al., 2003). In addition, it is now recognized that neonates may experience withdrawal symptoms after exposure to nicotine. Pregnant women must be informed about the harmful effects of smoking on their unborn baby’s health. These include IUGR, miscarriage, PROM, placenta previa, perinatal death, LBW, deficits in learning and behavior, and sudden infant death syndrome (SIDS) (Law et al., 2003). The positive association between maternal smoking and SIDS reflects in utero exposure and passive exposure postnatally. Mothers and all others should refrain from smoking near the infant. Smoking cessation during pregnancy greatly decreases the chance of fetal complications; therefore women should be counseled regarding smoking cessation programs.

In conclusion, the newborn at risk is frequently the victim of poor nutrition, smoking, alcohol, and drug abuse. Prevention of these factors begins with proper education, prenatal care, and involvement of the parents. Furthermore, the interdisciplinary team must take a holistic approach to provide the best care possible for these infants.
significant differences were noted in mental, psychomotor, or language scores (Singer et al., 2004). In a large controlled study of children exposed to cocaine and opiates in utero, only significant differences in the expressive, receptive, and total language, motor, and cognitive scores in some studies (Singer et al., 2002); however, in one study there were no significant differences in the expressive, receptive, and total language scores (Singer et al., 2004). In a large controlled study of children exposed to cocaine and opiates in utero, only subtle deficiencies in mental and psychomotor functioning were noted at 3 years of age (Messinger et al., 2004). No significant differences were noted in mental, psychomotor, or behavioral functioning. The environmental factors to which these children were exposed were perceived as an important factor in their development. Further long-term studies of exposed infants were recommended (Messinger et al., 2004).

Nursing care of cocaine-exposed infants is the same as that for other drug-exposed infants. Because they have increased flexor tone, these infants respond to swaddling in a semiflexed position (Astrip & Diehl-Jones, 2001). Positioning, infant massage, and limited tactile stimulation have been shown to be effective interventions. Cocaine enters the breast milk; mothers should be cautioned about this hazard to their infants. Referral to early intervention programs that offer comprehensive care, including child health care, parental drug treatment, individualized developmental care, and parenting education, is essential in promoting the optimum outcome for these children. Because affected children often live in an impoverished environment, they are at high risk for cognitive delays, lack of child health care, and inadequate nutrition.

Phencyclidine ("Angel Dust")
Phencyclidine (PCP) increases the risk of injury to the pregnant woman and therefore also to her fetus. The user may be unaware that she is ingesting PCP because it often is misrepresented as another drug of abuse or is mixed with other drugs.

PCP crosses the placenta and is found in breast milk. Literature about the effects on infants is limited. Infants exposed to PCP may exhibit abnormal motor behavior such as irritability, jitteriness, and hypertonicity.

Heroin
Heroin crosses the placenta and often results in IUGR. Heroin may have a direct growth-inhibiting effect on the fetus, but the exact mechanisms of growth inhibition are not clear. There is an increased rate of stillbirths but not of congenital anomalies.

Many of the medical complications attributed to heroin ingestion result from prematurity. Other risks include physical dependence in the fetus and the risk of exposure to infections, including hepatitis B and C virus and HIV.

Drug withdrawal in the mother is accompanied by fetal withdrawal, which can lead to fetal death. Maternal detoxification in the first trimester carries an increased risk of miscarriage. Detoxification is not recommended after the thirty-second week because of possible withdrawal-induced fetal distress. Heroin withdrawal occurs in 50% to 75% of infants born to addicted mothers, usually within the first 24 to 48 hours of life (Rosen & Bateman, 2002). The signs depend on the length of maternal addiction, the amount of drug taken, and the time of injection before birth. The infant whose mother is taking methadone may not demonstrate signs of withdrawal until a week or so after birth. The symptoms of
EVIDENCE-BASED PRACTICE
Kangaroo Care for Low-Birth-Weight Infants

BACKGROUND

• Low-birth-weight (LBW) babies (~2500 g, regardless of gestational age) are at greater risk for diseases; mortality; and possibly diseases as adults. Most infant deaths occur in this group worldwide. Medical care is costly and scarce in developing countries. A low-technology, low-cost intervention to improve the outcomes for LBW infants is an important advance. Kangaroo mother care (KMC) combines skin-to-skin contact between mother and infant, frequent breastfeeding, and early discharge from the hospital. Skin-to-skin contact has been shown to significantly increase and stabilize infant temperature, decrease respirations, increase blood glucose, and improve breastfeeding duration and lasting maternal-infant bonding. (See “Early Maternal Skin-to-Skin Contact with Healthy Infants,” the Evidence-Based Practice box in Chapter 18.) Babies in KMC are secured between their mother’s breasts in an upright position, day and night. Infants are not eligible for this intervention until they have demonstrated respiratory, temperature, and feeding stabilization (exclusive breastfeeding or a combination of gavage and breastfeeding).

OBJECTIVES

• The review committee sought evidence that would assess the beneficial and adverse effects of KMC on infants born weighing less than 2500 g, regardless of gestational age. Specific research questions included the effect of KMC on mortality, illness, infant growth, infection, admission to neonatal intensive care units (NICUs), breastfeeding, length of stay, costs, and satisfaction of parents and staff.

METHODS

Search Strategy

• The reviewers searched the Cochrane Library, MEDLINE, EMBASE, LILACS, PPOLINE, and CINAHL databases. Key words were kangaroo mother care, skin-to-skin, infants, and low-birth-weight infants. The reviewers found three randomized, controlled trials, involving 1362 infants from Ecuador, Colombia, Ethiopia, Indonesia, and Mexico, published from 1994 to 1998. All three studies used skin-to-skin contact and exclusive or nearly exclusive breastfeeding. Early hospital discharge was considered in only one study. Controls received standard neonatal care, including incubator use.

Statistical Analyses

• Statistical analyses allowed comparison of data at 41 weeks corrected gestational age, discharge, 6 months corrected age, and 12 months corrected age. Discharge may have occurred before 41 weeks corrected gestational age. “Corrected age” is counted not from the preterm birth, but from 41 weeks corrected gestational age, which would have been the term due date. Reviewers calculated relative risks for dichotomous (categoric) data, and weighted mean differences for continuous data. The authors accepted differences outside the 95% confidence intervals as significant.

FINDINGS

• No difference was found in infant mortality between the KMC and the control groups. Most mortality occurred during the stabilization process before eligibility for the study. Reviewers found a significant decrease in nosocomial (hospital acquired) infection at 41 weeks corrected gestational age. There was significantly less severe illness and lower hospital costs in the KMC group. No difference in readmissions between groups was noted. Weight and head circumference at discharge were significantly higher in the KMC group, but the difference was lost by term and 12 months. There was no difference between psychomotor skills at 12 months. Mothers felt competent and significantly more satisfied with their caregiving in the KMC group, but felt less social support regarding the NICU, although both groups were similar in perception of social support from the hospital, worry, stress, sensitivity, and infant responsiveness from mother. Infant temperatures were more stable in the KMC group. Hospital length of stay was variable, with one study reporting the KMC group had a shorter stay, and another study reporting longer stays than controls. Cost was lower for KMC, but there was not enough information to determine if this was significant. Most of the cost came during the stabilization period before enrollment in the study.

LIMITATIONS

• Patients, staff, and evaluators were fully aware of the group into which they were randomized, which could introduce bias or some other confounding influence. The definition of stabilization was not clarified. This could affect the outcomes because a more immature infant is more fragile. Missing and incomplete information regarding costs limited the ability to analyze this important outcome. All three studies were carried out in developing countries. Kangaroo Care for Low-Birth-Weight Infants

CONCLUSIONS

• The authors conclude that KMC appears to both reduce severe infant morbidity and to have no adverse outcomes, but the available research has methodologic problems that limit its usefulness.

IMPLICATIONS FOR PRACTICE

• The reviewers conclude that evidence to recommend the routine use of KMC in LBW infants is insufficient.

IMPLICATIONS FOR FURTHER RESEARCH

• Well-designed, randomized controlled trials that account for lack of concealment and dropouts can provide higher quality evidence to recommend this promising intervention. While developing countries stand to benefit from evidence that this low-technology, low-cost method can benefit LBW infants, it would be informative to have data from developed countries for comparison.

infants whose mothers used heroin or methadone are similar. Initially the infant may be depressed. The withdrawal syndrome may manifest as a combination of any of the following signs:

- Infant may be jittery and hyperactive.
- Cry is shrill and persistent.
- Infant may yawn or sneeze frequently.
- Tendon reflexes are increased but the Moro reflex is decreased.
- Neonate may exhibit poor feeding and sucking, tachypnea, vomiting, diarrhea, hyperthermia or hypothermia, and sweating.
- Infant may exhibit abnormal sleep cycle, with absence of quiet sleep and disturbance of active sleep.

The risk of SIDS is 5 to 10 times higher for infants with significant withdrawal problems than for infants in the general population.

If withdrawal is not treated, vomiting, diarrhea, dehydration, apnea, and convulsions may develop. Death may follow. Therapy is individualized. Dehydration and electrolyte imbalance are prevented or treated. Usually the following drugs are given, singly or in combination: phenobarbital, diluted tincture of opium (paregoric), methadone, or morphine.

Buprenorphine, a partial morphine agonist, reduces opiate use. It is being used in Europe and will soon be used in the United States. Infants born to mothers on buprenorphine have a lower incidence of small size for gestational age and a milder and shorter course of abstinence syndrome (Rosen & Bateman, 2002).

NURSE ALERT The use of naloxone (Narcan) is contraindicated in infants born to narcotic addicts because it may exacerbate narcotic abstinence syndrome and cause seizures.

Methadone

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, motting, and nasal stuffiness (Jansson, Vezer, & 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 AAP Committee on Drugs (2001) has revised its statement regarding breastfeeding for mothers who are in a methadone treatment program, suggesting that such mothers be allowed to breastfeed regardless of the methadone treatment 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 (Rosen & Bateman, 2002). 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.

Miscellaneous Substances

Methamphetamine

The fetal and neonatal effects of maternal use of methamphetamines in pregnancy are not well known but appear to be dose related (Smith et al., 2003). LBW, preterm birth, and perinatal mortality may be consequences of higher doses used throughout pregnancy. In addition, a higher incidence of cleft lip and palate and cardiac defects has been reported in infants exposed to methamphetamines in utero (Plessinger, 1998).

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

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 et al., 2003). After birth, infants may experience bradycardia or tachycardia that resolve as the drug is cleared from the infant’s 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 be seen during early childhood.

Phenobarbital

Phenobarbital crosses the placenta readily and is subsequently found in high levels in the fetal liver and brain. Because of its slow metabolic rate, withdrawal onset is generally 2 to 14 days after birth and duration is about 2 to 4 months. Irritability, crying, hiccuping, and sleepiness mark the initial response. During the second stage, the infant is extremely hungry, regurgitates and gas frequently, and demonstrates episodic irritability, sweating, and a disturbed sleep pattern.
Caffeine

Caffeine has not been implicated as a teratogen in humans. After a thorough review of the literature in print, Christian and Brent (2001) concluded that caffeine is a potential teratogen only when used with alcohol or tobacco or in very large amounts. Most published studies indicate an increased risk of fetal growth delay in women who consume more than 300 mg/day (three to four cups of coffee) (Andres, 2004).

Polydrug use

D’Apolito and Hepworth (2001) studied a small group (14) of infants exposed to multiple drugs (polydrug) in utero; these included opioids, stimulants, depressants, and sedatives. The most common symptoms observed were increased tone, increased respiratory rate, disturbed sleep, fever, frantic and increased sucking, and loose or watery stools. These findings are significant for nurses working in neonatal and obstetric areas; the presence of such findings may alert the nurse so documentation of events (using the Neonatal Abstinence Scoring [NAS] tool or other objective measure) may take place and therapy may be promptly implemented. Initial nursing interventions such as providing a quiet environment and offering a pacifier for frantic and excessive sucking may be implemented independently. It is important not to overfeed infants who demand frequent sucking as part of the withdrawal process.

CARE MANAGEMENT

Assessment and Nursing Diagnoses

Assessment of the newborn requires a review of the mother’s prenatal record. A medical and social history of drug abuse and detoxification is noted. The infant may have IUGR or be preterm with LBW.

The woman who is abusing chemical substances may have infections that compound the risk to the infant, including hepatitis B; septicemia; and STIs, including HIV-positive status.

The nurse often is the first to observe the signs of drug withdrawal in the infant. In many cases the newborn may be discharged before the appearance of any manifestations of withdrawal. The infant is assessed by means of the guidelines discussed in Chapter 19. The infant’s gestational age and maturity are noted. In utero exposure to some drugs results in observable malformations or dysmorphism (abnormality of shape). Neonatal behavior may arouse suspicion.

Neonatal abstinence syndrome is the term used to describe the set of behaviors exhibited by the infant exposed to chemical substances in utero (Table 27-7). Fig. 27-10 provides an example of a Neonatal Abstinence Scoring system for assessing withdrawal symptoms. Because many women are polydrug users, the newborn initially may exhibit a variety of withdrawal manifestations. 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 (NNNS) was developed by the National Institutes of Health (NIH) and 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 (see Fig. 27-10), and a complete neurologic examination, which includes evaluation of primitive reflexes and active and passive tone (Law et al., 2003).

Newborn urine, 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 is reported to provide more screening accuracy than urine, because drug metabolites accumulate in meconium (Ostrea, 2001). Urine toxicology screening has less accuracy because it reflects only recent substance intake by the mother (Hurits & Choo, 2002). Meconium and hair testing for drug metabolites have the advantages of ease of collection, noninvasiveness, and greater accuracy.

Nursing diagnoses, which depend on the assessment findings, are tailored to the individual needs of the neonate and the family. Following are examples of nursing diagnoses.

Neonate

- Risk for infection related to
  - Maternal risk behaviors that include sexual activity
  - Prolonged rupture of membranes
  - IUGR, preterm birth
- Risk for disorganized infant behavior related to
  - Chemical effects of maternal substance abuse
  - Caregiver cue misreading
  - Caregiver cue deficient knowledge
  - Sensory overstimulation
- Disturbed sleep pattern related to
  - Drug, chemical withdrawal

| TABLE 27-7 Signs of Neonatal Abstinence Syndrome |
|------------------------------ |------------------ |
| **SYSTEM**                  | **SIGNS**        |
| Gastrointestinal            | Poor feeding, vomiting, regurgitation, diarrhea, excessive sucking |
| Central nervous             | Irritability, tremors, shrill cry, incessant crying, hyperactivity, little sleep, excoriaisons on face, convulsions |
| Metabolic, vasomotor, respiratory | Nasal congestion, tachypnea, sweating, frequent yawning, increased respiratory rate >60/min, fever >37.2°C |

TABLE 27-7

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>SIGNS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Poor feeding, vomiting, regurgitation, diarrhea, excessive sucking</td>
</tr>
<tr>
<td>Central nervous</td>
<td>Irritability, tremors, shrill cry, incessant crying, hyperactivity, little sleep, excoriaisons on face, convulsions</td>
</tr>
<tr>
<td>Metabolic, vasomotor, respiratory</td>
<td>Nasal congestion, tachypnea, sweating, frequent yawning, increased respiratory rate &gt;60/min, fever &gt;37.2°C</td>
</tr>
</tbody>
</table>
### Neonatal Abstinence Scoring System

<table>
<thead>
<tr>
<th>System</th>
<th>Signs and Symptoms</th>
<th>Score</th>
<th>AM</th>
<th>PM</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>CENTRAL NERVOUS SYSTEM DYSFUNCTIONS</td>
<td>Excessive High Pitched (Or Other) Cry</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Continuous High Pitched (Or Other) Cry</td>
<td>3</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Sleeps &lt;1 Hour After Feeding</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sleeps &lt;2 Hours After Feeding</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sleeps &lt;3 Hours After Feeding</td>
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<tr>
<td></td>
<td>Hyperactive Moro Reflex</td>
<td>2</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Markedly Hyperactive Moro Reflex</td>
<td>3</td>
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<tr>
<td></td>
<td>Mild Tremors Disturbed</td>
<td>1</td>
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<tr>
<td></td>
<td>Moderate-Severe Tremors Disturbed</td>
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<tr>
<td></td>
<td>Mild Tremors Undisturbed</td>
<td>3</td>
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<td></td>
<td>Moderate-Severe Tremors Undisturbed</td>
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<tr>
<td></td>
<td>Increased Muscle Tone</td>
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<td>Excoriation (Specific Area)</td>
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<tr>
<td></td>
<td>Myoclonic Jerks</td>
<td>3</td>
<td></td>
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<tr>
<td></td>
<td>Generalized Convulsions</td>
<td>5</td>
<td></td>
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<tr>
<td>METABOLIC/VASOMOTOR/RESPIRATORY DISTURBANCES</td>
<td>Sweating</td>
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<td></td>
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<td></td>
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<tr>
<td></td>
<td>Fever &lt;100° (99-100.8° F / 37.2-38.2° C)</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Fever &gt;101° (38.4° C and Higher)</td>
<td>2</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Frequent Yawning (&gt;3 or 4 Times/Interval)</td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td>Nasal Stuffiness</td>
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<td></td>
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<tr>
<td></td>
<td>Sneezing (&gt;3 or 4 Times/Interval)</td>
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<tr>
<td></td>
<td>Nasal Flaring</td>
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<tr>
<td></td>
<td>Respiratory Rate &gt;60/min</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Respiratory Rate &gt;60/min with Retractions</td>
<td>2</td>
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<td>GASTROINTESTINAL DISTURBANCES</td>
<td>Excessive Sucking</td>
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<tr>
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<td>Poor Feeding</td>
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<tr>
<td></td>
<td>Regurgitation</td>
<td>2</td>
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<tr>
<td></td>
<td>Projectile Vomiting</td>
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<td></td>
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<tr>
<td></td>
<td>Loose Stools</td>
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<td></td>
<td>Watery Stools</td>
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<tr>
<td>TOTAL SCORE</td>
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<td></td>
<td></td>
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<tr>
<td>INITIALS OF SCORER</td>
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**Fig. 27-10** Neonatal Abstinence Scoring (NAS) system, developed by L. Finnegan. (From Nelson, N. (1990). Current therapy in neonatal-perinatal medicine (2nd ed.). St. Louis: Mosby.)
**Expected Outcomes of Care Neonate**

- The neonate will remain free of infection.
- Early manifestations of infection (viral or bacterial) will be recognized, and appropriate therapy to minimize effects of disease will be implemented.
- Newborn manifestation of withdrawal (NAS) will be recognized and appropriate therapy implemented to provide infant state regulation.
- Infant will receive appropriate physical and emotional care to minimize effects of maternal chemical substance use.
- Neonate will have steady patterns of uninterrupted sleep throughout the day.
- Neonate will demonstrate appropriate growth and development.

**Parent(s)**

- Parent(s) will demonstrate ability to consistently meet basic caregiving needs of neonate.
- Parent(s) will continue to participate in substance abuse program to enhance ability to cope with life and effectively parent the newborn.
- Parent(s) will receive counseling and information from health care staff regarding infant behavior, cues requiring comfort and feeding, signs of withdrawal, and general baby care.
- Parent(s) will recognize pattern of self-destructive behavior (substance abuse) and seek intervention.

**Plan of Care and Interventions**

Planning for care of the infant born to a substance-abusing mother presents a challenge to the health care team. Parents are included in the planning for the newborn’s care and are also encouraged to plan for their own care. A multidisciplinary approach is needed that includes home health or community resource personnel (e.g., regulatory agencies such as child protective services). Education and social support to prevent the abuse of drugs provide the ideal approach. However, given the scope of the drug abuse problem, total prevention is unrealistic.

Nursing care of the drug-exposed neonate involves supportive therapy for fluid and electrolyte balance, nutrition, infection control, and respiratory care. Swaddling, holding, reducing environmental stimuli, and feeding as necessary may be helpful in easing withdrawal (Plan of Care). Specific suggestions for providing care to infants experiencing withdrawal are listed in the Teaching Guidelines box.

Pharmacologic treatment is usually based on the severity of withdrawal symptoms, as determined by an assessment tool (see Fig. 27-10). Drug therapies to decrease withdrawal side effects include administration of phenobarbital, morphine, diluted tincture of opium, or methadone (Coyle, Ferguson, Lagoose, Oh, & Lester, 2002; Johnson, Gerada, & Greenough, 2003). 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, & Greenough, 2003).

After the presence of neonatal abstinence syndrome is identified in an infant, nursing care is directed toward treatment of the presenting signs, decreasing stimuli that may precipitate hyperactivity and irritability (e.g., dimming the lights, decreasing noise levels), providing adequate nutrition and hydration, and promoting maternal-infant relationships. Appropriate individualized developmental care is implemented to facilitate self-consoling and self-regulating behaviors. Irritable and hyperactive infants have been found to respond to physical comforting, movement, and close contact. Wrapping infants snuggly and rocking and holding them tightly limits their ability to self-stimulate. The infant’s arms should remain flexed with hands in close proximity of the mouth for sucking as is appropriate; sucking on fingers or hands is a form of self-control and comfort. Arranging nursing activities to reduce the amount of disturbance helps decrease exogenous stimulation.

Loose stools, poor intake, and regurgitation after feeding predispose infants with neonatal abstinence syndrome to malnutrition, dehydration, and electrolyte imbalance. Frequent weighing to detect fluid losses or caloric intake, careful monitoring of intake and output, electrolytes, and additional caloric supplementation may be necessary. In addition, these infants burn up energy with continual activity and increase oxygen consumption at the cellular level. It takes considerable time and patience to ensure that they receive a sufficient caloric and fluid intake.

Hyperactive infants must be protected from skin abrasions on the knees, toes, and cheeks that are caused by rubbing on bed linens while in a prone position while awake. The incidence of SIDS in children who have experienced neonatal abstinence syndrome is high, and parents should be reminded that the supine position for sleep is preferred. Monitoring and recording the activity level and its relationship to other activities, such as feeding and preventing complications, are important nursing functions.

Breastfeeding is encouraged in mothers who are not using illicit substances, are negative for HIV infection, and are compliant with a methadone program. Breastfeeding promotes maternal-infant bonding, and the small amount of methadone

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CD: Plan of Care—The Drug-Exposed Newborn

NURSING DIAGNOSIS Risk for injury related to irritability, hyperactivity, and disorganization

Expected Outcome Infant exhibits age-appropriate state modulation regulation and stability (i.e., quiet alert state; sleep/wake state; ability to withdraw from state and to respond to stimuli and interact appropriately)

Nursing Interventions/Rationales
- Position infant supine with knees and arms flexed and place a blanket next to face to promote containment and comfort and minimize frantic and frantic activity.
- Encourage mother to interact with infant and to become involved in care routines; show respect, provide support, and encourage maternal participation in a substance abuse counseling (and methadone maintenance, as appropriate) program to enhance maternal coping skills for effective caretaking of affected newborn.
- Involve family in care of infant to provide support for the mother.

NURSING DIAGNOSIS Ineffective maternal coping, anxiety, powerlessness, related to drug use, and infant distress during withdrawal

Expected Outcome Mother will accept newborn’s condition and participate in care activities, showing evidence of maternal-infant bonding process.

Nursing Interventions/Rationales
- Explain effects of maternal drug use on newborn and the withdrawal process to facilitate understanding of the effects of drug use.
- Encourage open communication (e.g., inform mother of ongoing condition, procedures, and treatment; answer questions; correct misperceptions; actively listen to her concerns) to provide a sense of respect, provide support, and encourage a sense of control.
- Encourage mother to interact with infant and to become involved in care routines to foster emotional connection.
- Explain how to perform care procedures, how to avoid overstimulation, and how to hold and comfort infant to enhance mother’s care abilities and her sense of confidence and control.
- If the infant demonstrates signs of withdrawal, explain to mother the infant’s inability to interact, gaze aversion, arching back, and lack of response to cuddling to enhance understanding of infant behaviors.
- Make appropriate referrals to social agencies for treatment of maternal substance abuse, infant development programs, and other needed support services to ensure adequate resources for care of self and infant.
- Encourage maternal participation in a substance abuse counseling (and methadone maintenance, as appropriate) program to enhance maternal coping skills for effective caretaking of affected newborn.
- Involve family in care of infant to provide support for the mother.

Note: These first two interventions take precedence over all others because manifestations of withdrawal may vary from one infant to another.

• Administer medications to decrease CNS irritability.
- Decrease environmental stimuli that may trigger irritability and hyperactive behaviors.
- Plan care activities carefully to allow for appropriate interaction as per infant’s behavioral cues.
- Wrap infant snugly and hold infant tightly to reduce self-stimulation behaviors.
- Position to avoid eye contact, swaddle infant, use vertical rocking techniques, and use a pacifier to counter poor organizational response to stimuli and depressed interactive behaviors.
- Monitor activity level, note the relationship between activity level and external stimulation, and stop external stimulation if it causes activity increase.
- Provide scheduled periods of rest, decreased overhead lighting, and no physical care to allow time for recovery of quiet state after periods of care.
- Help mother understand that infant behavioral cues are not a sign of rejection of her caretaking abilities to facilitate loss of maternal-infant interaction, decrease maternal guilt, and enhance environment conducive to infant growth (promote infant’s sense of trust).

NURSING DIAGNOSIS Imbalanced nutrition: less than body requirements related to central nervous system (CNS) irritability, disorganized sucking patterns, vomiting, and loose or watery stools

Expected Outcome Infant exhibits appropriate weight gain.

Nursing Interventions/Rationales
- Observe for feeding cues indicating readiness for interaction (quiet alertness, rooting) and feed frequent small amounts and burp well to ensure adequate re-ingestion of needed nutrients.
- Monitor weight daily and maintain strict intake and output and weight gain.
- Modify environment of feeding area as necessary to decrease stimulus that detract from feeding process and interaction with caregiver.

Passed through breast milk has not proved to be harmful to the neonate (Hale, 2002; Berghella et al., 2003; Philipp, Merewood, & O’Brien, 2003). Because many new drugs are being manufactured, it is recommended that the reader consult with updated references regarding the safety of medications for breastfeeding infants (see Table 20-4 on p. 631) (Lawrence & Lawrence, 2005; see also AAP [2001] for a complete list of drugs that should be avoided with breastfeeding).
Rh incompatibility
Rh incompatibility, or isoimmunization, occurs when an RhD-negative mother has an RhD-positive fetus that inherits the dominant Rh-positive gene from the father. The Rh blood group consists of several antigens (because D is the most prevalent Rh factor, there is a 50% chance that each infant born of the union will be Rh positive and a 50% chance that each will be Rh negative. An Rh-negative fetus is in no danger because it has the same Rh factor as the mother. An Rh-negative fetus with an Rh-positive mother is also in no danger. Only the Rh-positive offspring of an Rh-negative mother is at risk. From 10% to 15% of all Caucasian couples and about 5% of African-American couples have Rh incompatibility. Incompatibility is rare in Asian couples. The incidence of Rh sensitization and resulting hemolytic disease of the newborn have decreased dramatically since the development of Rh(D) immune globulin in 1968.

The pathogenesis of Rh incompatibility is as follows: hematopoiesis in the fetus, or the formation of blood cells, begins as early as the eighth week of gestation; in up to 40% of pregnancies, these cells pass through the placenta into the maternal circulation. When the fetus is Rh positive and the mother Rh negative, the mother forms antibodies against the fetal blood cells: first IgM antibodies, which are too large to pass through the placenta, and then IgG antibodies, which can cross the placenta. The process of antibody formation is called maternal sensitization. Sensitization may occur during pregnancy, birth, miscarriage or abortion, amniocentesis, CVS, and PUBS. Usually women become sensitized in their first pregnancy with an Rh-positive fetus but do not produce enough antibodies to cause lysis (destruction) of the fetal blood cells. In subsequent pregnancies, antibodies form in response to repeated contact with the antigen from the fetal blood, and lysis results. In approximately 10% to 15% of sensitized mothers, there is no hemolytic reaction in the newborn. In addition, some Rh-negative women, even though exposed to Rh-positive fetal blood, are immunologically unable to produce antibodies to the foreign antigen (Neal, 2001).

Severe Rh incompatibility results in marked fetal hemolytic anemia because the fetal erythrocytes are destroyed by maternal Rh-positive antibodies. Although the placenta usually clears the bilirubin generated by the RBC breakdown, in extreme cases fetal bilirubin levels increase. The fetus compensates for the anemia by producing large numbers of immature erythrocytes to replace those hemolyzed—hence the name for this condition: erythroblastosis fetalis. In hydrops fetalis, the most severe form of this disease, the fetus has marked anemia, as well as cardiac decompensation, cardiomegaly, and hepatosplenomegaly. Hypoxia results from the severe anemia. In addition, because of the decreased intravascular oncotic pressure involved, fluid leaks out of the intravascular space, resulting in generalized edema as well as effusions into the peritoneal (ascites), pericardial, and pleural (hydrothorax) spaces. The placenta is often edematous, which, along with the edematous fetus, can cause the uterus to rupture.

Intrauterine or early neonatal death may occur as a result of hydrops fetalis, although intrauterine transfusions and early birth of the fetus may avert this. Intrauterine transfusion involves the infusion of Rh-negative, type O blood into the umbilical vein. The frequency of intrauterine transfusions may vary according to institution and fetal hydropic status, but it may be as often as every 2 weeks until the fetus reaches pulmonary maturity at approximately 37 to 38 weeks of gestation (Moise, 2002).

ABO incompatibility
ABO incompatibility is more common than Rh incompatibility but causes less severe problems in the affected
infant. It occurs if the fetal blood type is A, B, or AB and the maternal type is O. It occurs rarely in infants with type B blood born to mothers with type A blood. The incompatibility arises because naturally occurring anti-A and anti-B antibodies are transferred across the placenta to the fetus. Unlike the situation that pertains to Rh incompatibility, first-born infants may be affected because mothers with type O blood already have anti-A and anti-B antibodies in their blood. Such a newborn may have a weakly positive direct Coombs’ test (also referred to as a direct antiglobulin test [DAT]). The cord bilirubin level usually is less than 4 mg/dl, and any resulting hyperbilirubinemia usually can be treated with phototherapy. Exchange transfusion is required only occasionally. Although ABO incompatibility is a common cause of hyperbilirubinemia, it rarely precipitates significant anemia resulting from the hemolysis of RBCs.

Other

It is not within the scope of this text to discuss the many potential causes of hemolytic jaundice in childhood. However, in some populations there is a high incidence of glucose-6-phosphate dehydrogenase deficiency (G6PD), which may cause an exaggerated jaundice in a newborn within 24 to 48 hours of birth. G6PD red cells hemolyze at a greater rate than healthy red cells, thus overwhelming the immature neonatal liver’s ability to conjugate the indirect bilirubin. Some of the triggers that potentiate hemolysis include vitamin K, acetaminophen, aspirin, sepsis, and exposure to certain chemicals (Reiser, 2004). Treatment is the same as for any newborn with rapidly rising serum bilirubin levels. Other metabolic and inherited conditions that increase hemolysis and may cause jaundice in the infant include galactosemia, Criglar-Najjar syndrome and hypothyroidism.

COLLABORATIVE CARE

At the first prenatal visit of an Rh-negative woman with a fetus who may be Rh positive, an indirect Coombs’ test should be done to determine whether she has antibodies to the Rh antigen. In this test the maternal blood serum is mixed with Rh-positive RBCs. If the Rh-positive RBCs agglutinate or clump, this indicates that maternal antibodies are present or that the mother has been sensitized. The dilution of the specimen of blood at which clumping occurs determines the titer, or level, of maternal antibodies. This titer indicates the degree of maternal sensitization. A level of 1:8 rarely results in fetal jeopardy. If the titer reaches 1:16, the risk of maternal sensitization, is measured. If the titer is 1:64, an exchange transfusion is indicated. In addition, the prevention of of prompt therapy for perinatal asphyxia, acidosis, cold stress, sepsis, and hypoglycemia will decrease the newborn’s risk for severe hemolytic disease and his or her susceptibility to kernicterus. Early feeding is also initiated to stimulate stooling and thus facilitate the removal of bilirubin.

Exchange Transfusion

Exchange transfusions are needed infrequently because of the decrease in the incidence of severe hemolytic disease in newborns resulting from isoimmunization. Other factors must always be considered as well, particularly the clinical condition of the infant, because it is a procedure with potential complications. Guidelines for the initiation of exchange transfusion in relation to serum bilirubin levels in infants of ≥ 35 weeks of gestation may be found in the 2004 AAP Clinical Practice Guideline.

Exchange transfusion is accomplished by alternately removing a small amount of the infant’s blood and replacing it with an equal amount of donor blood. If the infant has Rh incompatibility, type O Rh-negative blood is used for transfusion, so the maternal antibodies still present in the infant do not hemolyze the transfused blood. Depending on the infant’s size, maturity, and condition, amounts of 5 to 20 ml of the infant’s blood are removed at one time and replaced with warmed donor blood. The total amount of blood exchanged approximates 170 ml/kg of body weight, or 75% to 85% of the infant’s total blood volume. Preservatives in donor blood lower the infant’s serum calcium level; therefore, calcium gluconate is often given during the exchange transfusion. The neonate is monitored closely for signs of a blood transfusion reaction as well as hypotension, temperature instability, and cardiorespiratory compromise.

CONGENITAL ANOMALIES

Congenital anomalies (structural defects) occur in approximately 2% of all live births (Hudgins & Cassidy, 2002), but this number increases to about 6% by 5 years, when more anomalies are diagnosed. In addition, the incidence of congenital malformations in fetuses that are miscarried is higher than that in infants who are born alive, thus also adding to
the overall incidence. Major congenital defects are the leading cause of death in infants younger than 1 year of age in the United States and account for 20% of neonatal deaths. Although the incidences of other causes of neonatal mortality have decreased, the death rate associated with most congenital anomalies has essentially remained stable since 1932.

The most common major congenital anomalies that cause serious problems in the neonate are congenital heart disease, choanal atresia, neural tube defects, cleft lip or palate, clubfoot, and developmental dysplasia of the hip (DDH). These are thought to result from the interaction of multiple genetic and environmental factors. Some of the most common malformations include lack of a helical fold of the pinna, complete or incomplete simian creases, and a capillary hemangioma other than on the face or posterior aspect of the neck.

Ways of detecting and preventing some of these anomalies are being improved continuously, as are some surgical techniques for the care of the fetus with certain anomalies. Promoting the availability of these services to populations at risk challenges community health care systems. An interdisciplinary team approach is vital for providing holistic care: the surgical treatment, rehabilitation, and education of the child, as well as psychosocial and financial assistance for the parents. Parental disappointment and disillusion add to the complexity of the nursing care needed for these infants.

Central Nervous System Anomalies

Most congenital anomalies of the CNS result from defects in the closure of the neural tube during fetal development. Although the cause of NTDs is unknown, they are thought to stem from the interaction of many genes that may be influenced by factors in the fetal environment. Environmental influences such as treatment with valproic acid (an anticonvulsant) or methotrexate (a chemotherapeutic agent) and alcohol and tobacco consumption have been implicated. Maternal folic acid deficiency has a direct bearing on failure of the neural tube to close; therefore, folic acid supplementation is recommended for women of childbearing age. In the United States, rates of NTDs have declined from 1.3 per 1000 births (1970) to 0.3 per 1000 births after the introduction of mandatory food fortification with folic acid in 1998 (Honein, 2001). Increased use of prenatal diagnostic techniques and termination of pregnancies have also affected the overall incidence of NTDs. Although a neural tube defect is usually an isolated defect, it can occur with some chromosomal abnormalities and syndromes and also with other defects such as cleft palate, ventricular septal defect, tracheoesophageal fistula, congenital diaphragmatic hernia, imperforate anus, and renal anomalies.

**Encephalocele and anencephaly**

Encephalocele and anencephaly are abnormalities resulting from failure of the anterior end of the neural tube to close. An encephalocele is a herniation of the brain and meninges through a skull defect. Treatment consists of surgical repair and shunting to relieve hydrocephalus, unless a major brain malformation is present. Some of these infants will have some degree of cognitive deficit. Anencephaly is the absence of both cerebral hemispheres and of the overlying skull. It is a condition that is incompatible with life; many of the infants are stillborn or die within a few days of birth. Comfort measures are provided until the infant eventually dies of temperature instability and respiratory failure.

**Spina bifida**

Spina bifida, the most common defect of the CNS, results from failure of the neural tube to close at some point. There are two categories of spina bifida: spina bifida occulta and spina bifida cystica. Spina bifida occulta is a malformation in which the posterior portion of the laminae fails to close but the spinal cord or meninges do not herniate or protrude through the defect (Fig. 27-11, B). It is usually asymptomatic and may not be diagnosed unless there are associated problems. Spina bifida cystica includes meningocele and...
Hydrocephalus

Hydrocephalus is a condition in which the ventricles of the brain are enlarged as a result of an imbalance between the production and absorption of the CSF. Congenital hydrocephalus usually arises as a result of a malformation in the brain or an intrauterine infection. About one third of all cases of congenital hydrocephalus result from stenosis of the aqueduct of Sylvius in the brain. Hydrocephalus often occurs in conjunction with a myelomeningocele, which blocks the flow of CSF.

An infant with congenital hydrocephalus initially has a bulging anterior fontanel and a head circumference that increases at an abnormal rate, resulting from the increase in CSF pressure. Enlargement of the forehead with depressed eyes that are rotated downward, causing a “setting sun” sign, occurs as the condition worsens. If the surgical shunting of excess CSF from the brain is not done soon after birth, the resulting increasing ICP will lead to irreversible neurologic damage, as evidenced by pearly widening sutures and fontanels, dilated scalp veins, lethargy, poor feeding, vomiting, irritability, opisthotonic positioning, and a high-pitched, shrill cry.

Nursing actions appropriate to the needs of a newborn with hydrocephalus include care similar to that for any high-risk newborn. Measurement of the head circumference and neurologic assessments are done frequently. If the infant’s head is large, the placement of sheepskin or a special pressure-sensitive air mattress under the infant and frequent position changes are necessary to prevent skin breakdown.

Critical Thinking Exercise

Birth of a Child with a Congenital Anomaly

Marjorie, a 43-year-old mother, has just given birth to a daughter, Monica, who has a small ventricular septal defect (VSD). The infant has a loud murmur and circumoral cyanosis when crying vigorously but is pink at all other times. Marjorie knows that Monica has a heart defect but has not yet seen her. You are taking Marjorie to the nursery to see the baby for the first time. How will you introduce Monica to Marjorie? How will you describe her to Marjorie? How will you describe Monica’s defect and its treatment?

Evidence—Is there sufficient evidence to draw conclusions about what you should tell Marjorie?

1. Evidence—Is there sufficient evidence to draw conclusions about what you should tell Marjorie?

2. Assumption—What assumptions can be made about the following factors:
   a. The parents’ feelings about the infant and her physical defect
   b. Marjorie’s initial reaction to the sight of her daughter
   c. Treatment for the defect
   d. Long-term outcome for Monica

3. What implications and priorities for nursing care can be drawn at this time?

4. Does the evidence objectively support your conclusion?

5. Are there alternative perspectives to your conclusion?
fore mental retardation is common. Microcephaly can be
the result of an autosomal dominant disorder; a chromosomal
abnormality; fetal exposure to teratogens such as radiation;
and congenital infections such as rubella, toxoplasmosis, or
CMV. Infants with microcephaly require supportive nursing
care and medical observation to determine the extent of the
psychomotor retardation that almost always accompanies
this abnormality. There is no treatment. Parents need sup-
port to learn to care for a child with cognitive impairment.

Cardiovascular System Anomalies
Congenital heart defects (CHDs) are anatomic abnormali-
ties of the heart that are present at birth, although they may
not be diagnosed immediately. Some type of congenital car-
diovascular problem is present in approximately 3.7 to 8 of
every 1000 live births (Carey, 2002) and approximately two
or three newborns will be symptomatic with heart disease in
the first year of life (Bernstein, 2004). Ventricular septal de-
fects, constituting more than 20% to 25% of all CHDs, are
the most common type of cyanotic lesion. Tetralogy of
Fallot, constituting 5% to 7% of all CHDs, is the most com-
mon type resulting in cyanosis (Fig. 27-12). After prematurity,
CHDs, often in association with other congenital anomalies,
are the next major cause of death in the first year of life.

The cause of the CHD is unknown in more than 90% of
the cases. Maternal factors associated with a higher incidence
of CHD include maternal rubella, alcohol intake, diabetes
mellitus, systemic lupus erythematosus, phenylketonuria,
poor nutrition, and antiepileptic medication use.

Genetic factors are implicated in the pathogenesis of
CHD. As a general rule, these defects are thought to be mul-
tifactorial in origin, involving both genetic and environ-
mental influences; however, a familial occurrence of virtu-
ally all forms of CHD has been noted.

Chromosomal abnormalities may also be associated with
CHDs. For example, 50% of children with trisomy 21, or
Down syndrome, have a cardiac defect. Most children who
have trisomy 18, the second most common chromosomal
abnormality, have a cardiac anomaly.

Some CHDs are often evident immediately after birth,
especially those defects that cause central cyanosis (e.g., trans-
position of the great vessels) despite 100% oxygen admin-
istration. Infants with these anomalies are transferred directly
to a neonatal intensive care nursery or pediatric intensive care
unit.

Screening for congenital anomalies of the respiratory system
is necessary even in infants who are apparently normal at
birth. Respiratory distress at birth or shortly thereafter may
be the result of lung immaturity or anomalous development.
Congenital laryngeal web and bilateral choanal atresia are
readily apparent at birth. Respiratory distress caused by
congenital diaphragmatic hernia and TEF may appear im-
mediately or be delayed, depending on the severity of the
defect.

Laryngeal web and choanal atresia
A laryngeal web, which is uncommon, results from the
incomplete separation of the two sides of the larynx and is
most often between the vocal cords. Choanal atresia (Fig. 27-
13) is the most common congenital anomaly of the nose;
it is a bony or membranous septum located between the
nose and the pharynx. Inability to pass a suction catheter
through the nose into the pharynx or cyanosis without
Atrial septal defect (ASD)

An ASD is an abnormal opening between the right and left atria. Basically, three types of abnormalities result from incorrect development of the atrial septum. An incompetent foramen ovale is the most common defect. The high ostium secundum defect results from abnormal development of the septum secundum. Improper development of the septum primum produces a basal opening known as an ostium primum defect, frequently involving the atrioventricular valves. In general, left-to-right shunting of the blood occurs in all atrial septal defects.

Ventricular septal defect (VSD)

A VSD is an abnormal opening between the right and left ventricles. VSDs vary in size and may occur in either the membranous or muscular portion of the ventricular septum. Because of higher pressure in the left ventricle, a shunting of blood from the left to the right ventricles occurs during systole. If pulmonary vascular resistance produces pulmonary hypertension, the shunt of blood is then reversed from the right to the left ventricle, with cyanosis resulting.

Aortic stenosis (AS)

AS is a narrowing or stricture of the aortic valve, causing resistance to blood flow in the left ventricle, decreased cardiac output, left ventricular hypertrophy, and pulmonary vascular congestion. AS can be valvular, subvalvular, or supravalvular (rare). The most serious sequelae relate to the left ventricular hypertrophy (increased end-diastolic pressure, pulmonary hypertension, decreased coronary artery perfusion).

Patent ductus arteriosus (PDA)

PDA is a vascular connection that, during fetal life, bypasses the pulmonary vascular bed and directs blood from the pulmonary artery to the aorta. Functional closure of the ductus normally occurs soon after birth. If the ductus remains patent after birth, the direction of blood flow in the ductus is reversed by the higher pressure in the aorta.

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Atrioventricular canal (AVC) defect

An AVC is an incomplete fusion of the endocardial cushions. It consists of a low atrial septal defect that is continuous, with a high ventricular septal defect and clefts of the mitral and tricuspid valves, creating a large central atrioventricular valve that allows blood to flow between all four chambers of the heart. Flow is generally from left to right. It is the most common cardiac defect in children with Down syndrome.

Coarctation of the aorta (COA)

COA is characterized by localized narrowing of the aorta near the insertion of the ductus arteriosus, resulting in increased pressure proximal to the defect (head and upper extremities) and decreased pressure distal to the defect (body and lower extremities).

Pulmonic stenosis (PS)

PS is a narrowing at the entrance to the pulmonary artery, causing resistance to blood flow in the right ventricle, increased pressure in the pulmonary artery, and decreased pulmonary blood flow. Pulmonary atresia is the extreme form of PS, in which blood flow to the lungs is absent.

Tetralogy of Fallot (TOF)

TOF is characterized by the combination of four defects: (1) pulmonary stenosis, (2) ventricular septal defect, (3) overriding aorta, and (4) hypertrophy of the right ventricle. This is the most common defect, causing cyanosis in children surviving beyond 2 years of age. The severity of symptoms depends on the size of the ventricular septal defect, the degree of pulmonary stenosis, and the degree to which the aorta overrides the septal defect.

Fig. 27-12 Congenital heart abnormalities. (Modified from Hockenberry, M. [2003]. Wong’s nursing care of infants and children [7th ed.]. St. Louis: Mosby.)
obvious respiratory distress usually leads to its detection. Nearly half of the infants with choanal atresia have other anomalies. Infants with either a laryngeal web or choanal atresia require emergency surgery.

Congenital diaphragmatic hernia

Congenital diaphragmatic hernia (CDH) results from a defect in the formation of the diaphragm, allowing the abdominal organs to be displaced into the thoracic cavity. It occurs in approximately 1 in 5000 live births. However, if stillbirths resulting from this defect are included, the incidence increases to 1 in 2000 (Hartman, 2004). Herniation of the abdominal viscera into the thoracic cavity may cause severe respiratory distress and constitutes a neonatal emergency (Fig. 27-14). The defect and herniation may be so extensive that the viscera present in the thoracic cavity during embryonic life have prevented the normal development of pulmonary tissue. The defect is usually on the left because that side of the diaphragm fuses last.

Hypoplastic left heart syndrome (HLHS)

HLHS is characterized by under-development of the left side of the heart, resulting in a hypoplastic left ventricle and aortic stenosis. Most blood from the left atrium flows across the patent foramen ovale to the right atrium, to the right ventricle, and out the pulmonary artery. The descending aorta receives blood from the patent ductus arteriosus supplying systemic blood flow. Most CDHs are discovered prenatally on ultrasound. Hernias may be repaired by fetal surgery in some research institutions. Intrauterine surgical correction of CDH has met with poor neonatal outcomes in many cases, primarily as a result of tocolysis failure and preterm birth. At birth, most affected infants have severe respiratory distress, and respiratory assessment reveals worsening distress as the bowel fills with air. Typically the breath sounds are diminished and bowel sounds are heard in the chest. Heart sounds may be heard on the right side of the chest because the heart has been displaced there by the abdominal contents. Physical examination reveals a flat or scaphoid abdomen and a prominent ipsilateral chest. Diagnosis can be made on the basis of the x-ray finding of loops of intestine in the thoracic cavity and the absence of intestine in the abdominal cavity. Preoperative nursing interventions include participating in the stabilization of the infant’s condition until surgical repair can be done. Inhaled nitric oxide (NO) has been used in many centers with moderate success to treat the accompanying persistent pulmonary hypertension (Bradshaw,
2004). Gastric contents are aspirated and suction applied to decompress the GI tract and prevent further cardiothoracic compromise. Oxygen therapy, mechanical ventilation, and the correction of acidosis are necessary in infants with early clinical respiratory distress from CDH. Extracorporeal membrane oxygenation (ECMO) may be used in infants with severe circulatory and respiratory complications. The prognosis depends largely on the degree of fetal pulmonary development, but the prognosis in severe cases is often poor. The overall survival rate for infants who are symptomatic within the first few hours of life is about 50%, although it has improved recently with the advent of inhaled NO, improved management of high-frequency ventilation, and ECMO.

**Gastrointestinal System Anomalies**

Anomalies in the GI system can occur anywhere along the GI tract, from the mouth to the anus. Some anomalies, such as cleft lip, omphalocele, and gastroschisis, are apparent at birth. Others, including cleft palate, esophageal atresia, intestinal obstruction, and imperforate anus become apparent as the infant is further assessed or becomes symptomatic.

**Cleft lip and palate**

Cleft lip or cleft palate is a commonly occurring congenital midline fissure, or opening, in the lip or palate resulting from failure of the primary palate to fuse (Fig. 27-15). One or both deformities may occur. Multiple genetic and, to a lesser extent, environmental factors (e.g., maternal infection, radiation exposure, alcohol ingestion, and treatment with medications such as corticosteroids, some tranquilizers, and antiepileptics) appear to be involved in their development.

Cleft lip with or without cleft palate occurs approximately 1 in 800 live births. The incidence of cleft palate alone is 1 in 2000 live births. Cleft lip with or without cleft palate is more common in males, and cleft palate alone is more common in females. The defect appears more often in Asians and certain tribes of Native Americans than in Caucasians, and less often in African-Americans.

Treatment of the infant with cleft lip is surgical; repair usually occurs between 6 and 12 weeks of age. Cleft palate repair is generally postponed until 12 to 18 months of age to take advantage of palatal changes that take place with normal growth.

Feeding is difficult because the cleft lip renders the newborn unable to maintain a seal around a nipple; the cleft palate renders the infant unable to form a vacuum to maintain suction when feeding. In addition, the inability to suck and swallow normally allows milk to pool in the nasopharynx, which increases the likelihood of aspiration. Furthermore, as the infant attempts to suck, milk often comes out through the cleft and out of the nares. Although the degree of difficulty depends on the size of the cleft, feeding problems are greater in infants with a cleft palate than in those with a cleft lip alone (Fig. 27-15, D). Breastfeeding can be successful in some infants. There are special nipples, bottles, and appliances available to aid in feeding (Fig. 27-16).
general, parents of infants with these defects need a great deal of education and support as they learn to feed their baby, to prevent what should be a normal part of infant care from becoming a very frustrating experience.

Parents of infants with a cleft lip or palate need much support, particularly in the case of a cleft lip because this is both a cosmetic and functional defect. Recognizing that this may interfere with normal parent-infant bonding in the neonatal period, the nurse must assess for this and intervene appropriately.

Esophageal atresia and tracheoesophageal fistula

Esophageal atresia (EA) and tracheoesophageal fistula (TEF) often occur together, although they can also occur singly. EA is a congenital anomaly in which the esophagus ends in a blind pouch or narrows into a thin cord, thus failing to form a continuous passageway to the stomach (Fig. 27-17). TEF is an abnormal connection between the esophagus and trachea.

Maternal hydramnios is a common finding, particularly if the fetus has an EA without TEF. The infant with EA or TEF may also show some fetal growth restriction and will therefore be SGA; in addition, the presence of a midline defect such as EA or TEF is often accompanied by another significant embryonic defect such as a cardiac anomaly, cleft...
lip and/or palate, or vertebral, genitourinary, or abdominal wall defect (Bensard, Calkins, Partrick, & Price, 2002). Variations of the anomalies are possible, depending on the presence or absence of a TEF, the site of the fistula, and the location and degree of the esophageal obstruction (see Fig. 27-17).

Infants with EA and TEF may show significant respiratory difficulty immediately after birth. EA with or without TEF results in excessive oral secretions, drooling, and feeding intolerance. When fed, the infant may swallow, but then cough and gag and return the fluid through the nose and mouth. Respiratory distress can result from aspiration or from the acute gastric distention produced by the TEF. Choking, coughing, and cyanosis occur after even a small amount of fluid is taken by mouth.

Nursing interventions are supportive until surgery is performed. Any infant with excessive oral secretions and respiratory distress should not be fed orally until further evaluation is carried out. The infant is placed in the position least likely to cause aspiration of either mouth or stomach secretions. A double-lumen catheter is placed in the proximal esophageal pouch and attached to continuous suction to remove secretions and decrease the possibility of aspiration. Other supportive measures include maintaining thermoregulation, fluid and electrolyte balance intravenously, and acid-base balance and prevention of any further complications as a result of an associated defect. Surgical correction, done in one stage if possible, consists of ligating the fistula and anastomosing the two segments of the esophagus. The chances for survival in infants in a good risk category exceed 95%, depending on the presence of associated defects and the infant’s birth weight. Many EA and TEF infants will have postoperative issues related to feeding difficulties such as gastroesophageal reflux (GER) and esophageal strictures requiring periodic dilation.

Omphalocele and gastroschisis

Omphalocele and gastroschisis are two of the more common congenital defects that occur in the abdominal wall. They are rare, however, with omphalocele occurring in approximately 1 in 3000 to 10,000 live births, whereas the incidence of gastroschisis is 1 in 6000 live births (Blackburn, 2003).

An **omphalocele** is a covered defect of the umbilical ring into which varying amounts of the abdominal organs may herniate (Fig. 27-18). Although it is covered with a peritoneal sac, the sac may rupture during or after birth. Many infants born with an omphalocele are preterm, and more than half have other serious syndromes or defects involving the GI, cardiac, genitourinary, musculoskeletal, and nervous systems.

**Gastroschisis** is the herniation of the bowel through a defect in the abdominal wall to the right of the umbilical cord. No membrane covers the contents, as occurs with an omphalocele. Unlike infants with omphalocele, these infants have less than a 10% to 15% likelihood of associated anomalies, most of which are cardiac.
The preoperative nursing care is similar for infants with either defect. Exposure of the viscera causes problems with thermoregulation and fluid and electrolyte balance. Until closure is performed, the exposed viscera are covered with a moistened saline gauze and plastic wrap. In some cases the infant may be placed in an impermeable, clear plastic bowel bag to decrease insensible water losses, maintain thermoregulation, and prevent contamination of the exposed viscera (Bensard et al., 2002). Antibiotics, fluid and electrolyte replacement, gastric decompression, and thermoregulation are needed for physiologic support. If complete closure is impossible because of the small size of the abdominal cavity and the large amount of viscera to be replaced, a Silastic silo pouch (Dow Corning, Midland, MI) is created and sewn to the fascia of the abdominal defect. The defect is closed surgically after the reduction of contents is complete, which usually takes 7 to 10 days. Gastric decompression is necessary preoperatively to prevent aspiration pneumonia and to allow as much bowel as possible to be placed into the abdomen during surgery. Surgery is usually performed soon after birth. With surgical treatment, nutritional support, and medical management, the prognosis has improved for infants born with an abdominal wall defect. It is estimated that more than 80% of infants born with omphalocele survive, as do more than 90% of those born with gastroschisis, although residual feeding difficulties such as GER are not uncommon.

Gastrointestinal obstruction

Congenital intestinal obstruction can occur anywhere in the GI tract and takes one of the following forms: atresia, which is a complete obliteration of the passage; partial obstruction, in which the symptoms may vary in severity and sometimes not be detected in the neonatal period, or malrotation of the intestine, which leads to twisting of the intestines (volvulus) and obstruction. Esophageal atresia, discussed previously, is a type of GI obstruction. Meconium ileus is an obstruction caused by impacted meconium and is the earliest symptom of cystic fibrosis, a life-threatening chronic illness. Infants with this type of obstruction should be tested for cystic fibrosis because 95% of infants with meconium ileus have cystic fibrosis.

In addition to polyhydramnios in the pregnant woman, the infant shows the following cardinal signs and symptoms: bilious vomiting, abdominal distention, and failure to pass normal amounts of meconium in the first 24 hours.

Nursing care is aimed at supporting the infant until surgical intervention can be carried out to eliminate the obstruction. Oral feedings are withheld, a nasogastric tube is placed for suction, and IV therapy is initiated to provide needed fluid and electrolytes. In infants with an intestinal obstruction, surgery consists of resecting the obstructed area of bowel and anastomosing the nonaffected bowel. In recent years the survival rate for these infants has risen to 90% to 95% as a result of better treatments, improved neonatal intensive care, and an increased understanding of the total problem.

Imperforate anus

*Imperforate anus* is a term used to describe a wide range of congenital disorders involving the anus and rectum and, in many cases, the genitourinary system (Fig. 27-19). These anomalies have an incidence of approximately 1 in 5000 live births (Bensard et al., 2002). Occurring more in male than in female infants, they result from the failure of anorectal development in weeks 7 and 8 of gestational life. Such infants have no anal opening, and commonly there is also a fistula from the rectum to the perineum or genitourinary system. Types of anorectal malformations include the typical cloaca in females, which involves the vagina, colon, and urethra forming a single common passage in the perineum. Others include the low rectovaginal fistula (female) and rectourethral bulbar fistula (male). Extensive surgical repair is often required in stages for the more complex types of anorectal malformations. In some cases the anomaly may involve stenotic areas, or there may be a thin translucent membrane covering the anal opening. Treatment for such a membrane is excision followed by daily dilation, which parents are taught to do.

Musculoskeletal System

**Anomalies**

**Developmental dysplasia of the hip**

The broad term *developmental dysplasia of the hip* (DDH) describes a spectrum of disorders related to abnormal development of the hip that may develop at any time during fetal life, infancy, or childhood. A change in terminology from *congenital hip dysplasia* and *congenital dislocation of the hip* to developmental dysplasia of the hip more properly reflects a variety of hip abnormalities in which there is a shallow acetabulum, subluxation, or dislocation.

The incidence of hip instability of some kind is approximately 10 per 1000 live births. The incidence of frank dislocation or a dislocatable hip is 1 per 1000 live births (Wall,
and approximately 30% to 50% of infants with DDH are born in breech presentation (Thompson, 2004).

The cause of DDH is unknown, but certain factors such as sex, birth order, family history, intrauterine position, birth type, joint laxity, and postnatal positioning are believed to affect the risk of DDH. Predisposing factors associated with DDH may be divided into three broad categories: (1) physiologic factors, which include maternal hormone secretion and intrauterine positioning; (2) mechanical factors, which involve breech presentation, multiple fetus, oligohydramnios, and large infant size; other mechanical factors may include continued maintenance of the hips in adduction and extension that will in time cause a dislocation; and (3) genetic factors, which entail a higher incidence (6%) of DDH in siblings of affected infants and an even greater incidence (36%) of recurrence if a sibling and one parent were affected.

Three degrees of DDH are illustrated in Fig. 27-20 and are described as follows:

- **Acetabular dysplasia (or preluxation)—mildest form of DDH, in which there is neither subluxation nor dislocation. There is a delay in acetabular development evidenced by osseous hypoplasia of the acetabular roof that is oblique and shallow, although the cartilaginous roof is comparatively intact. The femoral head remains in the acetabulum.**

- **Subluxation**—The largest percentage of DDH, subluxation; implies incomplete dislocation of the hip and is sometimes regarded as an intermediate stage in the development from primary dysplasia to complete dislocation. The femoral head remains in contact with the acetabulum, but a stretched capsule and ligamentum teres cause the head of the femur to be partially displaced. Pressure on the cartilaginous roof inhibits ossification and produces a flattening of the socket.

- **Dislocation**—The femoral head loses contact with the acetabulum and is displaced posteriorly and superiorly over the fibrocartilaginous rim. The ligamentum teres is elongated and taut.

DDH is often not detected at the initial examination after birth; therefore all infants should be carefully monitored for hip dysplasia at follow-up visits throughout the first year of life. In the newborn period dysplasia usually appears as hip joint laxity rather than outright dislocation. Subluxation and the tendency to dislocate can be demonstrated by the Ortolani or Barlow tests. The Ortolani and Barlow tests are most reliable from birth to 2 or 3 months of age. Other signs of DDH are shortening of the limb on the affected side (Galeazzi sign, Allis sign), asymmetric thigh and gluteal folds, and broadening of the perineum (in bilateral dislocation) (see also Fig. 18-12).

The Ortolani and Barlow tests must be performed by an experienced clinician to prevent fracture or other damage to the hip. If these tests are performed too vigorously in the first 2 days of life, when the hip subluxates freely, persistent dislocation may occur.

Treatment is begun as soon as the condition is recognized, because early intervention is more favorable to the restoration of normal bony architecture and function. The longer treatment is delayed, the more severe the deformity, the more difficult the treatment, and the less favorable the prognosis. The treatment varies with the age of the child and the extent of the dysplasia. The goal of treatment is to obtain and maintain a safe, congruent position of the hip joint to promote normal hip joint development and ambulation.

The hip joint is maintained by dynamic splinting in a safe position with the proximal femur centered in the acetabulum in an attitude of flexion. Of the numerous devices available, the Pavlik harness is the most widely used, and with time, motion, and gravity, the hip works into a more abducted, reduced position (Fig. 27-21). The harness is worn continuously until the hip is proved stable on clinical and radiographic examination, usually in about 3 to 5 months.
The former practice of double- or triple-diapering for DDH is not recommended because it promotes hip extension, thus preventing proper hip development.

Clubfoot

Congenital clubfoot is a complex deformity of the ankle and foot that includes forefoot adduction, midfoot supination, hindfoot varus, and ankle equinus. Deformities of the foot and ankle are described according to the position of the ankle and foot. The more common positions involve the following variations:

- **Talipes varus**—An inversion or a bending inward
- **Talipes valgus**—An eversion or bending outward
- **Talipes equinus**—Plantar flexion in which the toes are lower than the heel
- **Talipes calcaneus**—Dorsiflexion, in which the toes are higher than the heel

Most cases of clubfoot are a combination of these positions, and the most frequently occurring type of clubfoot (approximately 95%) is the composite deformity talipes equinovarus, in which the foot is pointed downward and inward in varying degrees of severity. Unilateral clubfoot is somewhat more common than bilateral clubfoot and may occur as an isolated defect or in association with other disorders or syndromes, such as chromosomal aberrations, arthrogryposis (a generalized immobility of the joints), cerebral palsy, or spina bifida.

The goal of treatment for clubfoot is to achieve a painless, plantigrade (able to walk on the sole of the foot with the heel on the ground), and stable foot. Treatment of clubfoot involves three stages: (1) correction of the deformity, (2) maintenance of the correction until normal muscle balance is regained, and (3) follow-up observation to avert possible recurrence of the deformity. Some feet respond to treatment readily; some respond only to prolonged, vigorous, and sustained efforts; and the improvement in others remains disappointing even with maximum effort on the part of all concerned.

Serial casting is begun shortly after birth, before discharge from the nursery. Successive casts allow for gradual stretching of skin and tight structures on the medial side of the foot. Manipulation and casting are repeated frequently (every week) to accommodate the rapid growth of early infancy. In some cases daily manipulation and stretching of tissues is accomplished with taping and splitting of the affected extremity; a continuous passive motion machine may be used several hours daily to stretch and strengthen muscle groups involved (Faulks & Luther, 2005). The extremity or extremities are often casted or splinted until maximum correction is achieved, usually within 8 to 12 weeks. A Denis Browne splint may be used to manage feet that correct with casting and manipulation.

**Polydactyly**

Occasionally hands or feet have extra digits. In some instances, polydactyly is hereditary. If there is little or no bone involvement, the extra digit is tied with silk suture soon after birth. The finger falls off within a few days, leaving a small scar. When there is bone involvement, surgical repair is indicated.

**Genitourinary System Anomalies**

**Hypospadias and epispadias**

Hypospadias constitutes a range of penile anomalies associated with an abnormally located urinary meatus. The meatus can open below the glans penis or anywhere along the ventral surface of the penis, the scrotum, or the perineum (Fig. 27-22). It is the most common anomaly of the penis, affecting approximately 1 in 125 live births (Stokowski, 2004). It is classified according to the location of the meatus and the presence or absence of chordee, which is a ventral curvature of the penis.

![Fig. 27-21 Treatment for developmental hip dysplasia with Pavlik harness. (From Ball, J. [1998]. Mosby’s pediatric patient teaching guides. St. Louis: Mosby.)](image1)

![Fig. 27-22 Classification of hypospadias by position of the urethral meatus.](image2)
Mild cases of hypospadias (Fig. 27-23) are often repaired for cosmetic reasons and involve a single surgical procedure. In more severe cases, several operations are required to reconstruct the urethral opening and correct the chordee, thereby straightening the penis. The goals are to improve the appearance of the genitalia and make it possible for the child to be able to urinate in a standing position and have a sexually adequate organ. These infants are not circumcised because the foreskin may be needed during surgical repair. Repair is done early, between 4 and 8 months of life (Stokowski, 2004).

Epispadias, a rare anomaly, results from failure of urethral canalization. About 55% of the affected infants are males who have a widened pubic symphysis and a broad spadelike penis with the urethra opened on its dorsal surface. In females there is a wide urethra and a bifid (split in two) clitoris. Severity ranges from mild anomaly to a severe one that is associated with extrophy of the bladder. Surgical correction is necessary, and affected male infants should not be circumcised.

Exstrophy of the bladder

The most common bladder anomaly is exstrophy (Fig. 27-24), which often occurs in conjunction with epispadias. It is rare, occurring in only about 1 in 35,000 to 40,000 live births (Elder, 2004). It results from the abnormal development of the bladder, the abdominal wall, and the symphysis pubis that causes the bladder, urethra, and ureteral orifices to all be exposed. The bladder is visible in the suprapubic area as a red mass with numerous folds, with urine draining from it onto the infant’s skin.

Immediately after birth the exposed bladder is covered with a sterile, nonadherent dressing to protect it until closure can be performed. It is recommended that reconstructive surgery be started in the neonatal period, preferably with the bladder being closed during the first or second day of life.

Ambiguous genitalia

Ambiguous genitalia in the newborn (Fig. 27-25) often is discovered by the nurse during a physical assessment. Erroneous or abnormal sexual differentiation may be a genetic defect, such as congenital adrenal hypoplasia, which can be life-threatening because it involves deficiency of all adrenocortical hormones. Other possible causes of sexual ambiguity include chromosomal abnormalities, defective sex hormone synthesis in males, and the placental transfer of masculinizing agents to female fetuses. Gender assignment should be based on data gathered from the following sources: maternal and family history, including the ingestion of steroids during pregnancy and relatives with ambiguous genitalia or who died during the neonatal period; physical examination; chromosomal analysis (results are available in 2 to 3 days); endoscopy, ultrasonography, and radiographic contrast studies; biochemical tests, such as analysis of urinary steroid excretion, which helps detect several of the adrenal cortical syndromes; and, in some instances, laparotomy or gonad biopsy.

Therapeutic intervention, including any counseling and surgery, should be started as soon as possible. Any child born with ambiguous genitalia should not receive sex assignment until the appropriate sex of rearing may be properly assessed and assigned. An appropriate sex assignment should be based on the following: age at presentation, potential for mature sexual function, potential fertility, and the long-term psychological and intellectual impact on the child and family. Parents need much support as they learn to deal with this very challenging situation.

Teratoma

A teratoma is an embryonal tumor that may be solid, cystic, or mixed. It is composed of at least two and usually three types of embryonal tissue: ectoderm, mesoderm, and endoderm. A teratoma in the newborn may occur in the skull, mediastinum, abdomen, or sacral area; more than half are
located in the sacrococcygeal area. The treatment of choice for such neonates is complete surgical resection. Approximately 80% of all teratomas are benign, and no additional therapy is needed after complete resection done in the neonatal period. If the tumor is not surgically resected before the infant is 1 to 2 months old, the likelihood of the teratoma becoming malignant increases rapidly.

Any deviations from normal are reported to the primary health provider immediately. A thorough assessment of all body systems follows, with identification of both visible anomalies and those that might not be visible.

SOME INFANTS HAVE MULTIPLE CONGENITAL ANOMALIES. A RECOGNIZED PATTERN OF MALFORMATIONS IS REFERRED TO AS A SYNDROME.

The most common is Down syndrome, with the diagnosis confirmed early in the neonatal period.

Genetic Diagnosis
Diagnostic procedures for the detection of genetic disorders are performed after birth at any time from the postnatal period through adulthood. Many tests are available for various disorders; only the most commonly used ones are discussed here.

Newborn Screening
The most widespread use of postnatal testing for genetic disease is the routine screening of newborns for inborn errors of metabolism (IEMs) such as phenylketonuria (PKU), galactosemia, hemoglobinopathy (sickle cell disease and thalassemias) and hypothyroidism; these are the minimum mandatory newborn screening tests in most states in the United States. An inborn error of metabolism is the term applied to a large group of disorders caused by a metabolic defect that results from the absence of or change in a protein, usually an enzyme, and mediated by the action of a certain gene. These defects can involve any substrate produced from protein, carbohydrate, or fat metabolism. IEMs are recessive disorders, and a person must receive a defective gene from each parent for them to occur. The parents usually are unaffected because their normal dominant gene directs the synthesis of sufficient protein to meet their metabolic needs under normal circumstances. With the advent of new biochemical techniques, it is now possible to detect the abnormal gene responsible for causing an increasing number of these disorders early in the neonatal period so appropriate therapies to prevent further morbidity may be implemented.

A new screening test, tandem mass spectrometry, has the potential for identifying more than 20 IEMs, in addition to the standard ones. With tandem mass spectrometry, earlier identification of IEMs may prevent further developmental delays and morbidities in affected children.

PKU results from a deficiency of the enzyme phenylalanine dehydrogenase. The test for PKU is not reliable until the newborn has ingested an ample amount of the amino acid phenylalanine, a constituent of both human and cow’s milk. The nurse must document the initial ingestion of milk and perform the test at least 24 hours after that time. Early infant discharge from the hospital has the potential to cause neonates with a disorder such as PKU not to be screened as often as in the past. In response to this, the AAP (1996) made the following recommendations:

- Collect the initial specimen as close as possible to discharge and no later than 7 days after birth.
- Designate a primary care provider for all newborns before discharge for adequate newborn screening follow-up.
- Obtain a subsequent sample before 2 weeks of age if the initial specimen is collected before the newborn is 24 hours old.

If the infant is found to have PKU, a diet low in phenylalanine is begun soon after birth. Breastfeeding or partial breastfeeding may be possible for some infants if the phenylalanine levels are monitored carefully and remain within acceptable limits. Many affected children have some intellectual impairment. Successful management and outcome is largely dependent on early identification of the condition, modifying the diet, and compliance with the treatment regimen throughout the entire life cycle.

Galactosemia, caused by a deficiency of the enzyme galactose-1-phosphate uridylyltransferase, results in the inability to convert galactose to glucose. Galactosemia can be detected by measuring the blood levels of galactose in the urine of newborns suspected of having the disease who have ingested...
formula containing galactose. Early symptoms are vomiting, weight loss, and CNS symptoms, including poor feeding, drowsiness, and seizures. If the disorder goes untreated, the galactose levels will continue to increase and the affected infant will show failure to thrive, mental retardation, cata-
aracts, jaundice, hepatomegaly, and cirrhosis of the liver, with death possibly occurring in the first month of life. Therapy consists of eliminating galactose from the diet; this condi-
tion precludes breastfeeding because lactose is present in breast milk.

Congenital hypothyroidism results from a deficiency of thyroid hormones; it affects approximately 1 of every 3500 to 4000 newborns (Rose, 2002). All states in the United States routinely screen for hypothyroidism. This involves measuring thyroxine (T4) in a drop of blood obtained from a heel stick at 2 to 5 days of age. At this time the normally expected increase in T4 would be lacking in newborns with hypothyroidism. It is more often included as part of the new-
born screen done in the first 24 to 48 hours or before dis-
charge. Neonatal screening consists of an initial filter paper blood spot T4 measurement followed by measurement of thyroid-stimulating hormone (TSH) in specimens with low T4 values. Early screening may have false-positive results. Treatment is thyroid replacement. In the newborn, thyroid function test results are elevated in comparison with values in older children; therefore it is important to document the timing of the tests. In preterm and sick full-term infants thy-
roid function test results are usually lower than in the healthy full-term infant. Therefore it is important to document the
timed of the tests. In preterm and sick full-term infants thy-
roid function test results are usually lower than in the healthy full-term infant; T4 and TSH levels may be evaluated again after 30 weeks (corrected age) in newborns born before that
time and after resolution of the acute illness in the sick full-
term infant.

Cytogenetic Studies
Abnormalities can occur in either the autosomes or the sex chromosomes. Chromosomal disorders may sometimes be diagnosed on the basis of the clinical manifestations alone. However, an infant may have a clinical appearance that is only suggestive of a problem. Cytogenetic studies then need to be done to confirm or rule out a suspected diagnosis. Newer techniques in molecular cytogenetic analysis make possible a more precise identification of risk for having a fe-
tus affected with a genetic defect such as PKU.

Disorders in the number or structure of chromosomes can be diagnosed by a karyotype (see Fig. 8-1, B), which is a pho-
tographic enlargement of the chromosomes arranged by their
numbered pairs.

Dermatoglyphics
Dermatoglyphics is the study of the patterns formed by the ridges in the skin on the digits, palms, and soles. These pat-
terns, formed early in development, are strongly correlated with the effects of chromosomes. Many disorders that affect multiple body systems also affect these dermal ridges. The ad-
dition or deletion of genetic material produces alterations in the loops, swirls, and arches of the finger and toe prints, in the palm lines, and in the flexion creases on the palms of the hands and soles of the feet. Characteristic dermato-
glyphic patterns have been noted for almost all the chro-
mosomal abnormalities, such as Down syndrome.

An infant with Down syndrome may have a single, pal-
mar crease; a single flexion crease of the fifth digit; and an
increased distance between the first and second toes (Matthews & Robin, 2002). The characteristic dermato-
glyphic feature in a child with Turner syndrome is the large size of the dermal patterns on the fingers and toes. Certain fingerprint patterns may also be found in those people who have cardiac valvular problems later in life. Asymmetry of palmar ridges has been reported in congenital anomalies such as cleft lip and palate and congenital vertebral anomaly.

Interventions
A collaborative health team approach that includes spe-
cialists and community service representatives is needed in
the care of infants with some disorders. Surgical intervention
in the neonatal period may be necessary for the infant re-
quiring either immediate correction or a palliative procedure to relieve the symptoms of the anomaly until definitive cor-
rection can be done. There is a higher morbidity and mor-
tality in neonates than in older children or adults undergo-
ing similar procedures. However, despite these problems
unique to neonates, advances in surgical techniques, anes-
thesia, and the nursing care given in intensive care nurseries
have together been responsible for decreasing the risk of sur-
gery in neonates.

The health care team must be highly skilled to meet the
needs of these infants. These needs are similar to those of
other high risk infants. In addition to stabilization of the in-
fant’s condition (oxygenation and perfusion of tissues), other
preoperative interventions, such as nasogastric tube place-
ment for abdominal decompression, pain management, and
the maintenance of fluid and electrolyte balance, are im-
plemented to manage specific problems.

Postoperative care of the newborn
Postoperatively, the infant is returned to the intensive
""
Care of parents and family

While the infant is receiving optimal care, the parents also have needs that must be met as they deal with the crisis of having an infant with an abnormal condition. Their reactions are carefully assessed and are likely to be those typical of a grief response. Facilitating their understanding of the information given them about their infant’s condition is a vital nursing intervention. A newly diagnosed disorder often implies the need for the implementation of a therapeutic regimen. For example, the disorder may be an inborn error of metabolism, such as PKU, which requires consistent and rigid adherence to a diet. The family may need help with selecting the required formula and in receiving counseling from a clinical dietitian. The importance of maintaining the diet, keeping an adequate supply of special preparations, and avoiding the use of unauthorized substitutions must be impressed on the family.

Referral to appropriate agencies is another essential component of the follow-up management, and the nurse should make the parents aware of all possible sources of aid, including pertinent literature, parent groups, and national organizations. Many organizations and foundations, such as the March of Dimes, provide services and counseling for families of affected children. There are also numerous parent groups that families can join. There they can share experiences and derive mutual support in coping with problems similar to those of other group members. Nurses must be familiar with the services available in their communities that provide assistance and education to families with these special problems.

A major nursing function is providing emotional support to the family during all aspects of the care of the child born with a defect or disorder. The feelings stemming from the real or imagined threat posed by a congenital anomaly are as varied as the people being counseled. Responses may include apathy, denial, anger, hostility, fear, embarrassment, grief, and loss of self-esteem.

Parents benefit from seeing before-and-after pictures of other babies born with the same defect. Coupled with other verbal and nonverbal supportive care, this visual reassurance may be effective in allaying their concerns.

Families need much information, guidance, and support as they make decisions regarding the care of their infant. Once they have been given the facts and possible consequences and all the assistance they need in problem solving, the final decision regarding a course of action must be their own. It is then incumbent on health care providers to support the decision of the family.

Nurses frequently encounter children with genetic diseases and families in which there is a risk that a disorder may be transmitted to or occur in an offspring. It is a responsibility of nurses to be alert to situations in which persons could benefit from a genetic evaluation and counseling to be aware of the local genetic resources, to aid the family in finding services, and to offer support and care for children and families affected by genetic conditions. Local genetic clinics can be located through several sites, such as Gene Tests (www.genetests.org), a publicly funded medical genetics information resource developed for physicians and other health care providers, which is available at no cost to all interested persons. Another resource is the National Society of Genetic Counselors (www.nsgc.org), which lists genetic counselors by states in the United States. See also Chapter 7.

Key Points

- The identification of maternal and fetal risk factors in the antepartum and intrapartum periods is vital for planning adequate care of high risk infants.
- A small percentage of significant birth injuries may occur despite skilled and competent obstetric care.
- Infection in the newborn may be acquired in utero, at birth, in breast milk, and from within the nursery.
- The most common maternal infections during early pregnancy that are associated with various congenital malformations are represented by the acronym TORCH.
- HIV transmission from mother to infant occurs transplacentally at various gestational ages, perinatally through maternal blood and secretions, and through breast milk.
- Preterm infants are at risk for problems related to the immaturity of organ systems.
- Hyperbilirubinemia has a variety of etiologic factors, including maternal-fetal Rh and ABO incompatibility.
- The injection of Rh(D) immune globulin in Rh-negative and Coombs’ test-negative women minimizes the possibility of isoimmunization.

COMmUNITY ACTIVITY

Using the local telephone directory, identify sources of referral in your community for infants with congenital anomalies or who have been exposed to drugs such as cocaine or heroin. How many sources were you able to find? How difficult was it for you to locate these sources? Select one of the agencies and call to inquire (after identifying yourself as a nursing student) about (a) their source of funding, (b) whether those without insurance can access their services, (c) who can make referrals, (d) the number of children seen each year, and (e) the major problems seen. What have you learned from this exercise? Are there enough services available in your community to assist parents who need these services?
UNIT SEVEN
COMPLICATIONS OF CHILDBEARING

Key Points—cont’d

• The nurse often first observes signs of newborn drug withdrawal (neonatal abstinence syndrome) and acquires information from the maternal history.
• Major congenital defects are now the leading cause of death in infants under one year of age.
• The curative and rehabilitative problems of a child with a congenital disorder are often complex, requiring a multidisciplinary approach to care.
• Parents often need special instruction (e.g., cardiopulmonary resuscitation, oxygen therapy, or meeting nutrition requirements) before they take a high risk infant home.
• The supportive care given to the parents of infants with an abnormal condition must begin at birth or at the time of diagnosis and continue for years.

Answer Guidelines to Critical Thinking Exercise
Birth of a Child with a Congenital Anomaly

1. Yes, evidence is sufficient to draw conclusions about what you should tell Marjorie about the treatment and prognosis for Monica. The defect is not visible; symptoms are minimal. Breastfeeding is possible if Marjorie desires.

2. a. While a VSD is not visible, it can be serious. At times, parents may think a defect is not serious if they cannot see it. In Monica’s case the pediatrician thinks that the small defect will close spontaneously.
   b. When the infant is quiet and has good color, Marjorie may find it hard to believe that anything is wrong; she may use denial to cope with the situation. The nurse can assist Marjorie to accept the reality of the defect.
   c. Many small defects close spontaneously during the first year of life. Most children with small defects remain asymptomatic and require no treatment.

3. d. Infective endocarditis is a long term risk. Some adults with small VSDs have an increased incidence of subaortic stenosis, arrhythmias, and exercise in tolerance.
   The priority for nursing care at this time is to explain the VSD and possible sequelae. Marjorie will need assurance that follow-up will occur. She should be educated about the symptoms of adverse events including congestive heart failure and endocarditis. It is important to include the father of the baby and her family in the teaching. Reassurance and support is essential. Referral to a social worker may be useful if Marjorie does not have the resources to pay for the follow-up care.

4. Treatment of the child with a small VSD is symptomatic and supportive.

5. Although unlikely, parents may reject children with defects or be overprotective and not permit them to participate in activities of children without similar defects even when there is no reason to limit such activities.

Resources

Advances in Neonatal Care
Elsevier
360 Park Ave., South
New York, NY 10010
AIDS Network Hotline
800-424-2477
American Academy of Pediatrics (AAP)
141 Northwest Point Blvd.
Elk Grove, IL 60007-1098
847-228-5005
www.aap.org
American Cleft Palate Association
1216 Grandview Ave.
Pittsburgh, PA 15211
412-681-1376
800-224-5318 (800-24-CLEFT)
www.cleftline.org
American Society of Plastic Surgeons
Plastic Surgery Educational Foundation
Plastic Surgery Information Service: FAQs
www.plasticsurgery.org/faq/cleft.htm
Centers for Disease Control and Prevention (CDC)
1600 Clifton Rd., NE
Atlanta, GA 30333
404-329-1819
404-329-2286
www.cdc.gov

d. Infective endocarditis is a long term risk. Some adults with small VSDs have an increased incidence of subaortic stenosis, arrhythmias, and exercise in tolerance.

3 The priority for nursing care at this time is to explain the VSD and possible sequelae. Marjorie will need assurance that follow-up will occur. She should be educated about the symptoms of adverse events including congestive heart failure and endocarditis. It is important to include the father of the baby and her family in the teaching. Reassurance and support is essential. Referral to a social worker may be useful if Marjorie does not have the resources to pay for the follow-up care.

4 Treatment of the child with a small VSD is symptomatic and supportive.

5 Although unlikely, parents may reject children with defects or be overprotective and not permit them to participate in activities of children without similar defects even when there is no reason to limit such activities.

Gene Test (funded by the NIH)
9721 Third Ave., NE
Suite 602
Seattle, WA 98115
206-616-4033
206-221-4679 (fax)
genetests@genetests.org
www.genetests.org
HEST (Helga’s European Specialty Toys) (Down syndrome dolls used to teach children about disabilities and for children with Down syndrome)
www.downsyndromedolls.com
Journal of Genetic Counseling
Kluwer Academic Publishers
P.O. Box 122
3300 AH Dordrecht
The Netherlands
+31 (0) 78 637 60 50
+31 (0) 78 637 64 74 (fax)
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Journal of Perinatal and Neonatal Nursing
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