OBJECTIVES

1. Identify the risk factors and maternal and fetal history for complications related to gestational age and birthweight issues in the neonate such as prematurity, postmaturity, and small for gestational age (SGA) and large for gestational age (LGA) infants.
2. Describe physical characteristics of preterm, late preterm, postterm, SGA, and LGA infants.
3. Recognize potential problems related to preterm, late preterm, postterm, SGA, and LGA infants.
4. Describe the immediate assessment parameters and management of the premature infant.
5. Identify risk factors and the maternal and fetal history that are predictive of respiratory distress syndrome (RDS).
6. Describe the specific pathophysiology, assessment parameters, and management of RDS.
7. Identify risk factors and the maternal and fetal history that are predictive of transient tachypnea of the newborn (TTN).
8. Describe the specific pathophysiology, assessment parameters, and management of TTN.
9. Identify risk factors and the maternal and fetal history that are predictive of meconium aspiration syndrome (MAS).
10. Describe the specific pathophysiology, assessment parameters, and management of MAS.
11. Identify risk factors and the maternal and fetal history that are predictive of persistent pulmonary hypertension of the newborn (PPHN).
12. Describe the specific pathophysiology, assessment parameters, and management of PPHN.
13. Describe the maternal and neonatal factors that contribute to jaundice in the neonate, and distinguish among the various causal factors and related outcomes of jaundice in the neonate.
14. Describe the physiologic process of the production, conjugation, and elimination of bilirubin in the neonate; the differences between conjugated and unconjugated bilirubin; and assessment parameters and management of hyperbilirubinemia.
15. Describe the maternal and neonatal factors that contribute to congenital anomalies in the neonate, and distinguish the differences among the various causal factors and related outcomes.
16. Describe the specific pathophysiology, assessment parameters, and management of common congenital anomalies in the neonatal period.

INTRODUCTION

Most newborns will transition to extrauterine life without a problem. However, even when transition is uneventful, the first 48 hours of life is when vigilant observation and anticipatory caregiving are essential. Most infants with serious illness present at birth or within the first 48 hours during the transition to extrauterine life. The challenge for the caregiver is to be able to discriminate the subtle signs of disease from the dynamically changing characteristics of normal transition and adaptation to the environment. Without excellent nursing care and good family education, early discharge might lead to some of these infants not being identified and thus being susceptible to poorer outcomes. Some of the most common disease processes that can appear in the newborn period include gestational age and birthweight-related issues, hypothermia, hypoglycemia, RDS, TTN, MAS, PPHN, sepsis, congenital anomalies, hyperbilirubinemia, and drug exposure of the infant. Newborn sepsis is discussed in Chapter 20, and the drug-exposed infant is covered in Chapter 25. For ease of discussion, gestational age and birthweight-related issues are addressed first followed by prematurity, respiratory and cardiac conditions, hyperbilirubinemia, and the most common congenital anomalies. However, it is important to note that these processes can occur concurrently, and in reality often infants diagnosed with any one of the previously noted newborn disease processes are more susceptible to the others.
SMALL FOR GESTATIONAL AGE INFANTS

INTRODUCTION

A. An infant is defined as SGA when the weight is below the 10th percentile (Anderson & Hay, 2005; Townsend, 2005).

B. The SGA infant may also be known as having intrauterine growth restriction (IUGR).

C. Not all IUGR infants are SGA; IUGR from placental insufficiency usually reduces birthweight more than length and to a greater degree than head circumference; the greater the severity of IUGR, the greater is the deviation of weight, length, and (less so) head circumference as compared with population norms (Anderson & Hay, 2005; Rosenberg, 2008).

D. The SGA infant can be preterm, term, or postterm.

E. Conditions (alone or in combination) associated with SGA babies are as follows (Hendrix & Berghella, 2008):

1. Maternal conditions
   a. Chronic hypertension (associated with a four- to eightfold increase in the incidence of abruptio placentae)
   b. Anemia
   c. Cardiorespiratory disease
   d. Drug exposure (diethylstilbestrol, antineoplastics, narcotics, and illicit drugs)
   e. Smoking (frequently associated with abruptio placentae, placenta previa, prematurity, and respiratory distress) and alcohol consumption
   f. Young adolescent (10 to 14 years of age) or advanced maternal age (older than 35 years)
   g. Asthma

2. Fetal conditions
   a. Chromosomal abnormalities
   b. Heart disease and hemolytic disease
   c. Intrauterine infection: toxoplasmosis, rubella, cytomegalovirus, and herpes simplex (TORCH)
   d. Malformations
   e. Multiple gestation

3. Factors affecting the intrauterine environment
   a. Preeclampsia or eclampsia
   b. Decreased uteroplacental blood flow
   c. Diabetes mellitus
   d. Morphologic abnormalities
   e. Chorioamnionitis

4. Placental conditions
   a. Abruptio placenta
   b. Placenta previa

5. Environmental conditions
   a. High altitude
   b. Therapeutic x-ray exposure

F. Conditions altering fetal growth produce insults that affect all organ systems and are known to produce two patterns of growth that depend on the timing of the insult to the developing embryo or fetus (Rosenberg, 2008).

1. Conditions affecting early gestation (generally less than 28 weeks) occur at a time when rapid cell proliferation (hyperplasia) occurs.
   a. An insult at this stage results in organs with cells of normal size but fewer numbers of cells than if the insult had not occurred.
   b. Infants are symmetrically grown (weight, length, and head circumference plot similarly on a growth curve) and all organ systems are small.
   c. Generally these infants have the poorest long-term prognosis and commonly have chromosomal abnormalities; postnatal nutrition is unable to correct for growth deficits; symmetrically grown SGA babies may never catch up in size when compared with unaffected children.

2. Later in gestation (greater than 28 weeks), growth occurs as a combination of rapid cell proliferation (hyperplasia) but also as a result of increases in cell size (hypertrophy).
   a. An insult at this stage typically results in intrauterine malnutrition; organ systems have normal numbers of cells that are smaller.
b. The brain and heart are larger in proportion to body size as a whole, whereas the liver, spleen, adrenals, thymus, and placenta are small.

c. This type of infant is asymmetrically grown in that head size and length are spared, but overall weight and organ sizes are diminished.

d. Generally, the asymmetrically IUGR infant has a better prognosis than one who is symmetrically IUGR; in utero malnutrition, however, is associated with increased risk of intrauterine death (Rosenberg, 2008; Townsend, 2005).

e. Optimal postnatal nutrition generally restores normal growth potential because the number of body cells is normal.

G. The SGA infant may present with problems from the moment of birth (Rosenberg, 2008; Townsend, 2005).

1. Fewer reserves are available to help the fetus tolerate the rigors of labor and delivery, leading to the development of fetal asphyxia or meconium passage in utero and the need for resuscitation at delivery.
   a. Uteroplacental circulation is often impaired. A small placenta may have diminished capability for gas exchange, nutrient delivery, and removal of waste products from the fetal circulation.
   b. Cardiac glycogen stores may already be reduced, leading to the development of fetal bradycardia.
   c. Uterine contractions may add an additional hypoxic stress on the chronically hypoxic fetus with a marginally functioning placenta.

2. The combination of intrapartum and neonatal asphyxia places the infant at increased risk for a continuum of central nervous system insults that are the sequelae of perinatal asphyxia.

3. Decreased glycogen stores increase the potential for early development of hypoglycemia and temperature instability in the transition period (see later discussion of preterm infants).

4. Polycythemia frequently occurs as a result of chronic subacute hypoxia and dehydration.

H. Congenital anomalies are more frequently associated with intrauterine insult early in gestation during organogenesis; mortality rates for term SGA infants are five times that of term, appropriately grown infants resulting from the occurrence of major congenital anomalies (Mandruzzato et al, 2008).

I. The SGA infant is more frequently exposed to intrapartum infections such as rubella, cytomegalovirus (CMV), and toxoplasmosis; risk for impaired fetal gas exchange related to inadequate umbilical cord perfusion, hypoxia, and hypercarbia (Rosenberg, 2008).

J. Immune function in the SGA infant may be depressed as in older children with postnatal onset of malnutrition (Anderson & Hay, 2005; Mandruzzato et al, 2008).

K. The prognosis for SGA infants must consider adverse perinatal consequences in addition to being SGA; when perinatal problems are minimal or avoided because of early optimal obstetric intervention, the SGA neonate may still demonstrate developmental handicaps, especially in head growth restriction (Rosenberg, 2008).

L. Socioeconomic status and environment are the major determinant of developmental outcome at 2 years of age and older; SGA infants born to families of higher socioeconomic status demonstrate fewer developmental differences on follow-up, whereas those born to poorer families have significant developmental handicaps (Mandruzzato et al, 2008).

CLINICAL PRACTICE

A. Assessment

1. History (Rosenberg, 2008; Townsend, 2005)
   a. Antenatal findings
      (1) Maternal weight gain
      (2) Age and socioeconomic status
      (3) Maternal illnesses or conditions
         (a) Renal
         (b) Cardiac; hypertension
         (c) Phenylketonuria (PKU)
      (4) Substance use or abuse such as alcohol, illicit drugs, or tobacco
Pregnancy conditions
   (a) Oligohydramnios
   (b) Multiple gestation
(6) Elevated TORCH titers or other signs of infection

b. Intrapartum findings
   (1) Length of gestation
   (2) Color, consistency, and amount of amniotic fluid
   (3) Fetal heart rate patterns suggestive of distress

2. Physical findings (Rosenberg, 2008; Townsend, 2005)
a. Soft-tissue wasting and dysmaturity
   (1) Decreased amount of breast tissue
   (2) Diminished subcutaneous fat tissue
   (3) Loose, dry, and cracked skin, with decreased turgor
   (4) Diminished muscle mass especially noticeable in the buttocks and extremities
   (5) Scaphoid abdomen resulting from shrinkage of the abdominal contents
b. Smaller-than-average weight, length, and head circumference
   (1) The symmetric IUGR infant is smaller in all growth parameters (weight, length, and head circumference).
   (2) The asymmetric IUGR infant is smaller-than-average weight and average head circumference and length.
      (a) Large head-to-body ratio
      (b) Poor head control

3. Presenting behavioral findings seen at or soon after delivery depend on the occurrence of asphyxia (postasphyxial encephalopathy) (Rosenberg, 2008; Towsend, 2005).
a. Mild degree (duration less than 24 hours) exhibited by hyperalertness and sympathetic overactivity
b. Moderate degree exhibited by lethargy, stupor, hypotonia, suppressed primitive reflexes, and seizures
   c. Severe degree manifested by coma, flaccid tone, suppressed brainstem function, seizures, and increased intracranial pressure

4. Placental examination (Hendrix & Berghella, 2008)
a. Abnormal cord insertion
b. Placental hemangiomas, multiple infarcts, or chronic abruptio placentae
c. Placenta previa

5. Diagnostic procedures (Townsend, 2005)
a. Weight, length, and head circumference
b. Gestational age assessment and plotting of growth parameters on curve
c. Serial bedside glucose assessment
d. Assessment for infection (see Chapter 20 for a complete discussion of sepsis)
   (1) Complete blood count (CBC) with differential and platelets (also assess for polycythemia)
   (2) Viral studies
      (a) TORCH titer
      (b) Urine for CMV titer and culture
      (c) Nasopharyngeal culture for rubella
   (3) Possible lumbar puncture
   (4) Possible total and direct bilirubin levels
   (5) Coagulation studies if indicated by thrombocytopenia or petechia

B. Interventions/Outcomes (Rosenberg, 2008; Townsend, 2005)
1. Interventions are described for the most common problems: birth asphyxia; respiratory distress; temperature instability; blood glucose instability; nutritional support; polycythemia; infection related to possible exposure to intrauterine infection (see Chapter 20 for complete discussion of newborn sepsis).
a. Anticipate the need for and provide neonatal resuscitation according to Neonatal Resuscitation Program (NRP) guidelines as indicated by condition at the time of delivery.
b. Monitor and record trends in transition vital signs, blood pressure, and clinical parameters; anticipate clinical manifestations such as tachypnea, respiratory distress, acidosis, cardiovascular instability, cyanosis, and hypoxemia.
c. Provide oxygen as indicated based on pulse oximeter saturation monitoring, blood gas values, and close observation.

d. Provide stabilization care in a neutral thermal environment (NTE), and allow the infant to stabilize and self-correct mild acidosis, clear lung fluid, stabilize blood glucose, and stabilize blood pressure.

e. Monitor infant’s body temperature: axillary should be in the range of 36.4° to 37° C (97.6° to 98.6° F).

f. Examine the environment for potential sources of heat loss to prevent cold stress; for example, prewarm equipment, and avoid exposure to drafts.

g. Monitor incubator or warmer bed temperature and heater output; the nurse should be concerned if heater output is constant.

h. Monitor blood glucose levels if temperature instability occurs (to determine if hypoglycemia is causing temperature instability); anticipate blood glucose instability and hypothermia if the infant is fasting or as the infant transitions to bolus feedings; administer intravenous glucose (see discussion of hypoglycemia under care of preterm infant).

i. Initiate early and frequent oral feedings (every 2 to 3 hours) if not contraindicated by respiratory status; provide a high-calorie formula (>20 calories/ounce [30 mL]) as ordered to provide additional nutrients.

j. Obtain serum hemoglobin (normal 15 to 21.5 g/dL) and hematocrit (normal 45% to 65%) levels.

k. Observe for signs and symptoms of polycythemia.
   1. Ruddy appearance
   2. Cyanosis; may be more pronounced with activity or crying
   3. Tachypnea
   4. Persistent hypoglycemia
   5. Apnea or bradycardia
   6. Jaundice

l. Consider partial exchange transfusion for polycythemia when an infant is symptomatic to relieve capillary congestion and hyperviscosity.

2. Outcomes
   a. Infant’s 5-minute Apgar score is 7 to 10.
   b. Vital signs, blood pressure, blood glucose, and clinical parameters are stable.
   c. Oxygen saturation is maintained within normal limits.
   d. Normal body temperature is maintained.
   e. Neutral thermal environment is maintained.
   f. Infant shows no signs of cold stress, for example, increased oxygen consumption, hypoglycemia, and/or respiratory distress.
   g. Blood glucose levels are maintained at greater than 40 mg/dL.
   h. Oral feedings are tolerated well.
   i. Intravenous (IV) dextrose infusion, if indicated, maintains blood sugar within normal limits.
   j. Infant’s initial weight loss stabilizes within 3 to 5 days of life, and weight increases thereafter at an average of at least 15 to 30 g (0.5 to 1 ounce) per day.
   k. Serum hematocrit is less than 65%.
   l. Signs and symptoms of hypoglycemia and polycythemia are absent.
   m. Neonate’s intake is sufficient to achieve a urine output greater than 1.5 mL/kg/hr.

HEALTH EDUCATION

A. Inform parents of possible causes of IUGR.
B. Assist parents with guilt if chronic illness is a factor or if mother used substances known to compromise fetal growth.
C. Make parents aware of the discharge parameters for their newborn.
D. Instruct parents or family members on managing infant at home.
   1. Preparation of higher-caloric formula or frequent breastfeeding
   2. Performance of gavage feeding
   3. Use of developmental therapist to screen for developmental milestones and help optimize development
LARGE FOR GESTATIONAL AGE INFANTS

INTRODUCTION

A. The LGA infant is one whose weight is above the 90th percentile for gestational age (Townsend, 2005).

B. LGA babies may be preterm, term, or postterm.

C. Birthweight more than 4000 grams (8 pounds, 14.5 ounces) often reflects a genetic predisposition, except for the infant of a diabetic mother (IDM).
   1. Large parents tend to have large babies.
   2. Some Native Americans are more likely to have LGA infants.

D. Large size of the fetus may predispose the mother to an operative delivery.

E. If an LGA infant is born vaginally, the incidence of operative vaginal delivery (forceps or vacuum-assisted delivery) is higher than in the non-LGA infant; birth trauma is higher when compared with non-LGA babies and may include:
   1. Fracture of the clavicle or humerus
   2. Brachial plexus injuries
   3. Facial palsy
   4. Depressed skull fracture
   5. Cephalohematoma

F. The LGA fetus may show evidence of nonreassuring fetal heart rate patterns during a prolonged and difficult second stage of labor; neonatal respiratory depression may occur at the time of the delivery.
   1. Shoulder or body dystocia may occur.
   2. Particulate meconium-stained amniotic fluid may occur with risk of aspiration.

G. LGA infants are at risk for hypoglycemia related to early depletion of glycogen stores (see Chapter 22 for a complete discussion regarding the IDM).

CLINICAL PRACTICE

A. Assessment

1. History
   a. Maternal
      (1) Previous delivery of an LGA neonate
      (2) Large weight gain during pregnancy
      (3) Diabetes (classes A through C) during the pregnancy
      (4) Prolonged or difficult labor and birth, particularly a long second stage
      (5) Ultrasonography that confirms fetal macrosomia
   b. Infant
      (1) Birthweight above the 90th percentile for gestational age
      (2) Type of delivery
         (a) Cesarean birth
         (b) Vaginal delivery with possible shoulder or body dystocia
         (c) Vacuum extraction or forceps-assisted delivery
      (3) Apgar scores at 1 and 5 minutes suggestive of neonatal respiratory depression
      (4) Particulate meconium-stained amniotic fluid

2. Physical findings (Townsend, 2005)
   a. Weight greater than 90th percentile for gestational age
   b. Presence of caput succedaneum on the head
      (1) Localized soft tissue swelling over the presenting scalp area
      (2) Is present at birth and does not increase in size
      (3) Typically disappears within 12 to 48 hours
   c. Presence of a cephalohematoma
      (1) Increased incidence with vacuum extraction
      (2) Soft, fluctuant swelling in which the margins are limited to a cranial bone; does not cross suture lines
      (3) Increases in size for 2 to 3 days after birth
(4) Disappears 6 to 8 weeks after birth
(5) May be associated with complications
   (a) Jaundice, hyperbilirubinemia, or both
   (b) May be accompanied by a skull fracture with resultant subdural or subarachnoid hemorrhage
   (c) May be accompanied by intracranial hemorrhage
d. Evidence of facial nerve damage, resulting from intrapartum pressure on facial nerves related to abnormal fetal position or forceps trauma
   (1) The eye on the affected side does not completely close as it normally does while the infant is crying.
   (2) The forehead does not wrinkle.
   (3) The side of the face is smooth.
   (4) The corner of the mouth droops.
(e) Evidence of brachial plexus injury as a result of overextension and torsion of the neck at the time of delivery resulting in overstretching, hemorrhage or tearing, or complete avulsion of the cervical nerve roots from the spinal cord
   (1) Erb’s palsy (the most common type) as a result of upper cervical nerve root damage (C-5 and C-6)
      (a) Muscles of the upper arm are paralyzed.
      (b) The affected arm hangs limp, adducted, and internally rotated at the shoulder; movements that cannot be accomplished are:
         [i] Abduction and external rotation at the shoulder
         [ii] Flexion at the elbow and supination
      (c) Affected arm is pronated at the elbow and wrist is flexed, with strong palmar grasp present.
      (d) Deep tendon reflexes are absent.
      (e) Moro response is unilateral.
      (f) Occasionally associated with unilateral diaphragmatic paralysis, as evidenced by:
         [i] Asymmetry of chest expansion
         [ii] Tachypnea
         [iii] Cyanosis and/or dyspnea
   (2) Klumpke’s palsy (rare) as a result of lower cervical root damage (C-8 to T-1 nerve roots)
      (a) The condition is limited to the wrist and hand.
      (b) The grasp reflex is abolished, the hand is held limply flexed, and voluntary movements of the wrist cannot be made.
      (c) Often associated with this are the manifestations of paralysis of the cervical sympathetic nerve (Horner’s syndrome) on the same side.
         [i] Miosis of the pupil
         [ii] Slight lid droop
         [iii] Variations in local temperature, color, and sweating may appear later.
   (3) Complete brachial palsy (rare) as a result of injury to all roots from C-5 to T-1, producing entire paralysis of the arm and complete loss of sensation
(f) Evidence of clavicular or humeral fracture, either complete or incomplete
   (1) Decreased movement of affected side or arm may be seen when startle reflex is elicited.
   (2) Infant may cry in pain when affected area is manipulated; crepitus may be elicited.
   (3) Visible angulation or hematoma over the fracture site
   (4) Hypermobility of the bone
   (5) X-ray study confirms the diagnosis.
g. Evidence of hypoglycemia (see later discussion under Preterm Infant)
h. Signs of respiratory distress (see later discussion of respiratory distress)
   (1) Effort, character, and rate of respirations (increased, labored, greater than 60 breaths per minute)
   (2) Retractions: supraclavicular, intercostal, and substernal
   (3) Nasal flaring
   (4) Grunting
3. Diagnostic procedures
   a. Serum glucose
   b. X-ray study to assess for skeletal birth injuries
   c. X-ray study to assess for cause of respiratory distress
   d. Ultrasound scan or computed tomography (CT) for possible head injuries if cephalohematoma or depressed skull fracture is noted

B. Interventions/Outcomes
   1. Interventions are described for the most common problems: birth asphyxia; meconium aspiration (see later discussion); respiratory distress (see later discussion); birth trauma; temperature instability; blood glucose instability; nutritional support; hyperbilirubinemia; infection (see Chapter 20 for complete discussion of newborn sepsis).
      a. See discussion of hypoglycemia under care of preterm infant.
      b. See discussion of nutritional support under care of SGA infant
      c. See discussion of meconium aspiration.
      d. See discussion of respiratory distress.
      e. See discussion of temperature instability under care of preterm infant.
      f. On initial and repeat physical examination, note the following:
         (1) Size and position of caput or cephalohematoma
         (2) Evidence of skeletal bone fracture
         (3) Evidence of facial palsy or brachial plexus injury
      g. Observe for jaundice secondary to bruising or trauma (see discussion of hyperbilirubinemia).
      h. Provide treatment for any incidence of palsy.
         (1) Begin physical therapy and splinting early to prevent formation of contractures.
         (2) Provide gentle range-of-motion exercises to the affected extremity periodically.
         (3) Teach parents how to handle the infant without causing additional injury, provide range-of-motion exercises, and put on and take off splints.
      i. Provide treatment for fractured clavicle.
         (1) Obtain x-ray film for confirmation.
         (2) Immobilize affected arm and shoulder.
         (3) Support back and arm when lifting the infant.
         (4) Teach parents to expect a small bump over the fracture site to appear as healing occurs.
   2. Outcomes
      a. Infant’s 5-minute Apgar score is 7 to 10.
      b. Vital signs, blood pressure, blood glucose, and clinical parameters are stable.
      c. Oxygen saturation is maintained within normal limits.
      d. Normal body temperature is maintained.
      e. Neutral thermal environment is maintained.
      f. Infant shows no signs of cold stress; for example, increased oxygen consumption, hypoglycemia, and/or respiratory distress.
      g. Blood glucose levels are maintained at greater than 40 mg/dL.
      h. Oral feedings tolerated well
         i. IV dextrose infusion, if indicated, maintains blood sugar within normal limits.
      j. Infant’s initial weight loss stabilizes within 3 to 5 days of life, and weight increases thereafter at an average of at least 15 to 30 g (0.5 to 1 ounce) per day.
      k. Effects of trauma are minimized.
      l. Discomfort related to fracture is minimized or improved.
      m. Bilirubin levels remain within normal range or return to normal if phototherapy is instituted.

HEALTH EDUCATION

A. Remind parents of the infant’s immaturity and fragility despite her or his large size.
B. If the delivery was traumatic for the mother, she may need extra recuperation time before assuming total care of the infant.
C. Assist the parents to lift, position, and care for their large infant, especially for breastfeeding.
D. Instruct parents or family members regarding birth trauma, expected resolution, handling or treatment, and follow-up.
POSTTERM INFANTS

INTRODUCTION

A. A postterm pregnancy is one that extends beyond 41 completed weeks' gestation (Avery & Richardson, 1998).

B. Postterm neonates may be LGA, average for gestational age (AGA), SGA, or dysmature, depending on placental function.
   1. If the placenta continues to function well, the fetus will continue to grow for the extra time in utero, which results in an LGA neonate with typical problems of LGA neonates (as previously stated).
   2. If placental function decreases, the fetus may not receive adequate nutrition and wasting of subcutaneous fat, muscle, or both occurs (Doherty & Norwitz, 2008).
      a. As the placenta loses its ability to nourish the fetus (placental insufficiency), the fetus uses stored nutrients for nutrition and wasting occurs; the body is lean, with thin extremities and little subcutaneous fat.
      b. This condition occurs in three forms
         (1) Chronic placental insufficiency
            (a) No meconium staining occurs.
            (b) Infant appears malnourished with skin changes.
            (c) Infant has an apprehensive look, reflecting hypoxia.
         (2) Acute placental insufficiency
            (a) Infant has a malnourished and apprehensive appearance.
            (b) Green meconium staining of the skin, umbilical cord, and placental membranes occur.
         (3) Subacute placental insufficiency
            (a) Skin and nails are stained golden yellow (resulting from breakdown of green meconium to hydrolyzed meconium, which is golden or yellow).
            (b) Umbilical cord, placenta, and placental membranes may be greenish brown.

C. Because of the incidence of placental degeneration, postterm neonates are susceptible to perinatal asphyxia and meconium passage (Doherty & Norwitz, 2008).
   1. Prenatal asphyxia often results in meconium passage in utero with or without fetal gasping.
   2. Aspiration of particulate meconium is highly likely to occur at the time of delivery with the first breath.
   3. The maternal care providers and neonatal resuscitation team plan together to provide management of the meconium (see later discussion of MAS). NRP guidelines are used to guide resuscitation care as needed.
   4. Intrauterine hypoxia may trigger increased red blood cell (RBC) production, leading to polycythemia, which results in the following:
      a. Sluggish perfusion of organ systems
      b. Hyperbilirubinemia resulting from breakdown of excessive numbers of RBCs

D. Postterm neonates are susceptible to hypoglycemia because of the rapid depletion of glycogen stores.

E. Postterm neonates experience skin and integument changes (Townsend, 2005).
   1. The skin is parchment-like and scaly.
   2. Loss of perfusion to the skin during prenatal asphyxia causes the top three layers of skin to die and slough, causing a macerated appearance.
   3. Loss of subcutaneous fat predisposes the neonate to increased extrarenal fluid loss and increased risk for hypothermia.
   4. Hair is abundant; nails are abnormally long; Wharton’s jelly is decreased, and the umbilical cord is thin.

F. Amniotic fluid volume is decreased, leading to potential fetal distress while in labor (Doherty & Norwitz, 2008).
   1. Asphyxial renal changes cause fetal urine production to decrease; a low amniotic fluid index (AFI) may be present (AFI <5 suggests severe oligohydramnios).
2. In utero umbilical cord compression is more likely to occur if the amount of amniotic fluid to cushion the cord is reduced and the cord is thin, and is exhibited as decelerations, bradycardia, or both.

3. Prenatal passage of meconium in the circumstance of reduced amniotic fluid volume means that the meconium is thicker and the risk of aspiration is increased.

**CLINICAL PRACTICE**

A. **Assessment**

1. **History** (Townsend, 2005)
   a. Estimated day of conception (EDC)
   b. Gestational age assessment based on prenatal ultrasonography, if available
   c. Color, consistency, and amount of amniotic fluid
   d. Placental grading, if available (see Chapters 3 and 8 for further discussion regarding placental functioning and grading)
      (1) Grades are based on deposits of calcium in the placenta that may interfere with adequate transfer of nutrients and oxygen to the fetus.
      (2) Grades II and III are mature.
   e. Irregular fetal heart rate patterns in labor
      (1) Variable decelerations, which are often the result of decreased amniotic fluid volume
      (2) Late decelerations and decreased or absent variability, which are indicative of nonreassuring fetal heart rate patterns
      (3) Bradycardia
   f. Apgar scores
   g. Cord blood gases

2. **Physical findings** (Townsend, 2005)
   a. Skin is leathery, wrinkled, cracked, and peeling and frequently stained with meconium.
   b. Vernix is absent except in protected areas (scant amounts in neck and groin creases only).
   c. Fingernails are long and frequently meconium stained.
   d. Lanugo is absent.
   e. Creases cover the entire soles of the feet.
   f. Breast buds are large (greater than 1 cm in diameter) and the areolae are full and raised.
   g. Ear cartilage is thick and firm; ears stand away from the head.
   h. Has a wide-eyed and alert appearance, with more time spent in alert states.
   i. Postterm SGA neonates frequently appear hungry, with frantic rooting and fist sucking.
   j. Postterm LGA infants may be lethargic and have poor sucking ability.
   k. Signs and symptoms of respiratory distress may be present.
   l. Signs of birth trauma may be present in large infants.

3. **Diagnostic procedures**
   a. Gestational age assessment plotted by growth parameters on growth curve
   b. Bedside blood glucose test monitoring
   c. Chest x-ray film to evaluate possible aspiration
   d. If respiratory distress is present, monitor oxygenation.
      (1) Oxygen saturation monitoring
      (2) Arterial blood gas assay

B. **Interventions/Outcomes**

1. Interventions are described for the most common problems: glucose instability; nutritional support; meconium aspiration; respiratory distress; temperature instability; poor skin integrity
   a. See discussion of hypoglycemia under Care of Preterm Infant.
   b. See discussion of nutritional support under Care of SGA Infant.
   c. See discussion of meconium aspiration.
   d. See discussion of respiratory distress.
   e. See discussion of temperature instability under Care of Preterm Infant.
   f. Skin integrity may be decreased related to the absence of protective vernix and prolonged exposure to amniotic fluid (see discussion of skin integrity under Care of the Preterm Infant).
HEALTH EDUCATION

A. Inform parents of the consequences or sequelae of resuscitation as it applies to their newborn.
B. Refer to neurodevelopmental follow-up as indicated.
C. Provide parents or family members with information about any trauma sustained at birth.
D. Explain that postterm infants may need to feed more frequently (i.e., every 2 to 3 hours).

PRETERM INFANTS

INTRODUCTION

A. A preterm infant is one who is born before the end of 37 completed weeks’ gestation (Moos, 2004).
B. Preterm infants, particularly those born before 34 weeks’ gestation, represent a prototype of high-risk infants because of immaturity of all organ systems, numerous physiologic handicaps, and significant morbidity and mortality (Townsend, 2005). Late preterm infants are also at risk and need to be followed more closely because findings suggest that short- and long-term development is affected (Engle, Tomashek, & Wallman, 2007; Kelly, 2006).
C. Risk factors (Moos, 2004)
   1. Premature birth is frequently associated with maternal social deprivation and socioeconomic risk factors that promote catecholamine release, leading to decreased uterine blood flow and uterine irritability.
      a. Poverty, work away from home, teen pregnancy, and single motherhood have been identified as high-risk factors for preterm delivery (Balchin & Steer, 2007).
      b. Race (especially African American) continues to be a major risk factor for prematurity.
      c. Smoking and the use of illicit drugs, such as cocaine and crystal methamphetamine, have direct effects on placental and uterine blood flow and are commonly associated with uterine contractions, maternal hypertension, and placental abruption.
   2. Many women who deliver prematurely after spontaneous premature labor have an intraamniotic infection; bacterial vaginosis is a major risk factor for premature delivery, and early diagnosis and treatment of such infections have been shown to reduce the incidence of premature delivery.
   3. As the number of fetuses per pregnancy increases, the mean gestational age at delivery decreases; the mechanism of prematurity probably relates to the increase in intrauterine volume as well as the increased rate of volume change with multiple gestation.
   4. Bicornuate uterus and septate uterus are associated with increased incidence of prematurity.
   5. Prenatal maternal complications increase the risk for preterm birth.
      a. Maternal cardiorespiratory disease, hypoxia, hemorrhage, shock, hypotension, and hypertension
      b. Severe maternal anemia
      c. Maternal diabetes may result in preterm delivery because of fetal and maternal indications (Barnes-Powell, 2007).
      d. Abnormal placental conditions affect oxygen transfer from mother to fetus and result in asphyxial insult to the developing fetal lung.
D. Clinical problems of the premature neonate are directly associated with the degree of organ maturity at birth; prematurity is not a disease but rather a lack of organ maturity.
   1. Without full development, organ systems are not usually capable of functioning at a level needed to maintain extrauterine homeostasis.
   2. The more immature or lower the gestational age, the greater the risk of complications and system failure.
E. The respiratory system is one of the last to mature; therefore, the preterm infant is at risk for numerous respiratory problems (see later discussion of respiratory distress for complete discussion of issues).
F. The cardiovascular system undergoes transition at birth from the fetal to the neonatal circulatory pattern; preterm delivery can adversely affect this transition (Lott, 2007).
1. Transition is a response, in part, to the increased level of oxygen in the circulation once air breathing has begun; if oxygen levels remain low, the fetal pattern of circulation may persist, causing blood flow to bypass the lungs (Agarwal, Deorari, & Paul, 2008).
   a. Preterm infants have a high incidence of patent ductus arteriosus (PDA) (Dagle et al., 2009) (see more detailed discussion of PDA later).
   b. The foramen ovale may remain open if pulmonary vascular resistance is high.
2. The heart is relatively protected from hypoxia in utero; injury, if present, is generally reflected after delivery as cardiomegaly, with signs of cardiovascular insufficiency.
3. Preterm infants may have impaired regulation of blood pressure in the face of apnea, bradycardia, mechanical ventilation, and other types of neonatal intensive care unit (NICU) care (Blackburn, 2007).
   a. Fluctuations in cerebral blood flow are common. These fluctuations predispose the fragile blood vessels in the brain to rupture, causing intracranial hemorrhage.
   b. Fluctuations can cause loss of brain blood flow, resulting in ischemia. These fluctuations also predispose the preterm infant to develop retinopathy of prematurity.

G. The immune system is both immature and inexperienced, making the preterm infant susceptible to infections (Blackburn, 2007).
1. Immunologic ability depends in part on immunoglobulins (Ig), such as IgG, IgM, and IgA.
2. Preterm infants often have a deficiency of IgG because of delivery before transplacental transfer (occurs at approximately 34 weeks’ gestation).
3. IgA (the primary Ig in colostrum) is not available to the preterm infant if he or she does not receive breast milk or colostrum.
4. On occasion, preterm delivery comes about as a result of maternal infection with pathogenic bacteria; the preterm infant is especially prone to developing group B beta-hemolytic Streptococcus infection.
5. The risk for infection in the preterm infant is also increased because of disruption of skin integrity and instrumentation in the course of NICU care.

H. The immature liver may be highly inefficient in conjugating bilirubin, leading to hyperbilirubinemia; drug metabolism in the liver may be markedly altered, increasing the risk of drug intolerance (Blackburn, 2007; see more detailed discussion of hyperbilirubinemia later).

I. The preterm infant has great difficulty maintaining body temperature (McGrath, 2007b).
1. The preterm infant is at great risk for excessive heat loss resulting from the following:
   a. Decreased or inadequate subcutaneous fat
   b. Large head-to-body ratio
   c. Lack of muscle tone and flexion
   d. Increased transepidermal evaporative losses
2. Brown fat is not available or is inadequate to generate heat because sufficient stores are not available for use until after approximately 30 weeks’ gestation.
3. Cold stress quickly depletes what brown fat and glycogen stores are present, resulting in the following:
   a. Increased metabolic needs
   b. Increased oxygen consumption
   c. Consequences that include metabolic acidosis, hypoxemia, and hypoglycemia
4. Poor nutrient intake is commonly associated with temperature instability.

J. The preterm renal system is immature, resulting in the following (Blackburn, 2007):
1. Decreased ability to concentrate urine
2. Lack of selectiveness in filtration
3. Decreased glomerular filtration rate (GFR)
   a. Decreased drug clearance
   b. Increased likelihood of fluid retention
   c. Increased likelihood to develop fluid and electrolyte disturbances

K. Periventricular intraventricular hemorrhage (PIVH) and ischemic changes are of particular significance in the preterm infant weighing less than 1500 g (3 lb 5 oz); more severe cases of PIVH tend to have poorer long-term neurodevelopmental outcomes (Volpe, 2008).

L. Necrotizing enterocolitis (NEC) is of particular significance in preterm infants with birthweights less than 1500 g (3 pounds, 5 ounces) with time of onset inversely related to gestational age and birthweight; signs and symptoms often begin with feeding intolerance and proceed to the classic signs and symptoms (similar to sepsis) and abdominal x-ray changes (Bradshaw, 2009).
M. Hypocalcemia occurs in 30% to 90% of preterm infants.
N. Hypoglycemia is common among premature infants (Barnes-Powell, 2007; Stanley, 2006).

1. Functionally, hypoglycemia is defined as a serum glucose concentration of approximately 40 mg or less.

2. Perinatal conditions commonly associated with hypoglycemia are common among preterm, late preterm, and SGA infants.
   a. Diabetic mother
   b. Prematurity or SGA status
   c. Perinatal stress or hypoxia
   d. Cold stress
   e. Congenital heart disease or congestive heart failure
   f. Maternal drug therapy (beta-sympathomimetics, propranolol)

3. Signs and symptoms, if present, are nonspecific and appear at various serum glucose concentrations in different infants; they are often confused with infection and respiratory distress.
   a. Abnormal cry
   b. Lethargy
   c. Apnea
   d. Hypothermia
   e. Hypotonia
   f. Jitters
   g. Tremors
   h. Tachypnea
   i. Seizures
   j. Cardiac arrest

CLINICAL PRACTICE

A. Assessment

1. Maternal historical risk factors (Balchin & Steer, 2007)
   a. Premature labor treated with bedrest and tocolytics
   b. Multiple gestation
   c. Infections (see Chapter 20 for more detailed discussion of newborn sepsis)
   d. Antepartum bleeding
   e. Pregnancy-induced hypertension (PIH)
   f. Premature rupture of membranes (PROM)
   g. Cervical insufficiency or incompetence
   h. Psychosocial stress or high-risk maternal behaviors
   i. No prenatal care
   j. Poor nutrition
   k. Use of illicit drugs
   l. Domestic abuse
   m. Motor vehicle accident

2. Physical findings (Kelly, 2006)
   a. Neurologic: hypotonic resting posture (predominance of extensor muscle tone and underdevelopment of flexor muscle tone)
   b. Head
      (1) Larger in proportion to the body compared with the term infant
      (2) Skull bones soft and spongy, especially along suture lines
      (3) Fontanelles wide and soft with overriding sutures
      (4) Ears lacking development of cartilage
      (5) Scalp hair matted and woolly in appearance
   c. Skin
      (1) Skin is thin and edematous at early gestations, but thickness and opacity increase with advancing gestational age.
      (2) Transparent in early gestations so that the underlying capillary bed shows through, giving the infant a ruddy look; veins are readily visible.
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(3) Lanugo is fine and barely visible at early gestations, is thickest and most abundant between 28 and 30 weeks, and slowly disappears, beginning in the lower back as gestation advances.

(4) Skin is susceptible to breakdown because of decreased cohesion between the dermis and epidermis.

d. Breasts and nipples
(1) Are barely visible at early gestations.
(2) Areola becomes raised at about 34 weeks and increases in size as the breast bud enlarges.
(3) A small bud is palpable at about 36 weeks’ gestation and slowly increases in size with advancing gestational age.

e. Sole creases develop first in the anterior third of the sole and slowly advance downward with advancing gestational age.

f. Genitalia
(1) The preterm male has a small scrotum with few rugae and testes that are high in the inguinal canal; presence of rugae increases, and testes descend into the scrotum with advancing gestational age.
(2) The preterm female has a prominent clitoris and labia minora; the labia majora enlarge with advancing gestational age.

g. Thermal instability (putting the infant at risk for heat loss) results from the following (McGrath, 2007b):
(1) Larger surface-to-weight ratio
(2) Immature muscle tone and decreased muscular activity
(3) Diminished stores of white fat and brown fat
(4) Poor nutrient intake
   (a) Infant has a scrawny appearance.
   (b) Lack of insulating properties of white fat allows for more rapid transfer of heat from the infant’s core to the environment.
   (c) Reduced amounts of brown fat (deposited between 30 and 36 weeks’ gestation) mean that chemical thermogenesis in the preterm infant (the usual, nonshivering method of heat production in newborns) is unreliable.

h. An immature respiratory control center is exhibited by periods of apnea, periodic breathing, or both.
(1) Apnea is an absence of respiration lasting more than 20 seconds accompanied by a fall in heart rate (usually to 80 beats per minute [bpm] or less) and with resultant cyanosis, hypotonia, and metabolic acidosis.
(2) Periodic breathing is exhibited as breathing pauses, sometimes lasting more than 20 seconds, but without bradycardia, cyanosis, hypotonia, or acidosis.

i. RDS, or hyaline membrane disease, may be present, is inversely related to gestational age, and is compounded when asphyxia is present (see Care of Infant with Respiratory Distress).

j. Hypoglycemia may be present because of a lack of glycogen stores necessary to meet the infant’s metabolic demands (see earlier discussion of hypoglycemia), exhibited by (Stanley 2006):
(1) Lethargy
(2) Tachycardia
(3) Increased respiratory effort
(4) Jitteriness

k. Presence of a patent ductus arteriosus (PDA), which may be intermittent, as evidenced by:
(1) Systolic cardiac murmur in area of upper left sternal border
(2) Desaturation on pulse oximetry with or without color change
(3) Increase in peripheral pulses
(4) Increase in respiratory rate

l. Signs and symptoms of infection may be present (see Chapter 20 for further discussion of newborn sepsis).

3. Diagnostic procedures
a. Heart and respiratory rates
b. Axillary, skin, and rectal temperatures
c. Oxygen saturation levels by pulse oximetry and arterial blood gas assays
   (1) Oxygen saturation should be 90% to 92% in most cases.
   (2) Normal PaO₂ should be in the 50s to 60s.

d. Blood
   (1) Glucose
   (2) CBC with differential
   (3) Electrolytes including calcium, blood urea nitrogen (BUN) and serum creatinine
   (4) Bilirubin concentrations
   (5) Cultures

e. Urine output (normal 1 to 3 mL/kg/hr); specific gravity (normal 1.002 to 1.010)

f. Chest radiographs

g. Head and abdominal circumference

h. Daily weights

B. Interventions/Outcomes

1. Interventions for common problems of prematurity include those for temperature instability;
   blood glucose instability; respiratory distress (see later discussion); nutritional support; PDA
   (see later discussion); hyperbilirubinemia (see later discussion); renal insufficiency;
   environmental stress; infection related to possible exposure to intrauterine infection (see
   Chapter 20 for complete discussion of newborn sepsis).

a. Monitor temperature.
   (1) Place neonate on servo-control mode under radiant warmer or in isoolate with
      thermistor (located over the right upper quadrant of the abdomen) to achieve NTE.
   (2) Monitor heater output (be aware that continuous high heater output on servo-
      control mode in the face of normal skin surface temperatures is an alert to potential
      physiologic alterations in the neonate) (McGrath, 2007b)
   (3) Measure axillary, skin, and core temperatures as necessary (axillary temperatures
      are preferable and are as accurate as a core temperature if taken correctly) (McGrath, 2007b)

b. Evaluate the environment for potential sources of heat loss or gain through conduction,
   convection, radiation, and evaporation.
   (1) Do not bathe the neonate without first evaluating the consequences of cold stress on
      the neonate’s clinical condition.
   (2) Prewarm linens and equipment that will come into contact with the neonate.
   (3) Keep neonate’s head covered with a cap.
   (4) Remain vigilant to the presence of radiant heat loss to cold walls or windows and
      convection heat loss in the path of air conditioning vents.

c. Anticipate and prevent blood glucose instability (Stanley, 2006).
   (1) Bedside blood glucose levels (e.g., Accuchek, One Touch, Chemstrip) should be
      routinely measured at specific intervals (every 1 to 4 hours) in premature infants and
      others who have risk factors for hypoglycemia through the first several days of life.
   (2) Serum levels should be checked when the bedside value is less than 40 mg/dL in
      symptomatic infants.
      (a) If greater than 32 weeks’ gestation with no respiratory distress offer formula or
         D₅₀W (to raise and sustain the blood glucose level) every 2 to 3 hours.
      (b) Gavage feed an infant who refuses to suck, has tachypnea (respiratory rate greater
         than 60), or has poor coordination of suck, swallow, and breathing.
      (c) Administer parenteral D₅₀W if blood glucose does not respond to oral feeding.
   (3) Continuous IV glucose infusion at maintenance rates (70 to 80 mL/kg/day), started
      early in symptomatic premature infants who are not yet hypoglycemic, will preclude
      the need for bolus glucose infusions that often result in flip-flopping serum glucose
      concentrations.
   (4) Bolus IV glucose (D₅₀W at 2 mL/kg over several minutes) followed by a continuous
      infusion at maintenance rates often restores the serum glucose level within several
      minutes without producing unwanted flip-flop of the serum glucose level.

d. Provide nutritional support as appropriate for body requirements and gestational
   immaturity
   (1) Monitor neonate’s nutritional parameters (Kelly, 2006)
   (2) Intake (IV and oral) and output (urine, stool, or other) on an hourly basis
   (3) Body weight on a daily basis
(4) Head circumference and length on a weekly basis; document weight, length, and head circumference on a weekly basis on standard growth chart to assess for trends.

(5) Offer oral feedings, as tolerated and as ordered.
   (a) Oral feed if suck, swallow, and breathing coordination is present, neonate is in an awakened state, and stamina is good
   (b) Via breastfeeding as tolerated
   (c) Via intermittent bolus gavage (via intermittent insertion or indwelling tube)
   (d) Via continuous gavage (via indwelling tube)

(6) Monitor fluid and electrolyte levels regularly as needed.
   (a) Weight
   (b) Bedside monitoring of serum glucose level
   (c) Serum electrolyte values
   (d) Physical examination to determine hydration status

(7) Offer opportunities for nonnutritive sucking on a premature-size pacifier and social interaction as tolerated during feedings.

(8) Monitor for tolerance of feedings, noting characteristics, amount, and frequency of alterations.
   (a) Vomiting or regurgitation
   (b) Abdominal distention
   (c) Gastric residual (aspirated before next feeding): observe for color (bloody, bile-stained, or other); partially digested or not; and/or mucusy or not
   (d) Stools: observe for water loss; bloody; and/or explosive
   (e) Apnea or bradycardia related to reflux
   (f) Signs and symptoms of hypoglycemia
      [i] Jitters and tremors
      [ii] Lethargy and hypotonia
      [iii] Abnormal cry and tachypnea
      [iv] Bedside glucose screening test result less than 40 mg/dL

(9) Provide total parenteral nutrition (TPN) and lipids, when enteral feedings are contraindicated, administered via peripheral vein IV (maximum dextrose concentration is 12.5%) or via central catheter (central line or percutaneous central line).

e. Support skin integrity because of skin immaturity.
   (1) Turn and reposition neonate every 3 to 4 hours and check skin integrity if tolerated.
   (2) Keep skin clean, dry, and free from abrasions.
   (3) Never use oil-based lotions or creams, alcohol, or benzoin on the very low-birthweight (VLBW) neonate or on dry skin of any neonate.
   (4) Wipe off povidone-iodine (Betadine) with sterile water and cotton balls after procedures when it is used.
   (5) Use hydrogel products to affix thermistor probes or leads; do not use tape on VLBW neonates.
   (6) Remove hydrogel products and tape with sterile water and soak them off; if adhesive remover must be used, wash it off with sterile water immediately after.
   (7) Use baby soap, sterile water, and cotton balls for diaper care.
   (8) Consider using commercial skin barrier products whenever repeated taping to the skin is required and over areas of skin breakdown.
   (9) Use positioning aids (e.g., bolster, nest, rolled blanket) to position neonate for comfort and to relieve pressure over bony prominences.
   (10) Avoid friction or tearing of skin surfaces.

f. Evaluate environment for sources of excessive insensible water loss (IWL), and implement measures as needed to decrease IWL.
   (1) Use heat shield or plastic wrap blanket under radiant warmer.
   (2) Move to double-walled isolette as soon as possible after stabilization under radiant warmer.
   (3) Provide warmed, humidified oxygen as needed.
   (4) Ensure increased fluid intake under phototherapy as ordered.

g. Monitor urine output (check for mL/kg/hr) by weighing each diaper.
   (1) Dipstick for protein and blood and check for specific gravity (concentration),
Monitor for signs and symptoms of overhydration or fluid overload.
(a) Peripheral edema; taut and shiny skin
(b) Bounding pulses
(c) Increased blood pressure

Monitor for signs and symptoms of dehydration.
(a) Poor skin turgor
(b) Dry mucous membranes
(c) Sunken fontanelles

Estimate IWL when clinical conditions or therapies are present that increase IWL.
(a) Radiant warmer
(b) Phototherapy
(c) Respiratory distress
(d) Skin breakdown
(e) Ambient temperature above thermoneutrality
(f) Fever
(g) Increased activity

Monitor enteral and parenteral nutrition/fluid intake as ordered.

Provide for minimal stimulation with appropriate sedation, if needed, to conserve energy stores.

Activity intolerance secondary to prematurity compromises effective ventilation, oxygenation, and caloric use.

Caretaking activities should be clustered to provide adequate periods of rest, but care should be taken to provide breaks between procedures when the infant demonstrates signs of stress.

Minimal handling should be observed by all personnel; unnecessary touching should be avoided to provide maximum opportunity for the parents for bonding (see Health Education).

Infant should be handled gently, with slow, purposeful movements rather than abrupt, jerky movements.

Sedation may be ordered for infants whose spontaneous activity level puts the infant at risk for respiratory distress.

Prone positioning has been shown to optimize respiratory status and decrease stress in the preterm infant.

Outcomes

a. Neonate’s axillary temperature is maintained within normal limits (36.4° to 37.1°C [97.6° to 98.8°F]).

b. Optimal equipment to maintain thermal neutrality for infant is provided.

c. No signs of cold stress are observed.

d. The infant demonstrates intake of sufficient calories, as indicated by:
   (1) Weight gain of 15 to 30 g/day (0.5 to 1 ounce/day)
   (2) Head circumference growth averages 0.5 to 1 cm (0.2 to 0.4 inch) per week.
   (3) Spontaneous activity level that does not compromise weight gain

e. The infant does not exhibit signs and symptoms of feeding intolerance or hypoglycemia.

f. The infant is maintained on enteral feedings, as tolerated; if unable to tolerate enteral feedings, appropriate IV nutrition will be established and maintained.

g. The infant demonstrates signs and symptoms of adequate hydration (absence of underhydration or overhydration).
   (1) Urine output between 1 and 2 mL/kg/hr
   (2) Electrolyte values within normal limits (WNL)
   (3) Weight loss in the first few days of life, followed by a weight gain of 15 to 30 g/day (0.5 to 1 ounce/day)
   (4) Absence of excessive generalized edema
   (5) Urine specific gravity ranges from 1.002 to 1.010.
   (6) Protein and blood are absent in the urine.

h. The neonate is able to self-console by nonnutritive sucking on a pacifier and to show increased tolerance for social interaction.

i. The neonate maintains good skin integrity as evidenced by lack of abrasions, skin breakdown, or local irritation or infection.
LATE PRETERM INFANT

INTRODUCTION

A. The late preterm infant is born between 34 and 36 6/7 weeks’ gestation (Jorgensen, 2008a; 2008b).
B. Late preterm infants make up 70% of the preterm infant population and the number may be increasing due to obstetric practices, such as inductions, elective cesarean sections, and increased number of multiple gestation infants (Ramachandrappa & Jain, 2008; Yoder, Gordon, & Barth, 2008).
C. Late preterm infants are often the size of some full-term infants and are often treated as such.
D. Late preterm infants are not physiologically or behaviorally mature and experience issues similar to preterm infants (Engle et al, 2007).
   1. Temperature instability
   2. Difficulty with bottle and/or breastfeeding.
   3. Hyperbilirubinemia (see later discussion).
   5. Respiratory distress (see later discussion).
   6. Sepsis

CLINICAL PRACTICE

See discussion of preterm infants for management strategies, interventions and outcomes with these infants. Often not ill enough to justify care in the NICU, the late preterm neonate is often cared for in the normal newborn nursery; thorough assessment of risk and health status, interventions, and parent education for these babies should be individualized (Campbell, 2006; Pappas & Walker, 2010). Certain criteria should be in place before these infants are discharged home.

A. Discharge criteria (Pappas & Walker, 2010)
   1. Normoglycemic
   2. Temperature stability
   3. Stable or decreasing bilirubin
   4. Feeding to sustain growth
   5. Feeding plan in place and parent(s) educated on plan
   6. Scheduled home care and follow-up appointments (i.e., lactation support, weight checks, home treatment for hyperbilirubinemia).

HEALTH EDUCATION

Preterm Infants

A. Provide individualized instruction regarding the infant’s respiratory diagnosis, handling and treatment considerations, and monitoring and treatment that will be continued in the home.
B. Teach parents to prepare high-calorie formulas or supplementation to breastfeeding to ensure adequate weight gain and growth, and instruct them if the infant is to be fed around the clock on a fixed schedule.
C. Teach parents to prepare and administer discharge medications.
D. Inform parents about the extent, outcome, and prognosis after PIVH or ischemic changes because they may need referral for persistent neurodevelopmental delays.

Late Preterm Infants

A. Instruct parents when and how often to see primary health care provider.
B. Instruct parents in temperature assessment and maintenance at home; avoid over and under dressing
C. Feeding plan (minimum number or volume of feeding/day)
D. Assessment for jaundice and dehydration
E. Education on respiratory syncytial virus (RSV) prophylaxis and prevention
F. Long-term outcomes/risks:
1. Increased risk for Sudden Infant Death Syndrome (SIDS) (as much as 50%) (Darnall, Ariagno, & Kinney, 2006)
2. Increased risk for at least one hospital readmission within the first 6 to 12 months of life, especially if never admitted to the NICU (Cuevas, Silver, Brooten, Youngblut, & Bobo, 2005; Jain & Cheng, 2006)

RESPIRATORY DISTRESS

INTRODUCTION

A. Causes of Respiratory Distress. The cause of respiratory distress in the newborn may have its beginnings in the failure of one or more major body systems. Conditions that result in the structural and functional failure of a major body system result in mild to profound respiratory distress in the newborn, regardless of size and gestational age. The following is a discussion of the five most common body systems involved in the causes of respiratory distress in general and specifically RDS (Sinha, Gupta, & Donn, 2008).

1. Cardiac diseases
   a. Congenital heart disease (CHD)
   b. Congestive heart failure (CHF)
   c. Patent ductus arteriosus (PDA)

2. Hematologic disorders
   a. Anemia
   b. Hemorrhage
   c. Polycythemia

3. Metabolic disorders
   a. Acidosis
   b. Hypoglycemia
   c. Hyperglycemia
   d. Hypocalcemia
   e. Hypothermia
   f. Hyperthermia
   g. Hypermagnesemia
   h. Congenital hyperthyroidism

4. Central nervous system disorders
   a. Hemorrhage
      (1) Intracranial, subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH)
      (2) Intraventricular and periventricular hemorrhage (IVH and PVH)
   b. Infection
      c. Neonatal depression related to maternal drugs given during labor
         (1) Magnesium sulfate
         (2) Analgesics
   d. Neonatal substance-withdrawal syndrome
   e. Asphyxia

5. Respiratory disorders
   a. Most common conditions
      (1) RDS
      (2) TTN
      (3) Aspiration syndromes
         (a) MAS
         (b) Blood aspiration
         (c) Amniotic fluid aspiration
         (d) PPHN
   b. Pneumonia
   c. Spontaneous pneumothorax occurs in 1% to 2% of live births; only symptomatic in 1 in 1500 live births (Sinha et al, 2008). Symptoms may include tachypnea, minimal retractions,
grunting, nasal flaring, and cyanosis; diminished air entry on affected side, shifting of cardiac impulse, and muffled heart tones may also be noted.

**CLINICAL PRACTICE**

Although knowledge and understanding of these conditions are helpful in diagnosing and treating respiratory distress, this section focuses specifically on several respiratory conditions, including RDS, TTN, MAS, and PPHN.

**Respiratory Distress Syndrome**

**A. Introduction:** RDS is the major cause of respiratory distress in the newborn, and prematurity is the single most important risk factor (Sinha et al, 2008).

1. **RDS,** sometimes referred to as hyaline membrane disease (HMD), accounts for 20% to 30% of all neonatal deaths and approximately 50% to 70% of all premature deaths; RDS occurs in 60% of babies born at less than 28 weeks’ gestation, 50% of those born at 28 to 34 weeks, and in less than 5% of those born after approximately 34 weeks.
   a. Its incidence is inversely proportional to gestational age and occurs most frequently in infants of less than 1200 g (2 pounds, 10.5 ounces) birthweight and 30 weeks’ gestation.
   b. Surfactant deficiency is the principal factor leading to the development of RDS.
2. Prenatal maternal complications increase the risk for preterm birth and increase the risk of respiratory distress in the preterm infant by negatively affecting the fetal pulmonary circulation.
3. The ability to stabilize the chest wall is directly related to increasing gestational age (GA); therefore, the preterm infant is at risk for chest wall deformation and atelectasis, which can result in hypoxemia, hypercarbia, and apnea.
4. Immaturity of the respiratory system and its control centers places the preterm infant at risk for apnea of prematurity, a paradoxical response to low oxygen, high carbon dioxide, or both in which the preterm infant fails to breathe more quickly but instead stops breathing.
5. Other causes of respiratory problems in the preterm infant (Sinha et al, 2008)
   a. TTN, in which delayed clearance of fetal lung fluid occurs manifested by rapid breathing, retractions, grunting, and cyanosis
   b. PPHN, in which hypoxemia and acidemia caused by failure to completely change from the fetal to the neonatal circulatory pattern occurs
   c. Bronchopulmonary dysplasia (BPD), in which oxygen therapy and intermittent mandatory ventilation combine to bring about chronic lung disease
6. **Pathophysiology:** surfactant deficiency
   a. Surfactant is a complex mixture of phospholipids and proteins that binds to the alveolar surface of the lungs and forms a coating over the inner surface of the alveoli and decreases the surface tension, preventing collapse at the end of expiration (Halliday, 2008; Turell, 2008).
   b. Alveolar development occurs between 24 and 28 weeks’ gestation, which results in the following (Hermansen & Lorah, 2007):
      (1) Increases in pulmonary vascularization
      (2) Development of ability for gas exchange
      (3) Development and proliferation of type II respiratory cells responsible for surfactant production and synthesis
   c. Surfactant deficiency results in the following (Halliday, 2008; Turell, 2008)
      (1) Increased surface tension, leading to alveolar collapse
      (2) Diffuse atelectasis
      (3) Loss of functional residual capacity
      (4) Decreased lung compliance
      (5) Right-to-left intrapulmonary shunting through the ductus arteriosus with increased pulmonary vascular resistance (PVR)
7. **Complications of RDS** include the following:
   a. PDA incidence increases with decreasing gestational age (see later in this chapter for a complete discussion of PDA).
   b. Air leak syndromes (e.g., pneumothorax, pneumomediastinum, pneumopericardium) occur in 5% to 30% of infants with RDS (Sinha et al, 2008)
c. IVH occurs in approximately 15% to 20% of infants weighing less than 1500 g (3 lbs, 5 oz) (Volpe, 2008).

d. BPD occurs in approximately 23% of infants with RDS (Lefkowitz & Rosenberg, 2008).

e. Retinopathy of prematurity (ROP) occurring in infants with RDS is increasing and is currently estimated to be between 20% and 25%; most of these cases regress, with the incidence of severe disease estimated to be between 5% and 10% (Pollan, 2009); prevention of premature birth is the best preventive of ROP; after a preterm birth, oxygen should be used only in amounts sufficient to avoid hypoxia.

B. Assessment

   a. At less than 28 weeks’ gestation, 60% of neonates demonstrate clinical signs of RDS.
   b. At 28 to 32 weeks’ gestation, 50% of neonates demonstrate clinical signs of RDS.
   c. At 37 weeks’ gestation and older, 3% to 5% of neonates demonstrate clinical signs of RDS.
   d. At birth 10% to 15% of infants who weigh less than 2500 g (5 pounds, 8 ounces) demonstrate RDS; the highest incidence occurs among the group with the lowest birthweight (Hermansen & Lorah, 2007; Sinha et al, 2008)
   e. RDS is increased:
      (1) In males versus females (1.5 times’ higher incidence)
      (2) Among whites versus nonwhites
      (3) In IDMs; insulin is antagonistic to surfactant production
      (4) In the presence of asphyxia, regardless of gestational age
      (5) When birth is by cesarean section, especially in the absence of labor, related to the lack of a thoracic squeeze
      (6) In the second-born of twins, which may be related to the second-born’s longer stay in the birth canal, with the second twin receiving an excess of amniotic fluid with the birth of the first twin
   f. RDS is decreased with:
      (1) Prolonged or premature rupture of membranes (PROM)
      (2) IUGR
      (3) PIH
      (4) Maternal heroin addiction
      (5) Prenatal corticosteroids

2. Physical findings: Symptoms of RDS frequently occur within 4 to 24 hours after delivery; symptoms are usually apparent in the delivery room; typically, the clinical course of RDS worsens during the first 48 hours after birth, and respiratory function generally begins to improve within 72 hours after birth.
   a. Intercostal, subclavicular, and substernal retractions occur because of the compliant chest wall of the preterm infant, in addition to relatively noncompliant lungs (“seesaw” respirations).
   b. Expiratory grunting, heard as a result of partial vocal cord closure, increases transpulmonary pressures in an attempt to improve lung volume capacity.
   c. Nasal flaring is often present as the infant attempts to decrease nasal airway resistance.
   d. Tachypnea with a respiratory rate of greater than 60 breaths per minute is common.
   e. Decreased breath sounds or unequal breath sounds are usually heard.
   f. Poor air entry is heard on auscultation.
   g. Fine rales may be heard bilaterally or unilaterally.
   h. Generalized cyanosis may be seen because of impaired ventilation and intrapulmonary and intracardiac shunting.
      (1) Peripheral cyanosis alone is common in newborns and is usually not significant.
      (2) The degree of cyanosis depends on the following:
         (a) Hemoglobin concentration
         (b) Status of the peripheral circulation
         (c) Color of the infant and the available light to visualize the cyanosis
   i. Tachycardia with a heart rate of 150 to 180 bpm may occur.
   j. Hypothermia may occur despite an NTE.
   k. Hypoglycemia with a glucose level of less than 20 mg/dL in the preterm infant may be noted.
   l. Hypotension is a frequent indication of severe RDS.
   m. Hypotonia results in a limp and flaccid infant.
   n. Apnea occurs frequently in the severely compromised infant.
3. Diagnostic procedures
   a. Apgar scores may not reflect the severity of RDS.
   b. Maturity assessment
      (1) Lecithin/sphingomyelin (L/S) ratio of greater than 2:1 indicates mature lungs in the absence of a diabetic pregnancy (Hermansen & Lorah, 2007).
      (2) The presence of phosphatidylglycerol (PG) confirms maturity and is especially important to ascertain lung maturity in the presence of maternal conditions such as diabetes.
         (a) Is present at 37 weeks’ gestation and levels rise to term
         (b) If greater than 1%, indicates mature lungs
      (3) Shake test or foam stability index (FSI) may be performed on amniotic fluid at the maternal bedside to quickly determine the presence of surfactant.
   c. Chest x-ray initially may appear better than the clinical course would suggest; the classic chest x-ray film findings include the following:
      (1) Reticulogranular (ground-glass) pattern
      (2) Air bronchograms that demonstrate diffuse alveolar collapse surrounding open bronchi
      (3) Decreased lung volumes
      (4) Possible cardiomegaly
   d. Arterial blood gas (ABG) assays show hypoxemia and hypercapnia.

C. Interventions
1. Provide appropriate supportive measures for the neonate, to offer optimal respiratory support.
   a. Resuscitation in the delivery room should follow NRP guidelines. Oxygen should be provided only as needed and blended based on assessment of infant (Rabi, Rabi, & Yee, 2007).
   b. Ensure ready availability of bag and mask setup and bulb and wall suction in the event that the infant requires intermittent mandatory ventilation or suction to clear the airway.
   c. Assess the neonate’s respiratory effort with regard to rate, character, effort, and signs of respiratory distress.
   d. Maintain position of the neonate so that the upper airway is not obstructed.
   e. Intubate with the appropriate size of endotracheal tube (ETT); choice of tube size is determined by the neonate’s weight, as follows (Bachman, Marks, & Rimensberger, 2008):
      (1) Less than 1001 g: 2.5 mm
      (2) 1001 to 2000 g: 3 mm
      (3) 2001 to 3000 g: 3.5 mm
      (4) More than 3001 g: 4 mm
   f. Transfer infant to the NICU if mechanical ventilation is required.
   g. Administer continuous positive airway pressure (CPAP) to infants, as needed, who do not require mechanical ventilation. May be administered via ETT or nasal prongs (Bancalari & Claure, 2008). CPAP may resolve some of the atelectasis, decrease intrapulmonary shunting, and improve ventilation to alveoli already open.
   h. Place infant in appropriate concentration of oxygen via oxygen hood or nasal cannula as determined by an ABG assay if mechanical ventilation or CPAP is not needed.
      (1) Oxygen should always be warmed and humidified to prevent infant heat loss and drying of mucous membranes.
      (2) Oxygen concentrations should be monitored continuously and documented per facility protocol (Shiao & Ou, 2007).
   a. Cardiorespiratory monitor with 15-second apnea delay
   b. Oxygen saturation
3. Assist with obtaining and review diagnostic tests to determine likely causes of respiratory distress (including any or all of the following: hypoglycemia, pneumonia, and RDS).
4. Provide airway patency by the removal of secretions using accepted guidelines per facility protocol (Marechal, Barthod, Lottin, Gautier, & Jeulin, 2007).
   a. Use a catheter of appropriate size (a 5- or 6-mm catheter is recommended, if possible) to pass infant’s airway.
   b. Determine depth of suctioning by size of infant or length of ETT tube; carefully pass the suctioning catheter, which will prevent damage to the mucosa; never deep suction, but
rather, suction to 0.5 cm below the length of the ETT (Marechal et al, 2007; Youngmee & Yonghoon, 2003). Never deep suction the infant, which may cause damage to the tracheal tissues.

c. Set the vacuum gauge at 50 to 80 cm of water pressure.

d. Suction only as needed; assess need to suction by the following:
   (1) Breath sounds
   (2) Type and amount of secretions
   (3) Tolerance and clinical status

e. Have two people perform the procedure as necessary.

f. Preoxygenate no more than 10% above baseline oxygen requirements and hyperinflate before suctioning.
   (1) Begin 1 minute before suctioning.
   (2) Continue during and after procedure until the infant reaches presuctioning heart rate and oxygen saturation baseline.

g. Avoid repeated passes of the catheter if possible.

5. To minimize the risk of suctioning, continuously monitor the infant’s tolerance of the suctioning procedure by observing for the signs and symptoms of:
   a. Hypoxia resulting in decreased oxygen saturation
   b. Bradycardia, dysrhythmias, or both
   c. Mucosal ulceration and hemorrhage secondary to the trauma of repeated suctioning

6. Provide continuous monitoring of infant’s condition.
   a. All infants should be connected to continuous cardiorespiratory monitors to detect abnormalities in heart rate and rhythm, as well as apnea and bradycardia.
   b. Blood pressure should be monitored in all infants with RDS; normal blood pressure varies with size and gestational age.
   c. An ABG assay should be performed as needed.
   d. Ongoing infant respiratory status should be assessed every 1 to 4 hours as needed.
      (1) Auscultation of breath sounds
      (2) Quality of air entry
      (3) Respiratory effort and spontaneous respiratory rate
   e. Serial chest x-ray films should be obtained as appropriate to assess disease progression.

7. Administer exogenous surfactant per physician or facility protocol as needed to support lung function until the infant’s own supply is produced (Halliday, 2008; Turell, 2008).
   a. Administration in the delivery room (early) has been shown to be more beneficial than late administration (Halliday, 2008; Turell, 2008)
   b. Administration protocol should be drug specific; not all surfactants are the same; dose, timing, and delivery systems may vary, as well as cost.

8. Prevent or treat hypotension.
   a. Systemic hypotension results in pulmonary and systemic vasoconstriction and will prevent adequate tissue perfusion and gas exchange.
   b. Administer fluids to prevent or treat hypovolemia. If more pressure support is needed the neonatal team should be notified and the infant should be prepared for transfer.

9. Prevent or treat hypothermia (McGrath, 2007b).
   a. Hypothermia causes cold stress in the neonate, which results in vasoconstriction and subsequent lowering of PaO2
   b. Maintain an NTE: defined as the condition under which the amount of heat produced is equal to the least amount of heat lost to the environment, with the least metabolic stress.

10. Prevent or treat metabolic acidosis.
    a. Metabolic acidosis causes constriction of pulmonary vessels and decreased lung perfusion.
    b. Administer IV fluids, as indicated.
    c. Prevent excessive water loss.
    d. Request support from neonatal team if infant does not respond to above interventions.

11. Prevent or treat hypoglycemia (see Care of Preterm Infant).

12. Prevent or treat anemia (see Care of Preterm Infant).

13. Provide for minimal stimulation with appropriate sedation, if needed, to conserve energy stores (see Care of Preterm Infant).
D. Outcomes

1. The neonate is appropriately and adequately resuscitated in the delivery room with minimal asphyxia, hypothermia, shock, and acidosis.
2. The infant receives the appropriate ventilatory support.
   a. The appropriate size of ETT is used, based on weight.
   b. Mechanical ventilation, CPAP, or oxygen is administered as needed.
3. The infant’s vital signs are WNL.
   a. Heart rate between 120 and 180 bpm
   b. Respiratory rate between 40 and 60 breaths per minute
   c. Blood pressure WNL for size and gestational age
4. The neonate demonstrates minimal respiratory distress.
   a. Bilateral breath sounds
   b. Good air entry
   c. Consistent respiratory effort with spontaneous respirations
   d. Minimal retractions
   e. Absence of grunting and nasal flaring
   f. Absence of central or generalized cyanosis
5. The neonate demonstrates adequate pulmonary and tissue perfusion.
   a. Well oxygenated, with normal PaO₂
   b. Absence of metabolic acidosis
   c. Normal systemic blood pressure
   d. Absence of hypothermia
   e. Absence of hypoglycemia
   f. Normal hematocrit and hemoglobin levels
6. The infant receives minimal tactile stimulation.
   a. Caretaking activities will be grouped together.
   b. The infant will not be medically touched except to provide necessary care, and parental touch will be encouraged.
   c. Parents demonstrate knowledge of their infant’s activity tolerance by minimizing stress during bonding activities.
   d. The infant is handled gently and carefully.
   e. Sedation is given to infants who demonstrate excess activity resulting in failure to adequately ventilate or oxygenate and whose caloric expenditure is in excess of caloric intake.

Transient Tachypnea of the Newborn (TTN)

A. Introduction: TTN is sometimes referred to as wet lung syndrome, retained lung fluid (RLF), and RDS type II (Guglani, Lakshminrusimha, & Ryan, 2008).

1. Pathophysiology: excess fluid in the lungs, failure to clear normal fetal lung fluid, or both (Takaya et al, 2008).
   a. Fluid accumulates in the peribronchial lymphatics and bronchovascular spaces.
   b. Excess fluid may be related to the following:
      (1) Aspiration
         (a) Amniotic fluid
         (b) Secretions; tracheal fluid
      (2) Factors that promote the formation of interstitial lung fluid
         (a) Decreased plasma colloid osmotic pressure (hypoalbuminemia)
         (b) Increased interstitial colloid osmotic pressure (transudation of plasma proteins)
         (c) Increased capillary hydrostatic pressure
   c. Fetal lung fluid is normally cleared via the following:
      (1) Expulsion during delivery
      (2) Absorption after delivery related to pulmonary circulation and lymphatic drainage
   d. Failure to clear fetal lung fluid is usually caused by the lack of a “thoracic squeeze” to expel the fluid during delivery.
2. There is no known residual pulmonary dysfunction; however, spontaneous pneumothorax may occur.
B. Assessment

   a. TTN infants tend to be term or near term (36 weeks’ and longer gestation) with mature lungs, as indicated by L/S ratio.
   b. TTN is increased (Guglani et al, 2008):
      (1) In large infants with a birthweight of more than 4000 g (8 pounds, 14 ounces)
      (2) In infants born by cesarean section
      (3) In breech births
      (4) In the second-born of twins
      (5) When labor and delivery are rapid and preclude the opportunity for an effective thoracic squeeze (especially in the small infant)
      (6) With a maternal history of heavy sedation
      (7) In infants with polycythemia, delayed cord clamping, or both; hyperviscosity of blood leads to sluggish circulation in the pulmonary vessels.
      (8) In infants suffering from hypothermia at or shortly after birth
         (a) Hypothermia causes pulmonary vasoconstriction.
         (b) Vasoconstriction causes the infant to experience hypoxemia and increased oxygen consumption, which produces respiratory distress.

2. Physical findings: symptoms of TTN are usually present within the first hours of life (most often within 30 minutes); typically, the clinical course of TTN occurs during the first 12 to 72 hours of life and the disease is self-limiting.
   a. Transient tachypnea with a respiratory rate of 60 to 140 breaths per minute (rarely lasts longer than 48 to 96 hours)
   b. Grunting, nasal flaring and/or mild intercostal retractions
   c. Possible mild cyanosis
   d. Breath sounds that may be slightly decreased because of reduced air entry
   e. Absence of rales
   f. Chest that may appear hyperexpanded or barrel shaped

3. Diagnostic procedures: TTN is a diagnosis of exclusion.
   a. A diagnosis of TTN can be ascertained only after resolution of symptoms within the first 4 days.
   b. Chest x-ray film reveals the following:
      (1) Increased lung fluid, with fluid in the interlobar tissues
      (2) Prominent vascular marking (so-called hairy heart)
      (3) Flat diaphragm, with increased lung volume
      (4) Mild pleural effusions may be demonstrated.
      (5) Occasional presence of mild cardiomegaly
      (6) Occasionally, the initial chest x-ray film (within the first 3 hours of life) appears similar to RDS.
   c. ABG assay
      (1) Mild hypoxemia
      (2) PaCO₂ normal to mildly elevated (less than 50 mm Hg)
      (3) pH: usually normal

C. Interventions/Outcomes

1. Interventions
   a. Assisted ventilation is seldom required (Guglani et al, 2008; Takaya et al, 2008).
   b. Provide appropriate oxygen therapy to maintain ABG values WNL.
      (1) TTN infants rarely require more than 70% fraction of inspired oxygen (FIO₂) (usually 35% to 40%).
      (2) Providing CPAP for the first few hours with severe pulmonary involvement may be useful.

2. Outcomes
   a. The infant receives the appropriate concentration of oxygen.
   b. The infant’s ABG values are WNL.
   c. The infant demonstrates minimal respiratory distress.
      (1) Respiratory rate between 40 and 60 breaths per minute
      (2) Minimal retractions
      (3) Bilateral breath sounds with good air entry
Absence of grunting and nasal flaring
Absence of generalized cyanosis

Meconium Aspiration Syndrome

A. Introduction
1. Meconium aspiration is the most common of the aspiration syndromes (Walsh & Fanaroff, 2007; Wiedemann, Suagstad, Barnes-Powell, & Duran, 2008).
2. Pathophysiology: Two overlapping phenomena occur with MAS.
   a. Pneumonitis and pneumonia, with or without air leak
      (1) Bile salts and pancreatic enzymes and other particles in the meconium cause a chemical pneumonitis.
      (2) Meconium occludes the distal airways and acts as a ball-valve mechanism that allows air in but obstructs airflow out during expiration; this leads to air trapping and air leak (pneumothorax).
   b. Three general mechanisms in the fetus result in passage of meconium.
      (1) Direct hypoxic bowel stimulation
         (a) Passage of meconium into the amniotic fluid may be the result of some intrauterine insult that causes fetal distress.
         (b) Hypoxia and acidosis may result in relaxation of the anal sphincter and passage of meconium.
      (2) Spontaneous gastrointestinal (GI) motility: Spontaneous normal physiologic defecation may occur in the term or postterm infant.
      (3) Vagal stimulation with or without specific cause (often the result of cord compression)
   c. After the passage of meconium into the amniotic fluid, the fetus may swallow or aspirate the meconium into the mouth, pharynx, and trachea.
      (1) With hypoxia, the fetus demonstrates normal or irregular respiratory movements.
      (2) Intrauterine aspiration can occur in infants with overwhelming distress.
      (3) The greatest risk for aspiration is at delivery; if meconium-stained fluid is present in the mouth and pharynx when the infant draws the first breath (Wiedemann et al, 2008)
   d. In addition to the mechanical damage done by the meconium itself, asphyxia is by far the most damaging aspect of MAS (MAS infants without asphyxia do better clinically) (Walsh & Fanaroff, 2007).
      (1) Meconium aspiration may be the primary presenting event, but prolonged fetal asphyxia may have occurred before the meconium aspiration.
      (2) Asphyxia may lead to the following conditions:
         (a) Edema of the airways
         (b) Necrosis of the airways
         (c) Vascular collapse of the alveoli
         (d) Pulmonary hemorrhage
3. Incidence of MAS
   a. Approximately 12% of all births include the presence of meconium-stained amniotic fluid (Wiedemann et al, 2008).
   b. Meconium may be aspirated from the trachea in approximately 35% of these infants or approximately 4% of all live births.
   c. Infants who are depressed at birth or make poor attempts to take the first breath should be intubated in the delivery room and suctioning of the trachea should occur to remove meconium below the vocal cords; infants who do attempt to breathe and clear their own airway should be allowed to do so without intervention. Approximately one third of infants with meconium below the vocal cords become ill and require intensive care (Wiedemann et al, 2008).
4. Prevention
   a. Antenatal diagnosis and treatment of fetal asphyxia is critical to the prevention of MAS (Walsh & Fanaroff, 2007).
   b. Intrapartum amnioinfusion, particularly when oligohydramnios is an issue, should be considered for prevention (Walsh & Fanaroff, 2007).
      (1) Decreases rate of cesarean section
      (2) Decreases morbidities related to MAS
(3) Decreases cord compression
(4) Dilutes meconium and may decrease its toxicity

5. Complications of MAS include:
   a. Air leak syndrome such as pneumothorax, pneumomediastinum, or both occurs in 20% to 30% of MAS infants (Wiedemann et al., 2008).
   b. Pneumonia
   c. Infection
   d. PPHN
   e. Pulmonary institional emphysema (PIE)
   f. Less commonly, severe asphyxia, thrombocytopenia, pulmonary hemorrhage

B. Assessment
   1. History
      a. MAS infants tend to be primarily term, postterm, or SGA; the condition rarely occurs before 38 weeks' gestation (Wiedemann et al., 2008).
      b. Perinatal factors associated with or predisposing to MAS include (Bhat & Rao, 2008):
         (1) Prolonged labor
         (2) Fetal bradycardia and distress
         (3) Breech presentation
         (4) Presence of meconium-stained amniotic fluid
         (5) Delivery by cesarean section
         (6) Low Apgar scores (less than 6)
         (7) IUGR
         (8) Decreased fetal movements
   2. Physical findings (El Shahed, Dargaville, Ohisson, & Soll, 2007)
      a. Severity of MAS correlates with:
         (1) Consistency of meconium
            (a) Early in the labor, heavy “pea soup” consistency
            (b) Late in the labor, passage of large particles
         (2) Amount of meconium
            (a) More than 2 mL in pharynx
            (b) More than 2 mL in trachea
      b. Meconium staining of skin, umbilical cord, and nails
      c. Hyperexpansion of the chest (barrel shaped)
      d. Signs and symptoms of respiratory distress occur in up to 50% of MAS infants.
      e. Clinical signs of CHF
   3. Diagnostic procedures
      a. ABG assays show hypoxemia with respiratory or metabolic acidosis or both.
      b. Chest x-ray film
         (1) Lungs are hyperinflated (9 to 11 ribs expanded).
         (2) Nonuniform, coarse, and patchy infiltrates radiate from one hilum into the peripheral lung fields.
         (3) Infiltrates associated with focal areas of irregular aeration may be present; some appear atelectatic or consolidated, and others appear emphysematous (air trapping).
         (4) Pleural effusions may be seen in MAS infants.

C. Interventions/Outcomes
   1. Interventions
      a. Impaired ventilation and oxygenation in response to inadequate respiratory effort secondary to MAS
         (1) Perform amnioinfusion before delivery.
         (2) Provide appropriate delivery room care to prevent aspiration of any meconium found in the mouth and pharynx.
         (3) Once delivered, if the infant is depressed (i.e., anticipated 1-minute Apgar of less than 7), the vocal cords should be visualized with a laryngoscope, and the trachea should be suctioned; this selective approach has decreased the need for intubation by 40% without an increase in incidence of MAS (Bhat & Rao, 2008).
      b. Provide appropriate supportive measures for the neonate to offer optimal respiratory support.
         (1) Provide ventilatory support and transport to a tertiary nursery, if needed.
         (2) Provide for high oxygenation (PaO₂ of 75 to 90 mm Hg) to prevent vasoconstriction.
(3) Provide pharmacologic assistance, as needed, to achieve desired ventilation and oxygenation of the infant (Basu, Kumar, & Bhatia, 2007).
   (a) Surfactant inactivation can be overcome by instillation of exogenous surfactants; may require three or more doses (El Shahed et al, 2007).
   (b) Sedation to prevent hyperactivity that can compromise oxygenation
   (c) Vasodilators to increase pulmonary blood flow to allow for improved oxygenation

c. Provide continuous monitoring of infant’s condition.
   (1) Cardiorespiratory monitoring
   (2) Transcutaneous and oximetry monitoring
   (3) ABG assay
   (4) Ongoing respiratory status assessments
   (5) Serial chest films
d. Prevent and treat conditions that may occur secondary to inadequate ventilation and oxygenation.
   (1) Systemic hypotension may be treated with the following:
      (a) Additional fluids
      (b) Vasopressors
   (2) Persistent metabolic acidosis may be treated with the following:
      (a) Additional fluids
      (b) Buffer agents as needed, to maintain a pH between 7.45 and 7.50
   (3) Anemia: transfuse with blood products, as indicated.
   (4) Infection (Basu et al, 2007)
      (a) Meconium is an excellent growth medium for bacteria.
      (b) Administration of prophylactic antibiotics may be indicated.

2. Outcomes
   a. The infant receives adequate delivery room care to prevent MAS.
      (1) Suctioned on the perineum before delivery of the thorax
      (2) Intubation and tracheal suctioning after birth only if the infant is depressed
   b. The infant is appropriately ventilated.
      (1) Mechanical ventilation, as needed
      (2) CPAP
   c. The infant is adequately oxygenated, as evidenced by:
      (1) PaO$_2$ of 75 to 90 mm Hg
      (2) The infant does not progress to or demonstrate signs of the severe consequences of MAS (persistent pulmonary hypertension, pneumonia, or air leaks).
   d. The infant receives pharmacologic support, as needed, to achieve adequate ventilation and oxygenation.
   e. The infant is continuously monitored.
   f. The infant does not demonstrate conditions secondary to inadequate oxygenation.
      (1) Hypotension
      (2) Metabolic acidosis
      (3) Anemia
      (4) Infection

Persistent Pulmonary Hypertension of the Newborn (PPHN)

A. Introduction: PPHN is described as hypoxemia with “persistent physiologic characteristics of fetal circulation in the absence of recognizable cardiac, pulmonary, hematologic, or central nervous system disease” (Latini, DelVecchio, DeFelice, Verrotti, & Bossone, 2008, p. 1507).
1. Pathophysiology: failure to make the transition from high PVR and low pulmonary blood flow normally found in utero, to low PVR and high pulmonary blood flow normally found after transition to extraterine life
   a. Usually the foramen ovale and ductus arteriosus remain open, with high blood flow shunting through these ducts that is sometimes bidirectional.
b. Results in profound hypoxemia; mechanisms that maintain the fetal state are relatively unknown but may be related to alterations in nitric oxide, arachidonic acid metabolism, and systemic acidosis (Hernandez-Diaz, Van Mater, Werier, Lounik, & Mitchell, 2007).

c. Theories suggest that indomethacin and aspirin, which are cyclooxygenase blockers, may contribute because exposure of the fetus to these drugs may prevent the decrease in PVR that occurs after initiation of ventilation.

d. Research suggests that increased levels of immunoreactive endothelin-1, as well as insufficient production of endogenous endothelium-derived relaxin factor, may contribute to continued vasoconstriction (Silvani & Camporesi, 2007).

B. Assessment

1. History (risk factors) (Hernandez-Diaz et al, 2007)
   a. MAS
   b. Birth asphyxia, postdates
   c. RDS
   d. Pneumonia
   e. Metabolic acidosis
   f. Infection (group B Streptococcus)
   g. Acute hypoxia with delayed resuscitation, hypoglycemia, or hypothermia

2. Physical findings (Latini et al, 2008)
   a. Cyanosis over entire body or differential cyanosis may be seen.
   b. Respiratory distress with tachypnea
   c. Normal or decreased blood pressure (BP) or asymmetric BP with right arm being greater than left lower extremity
   d. Tricuspid insufficiency murmur may be present (harsh).

3. Diagnostic procedures
   a. Hypoxia test: preductal and postductal PaO2 with preductal and postductal oximetry
   b. Glucose status
   c. ABG assay: pH status; respiratory or metabolic or mixed acidosis usually present
   d. Chest x-ray film reveals variable heart size depending on cause of PPHN and whether heart defects are present; lung findings are also dependent on primary lung disease.
   e. Echocardiography may reveal:
      (1) Echo-increase in pulmonary artery (PA) pressure, shunting right to left or bidirectional at foramen ovale; may be shunting right to left at ductus or bidirectional at ductus.
      (2) Tricuspid insufficiency
      (3) Right ventricular hypertrophy may be present.

C. Interventions/Outcomes

1. Interventions
   a. Prevention is best, with early and effective resuscitation and correction of acidosis and hypoxia.
   b. Assisted ventilation with hyperoxia may be used as needed to increase passive oxygen diffusion across alveolar membrane and promote vasodilation. Call the neonatal team and transfer to NICU as needed to best support the infant (Latini et al, 2008).
   c. Decrease environmental stimulation because these infants are often quite sensitive to handling and stress from the environment.
      (1) Decrease activity.
      (2) Decrease noise and light.
      (3) Cluster caregiving.
      (4) Use sedation as needed.
      (5) Manage and treat before painful procedures.

2. Outcomes
   a. The infant receives the appropriate concentration of oxygen for PVR to decrease and pulmonary vascular blood flow to increase.
   b. The infant’s ABG values are WNL.
   c. The infant demonstrates minimal respiratory distress.
      (1) Respiratory rate between 40 and 60 breaths per minute
      (2) Bilateral breath sounds with good air entry
      (3) Absence of generalized cyanosis
HYPERBILIRUBINEMIA

INTRODUCTION

Causes of Hyperbilirubinemia

Physiologic jaundice is a condition that is common in the term newborn infant during the second or third day of life (second to ninth day in preterm neonates) and is not considered to be pathologic unless bilirubin levels exceed the normal physiologic limitations of a healthy neonate; pathologic hyperbilirubinemia, which can be defined only by serum concentrations of unconjugated bilirubin, has diverse causes that are frequently, but not always, interlinked; jaundice is a manifestation of bilirubin accumulation in extravascular tissues.

A. Incidence of physiologic jaundice during the first week of life

1. Almost all neonates have elevated serum bilirubin levels greater than 2 mg/dL (the normal level in adults is 1.3 mg/dL or less) (Maisel, 2006)
2. Elevated serum bilirubin levels greater than 5 mg/dL occur in 60% of neonates (Maisel, 2006)
3. Hyperbilirubinemia, as it occurs in physiologic jaundice, may confer some biologic advantage to the neonate.
   a. Beneficial effects of bilirubin molecules might be noted at a cellular level (Maisel, 2006).
   b. Bilirubin has a potent antioxidant effect.
   c. Neonates have deficient levels of most antioxidant substances.
4. Breastfeeding jaundice appears in the first days of life and is called such because it appears to be related to early ineffective breastfeeding practices that lead to (Maisel, 2006):
   a. Decreased volume of fluid intake
   b. Decreased caloric intake of fluid
   c. Dehydration
   d. Delayed passage of meconium
5. Breast milk jaundice occurs after 3 to 5 days of life, with a steady increase in serum bilirubin that usually peaks at approximately 2 weeks (5 to 10 mg/dL) and then decreases slowly; this type of jaundice appears to be related to the composition of the breast milk that results in enhanced enterohepatic circulation, although this is speculative (Maisel, 2006).

B. Bilirubin production and conjugation

1. Bilirubin has two forms:
   a. Unconjugated or indirect
      (1) Fat soluble
      (2) Toxic to tissues
   b. Conjugated or direct
      (1) Water soluble
      (2) Nontoxic to tissues
2. The majority of bilirubin comes from the destruction of hemoglobin.
3. Process of bilirubin conjugation
   a. Within the circulatory system, unconjugated bilirubin tightly bound to albumin is transported to the liver, where the conjugation process takes place.
   b. Bilirubin is then released from the albumin binding site and undergoes the following:
      (1) Transfer across the hepatocyte membrane
      (2) Cytoplasmic protein binding
         (a) Within the liver, bilirubin is bound to ligandin and other hepatic proteins.
         (b) This binding helps prevent a backup of bilirubin into the general circulation.
      (3) Transport (while bound to protein)
         (a) Smooth endoplasmic reticulum is the site of conjugation process.
         (b) Process of conjugation transforms the poorly soluble, unconjugated bilirubin into a water-soluble form that can be excreted by the neonate; this process requires oxygen and glucose.
      (4) Excretion (after conjugation) into the bile, into the intestine, and finally into stool
         (a) Some bilirubin is excreted through the kidneys as urobilinogen.
         (b) In the intestine, the enzyme glucuronidase may break the ester linkage of the bilirubin, causing it to become unconjugated.
         (c) The newly unconjugated bilirubin may be reabsorbed into the neonate’s circulation, necessitating a repetition of the entire conjugation process.
4. Neonatal hyperbilirubinemia can occur from the following:
   a. An increased load of circulating bilirubin resulting from:
      (1) Polycythemia
      (2) Isoimmune hemolytic disease
      (3) Structural and enzyme defects of RBCs
      (4) Drug toxicity (chemical hemolysis)
      (5) Extravascular hemolysis
   b. Impaired hepatic function (e.g., defective uptake, conjugation, or excretion)
      (1) Deficient glucuronyl transferase activity
      (2) Biliary obstruction or biliary atresia
      (3) Infection
      (4) Metabolic problems: galactosemia, breast milk jaundice and hypothyroidism
   c. Perinatal complications, such as asphyxia, hypothermia, and hypoglycemia
   d. Decreased albumin binding sites, because of preterm birth and/or competition with drugs having an affinity for the binding sites
   e. Delayed meconium passage
5. Rate of bilirubin level increase
   a. Must exceed 4 to 6 mg/dL before it is visible as jaundice.
   b. In physiologic jaundice:
      (1) Bilirubin level increase first appears after 24 hours of age in term neonates and after 48 hours of age in preterm neonates.
         (a) Reaches peak at day 3 or 4 and returns to a normal level by the end of day 7 in term neonates
         (b) Reaches peak at day 5 or 6 and returns to a normal level by the end of day 9 or 10 in preterm neonates
         (c) Pattern for breastfed infants is slightly different; peak level often occurs on day 4 and the decline may be slower.
      (2) Indirect bilirubin value does not usually exceed 12 mg/dL.
      (3) Direct bilirubin does not usually exceed 1 to 1.5 mg/dL.
      (4) Daily increases of bilirubin do not usually exceed 5 mg/dL.
   c. In pathologic jaundice, elevated bilirubin levels:
      (1) Appear within the first 24 hours of life
      (2) Persist beyond the age for return to a normal level in term and preterm neonates
      (3) No specific serum level can be used for diagnosis.
   d. Kernicterus is a preventable neurologic syndrome with lifelong sequelae; it is caused by severe and inadequately treated hyperbilirubinemia during the neonatal period (Bhutani & Johnson, 2009).
      (1) Bilirubin encephalopathy is the most serious complication of hyperbilirubinemia.
      (2) Yellow staining of brain tissue occurs and creates morphologic changes in brain cells, which results in irreversible damage.
      (3) Approximately one half of affected neonates do not survive.
      (4) Condition is generally thought to occur at bilirubin levels in excess of 20 mg/dL in full-term neonates.
      (5) Early signs include:
         (a) Extreme jaundice
         (b) Alterations in level of consciousness (lethargy)
         (c) Tone (hypotonia, then later, hypertonia)
         (d) Abnormal movement (opisthotonos)
         (e) Poor feeding
         (f) High-pitched crying
      (6) The long-term sequelae include:
         (a) Cerebral palsy
         (b) Sensorineural hearing loss
         (c) Gaze paresis
         (d) Dental dysplasia
         (e) Kernicterus
         (f) Mental retardation
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C. Phototherapy

1. Treatment is widely used to manage and control rising bilirubin levels (Stokowski, 2006).
   a. Un clothed neonate, with eye shields, is placed in an infant incubator within 45 to 50 cm (18 to 20 inches) of the bank of lights and turned every 2 hours.
   b. Treatment can also be performed with a fiberoptic blanket attached to an illuminator and wrapped around the neonate’s torso and extremities.
      (1) Allows use of an open crib
      (2) Eye shields are not needed.
      (3) Neonate can be held.
      (4) Can be used for home phototherapy (Stokowski, 2006)

2. Mechanism of phototherapy action
   a. Treatment is thought to reduce serum bilirubin levels by facilitating biliary excretion of unconjugated bilirubin.
   b. Treatment causes the formation of photosomers, which are more water soluble and therefore more easily excreted in stool and urine.
      (1) Unconjugated bilirubin is rapidly converted to photobilirubin and lumirubin.
      (2) Photobilirubin and lumirubin are rapidly taken up by the liver and transported into the bile.
      (3) Process occurs independent of hepatic conjugation of bilirubin.
   c. White, daylight, cool blue, and special blue are among the various types of phototherapy lights available.
      (1) Lights with high-energy output, ranging between 420 and 450 nm in the blue spectrum, are the most effective.
      (2) Optimal energy output light levels should be monitored to ensure that levels are maintained to achieve maximum efficiency.
   d. Home monitoring and treatment
      (1) Bilirubin levels can be determined in the hospital as well as during a home visit by using transcutaneous bilirubinometry (TcB).
         (a) Procedure uses a noninvasive, portable instrument.
         (b) Predicts serum bilirubin levels in the neonate by using reflective measurements on the skin to determine the amount of yellow in the skin.
         (c) Measurement can correlate well with serum bilirubin levels; however, error rates can be high because of a variety of neonatal factors (Maisel, 2006).
            [i] Gestational age of neonate
            [ii] Birthweight
            [iii] Skin pigmentation because of different ethnic origin
            [iv] Phototherapy treatment (Stokowski, 2006)
      (d) TcB should be used only as a screening tool to determine when a laboratory measurement of serum bilirubin is needed (Maisel, 2006).
         (2) In some circumstances, home phototherapy can be provided and thus avoid separation of neonate from parents.

D. Treatment controversies surrounding physiologic jaundice exist.

1. The trend has been to decrease interventions and to observe and manage neonates as outpatients.
2. Research has shown that healthy full-term neonates in the absence of significant hemolysis or other underlying medical conditions with serum bilirubin levels of approximately 18 mg/dL do not have any detrimental effects with an expectant observation treatment approach (Bhutani & Johnson, 2009; Maisel, 2006).
3. The American Academy of Pediatrics (AAP) Practice Guidelines for Management of Hyperbilirubinemia in the Healthy Term Newborn provides guidelines for identifying at-risk infants and intervention and treatment strategies; in April 2001, The Joint Commission (TJC) issued a Sentinel Event Alert regarding the increased incidence of kernicterus. Proposed risk reduction strategies include:
   a. PredischARGE bilirubin measurement, with standing orders allowing nurses to order total serum bilirubin (TSB) levels or TcB levels for newborns (Bhutani & Johnson, 2009; Maisel, 2006).
   b. Use of a percentile-based nomogram to predict the risk of hyperbilirubinemia and implement strategies for follow-up, which may be helpful in provision of care (Bhutani & Johnson, 2009) (Figure 17-1)
c. Follow-up for all newborns within 48 hours after discharge by a physician or trained health care provider who is experienced in the care of newborns to provide follow-up physical assessment and ongoing lactation support to ensure adequacy of intake for breastfed infants (Bhutani & Johnson, 2009; Maisel, 2006).

d. Providing parents with adequate educational materials at discharge regarding jaundice, feeding adequacy, and symptoms to watch for (Stokowski, 2006)

CLINICAL PRACTICE

A. Assessment

1. History

   a. Maternal

      (1) Mother’s blood type can have an effect on a neonate’s bilirubin levels by causing intravascular hemolysis via an antigen-antibody reaction.

      (a) Rh disease

      (b) ABO incompatibility

      (c) Positive Coombs’ test result

      (2) Insulin-dependent diabetic mother

      (a) Neonate has an increased RBC mass at birth.

      (b) Breakdown of a high number of aged RBCs results in excessive amounts of bilirubin in the neonate.

   (3) Maternal and paternal ethnicity

      (a) Asian and Native American neonates have a higher incidence of hyperbilirubinemia.

      (b) Mean serum levels of unconjugated bilirubin in Asian and Native American neonates are higher than the mean levels for neonates of other ethnic origins (Maisel, 2006).

         [i] Mean serum levels of unconjugated bilirubin for the former are 10 to 14 mg/dL.

         [ii] This mean is approximately double that of neonates of other ethnic origins.

      (c) African American and white neonates tend to have lower mean levels of bilirubin.
(4) Maternal drugs used during the last 3 months of pregnancy
   (a) The following drugs interfere with the binding of bilirubin to albumin in neonates:
      [i] Sulfonamides
      [ii] Salicylates
      [iii] Ibuprofen
   (b) Synthetic oxytocin (Pitocin) is associated with a higher incidence of neonatal jaundice.
(5) 70% possibility of recurrence in subsequent siblings of an infant diagnosed with breast milk jaundice (Maisel, 2006)
   (a) Breast milk jaundice occurs in 0.5% to 2% of breastfed term infants (Maisel, 2006).
   (b) Affected breast milk contains substances that inhibit the activity of glucuronyl transferase in the neonatal liver.
   (c) Bilirubin levels continue to rise on the fourth and fifth days, when physiologic jaundice should be subsiding.
      [i] Rise to peak concentrations of between 10 and 27 mg/dL between 10 and 15 days of age
      [ii] Gradually decrease from peak levels to normal levels between 3 and 12 weeks of age (Maisel, 2006)

b. Labor and delivery history
   (1) Use of the following drugs:
      (a) Synthetic oxytocin (Pitocin)
      (b) Diazepam
      (c) Bupivacaine (in epidural anesthesia)
   (2) Delayed umbilical cord clamping
      (a) Allows an excessive amount of blood to be transfused from the placenta to the neonate
      (b) Increase in quantity of RBCs results in the following:
         [i] Sequestration of greater numbers of aged RBCs
         [ii] Excessive amounts of bilirubin from breakdown of aged RBCs (Maisel, 2006)
   (3) Operative delivery: Ecchymosis and extravascular hemolysis may be increased by use of forceps or vacuum extractor.

c. Perinatal complications
   (1) Cephalohematoma
   (2) Cerebral hemorrhage
   (3) Pulmonary hemorrhage
   (4) Any occult bleeding
   (5) Maternal-fetal transfusion
   (6) Hypoxia
      (a) Causes increase in free fatty acids.
      (b) Free fatty acids compete with bilirubin for albumin-binding sites.
   (7) Infection and sepsis (bacterial, viral, or protozoal)
      (a) Hemolysis
      (b) Hemolytic anemia
   (8) Low Apgar scores at 1 minute and at 5 minutes may indicate a complication.

d. Postnatal complications
   (1) Caloric deprivation, causing weight loss, results from:
      (a) Infrequent feedings
      (b) Poor suck-and-swallow reflex
      (c) Decrease in gastric motility and enzymatic activity
   (2) Glucose-6-phosphate dehydrogenase (G6PD) deficiency
      (a) Is an enzymatic deficiency
      (b) Disrupts erythrocyte metabolism and causes hemolysis
      (c) Occurs primarily among African Americans, Filipinos, Sephardic Jews, Sardinians, Greeks, and Arabs (Maisel, 2006).
      (d) Manifests as jaundice on the second or third day of life and persists into the second or third week of life.

2. Physical findings
   a. Visible jaundice progresses in a cephalocaudal direction and can be seen first in facial skin, sclera, and gums, and later in the torso and lower extremities.
b. Bilirubin levels
   (1) Elevated umbilical cord blood bilirubin level
   (2) Pattern of jaundice onset, length, and duration

c. SGA condition that is associated with infection

d. Small head circumference (microcephaly) that is associated with infection

e. Cephalohematoma, ecchymosis, and abrasions are indicative of a traumatic delivery process.

f. Pallor that is suggestive of hemolytic anemia

g. Poor feeding or difficulty with breastfeeding

h. Lethargy; difficult to awaken for feedings

i. Petechiae that are suggestive of the following:
   (1) Congenital infection
   (2) Overwhelming sepsis
   (3) Severe hemolytic disease

j. Plethora that is associated with polycythemia

k. Vomiting that is suggestive of the following:
   (1) Sepsis
   (2) Pyloric stenosis

l. Hepatosplenomegaly that is suggestive of the following:
   (1) Chronic intrauterine infection
   (2) Hemolytic anemia

m. Congenital anomalies: Increased incidence of jaundice is found in infants with trisomies.

3. Diagnostic procedures

a. Maternal blood group and indirect Coombs’ test to rule out possibility of ABO or Rh incompatibility

b. Serologic assays to rule out congenital syphilis

c. Hemoglobin to rule out the following:
   (1) Anemia
   (2) Polycythemia: hemoglobin concentration higher than 22 g/dL

d. CBC with differential
   (1) Elevated reticulocyte count suggests hemolytic disease.
   (2) RBC morphology
      (a) Spherocytes suggest ABO incompatibility.
      (b) RBC fragmentation suggests disseminated intravascular coagulopathy (DIC).
   (3) WBC count
      (a) Less than 5000/mm$^3$ suggests infection.
      (b) Increase in bands to more than 2000/mm$^3$ suggests infection.
   (4) Platelets: Thrombocytopenia suggests infection.
   (5) Sedimentation rate values in excess of 5 mm$^3$ during first 48 hours is suggestive of infection or ABO incompatibility.
   (6) Elevated direct bilirubin (conjugated) level suggests infection or severe Rh incompatibility.

B. Interventions/Outcomes

1. Interventions

a. Increase fluid intake to offset the following:
   (1) Increase in metabolic rate caused by phototherapy (Stokowski, 2006)
   (2) Significant increase in water loss through the skin
   (3) Increase in water content of frequent stools
   (4) Water loss caused by hyperthermia

b. Offer neonate frequent feedings.
   (1) Feed as often as tolerated (approximately every 2 hours).
   (2) Increase calories to offset rapid intestinal transit time (frequent stooling) and decreased intestinal absorption of milk.

c. Maintain thermal homeostasis while infant is under phototherapy lights by using a servo-control mechanism such as an isolette or radiant warmer.
   (1) Hypothermia stimulates the release of free fatty acids, which compete for albumin binding sites.
   (2) Hyperthermia increases the neonate’s metabolic rate.
d. Monitor for impaired neonatal skin integrity related to diarrhea, urinary excretions of bilirubin, and exposure to phototherapy lights (Stokowski, 2006).
   (1) Diapering of the neonate may be accomplished by using a paper face mask with the metal strip removed.
      (a) Allows maximal skin exposure for phototherapy lights
      (b) Provides protection for genitals and bedding
      (c) Shields a minimum of jaundiced skin
   (2) It is typical for stools to be loose, greenish, frequent, and expelled with force.
   (3) It is important to protect neonate’s skin from excoriation by thoroughly removing urine and feces with each diaper change.
   (4) It is important to change diapers frequently.
   (5) It is important to keep neonate’s skin clean and dry by frequently checking bedding for dampness or stool soiling.

e. Protect the neonatal cornea from phototherapy light exposure through continuous wearing of protective eye shields.
   (1) Protect neonate’s eyes from phototherapy lights by applying eye shields to prevent eye damage.
   (2) Secure placement of the eye shields tightly enough to prevent slippage and accidental eye exposure but not so tightly that constraint and excessive pressure are placed on the neonate’s eyes.
   (3) Remove eye shields when neonate is not under phototherapy lights.
      (a) For feedings
      (b) For procedures not performed under phototherapy lights
   (4) Make sure the neonate’s eyes are closed when applying the eye shields.
   (5) Change eye shields frequently and watch for any signs of conjunctivitis, such as:
      (a) Purulent discharge
      (b) Edema

f. Assess for impaired parenting related to parent-infant separation secondary to phototherapy treatments (Stokowski, 2006).
   (1) Encourage parents to participate in the caretaking responsibilities of their neonate.
   (2) Encourage parents to hold their neonate for short periods.
   (3) Encourage parents to provide gentle stroking and touching of their neonate while he or she is under phototherapy lights.

2. Outcomes
   a. Fluid intake is increased.
   b. Neonate remains well hydrated while under phototherapy lights.
   c. Neonate maintains a normal temperature while under the phototherapy lights.
   d. Neonate is covered and kept warm when not under phototherapy lights (i.e., for feedings and other procedures).
   e. Neonate’s skin remains clean and dry while under phototherapy lights.
   f. No signs of redness or excoriation are present on the neonate’s skin.
   g. Neonate wears protective eye shields at all times while under phototherapy lights.
   h. No signs of excessive eye shield pressure are present on neonate’s skin around the eyes.
   i. No signs of conjunctivitis are present in neonate.
   j. Parents actively participate in caretaking activities with their neonate.
   k. Parents provide soothing tactile stimulation to their neonate while he or she is under phototherapy lights.

HEALTH EDUCATION

A. The technologic advances made in newborn care during the past 30 years have markedly decreased morbidity and mortality in this group of high-risk infants.
   1. Technologic advances have brought a heightened sensitivity to the psychological and emotional effect felt by the family of a sick neonate.
   2. This awareness has introduced the need for a family-centered approach to newborn care (McGrath, 2007a).
   3. In addition to the physiologic care of the sick neonate, health care workers need to address the psychologic needs of family members during this experience.
4. Parental attachment to the infant with respiratory distress, hyperbilirubinemia, or both, is especially difficult.
   a. The infant may be premature.
   b. Normal interaction may be severely curtailed.
   c. An infant who is sick enough to be in a special care or intensive care unit on a ventilator, or receiving oxygen support or phototherapy may not give cues adequate to arouse parental attachment.

5. Provide information in nontechnical terms to parents about serum bilirubin levels while the neonate is being monitored, and encourage their questions and concerns about their neonate’s condition.

B. The birth of a newborn who is sick represents a unique crisis for the family and perinatal health care team (McGrath, 2007a).
   1. The family must simultaneously adjust to the immediate situation and begin the normal developmental process of parenthood.
   2. Situational factors have an important bearing on the family’s ability to cope with this present crisis and can affect the overall outcome.
      a. The behavior and attitude of the hospital staff
      b. The sensitivity used in the transfer process either to another facility or the NICU
      c. The flexibility in unit visitation and extended family involvement
      d. The instruction received by the family related to their infant’s unique characteristics and behavior
      e. The staff’s sensitivity to the family’s responses and adaptation to crisis
      f. The use of emotionally supportive intervention programs in the nursery
      g. The development of appropriate discharge planning to provide adequate follow-up for the family

C. Discharge planning for the infant with respiratory distress or hyperbilirubinemia may include the following:
   1. Family education
      a. Use of equipment needed in the care of the infant (e.g., oxygen, suctioning devices, phototherapy)
      b. Praise for parents’ caretaking abilities of infant while in the hospital; parents are not visitors
      c. When and how to perform chest physiotherapy
      d. Dosage, route of administration, side effects, and planned duration of use of all medications
      e. Nutritional information to maintain adequate calorie and fluid balance
         (1) Type of formula (if used instead of breast milk)
         (2) How and when to feed the infant
         (3) Possible alternative feeding methods
         (4) If and when to provide oxygen during the feedings
      f. Support for mother’s or father’s attempts to feed the neonate and encourage frequent feedings to provide neonate with adequate hydration and increased calories
      g. The use of home monitoring, if needed
      h. Infant cardiopulmonary resuscitation (CPR)
      i. Recognition of signs and symptoms of illness in the newborn
      j. Normal newborn care
   2. Acquisition and maintenance of specialized equipment that may be needed to care for the infant at home, including:
      a. Oxygen and oxygen equipment
      b. Suction machine and supplies
      c. Home monitoring equipment
      d. Phototherapy
   3. All information and education given to the family should be in written form, if possible; in addition, whenever possible, return demonstrations by the family reinforce learning.
   4. Parents should be instructed about signs, symptoms, and treatment of hyperbilirubinemia that may occur at home as a result of early discharge program (Bhutani & Johnson, 2009; Maisel, 2006; Stokowski, 2006).
      a. Monitoring neonate’s behaviors that may be indicative of increasing bilirubin levels
      b. Blanching neonate’s skin to determine degree of jaundice
c. Placing the neonate’s bassinet or cradle near a window during the daytime to take advantage of natural sunlight
d. Monitoring feeding and level of consciousness
e. Maintaining sufficient hydration level in neonate
f. Advising when to take the neonate to the health care provider for further evaluation of bilirubin levels

D. Long-term follow-up of the infant with respiratory distress or hyperbilirubinemia is essential.
   1. Parents must have telephone numbers of the medical facility and personnel to call 24 hours a day in case of problems or equipment failure.
   2. Before hospital discharge, parents need to have information regarding follow-up appointments and referrals for long-term care.

COMMON CONGENITAL ABNORMALITIES

NEURAL TUBE DEFECTS

A. Hydrocephalus
   1. Introduction
      a. Congenital hydrocephalus is an enlargement of the cerebral ventricles or subarachnoid spaces.
      b. Incidence of congenital hydrocephalus is 0.4 to 3.16 per 1000 newborns (Landingham, Nguyen, Roberts, Parent, & Zhang, 2008).
   2. Clinical practice
      a. Assessment
         (1) History
            (a) Fetal ultrasound testing shows an enlarged head.
            (b) Cephalopelvic disproportion (CPD)
         (2) Physical findings
            (a) Head circumference greater than the 90th percentile equates to a 95% likelihood of ventricular dilation (Landingham et al, 2008).
            (b) Enlarged or full fontanelles
            (c) Wide or split suture lines
            (d) Excessive rate of head growth
            (e) Vomiting might occur.
      b. Diagnostic procedures
         (1) Sequential ultrasound testing to evaluate ventricular size and rate of dilation
         (2) Magnetic resonance imaging (MRI) and CT for evaluation of cerebral parenchyma
      c. Interventions/Outcomes
         (1) Interventions
            (a) Clean and dry skin creases after feeding or vomiting.
            (b) Place infant on a waterbed or an egg-crate mattress.
            (c) Reposition neonate’s head frequently.
            (d) Monitor any reddened areas, and position infant away from any potential problem areas.
         (2) Outcomes
            (a) Neonate’s skin remains intact and shows no signs of beginning breakdown.

B. Spina bifida
   1. Introduction
      a. Spina bifida is a general term used to describe defects in closure of the neural tube associated with malformations of the spinal cord and vertebrae. Spina bifida is the most common congenital malformation of the CNS (Landingham et al, 2008).
      b. Spina bifida cystica includes three main types of defects
         (1) Meningocele
            (a) Presence of a sac containing meninges and cerebrospinal fluid (CSF); however, the spinal cord and nerve roots are normal in their structure and positioned within the spinal canal.
            (b) Typically infants will not show neurologic deficits.
         (2) Myelomeningocele (MN)
            (a) Most common form of spina bifida cystica; occurrence rate is 1 to 1.5 per 1000 live births (Vogel & Sturm, 2008)
2. Clinical practice
   a. Assessment
      (1) History
         (a) Elevated maternal serum alpha-fetoprotein levels
         (b) Ultrasound visualization of the defect
         (c) Familial history of spina bifida
         (d) Hydrocephalic fetus
      (2) Physical findings
         (a) Presence of a spinal lesion
         (b) Enlarged head circumference: hydrocephalus
         (c) Lack of spontaneous movement of the lower extremities
         (d) Hip clicks secondary to congenital hip dislocation
         (e) Clubfoot and scoliosis
         (f) Flaccid or spastic muscles in the lower extremities
         (g) Urine and stool leakage
   b. Interventions/Outcomes (for immediate care in the delivery room and prior to transport to more acute care)
      (1) Interventions
         (a) Place neonate only in prone or side-lying position.
         (b) Place neonate into sterile bowel bag, secure top at level of axilla; or cover defect with warm, normal saline–moistened sterile gauze, and place plastic wrap over dressing.
         (c) Keep meconium or urine away from lesion.
         (d) Administer antibiotics per orders.
      (2) Outcomes
         (a) Membranous sac stays intact.
         (b) Aseptic environment is maintained around and over the lesion.
         (c) Neonate does not show any signs of infection.

GASTROINTESTINAL CONGENITAL ANOMALIES

A. Cleft lip and cleft palate
1. Introduction
   a. Cleft lip with or without cleft palate is the most common craniofacial anomaly affecting approximately 1 of every 600 live births (Johnson & Little, 2008).
   b. Cleft palate alone occurs in 1 of every 1000 newborns and occurs more frequently in females (Ciminello, Morin, Nguyen, & Wolfe, 2009).
   c. Cleft lip may be unilateral or bilateral. Cleft palate may involve just the soft palate or both the soft and the hard palates.
   d. Facial clefting is associated with an increased incidence of other abnormalities (Jaruratanairikul, Chichareon, Pattanapreechawong, & Sangsupavanich, 2007).
2. Clinical practice
   a. Assessment
      (1) History
         (a) Maternal use of phenytoin, alcohol, retinoic acid, cigarette smoking
         (b) Family history of cleft lip and/or palate in siblings
         (c) More than 300 recognized syndromes include cleft lip and palate as a characteristic (Jaruratanairikul et al, 2007).
      (2) Physical findings
         (a) Unilateral or bilateral visible defect
         (b) Flattening or depression of midfacial contour in cleft lip
         (c) Fissure connecting oral and nasal cavities in cleft palate
         (d) Difficulty in sucking
(e) Expulsion of formula or breast milk through the nares
(f) Dehydration
(g) Poor weight gain or weight loss

b. Interventions/Outcomes (for care in the newborn nursery; some infants may require more acute care)

(1) Interventions (Ciminello et al, 2009)
   (a) Feed with a special nipple and bottle set.
   (b) Burp frequently.
   (c) Feed in upright position with head and chest tilted slightly back to aid swallowing and discourage aspiration.
   (d) Limit feedings to 30 to 45 minutes to avoid poor weight gain due to fatigue.
   (e) Feed high-calorie-per-ounce formula to increase caloric intake.
   (f) Have mother attempt breastfeeding if only cleft lip is present.
   (g) Support parental coping and assist parents with grief over loss of idealized baby.
   (h) Encourage parents to verbalize feelings about the defect and the feeding frustrations.
   (i) Provide role modeling while interacting with the neonate so that parents can internalize positive interaction (Johnson & Little, 2008).
   (j) Refer parents to community agencies and support groups.

(2) Outcomes
   (a) Neonate is gaining weight appropriate to age.
   (b) Neonate is not vomiting feedings.
   (c) Neonate is not excessively fatigued after feeding.
   (d) Parents are able to freely verbalize their feelings and frustrations about their infant.
   (e) Parents are involved in the neonate’s care in the hospital and frequently seek information about the infant’s progress.
   (f) Parents exhibit bonding behaviors with their infant.

B. Abdominal wall defects

1. Introduction
   a. Omphalocele
      (1) Defect of the umbilical ring that allows evisceration of abdominal contents into an external peritoneal sac (Ledbetter, 2006)
      (2) Incidence is 1.5 to 3 per 10,000 live births (Ledbetter, 2006)
   b. Gastroschisis
      (1) Defect of the umbilical ring that allows evisceration of bowel through a defect in the abdominal wall with no membrane covering (Ledbetter, 2006)
      (2) The incidence is 0.4 to 3 per 10,000 live births (Ledbetter, 2006)

2. Clinical practice
   a. Assessment
      (1) History
         (a) Polyhydramnios
         (b) Visualization on fetal ultrasonography
         (c) Elevated maternal serum alpha-fetoprotein
         (d) Preterm labor in the case of gastroschisis
      (2) Physical findings
         (a) Visible defect over the abdominal area
            [i] Omphalocele is covered with a sac consisting of peritoneum and amniotic membrane.
            [ii] Gastroschisis defect exposes viscera because of lack of any covering.
            [iii] Gastroschisis occurs almost exclusively to the right of the umbilicus.
         (b) About 50% of newborns born with omphalocele have cardiac, gastrointestinal, genitourinary, musculoskeletal, and CNS anomalies (Ledbetter, 2006).
   b. Interventions/Outcomes (for immediate care in the delivery room or prior to transfer to more acute care)
      (1) Interventions
         (a) Place neonate feet first into sterile bowel bag, and secure top at level of axilla.
         (b) Monitor temperature closely.
         (c) Place in isolette, and maintain NTE.
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(d) Administer IV fluids and albumin per institutional protocol.
(e) Maintain nothing per mouth (NPO) status.
(f) Maintain integrity of sterile bowel bag or moist sterile dressing.
(g) Monitor glucose levels and electrolytes.
(h) Insert nasogastric tube to decompress bowel.

(2) Outcomes
(a) Omphalocele sac remains intact.
(b) Herniated viscera remains normal in color and moist.
   [i] Hydration is maintained.
   [ii] No further advancement of bowel herniation through defect
(c) Neonate’s temperature remains WNL.

C. Congenital diaphragmatic hernia
1. Introduction
   a. Congenital diaphragmatic hernia (CDH) is a malformation that consists of herniation of abdominal contents into the thorax cavity via a defect in the diaphragm. The exact etiology of CDH is not fully understood. CDH might be caused by maldevelopment of one or more components of the diaphragm occurring during the embryonic stage of development (Haricharan, Barnhart, Cheng, & Delzell, 2009).
   b. Incidence of CDH is approximately 2.5 to 3.8 per 10,000 live births with a mortality rate of 45% in live-born infants (Kays, 2006). CDH and neonatal lung lesions often occur together. Abdominal contents in the thorax cause a mediastinal shift that can result in impairment of cardiovascular function.
      (1) Interference with venous return to the heart
      (2) Reduction in cardiac output
      (3) Metabolic acidosis

2. Clinical practice
   a. Assessment
      (1) History
         (a) Term or postterm
         (b) Polyhydramnios common
      (2) Physical findings (Haricharan et al, 2009)
         (a) Large or barrel chest
         (b) Scaphoid abdomen (Kays, 2006)
         (c) Respiratory distress (ranges from mild to life threatening)
            [i] Difficulty in initiating respiration
            [ii] Gasping respirations
            [iii] Retractions and nasal flaring
            [iv] Cyanosis
            [v] Decreased or absent breath sounds on the side of the hernia
         (d) Displacement of the cardiac impulse to one side of the chest
         (e) Bowel sounds heard in the chest
         (f) Asymmetric chest expansion
      (3) Diagnostic procedures
         (a) Chest radiograph film: Diaphragmatic margin is absent on the defective side with presence of loops of intestine in the chest cavity, which might be gas filled, giving a multicystic appearance; mediastinal shift to the side opposite of defect (Haricharan et al, 2009)
         (b) Arterial blood gas assay: hypoxemia, respiratory acidosis, and metabolic acidosis
   b. Interventions/Outcomes (for immediate care and prior to transport to more acute care)
      (1) Interventions
         (a) Delivery room resuscitation
            [i] Neonate should be immediately intubated; bag-and-mask ventilation must be avoided because air can be forced into the intestine, which will further compromise lung space in the chest.
            [ii] Mechanical ventilation pressures should be kept at a minimal level to avoid pneumothorax.
            [iii] Administer 100% oxygen to increase the PaO₂ and decrease persistent pulmonary hypertension.
Hyperoxygenate to minimize hypoxemia.
Ventilate with small tidal volumes at a rapid respiratory rate to provide oxygenation and decrease risk of pneumothorax.
Administer IV vasopressors as ordered.
Sedate as needed to minimize oxygen needs.

Gastric decompression should be immediately initiated by inserting a large-bore nasogastric tube and advancing it as far as possible.

1. Positioning
   a. Elevate head of bed to minimize abdominal organ pressure on diaphragm.
   b. Turn the neonate onto the affected side to allow unaffected lung to expand.

3. Outcomes
   a. Neonate’s respiratory effort receives optimal support until emergency surgical intervention occurs (Haricharan et al, 2009).
   b. Neonate remains hyperoxygenated as evidenced by blood gases.
   c. Blood pressure remains within the ordered parameters.
   d. Adequate sedation is maintained.

CONGENITAL CARDIAC LESIONS

INTRODUCTION

The incidence of congenital cardiovascular malformations is approximately 5 to 8 per 1000 live-born infants. Genetic disorders that might cause heart defects are categorized into three major groups (Lott, 2007).

A. Chromosomal disorders, including Di George syndrome, trisomy 21 (Down syndrome), and Turner syndrome
B. Single-gene disorders, which might be either autosomal dominant or autosomal recessive
C. Polygenic disorders resulting from multiple genetic or environmental influences
D. Cardiac defects or lesions are categorized as:
   1. Acyanotic: Oxygenated blood is shunted to the body, but the infant remains “pink.”
   2. Cyanotic: Unoxygenated blood is shunted to the body causing the infant to be “blue.”

CLINICAL PRACTICE

A. Acyanotic defects
   1. Patent Ductus Arteriosus (PDA)
      a. Introduction
         (1) An anatomic and functionally open shunt exists between the pulmonary artery and the aorta.
         (2) PDA occurs in 5% to 10% of all cases of congenital heart disease in full-term neonates (Lott, 2007).
         (3) PDA occurs in 37% of infants weighing 501 to 1500 g (1 lb, 2½ oz to 3 lbs, 5 oz) (Dagle et al, 2009).
         (4) PDA becomes functionally closed within the first 12 hours of life in the full-term infant. The closure is complete by 2 to 3 weeks of life (Agarwal et al, 2008).
      b. Assessment
         (1) Physical findings
            a. A harsh systolic murmur, which becomes continuous, is heard at the left upper sternal border and posteriorly.
            b. An active precordium is common.
            c. Bounding peripheral pulses
            d. Pulse pressure is widened with a low diastolic pressure.
         (2) Diagnostic procedures
            a. Chest radiograph film: Findings depend on shunt size; in moderate or large shunts, heart enlargement might be present.
      c. Interventions/Outcomes (Sadowski, 2010)
         (1) Management depends on whether the shunt is hemodynamically significant. In premature infants, the PDA may prolong ventilator use beyond the dictates of the initial lung disease.
(2) Conservative measures are generally used initially, which may minimize exposure to pharmacologic agents. However, delaying treatment may decrease response to nonsteroidal antiinflammatory drugs.
   (a) Fluid restriction
   (b) Diuretics: If used with fluid restriction, may lead to electrolyte imbalance, dehydration, and caloric deprivation.
   (c) Positive end-expiratory pressure (PEEP) is useful in reducing left-to-right shunt via PDA.
(3) Medical management uses a nonsteroidal antiinflammatory agent that inhibits COX-1 and/or COX-2; treatment choices are indomethacin and ibuprofen lysine; usually used with preterm infants.
(4) Surgical management
   (a) Standard approach is surgical ligation via a thoracotomy.
   (b) New techniques include placement of a stainless-steel spring coil or Da occlusion device via cardiac catheterization or minimally invasive video-assisted thoracoscopic surgery that allows for PDA closure using two titanium clips placed with a trocar (Dutta & Albanese, 2006).

2. Atrial septal defects
   a. Introduction
      (1) An atrial septal defect (ASD) is an opening in the septum between the atria that occurs as a result of improper septal formation in early fetal cardiac development. An incompetent or malformed foramen ovale is the most common defect.
      (2) Atrial septal defects account for 5% to 10% of all congenital heart disease. Incidence in Down syndrome is 20% (Lott, 2007).
      (3) Permits shunting of blood between the two atria
   b. Assessment
      (1) Physical findings—dependent on severity of defect
         (a) Low-pitched diastolic murmur best heard at the left lower sternal border.
         (b) Widely split S2
      (2) Diagnostic procedures
         (a) Chest radiograph film: cardiomegaly and increased vascular markings due to increased blood flow to the lungs as a result of shunting of blood from the left side of the heart to the right side
         (b) Electrocardiogram: usually sinus rhythm with ostium primum and secundum, but can show prolonged P-R interval with right atrial hypertrophy and P-wave changes
   c. Interventions (Sadowski, 2010)
      (1) If defect is small, clinical follow-up is indicated because the defect may close spontaneously.
      (2) If the defect is large, or with intractable CHF, surgical repair is done with the patient on cardiopulmonary bypass.

3. Ventricular septal defect
   a. Introduction
      (1) A ventricular septal defect (VSD) is an opening in the septum between the right and left ventricles that results from imperfect ventricular formation during early fetal development.
      (2) VSD is the most commonly occurring form of CHD, with an incidence of 20% to 25% among neonates with cardiac defects. VSD frequently occurs in association with other congenital heart diseases.
   b. Assessment
      (1) Physical findings
         (a) Neonates with a small VSD usually show no signs other than a holosystolic murmur in the area of the left lower sternal border. The infant might have normal growth and development patterns.
         (b) An infant with a large VSD will also have a holosystolic murmur that is frequently accompanied by a thrill.
      (2) Diagnostic procedures
         (a) Chest radiograph film: might be normal with small VSD. If larger, will show cardiomegaly and increased vascular markings due to increased blood flow to the lungs as a result of the shunting from the left side of the heart to the right side.
(b) Electrocardiogram: left ventricular hypertrophy; right ventricular hypertrophy if pulmonary hypertension is present. Echocardiogram: direct visualization of left to right shunting.

c. Interventions (Sadowski, 2010)
(1) 50% to 75% of small lesions close spontaneously; 20% of large defects become smaller or close.
(2) With mild CHF, treatment consists of digoxin and diuretics.
(3) Surgery is indicated if patient has failure to thrive or intractable CHF.

4. Coarctation of the aorta
a. Introduction
(1) Coarctation of the aorta is a narrowing of the upper thoracic aorta that produces an obstruction to the flow of blood through the aorta.
(2) Simple coarctation presents with no other intracardiac lesions and can present with or without a PDA. Complex coarctation presents with other intracardiac lesions.
(3) Coarctation of the aorta accounts for 8% of all cases of congenital heart disease. About 30% of infants with Turner syndrome present with coarctation of the aorta (Lott, 2007).
(4) The coarctation can be juxtaductal, which is opposite to the location of the ductus arteriosus; preductal, which is proximal to the ductus arteriosus; or postductal, which is distal to the ductus arteriosus.

b. Assessment
(1) Physical findings depend on location of the coarctation.
(a) Diminished or absent femoral pulses
(b) Blood pressure more than 20 mm Hg; higher in upper extremities than in lower extremities (Wolfe, Boucek, Schaffer, & Wiggins, 1997)
(c) Blowing systolic murmur at the left upper sternal border and axilla
(d) Respiratory distress
(e) Pallor
(f) Poor weight gain during the first 2 to 6 weeks of life
(2) Diagnostic procedures
(a) Chest radiograph film: might show cardiomegaly with pulmonary venous congestion (Beekman, 2001)
(b) Electrocardiogram: might be normal. However, in symptomatic neonates, the electrocardiogram might show evidence of left ventricular hypertrophy.

c. Interventions (Sadowski, 2010)
(1) Aggressive medical management of CHF
(2) Prostaglandin E1 (PGE1) to dilate ductus arteriosus (preductal lesion)
(3) Palliative balloon angioplasty in critically ill neonate, followed by surgery
(4) Isolated postductal coarctation: control of CHF first, then delayed surgical correction
(5) Surgical correction

B. Cyanotic defects
1. Tetralogy of Fallot
a. Introduction. Tetralogy of Fallot (TOF) is the most common type of cyanotic heart lesion, accounting for 10% of all cases of congenital heart disease (Lott, 2007), characterized by a combination of four defects.
(1) Ventricular septal defect
(2) An overriding aorta
(3) Right ventricular outflow obstruction
(4) Hypertrophy of the right ventricle

b. Assessment
(1) Physical findings
(a) Respiratory distress, mainly tachypnea
(b) Cyanosis: Degree is directly related to the extent of right ventricular outflow obstruction.
   [i] If there is severe right ventricular outflow tract obstruction and the ductus is patent, the neonate might have minimal cyanosis.
Spontaneous closure of the ductus arteriosus results in a severe decrease in pulmonary blood flow and significant cyanosis.

Crying or feeding increases cyanosis and respiratory distress due to increased shunting of unoxygenated blood from the right side of the heart to the left as a result of pulmonary artery obstruction.

(c) Harsh systolic murmur located at the left upper and lower sternal borders (Siwik, Patel, & Zahka, 2001)
(d) Signs of congestive heart failure if the VSD is large
(e) As the ductus arteriosus closes, the infant might exhibit hypoxemia if there is significant pulmonary stenosis.

Diagnostic procedures
(a) Chest radiograph film: might be normal or decreased pulmonary vascular markings. Heart might have a boot-shaped appearance secondary to upturning of the apex related to right ventricular hypertrophy.
(b) Electrocardiogram: right atrial hypertrophy
(c) Echocardiogram: right ventricular wall thickening and visualization of the overriding aorta and the VSD

Interventions
(1) Current trend is complete repair of the TOF, which includes repair of pulmonary stenosis and closure of the VSD (Siwik et al, 2001).
(2) Palliative surgical interventions such as a Blalock-Taussig shunt and a central shunt can be used to control cyanosis. These shunts temporarily increase blood flow to the pulmonary artery from the aorta.

2. Transposition of the Great Arteries
   a. Introduction
      (1) Transposition of the great arteries (TGA) accounts for 5% of congenital heart disease (Lott, 2007).
      (2) The aorta originates from the right ventricle and the pulmonary artery originates from the left ventricle.
      (3) Survival is dependent on early diagnosis and aggressive treatment. An abnormal communication between the two separate circulations must be present or created if the infant is to survive. If the patent foramen ovale is closing, it should be reopened in the cardiac catheterization lab with a procedure called a balloon atrial septostomy. A PDA also maintains blood flow between the two independent circulations.
   b. Assessment
      (1) If untreated, the infant will become critically ill, including hypoxemia and heart failure, which may result in death.
      (2) As the communication between the independent circulations closes, the infant becomes prominently cyanotic.
      (3) Other symptoms might include progressive cyanosis that worsens with crying and feeding. Tachycardia, tachypnea, and a pansystolic murmur are present (Lott, 2007).
      (4) Diagnostic procedures
         (a) Chest radiograph may initially be normal in the neonate, but eventually shows an oval cardiac silhouette, mild cardiomegaly, and increased pulmonary vascular markings (Lott, 2007).
         (b) Electrocardiogram might show ventricular hypertrophy.
         (c) Echocardiogram is diagnostic and might show a small aorta and small left ventricle.
      c. Interventions
         (1) No supplemental oxygen to prevent closure of the PDA
         (2) Correction of acidosis, inotropic therapy, and PGE, via continuous intravenous infusion
         (3) Immediate transport to an appropriate facility with cardiac expertise. Surgically, a physiologic correction of the vessels, called an arterial switch, needs to be performed.

3. Hypoplastic left heart syndrome or single ventricle
   a. Introduction
Hypoplastic left heart syndrome includes various defects that are either valvular or vascular obstructive lesions on the left side of the heart that impede left-sided filling or emptying. As a result of the obstruction during intrauterine growth, a very small quantity of blood fills the left ventricle, causing hypoplasia of the left ventricle (Lott, 2007).

Mitral atresia or aortic atresia rapidly causes congestive heart failure, and death can occur within a couple of days when ductus arteriosus typically closes.

Infants with communicating atrial and VSDs might live longer, but require immediate surgical intervention.

Hypoplastic left heart syndrome accounts for 7% to 9% of all congenital heart defects (Lott, 2007).

These neonates are dependent on the PDA, and as it closes the neonate’s condition deteriorates rapidly, leading to death.

**Assessment**

1. Birthweight can be normal.
2. Neonate is commonly asymptomatic at birth but can become symptomatic within 1 to 2 days of life.
3. Respiratory distress includes tachypnea and dyspnea.
4. Diminished pulses, pallor, cyanosis, and mottling leading to vascular collapse and acidosis are seen as the PDA begins to close.
5. Most neonates have a soft, systolic, ejection murmur (Lott, 2007).

**Diagnostic procedures**

- Chest radiograph film: radiograph may be normal. Vascular markings are variable. Aortic arch to the right is common (Lott, 2007).
- Electrocardiogram: sinus tachycardia and right ventricular hypertrophy

**Interventions**

1. This lesion was once thought to be a lethal abnormality and inoperable, but now long-term, staged, palliative surgery is effective (Lott, 2007). Other options include cardiac transplantation.
2. The neonate requires immediate transfer to an appropriate pediatric facility with cardiac expertise.
3. Medical support might include inotropic therapy, PGE1 IV infusion to maintain a PDA, and hypoventilation (reduced oxygen concentration).
4. Maintenance of a delicate acid-base balance in the blood is essential for these fragile neonates.

**HEALTH EDUCATION**

**A. General parental and family adaptation to an ill newborn** (McGrath, 2007a)

1. Orient parents or family members to the nursery environment, including nursing, medical, and ancillary staff who will come into contact with their newborn; visiting and operational policies and procedures that will affect them; and telephone numbers to call to access care providers.
2. Help parents or family members recognize that grief is an appropriate response to the loss of the fantasized newborn.
   a. Discuss stages of grief.
   b. Teach that reactions to loss have individual and cultural differences.
   c. Be alert for inappropriate denial, which signals a dangerous lack of progress through the stages of grief.
3. Encourage parents or family members to express feelings and to deal openly with feelings of anger, fear, sadness, guilt, blame, frustration, and loss of self-esteem.
4. Encourage parents or family members not to become trapped in feelings of guilt, blame, or low self-esteem.
5. Remind parents or family members to support each other in their unique reactions to the situation.
6. Help parents or family members obtain accurate information about the infant’s treatment, care, prognosis, and outcomes.
7. Help mobilize support among the extended family, friends, and religious community.
   a. Provide information about local and national support groups.
   b. Refer the family to clergy and other social services as needed.
8. Promote attachment between parents or family members and infant.
9. Demonstrate safe methods of interaction and direct care that parents or family may provide.
10. Support and encourage the mother’s attempts to express breast milk, if she desires.
11. Help parents or family members interpret infant responses and recognize cues of satisfaction, overstimulation, and distress.
12. Teach parents to bathe, clothe, position, feed, and monitor the newborn according to individual needs, and support and encourage parents as they learn new skills.
13. Teach the parents or family members infant CPR.
14. Perform discharge planning and teaching from the time of admission.
   a. Offer parents a rooming-in option as available in anticipation of discharge.
   b. Assist parents or family members to plan for care to be provided in the home.
15. Follow up with telephone calls and home visits after discharge.

CASE STUDIES AND STUDY QUESTIONS

A 2300-g (5-pound, 1.5-ounce) infant girl is born to a 25-year-old gravida 1 woman, now para 1 (G1, P1) by spontaneous vaginal delivery. The infant’s length is 44 cm (17.5 inches), and her head circumference is 30.5 cm (12 inches). No abnormalities are noted on physical examination. Maternal history and a gestational age assessment reveal the neonate to be at approximately 38 weeks’ gestation and that she is SGA.

1. What might you expect to find in the mother’s history?
   a. Weight gain of 15.9 kg (35 pounds)
   b. A history of smoking one pack of cigarettes per day
   c. Documented class A diabetes

2. What implications do the infant’s measurements have?
   a. The insult occurred early in gestation, during the hyperplasia phase.
   b. The insult occurred late in gestation, during the hypertrophy phase.
   c. The insult occurred during labor and delivery.

3. To what should the nurse be alert when caring for this infant?
   a. Possible skull fracture
   b. Positive drug screen
   c. Hypothermia

A 4100-g (9-pound, 1.5-ounce) boy is born after a difficult forceps delivery. The prenatal history reveals an uncomplicated pregnancy of 40 weeks’ gestation. The infant’s length is 53.3 cm (21 inches), and his head circumference is 37 cm (14.6 inches). Gestational age assessment reveals the infant to be LGA.

4. On physical examination, what might you expect to find with this infant?
   a. Clubfoot
   b. Brachial plexus injury
   c. Diminished Babinski reflex

5. In actuality, the nurse finds the infant has a fractured clavicle, which is confirmed by x-ray. What may have led the nurse to this conclusion?
   a. Asymmetric startle reflex
   b. Extreme jitteriness
   c. History of forceps delivery

6. For what should the nurse be alert when caring for this infant?
   a. Hypothermia
   b. Respiratory distress
   c. Hypoglycemia

An 18-year-old G1, P1 woman is delivered of a 2000-g (4-pound, 6.5-ounce) boy by cesarean section. The infant is assessed to be AGA of 34 weeks. No abnormalities are noted on physical examination.

7. This infant is at risk for what condition?
   a. Hyperglycemia
   b. Premature closure of the ductus arteriosus
   c. Respiratory distress syndrome

8. What might the nurse expect to find in the mother’s history?
   a. Premature labor treated with tocolytics
   b. Gestational diabetes
   c. Exposure to rubella in the first trimester

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   b. Gestational diabetes
   c. Exposure to rubella in the first trimester

9. What might be detected in this infant during the first few days of life?
   a. Congenital syphilis
   b. Cephalohematoma
   c. Hyperbilirubinemia
A 32-year-old gravida 3, now para 3 (G3, P3) woman delivered a 3250-g (7-pound, 3-ounce) girl through meconium-stained fluid at 42 weeks' gestation. The infant’s initial presentation is that she is limp, cyanotic, has minimal respirations, and has a heart rate less than 100 bpm. She was intubated and suctioned by the neonatal team. Although she was suctioned through the endotracheal tube, no meconium was seen below the cords. With oxygen and stimulation, her Apgar scores at 1 and 5 minutes were 7 and 9, respectively.

10. What is the most serious consequence that might result from this delivery?
   a. Patent ductus arteriosus
   b. Meconium aspiration
   c. Hyaline membrane disease

11. What would the nurse expect to see on examination of this infant?
   a. Abundant lanugo
   b. Absence of sole creases
   c. Leathery, cracked, and wrinkled skin

12. What should the nurse do to protect this infant’s skin from further trauma?
   a. Avoid the use of tape except when absolutely necessary.
   b. Use powders and oils frequently.
   c. Wear gloves when handling the infant.

13. Why is hyperbilirubinemia of special concern in preterm infants?
   a. Immature liver function
   b. Poor vascular system
   c. Immature endocrine function

14. What physiologic factor contributes to greater risk for alterations in skin integrity in preterm infants?
   a. Immature immunologic system
   b. Malfunctioning of regulatory organs, such as the kidneys and respiratory tract
   c. Decreased cohesion between the dermis and epidermis

15. Gavage feedings are frequently needed to meet the nutritional needs of preterm infants because:
   a. Lactose enzyme activity is not adequate.
   b. Suck, swallow, and breathing reflexes are uncoordinated.
   c. Hyperbilirubinemia is likely.

16. A 42-week postterm neonate was born with greenish discoloration of the nails and skin and greenish secretions in the nasal passages. Why might the infant be transferred to a level 3 nursery?
   a. To determine the reason for the postmaturity
   b. To observe more closely for skin color changes
   c. To manage severe respiratory problems that develop

17. Factors that contribute to impaired fetal growth include:
   a. Class A and C maternal diabetes
   b. Grade III placenta
   c. Multiple births

18. What problem may the LGA infant experience?
   a. PDA
   b. Facial nerve damage
   c. Poor suck, swallowing, and breathing coordination

Ms. J., a gravida 2, para 1 (G2, P1) woman, delivered an infant boy vaginally at 35 weeks’ gestation. The infant weighed 2500 g (5 pounds, 8 ounces); the fetal heart rate appeared fine during labor, although an L/S ratio was reported at 1.8:1. Ms. J. has gestational diabetes.

20. Surfactant production for an infant with RDS is inhibited because of:
   a. Prematurity
   b. Infection
   c. Hypoglycemia

21. The best indicator of an infant’s need for oxygen is:
   a. Respiratory rate
   b. Skin color
   c. Arterial PaO₂

22. What characteristic of neonates with RDS?
   a. Have a deficiency of pulmonary surfactant
   b. Are postmature
   c. Have sternal excursions

23. Which statement is NOT true about RDS?
   a. It is characterized by atelectasis.
   b. It may be induced by hypothermia in a preterm infant.
   c. With adequate supportive care, it is self-resolving in approximately 72 hours.

24. In RDS, blood may not be well oxygenated because of all of the following except:
   a. A patent ductus arteriosus
   b. Decreased pulmonary resistance
   c. Atelectasis of the alveoli
25. Which prenatal factor is most likely to predispose the neonate to develop respiratory distress?
   a. Maternal diabetes
   b. Gestation of 34 weeks
   c. Fetal scalp pH of 7.20

26. True or False: Acidosis and hypothermia may lead to decreased pulmonary blood flow, which perpetuates decreased production of surfactant and may cause RDS.

27. A condition that usually occurs in term infants and cesarean section deliveries, is manifested by tachypnea, and is caused by retained lung fluid is called:
   a. Pneumonia
   b. TTN
   c. Meconium aspiration

28. When suctioning a limp neonate born through meconium-stained amniotic fluid, what is suctioned first?
   a. Both nares
   b. Stomach
   c. Trachea

29. An infant with PPHN and a PDA is at risk for developing:
   a. Pulmonary hemorrhage
   b. Hepatomegaly
   c. Pleural effusions

30. You are preparing to care for several infants recently born. Which infant is at greatest risk for TTN?
   a. Spontaneous vaginal delivery; 40 weeks’ gestation
   b. Cesarean section delivery; 41 weeks’ gestation
   c. Vaginal delivery with maternal anesthesia; 38 weeks’ gestation

Baby M. was delivered after a 16-hour induced labor. Maternal membranes were artificially ruptured, fluid was clear, and a Pitocin infusion was initiated. The mother was afebrile throughout the labor. The second stage of labor was 2 hours, 45 minutes. Review of the mother’s prenatal and labor history yielded the following information:

   Blood type is A+.
   Venereal disease research laboratory (VDRL) is nonreactive.
   Alpha-fetoprotein is normal.
   Average BP is 116-124/76-82.
   Total weight gain was 12.25 kg (27 pounds).
   Medications are prenatal vitamins, iron, and aspirin for stress headaches.
   Gestational age is 40 6/7 weeks.
   Nonstress test is reactive.

Baby M. had the umbilical cord wrapped twice around her neck and required stimulation to initiate breathing and administration of oxygen by face mask. Apgar scores were 7 at 1 minute and 8 at 5 minutes.

31. From this information, which factor places Baby M. at increased risk for hyperbilirubinemia?
   a. Ruptured membranes for 16 hours
   b. Postmaturity
   c. Labor induced with Pitocin

32. All of the following factors indicate that Baby M. is at greater risk for hyperbilirubinemia except:
   a. Gestational age of 40 6/7 weeks
   b. Mother taking aspirin for her headaches
   c. Respirations having to be stimulated and oxygen administered

On her second day of life, Baby M. required phototherapy treatment. Her mother was being discharged from the hospital and came to the nursery to breastfeed her infant before leaving. Baby M.’s mother was crying and did not want to go home without her infant. Baby M.’s father was trying to comfort his wife.

33. While Baby M. is under the phototherapy lights, it is important to:
   a. Keep the infant under the lights at all times so that there will be maximal effectiveness in the shortest period.
   b. Discontinue Baby M.’s breastfeeding because the fluid content of breast milk is deficient for a neonate undergoing phototherapy.
   c. Prevent hypothermia, hyperthermia, or both in Baby M.

34. Baby M.’s mother is crying, expresses fear about her infant’s health, and does not want to leave. Which intervention would be the least effective?
   a. Encourage the mother to come in to feed her infant as often as possible.
   b. Emphasize the temporary nature of hyperbilirubinemia, and explain the monitoring of Baby M.’s bilirubin levels.
   c. Remind the mother that newborns require demanding care, which is very fatiguing to a new mother, and that she should take this added opportunity to rest and recover.

Baby L. was born to a 21-year-old gravida 1, para 1 (G1, P1) Vietnamese mother. He was born vaginally after a difficult delivery due to shoulder dystocia. At admission, his weight was 4400 g
(9 pounds, 11.5 ounces). Physical and neurologic examination placed him at 40 weeks of gestation. His physical examination revealed an unequal Moro reflex with decreased movement of the left arm and crepitus at the left neck area. Bluish marking was also noted across the lower back.

35. What is the correct gestational classification for Baby L.?
   a. LGA with risk for hypoglycemia
   b. AGA with risk for hypothermia
   c. SGA with risk for hypoglycemia

36. The historical and physical findings for Baby L. might suggest:
   a. Torticollis
   b. Fractured clavicle
   c. Erb’s palsy

37. What is the most likely cause of the bluish marking across the lower back?
   a. Purpura
   b. Birth trauma
   c. Mongolian spot

Baby R. is a 6-hour-old female born to a 28-year-old mother with a history of pregnancy-induced hypertension. Baby R. was born 5 weeks premature and has a birthweight of 1450 g (3 pounds, 3 ounces). On admission to the newborn nursery, the infant’s respiratory rate was 74, and she was noted to have mild intercostal and substernal retractions with adequate air entry on auscultation. Auscultation of her heart revealed a harsh murmur that was best heard in the area of the left upper sternal border.

38. Baby R.’s history and presentation are most indicative of:
   a. Coarctation of the aorta
   b. Atrial septal defect
   c. Patent ductus arteriosus

39. All of the following statements regarding gastroschisis are true except:
   a. There is an increased incidence of preterm birth associated with gastroschisis.
   b. There is no skin covering the eviscerated organs.
   c. Intestinal atresias are frequently associated with gastroschisis.

40. A term infant develops severe respiratory distress immediately after birth. On physical examination, the chest is hyperexpanded, and the point of maximal impulse (PMI) is shifted to the right. What is the most likely cause for this infant’s respiratory distress?
   a. Diaphragmatic hernia
   b. Right pneumothorax
   c. Transposition of the great arteries

ANSWERS TO STUDY QUESTIONS

1. b
2. a
3. c
4. b
5. a
6. c
7. c
8. a
9. c
10. b
11. c
12. a
13. a
14. c
15. b
16. c
17. c
18. b
19. c
20. a
21. c
22. a
23. c
24. b
25. b
26. True
27. b
28. c
29. a
30. b
31. c
32. a
33. c
34. c
35. a
36. a
37. c
38. a
39. c
40. a

REFERENCES


