Chapter 108: Resuscitation of Neonates

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FIGURE 108-1.

INTRODUCTION AND EPIDEMIOLOGY

Resuscitation of the newborn is required to some extent in nearly 10% of all births. Extensive resuscitation is required in about 1%. Delivery room resuscitation is required for >50% of the high-risk population of very-low-birth-weight (<1500 grams) newborns. Worldwide, nearly 25% of neonatal deaths result from birth asphyxia.[1] With proper antenatal and intrapartum surveillance, the potential need for active resuscitation at birth can often be identified before birth. Unfortunately, the arrival of a newborn to the ED is never planned. This chapter reviews the principles of emergency resuscitation of neonates.

PATHOPHYSIOLOGY

The transition from intrauterine to extrauterine life is a treacherous time. Even the normal laboring process places significant stress on the placental-fetal unit. Blood flow and, therefore, oxygen delivery are transiently impaired during uterine contractions. Compression of the umbilical cord, when it occurs, further impairs circulatory flow. Although antenatal/intrapartum US imaging and fetal heart tone monitoring have permitted better surveillance of fetal well-being, prediction of fetal status at birth remains inexact. Maternal complications of pregnancy can predispose newborns to complications and include infections, chronic or gestational disease (e.g., diabetes, lupus), and illicit or prescribed medication use. Complications of labor, such as preterm delivery and/or prolonged rupture of membranes, maternal fever, breech or transverse fetal position, placental abruption, and umbilical cord problems such as a nuchal cord (cord wrapped around the neck) or true knots in the cord, can significantly heighten the risk to the fetus.

Once delivery occurs, the newborn still faces a variety of risks as the transition to extrauterine life unfolds. Requirements of this transition include the onset of respiration, absorption of lung fluid, reduction of pulmonary vasculature resistance to allow flow to the pulmonary vascular circuit, and closure of the ductus arteriosus and foramen ovale. Premature infants and infants who are small for gestational age are at risk for additional challenges in transitioning from fetal to infant physiology including insufficient pulmonary surfactant, fragile germinal matrices within the cerebral ventricles, and thin skin that impairs thermoregulation. The transition from the sterile intrauterine environment to the extrauterine world teeming with bacteria places yet another burden on the newborn.

CLINICAL FEATURES

HISTORY

Obtain a brief history from the mother, including the date of last menstrual period/estimation of gestational age, number of fetuses, number of previous pregnancies and living children, history of diabetes, hypertension or pregnancy-related problems, prenatal care (including known congenital anomalies), prolonged rupture of membranes, fever, and meconium-stained amniotic fluid.

PHYSICAL EXAMINATION

The need for resuscitation or routine newborn care (see "Treatment" below) is determined by the initial physical examination. For the term infant who is crying or breathing and who has good muscle tone at delivery, provide routine newborn care with the infant skin-to-skin on the mother. A slightly more detailed examination using the Apgar scoring system has been used for generations to assist medical personnel in assessing newborns both for the need for resuscitation and the response to resuscitation. Evaluate the newborn at 1 and 5 minutes after delivery for ***heart rate*** (absent = 0, <100/min = 1, >100/min = 2), ***respiratory effort*** (absent = 0, weak = 1, crying or normal = 2), ***muscle tone*** (limp = 0; some flexion = 1; active, fully flexed = 2), ***reflex irritability*** (no response = 0, grimace = 1, crying or active = 2), and ***color*** (blue or pale = 0, acrocyanosis = 1, completely pink = 2). If the 5-minute Apgar score is <7, continue Apgar scoring at 5-minute intervals until a score of 7 or more is reached. The expanded Apgar scoring system includes a section to document resuscitative measures.[2] For infants requiring resuscitation, monitor pulse oximetry with the probe placed on the newborn's right hand (preductal) (see targets for resuscitation below).

LABORATORY EVALUATION

Routine laboratory studies are not required for most term or preterm deliveries. However, obtain point-of-care glucose testing in infants born to diabetic mothers, infants who are small or large for gestational age, or depressed or irritable infants, or if there is poor response to the initial steps of resuscitation. Obtain a type and screen or crossmatch of blood in infants requiring significant resuscitation in the setting of suspected blood loss.

IMAGING

Routine imaging is not required for most deliveries. In rare circumstances, an x-ray of the chest and abdomen may be useful to confirm endotracheal tube placement, suspected pneumothorax, and some congenital defects (e.g., diaphragmatic hernia).

PREPARATION AND EQUIPMENT

EQUIPMENT

Table 108-1 lists equipment that may be needed during neonatal resuscitation. A compressed air source, an oxygen blender with flow meter, pulse oximetry for neonatal use, and laryngeal mask airways are standard resuscitation equipment.

TABLE 108-1

Neonatal Resuscitation Equipment

Radiant warmer with servocontrol temperature sensor
Prewarmed towels/receiving blankets
Wall suction, suction catheters, bulb syringes
Heated, humidified oxygen source
Compressed air source and oxygen blender
Cardiorespiratory monitor/monitor leads
Pulse oximeter
Bag (flow-inflating or self-inflating) *with* manometer
Masks (sizes 1, 2, 3, 4)
Laryngoscope (0, 1 blade)
Endotracheal tubes (2.5, 3.0, 3.5, 4.0)
Meconium aspirator
CO_2 detector
Nasogastric tubes (5F, 8F)
IV infusion equipment
IV fluids (10% dextrose in water, normal saline)
Umbilical catheter tray
Curved hemostat
Two iris curved forceps, no teeth

Scalpel handle/blade

Needle holder

Scissors

Syringes

2 × 2 gauze sponges

3.5F, 5F umbilical catheters

Three-way stopcock

Suture material

Umbilical tape

Povidone-iodine solution

UMBILICAL CORD CLAMPING

Do not clamp the umbilical cord of newborns (term or preterm) who do not require positive-pressure ventilation or immediate resuscitation for at least 1 to 3 minutes after birth. Delayed cord clamping reduces the need for blood transfusion, increases neonatal iron stores, and may decrease the risk of requiring treatment for hyperbilirubinemia.[1] For newborns requiring positive-pressure ventilation, the cord may be clamped and cut to allow effective ventilations to be performed.

ROUTINE NEWBORN CARE

Provide routine newborn care to term infants who are breathing or crying with good tone. Leave the newborn with the mother, provide warmth (skin-to-skin or blankets), clear the nose and mouth with bulb suction only if signs of obstructed breathing are noted, dry the baby, and provide ongoing assessment of respiratory effort and tone. Even before initiation of the ABCs (airway, breathing, circulation) of resuscitation, provide a neutral thermal environment for the newborn. Although vigorous term infants may be placed skin-to-skin with their mother for warmth, preterm or depressed newborns should be placed under a preheated radiant heat source. Place the infant on his or her back in the warmer. Then, gently dry the newborn with a warm towel while preparing to initiate resuscitation. Very-low-birth-weight newborns and those <29 weeks of estimated gestational age should be placed in polyethylene bags that have been developed for that purpose (plastic food wrap or a food-grade 1-gallon plastic bag may also be used). Avoid hyperthermia, which may precipitate apnea and worsen hypoxic-ischemic injury.

RESUSCITATION

Newborn resuscitation almost exclusively involves care of primary respiratory compromise (Table 108-2). Consensus guidelines[3] recommend a timed sequence of steps (30, 60, and >60 seconds). Most important is the rapid establishment of effective ventilation and determining the heart rate before initiating CPR.[4,5]

TABLE 108-2

Steps in Neonatal Resuscitation

Newborn Appearance	Management	Comments
Infant breathing, crying, good tone	Routine care: warm, dry, delay cord clamping 1–3 min, observe	Vigorous term babies may be warmed skin-to-skin with mother. Stimulate nonvigorous babies after drying by rubbing back vigorously several times.
Poor tone/respiratory effort *or* respiratory distress	Warm, open airway and clear nose and mouth if obstructed, dry, stimulate	
Labored breathing or persistent cyanosis with HR >100 beats/min	Clear nose and mouth, monitor O_2 saturation; provide O_2 only to maintain levels in Table 108-3. Consider CPAP.	Oxygen monitor should be placed on right upper extremity (preductal).

Newborn Appearance	Management	Comments
Apnea, gasping, or HR <100 beats/min	PPV Continue PPV for 30 s, taking corrective steps for ventilation if no improvement in HR	Provide PPV with BVM at a rate of 40–60 breaths/min using room air. Provide 30 cm H_2O pressure for term infants and 20–25 for preterm infants.
HR <60 beats/min	Initiate CPR:3:1 compression-to-ventilation ratio 90:30 compressions and ventilations per minute	Use thumb-encircling technique to provide chest compressions to lower one third of sternum.
HR <60 beats/min after appropriate ventilation and CPR	Administer epinephrine	May be given IO, IV, or through a UV or ETT
	Consider volume expansion if blood loss; treat hypoglycemia	

Abbreviations: BVM = bag-valve mask; CPAP = continuous positive airway pressure; ETT = endotracheal tube; HR = heart rate; IO = intraosseous; O_2 = oxygen; PPV = positive-pressure ventilation; UV = umbilical vein.

INITIAL STEPS (FIRST 30 SECONDS)

Within the first 30 seconds of birth, provide warmth, and dry and stimulate the baby. Current guidelines[3] no longer advise the routine suctioning of the newborn nose and mouth. **Infants who are spontaneously breathing, whether delivered through clear or meconium-stained amniotic fluid, do not require tracheal suctioning because tracheal suctioning can cause reflex bradycardia and apnea.** If the infant is not breathing initially, dry and provide stimulation by rubbing the back two to three times; if there is no response, open the airway using jaw thrust and towels beneath the shoulders to provide a sniffing position. If there appears to be obstruction from amniotic fluid, gently suction the nose and throat with a bulb or 8F catheter.

After these initial steps, assess the respiratory effort and heart rate.

ONGOING RESUSCITATION (30 TO 60 SECONDS)

After warming, drying, and stimulating, reassess the respiratory effort and the heart rate. If the infant begins breathing without significant effort and with good color, return to the mother for routine care. If the heart rate is >100 beats/min but there is persistent cyanosis or labored breathing, open the airway and suction the nose and mouth if there is a visible obstruction; attach pulse oximetry to the right hand or wrist

(preductal) and apply supplemental oxygen to achieve targeted preductal oxygen saturation goals as per Table 108-3.

TABLE 108-3

Targeted Pulse Oxygen Levels During Newborn Resuscitation

Time After Birth	Target Oxygen Saturation (preductal)
1 min	60%–65%
2 min	65%–70%
3 min	70%–75%
4 min	75%–80%
5 min	80%–85%
10 min	85%–90%

Apneic or depressed newborns delivered through meconium are at risk for meconium aspiration syndrome, but current evidence indicates that tracheal suctioning does not reduce morbidity or mortality.[3]

Naloxone is not recommended for treatment of neonatal respiratory depression, even after maternal opioid exposure or use.[3] Provide usual respiratory support and ventilation.

Initiate positive-pressure ventilation using a bag and mask for infants with a heart rate of <100 beats/min or who are gasping or remain apneic after the initial steps of newborn resuscitation. Begin resuscitation using room air because newborn blood oxygen levels, even in healthy newborns, take time to reach extrauterine values and excessive oxygenation is associated with increased mortality.[6] Table 108-3 provides subsequent oxygen saturation goals throughout neonatal resuscitation.

POSITIVE-PRESSURE VENTILATION

Provide positive-pressure ventilation with a self-inflating or flow-inflating infant bag or a T-piece resuscitator for all newborns with an HR <100 beats/min or who are gasping or apneic after 30 seconds. Bradycardia, even extreme, is typically the result of respiratory failure, and chest compressions or medications should not be initiated until effective ventilations have been provided. Administer positive-pressure ventilation if available. Use a manometer to monitor peak inspiratory pressures: a peak inspiratory pressure of 20 cm H_2O is usually sufficient, although initial peak inspiratory pressures as high as 30 to 40 cm H_2O may be required. Generally, flow-inflating bags are preferred, because they allow better control of inflation pressures. Self-inflating bags are superior if supplemental air or oxygen is unavailable. Be careful when using a self-inflating bag, because pop-off valve pressures, usually set at 30 to 40 cm H_2O, can be exceeded if excessive pressure is applied. T-piece resuscitators have the advantage of delivering a consistent pressure with each artificial breath. **Excessive inflation pressures can cause pneumothorax and**

compromise resuscitation. Provide 40 to 60 breaths/min. Good chest rise and an increase in heart rate (usually within 5 to 10 breaths) are the best indicators of effective ventilation.

Most infants will respond to initial positive-pressure ventilation as outlined above. The most likely reason for a poor response to positive-pressure ventilation is inadequate positive-pressure ventilation, and corrective steps should be taken to assure effective ventilation prior to further resuscitation measures. The American Heart Association recommends use of the pneumonic "MR SOPA," which stands for **M**ask (adjust to improve the seal), **R**eposition the head to open the airway, **S**uction the mouth then nose, **O**pen the mouth with a jaw thrust, and increase the **P**ressure until chest rise is noted (maximum peak inspiratory pressure 40 cm H_2O), and if none of these is effective, proceed to definitive **A**irway control (endotracheal intubation).

Infants with significant labored breathing may benefit from **continuous positive airway pressure ventilation** if the necessary equipment and expertise are available.

ENDOTRACHEAL INTUBATION

In the absence of improvement with bag-mask ventilation, endotracheal tube insertion and ventilation are indicated. Other potential indications for endotracheal intubation in the newborn include (1) concomitant need for chest compressions, (2) administration of endotracheal medications, (3) known or suspected congenital diaphragmatic hernia (to avoid inflating stomach/bowel situated in the chest), and (4) extremely low birth weight (<1000 grams).[7]

The technique for endotracheal intubation is discussed in detail in chapter 111, Intubation and Ventilation in Infants and Children. Tables 108-4 and 106-5 provide recommended equipment sizes for neonatal resuscitation. **If time allows, precut the endotracheal tube proximally at the 13-cm mark at the endotracheal tube adapter site.** This may eliminate the need to do so later, after the newborn has already been intubated. Uncut tubes allow for excessive "dead space" and are prone to kinking. A stylet is not essential but may allow for easier intubation by providing both greater rigidity and better concave curvature to the relatively soft endotracheal tube.

TABLE 108-4

Selection of Endotracheal Tube Size

Tube Size (mm)	Weight (grams)	Gestational Age (wk)
2.5	<1000	<28
3.0	1000–2000	28–34
3.5	2000–3000	34–38
4.0	>3000	>38

Confirm tube placement by direct visualization, observation, and auscultation of bilateral chest rise and breath sounds and by confirmation of end-tidal carbon dioxide detection. A rule of thumb for proper tube insertion depth is **6 + weight in kg at the lips** (e.g., 3-kg infant would be 6 + 3 = 9 cm at the lip).

ADVANCED RESUSCITATION: CIRCULATION (>60 TO 90 SECONDS)

If, despite assisted ventilation for 30 seconds, the newborn remains severely bradycardic with an HR <60 beats/min, start chest compressions. Deliver chest compressions to the lower one third of the sternum to a depth of about one third of the anteroposterior diameter of the chest. The compression phase should be slightly shorter than the relaxation phase to allow for cardiac filling. Avoid simultaneous compressions and ventilation. Deliver chest compressions and ventilations in a ratio of **three chest compressions to one breath for** a total of **90 compressions and 30 breaths/min**.

There are two techniques to perform chest compressions.[7] In the "two-thumb" technique, the chest is compressed with both thumbs with the fingers encircling the newborn's back. In the "two-finger" technique, the operator's second and third digits compress the lower one third of the sternum, often with the other hand supporting the newborn's back. The "two-thumb" technique seems to be superior in generating greater peak systolic pressures. The "two-finger" technique may be more practical if a colleague is simultaneously attempting umbilical vessel catheterization.

Stop chest compressions when the HR exceeds 60 beats/min. Once chest compressions have been discontinued, increase the ventilation rate to 40 to 60 breaths/min, because interference from the chest

compressions is no longer an issue. Slowly wean positive-pressure ventilation when the HR exceeds 100 beats/min and the newborn has begun to breathe spontaneously.

Medications and Volume Expansion

If bradycardia continues despite bag-mask ventilation followed by endotracheal intubation, adequate ventilation with 100% oxygen, and chest compressions for 45 to 60 seconds, then give epinephrine. Epinephrine is the primary drug used for neonatal resuscitation and should be administered IV or IO, although it can be given intracheally (use larger dose below) if vascular access cannot be obtained (see "Vascular Access" below for umbilical venous catheter placement). The dose of epinephrine is 0.01 to 0.03 milligram/kg IV/IO (0.1 to 0.3 mL/kg of 1:10,000 solution). Intratracheal dosing is 0.05 to 0.1 milligram/kg or 0.5 to 1 mL/kg of 1:10,000. Naloxone and sodium bicarbonate are no longer recommended for routine use in neonatal resuscitation. **Naloxone is contraindicated in the newborn when maternal narcotic addiction is suspected, because neonatal seizures may result. Sodium bicarbonate may worsen intracellular acidosis.**[8,9]

Consider **volume expansion** when there is known or suspected blood loss (pallor, poor perfusion, or weak pulses). Administer 10 mL/kg of 0.9% saline solution (normal saline) or O-negative blood. Volume should be given slowly (3 to 5 minutes), especially to premature infants who are at risk for intraventricular hemorrhage.

TABLE 108-5

Selection of Laryngoscopy Equipment

Tube Size/Gestational Age (wk)	Laryngoscope Blade Size	Suction Catheter Size
2.5/<28	Miller 0	5F or 6F
3.0/28–34	Miller 0	6F or 8F
3.5/34–38	Miller 0	8F
3.5–4.0/>38	Miller 0–1	8F or 10F

Hypoglycemia in neonates is associated with adverse outcomes following birth asphyxia, whereas hyperglycemia is not. Although there is no evidence-based target for serum glucose in newborns, administer 2 mL/kg of 10% dextrose in water IV/IO for glucose <25 milligrams/dL (1.38 mmol/L) in the first few hours of life (see "Hypoglycemia," under "Special Problems in the Newborn," below).

Postresuscitation Care

Newborns with any degree of asphyxia, even those who respond to resuscitative efforts, require a period of close observation. Transfer or admission to a special care nursery or neonatal intensive care unit will

depend on the degree of resuscitation required.

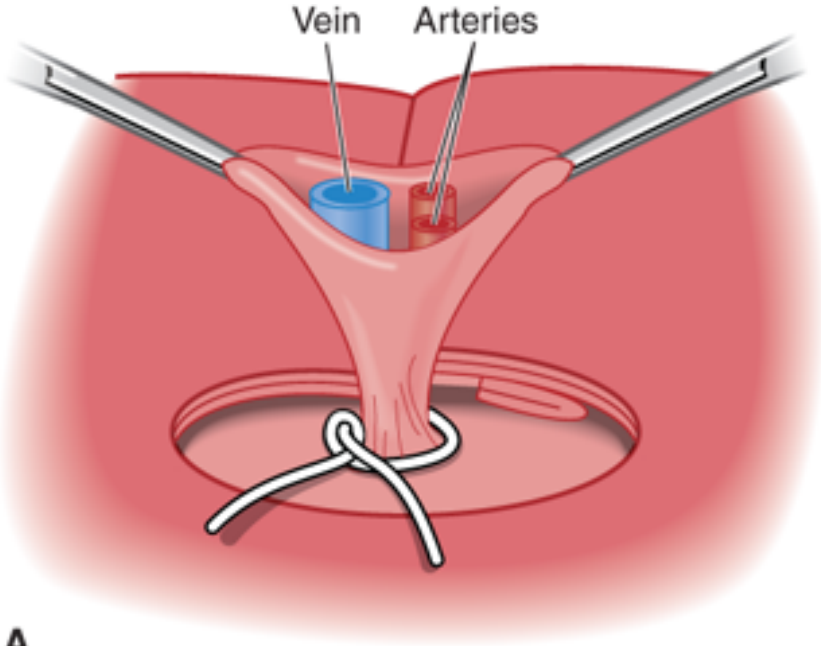
Several large trials have demonstrated significant decrease in mortality and improved 18-month neurologic outcomes among term (≥36 weeks of gestational age) newborns with moderate to severe hypoxic-ischemic encephalopathy treated with hypothermia.[7,10,11,12] Consider inducing hypothermia between 33.5°C and 34.5°C for term neonates requiring extensive resuscitative care,[13] and obtain emergency neonatologist consultation whenever possible.

VASCULAR ACCESS

Peripheral venous access is often difficult in the newborn, so intraosseous access is a good alternative (see "Vascular and Intraosseous Access"). The most readily available site for venous access in the newborn is the umbilical vein. The umbilical vein is easily differentiated from the two umbilical arteries by its larger orifice and thin wall versus the smaller, thicker-walled arteries (**Figure 108-1**).

FIGURE 108-1.

A. Inserting an umbilical catheter. **B**. Correct location of umbilical venous catheterization. When the results of the x-ray are known, reposition the catheter if necessary.



A

Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
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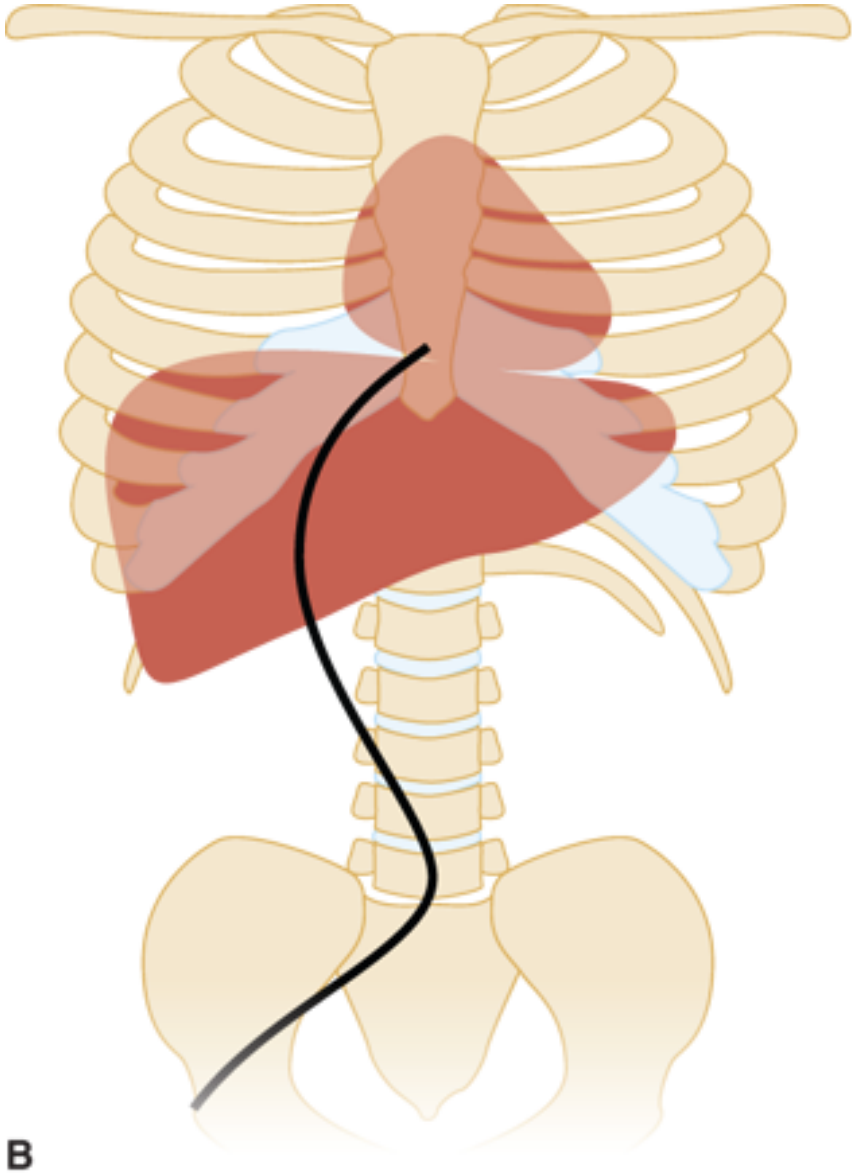

B

Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
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PREPARATION

Using sterile technique, snugly tie the umbilical cord at its base. The tie can be tightened as needed to avoid oozing through the umbilical vessels once the cord has been cut with a scalpel. Then, cut the cord below the umbilical clamp that was placed at the time of birth, leaving a residual umbilical stump of 1 to 2 cm.[14]

TECHNIQUE

Flush a 3.5F or 5F umbilical catheter with normal saline. Next, attach the preflushed catheter to a 3-mL syringe with a three-way stopcock. Advance the catheter into the umbilical vein until a free flow of blood is seen—this site will be below the level of the liver. At this point, the catheter can be used for volume expansion and medication administration.

Ultimately, the ideal position for the umbilical venous catheter is in the inferior vena cava above the liver and diaphragm, but below the heart, at the T7 to T8 level (Figure 108-1). To achieve this position, either measure the length from the xiphoid to the umbilicus and add 1 to 2 cm, *or* measure total body length, divide by 6, and add 1 to 2 cm. Positioning of the catheter can be confirmed with an x-ray after the patient has been resuscitated.

If placing an umbilical arterial catheter for monitoring purposes, the ideal final catheter positions are either between T6 and T10 (high line) or L2 and L4 (low line). To place a high line, measure total body length (cm), divide by 3, and add 1 to 2 cm *or* measure the shoulder to umbilicus length (cm) and add 1 to 2 cm.

WITHHOLDING AND DISCONTINUING NEONATAL RESUSCITATION

The ED is typically the site of precipitous, unplanned deliveries. If there is uncertainty about gestational age, infant weight, or viability, it is best to err on the side of resuscitation.

A fetus at <22 weeks of gestation and weighing <400 grams is not viable. At 22 weeks of gestation, survival is about 10%; at 23 weeks of gestation, survival is 35% to 40%; and at 24 weeks of gestation, survival is 60% to 65%.[19] Resuscitation should be initiated on newborns who are 23 weeks of gestation or older. Some centers are now routinely attempting resuscitation of newborns beginning at 22 weeks of gestation. It is difficult to identify with precision the week of gestation at borderline viability based exclusively on anatomic features.

DISCONTINUING NEONATAL RESUSCITATION

Newborns with no sign of life after 10 minutes of continuous and active resuscitation are virtually certain to suffer severe morbidity and/or mortality if continued resuscitation is successful in restoring vital signs. **Therefore, it is justified to cease resuscitative efforts after 10 minutes and, certainly, after 15 minutes of asystole.[7]**

SPECIAL PROBLEMS IN THE NEWBORN

NEONATAL CYANOSIS

Cyanosis is a common finding in the newborn. The first step is differentiating central cyanosis from peripheral cyanosis.

Clinical Features

Peripheral cyanosis, or acrocyanosis, is a normal finding in the first few days of life secondary to vasomotor instability and requires no specific evaluation or intervention. Central cyanosis is cyanosis involving the mucous membranes/lips, tongue, and skin. It indicates the presence of at least 4 to 5 grams/dL of unsaturated hemoglobin. The very anemic newborn may present as pale but not cyanotic if the level of unsaturated hemoglobin is below the 4 to 5 grams/dL threshold.

The breathing pattern often provides valuable clues to the cause of cyanosis (Table 108-6). On auscultation, unilaterally decreased breath sounds with retractions are associated with a pneumothorax or a space-occupying lesion of the chest. Bilaterally decreased breath sounds with retractions may suggest upper airway obstruction. Stridor also suggests an upper airway cause. Rales and rhonchi may be heard with pneumonia, respiratory distress syndrome, or meconium aspiration syndrome.

TABLE 108-6

Causes of Neonatal Central Cyanosis

Airway Obstruction (retractions and/or grunting respirations; stridor)	Cardiac Disorders (tachypnea*, no grunting or retractions)	Pulmonary Disorders (tachypnea* with grunting and/or retractions; rales or rhonchi)	CNS and Metabolic Disorders (slow, shallow respirations without retractions)
Choanal atresia	Transposition of great arteries	Respiratory distress syndrome	Intracranial hemorrhage
Laryngeal web/cyst	Tricuspid atresia	Meconium aspiration syndrome	Brain anomalies (Dandy-Walker malformation, congenital hydrocephalus)
Tracheal stenosis	Truncus arteriosus	Pneumonia	Central hypoventilation syndrome
Pierre Robin sequence	Total anomalous pulmonary venous return	Congenital diaphragmatic hernia	Polycythemia

Airway Obstruction (retractions and/or grunting respirations; stridor)	Cardiac Disorders (tachypnea*, no grunting or retractions)	Pulmonary Disorders (tachypnea* with grunting and/or retractions; rales or rhonchi)	CNS and Metabolic Disorders (slow, shallow respirations without retractions)
Cystic hygroma/goiter	Pulmonary atresia	Pulmonary hypoplasia	Hypoglycemia
	Coarctation of aorta	Congenital lobar emphysema	Sepsis/shock
	Hypoplastic left heart syndrome	Congenital cystic adenomatoid malformation	Methemoglobinemia
	Primary pulmonary hypertension of the newborn		

*Tachypnea defined as >60 breaths/min.

A thorough knowledge of the differential diagnosis for cyanosis allows for quick, organized assessment, diagnosis, and treatment (Table 108-6).

Obtain simultaneous preductal (e.g., right radial) and postductal (e.g., lower extremity) or arterial blood gases to help diagnose persistent pulmonary hypertension of the newborn: the postductal Pa_{O_2} is significantly lower than the preductal Pa_{O_2}. Using pre- and postductal pulse oximetry may serve the same purpose.

A discrepancy between upper and lower limb blood pressures or reduced femoral pulses may suggest coarctation of the aorta. Babies with coarctation of the aorta often develop new-onset tachypnea and absent femoral pulses later in the first day of life or into the second day of life. Femoral pulses are palpable at birth but disappear after the ductus arteriosus has closed with coarctation of the aorta.

Patients with hypoplastic left heart syndrome may present with poor pulses in all four limbs, poor perfusion, and tachypnea after the immediate newborn period once the ductus arteriosus has closed (see chapter 126, "Congenital and Acquired Pediatric Heart Disease").

Stepped Evaluation and Treatment

See **Table 108-7**.

TABLE 108-7

Steps to Evaluate and Treat Neonatal Central Cyanosis

1. Identify breathing pattern to characterize infant into airway obstruction, cardiac pulmonary, or CNS/metabolic pattern.
2. Obtain peripheral oxygen saturation pre- and postductal, check pulses in all 4 extremities, and obtain ABG.
3. Perform hyperoxia test. Deliver 100% oxygen for 5–10 min. Congenital cyanotic heart disease cannot increase oxygen saturation >20% or raise Pa_{O_2} to 100 mm Hg.
4. Obtain stat chest x-ray to identify pneumothorax, congenital diaphragmatic hernia, or pulmonary infiltrates, and assess cardiac size and shape and pulmonary vasculature for clues to congenital heart disease.
5. Establish vascular access in umbilical or peripheral vein, obtain POC glucose, CBC, and metabolic panel. Treat hypoglycemia (glucose <25 milligrams/dL [1.38 mmol/L]) with 10% dextrose in water, 2 mL/kg IV bolus, or 3.3 mL/kg/h (see "Hypoglycemia" section below).
6. Institute continuous positive airway pressure or intubate and ventilate if O_2 saturation does not improve with standard methods for oxygen delivery.
7. If oxygenation still does not improve, treat as presumptive congenital cardiac disease with prostaglandin E_1 starting at 0.05 microgram/kg/min and titrate to the minimum effective dose.

Abbreviations: ABG, arterial blood gas; POC, point of care.

Evaluation

The **hyperoxia test** is a quick method to help differentiate a cardiac from a noncardiac cause for cyanosis. Place the newborn in a 100% hood for 5 to 10 minutes. Cyanotic newborns with a pulmonary disorder can increase their oxygen saturation >20% and their Pa_{O_2} to >100 mm Hg. Those with a fixed shunt secondary to congenital cyanotic heart disease or the right-to-left shunting of persistent pulmonary hypertension of the newborn cannot do so.

Obtain a chest x-ray to identify pulmonary disease, abnormalities of pulmonary blood flow, and abnormalities of heart size. If physical examination and chest x-ray have not pointed to a particular diagnosis, an echocardiogram will be needed later on. An echocardiogram is not necessary in the ED during the initial resuscitation.

Treatment

Although oxygen therapy is the mainstay of treatment for cyanosis, treat the underlying cause. Provide positive-pressure ventilation (continuous positive airway pressure or endotracheal intubation and mechanical ventilation) to the cyanotic newborn with significant respiratory symptoms. Monitor blood gas and pulse oximetry. Establish vascular access, and initiate 10% dextrose in water at 3.3 mL/kg/h (80 mL/kg/24 h) if in the first 24 hours of life. Check serum glucose every 30 to 60 minutes until stable (see "Hypoglycemia" below). Administer empiric antibiotics for sepsis while obtaining appropriate labs (CBC

with differential count and platelets, blood culture, chest x-ray, and, possibly, urine culture and C-reactive protein).

If, after initial examination and testing, cyanotic heart disease cannot be ruled out, begin an infusion of **prostaglandin E_1 starting at 0.05 microgram/kg/min, and titrate to the lowest effective dose** to maintain ductal patency.

PNEUMOTHORAX

Pulmonary air leaks are seen more commonly in newborns with respiratory distress syndrome, meconium aspiration syndrome, pneumonia, pulmonary hypoplasia, and congenital diaphragmatic hernia. Pneumothoraces may occur in the otherwise normal newborn in the first few minutes after birth due to increased intrathoracic pressure created with the onset of respiration in the fluid-filled newborn lungs. Air can also dissect into the pulmonary interstitium, mediastinum, pericardium, peritoneum, and subcutaneous space. Pneumothoraces may also be iatrogenic secondary to overexuberant bagging during resuscitation, especially with already compromised lungs. Unfortunately, the disorders that most commonly lead to pneumothoraces are also associated with respiratory distress and cyanosis, which may delay the suspicion and diagnosis of the pneumothorax.

Clinical Features and Diagnosis

Tension pneumothorax requires rapid treatment to avoid severe respiratory compromise, cardiovascular collapse from impaired venous return to the heart, and, possibly, death. In the preterm newborn, tension

pneumothoraces are also highly related to subsequent intracranial hemorrhage, presumably secondary to venous backup into the cerebral circulation.

Tachycardia, tachypnea, and retractions are noted. On auscultation, breath sounds are decreased on the side of the pneumothorax. If there is a tension pneumothorax, heart sounds and point of maximum impulse may be displaced in the direction away from the pneumothorax. Transillumination of the chest with a bright light is another method to help rapidly establish pneumothorax, by "lighting up" the pleural air. Bedside US can also identify pneumothorax. Chest x-ray will confirm the diagnosis.

Treatment

The management of a pneumothorax depends on pneumothorax size and the tension it creates in the pulmonary space. A small, nontension pneumothorax can be observed without evacuation. In a term or near-term newborn, the nitrogen washout technique, placing the baby in a 100% oxygen hood for 6 to 12 hours, will usually accelerate clearance of the air leak. This technique is contraindicated in preterm newborns due to concerns of oxygen toxicity to the lungs and retinas.

Emergency evacuation of a tension pneumothorax may be performed with an 18- or 20-gauge 1-inch percutaneous catheter. Instill local anesthetic at the insertion site before the procedure if the patient is not in extremis. After elevating the neonate's affected side with towels under the back, insert the catheter into the fourth intercostal space at the anterior axillary line, which should correlate with the nipple line. Once the pleural space is penetrated, withdraw the needle, and attach the catheter to a three-way stopcock connected to a 10- or 20-mL syringe. Open the stopcock to the syringe, and aspirate the pleural air. More

than one syringe of air may be evacuated if a large pneumothorax is present. Clinical improvement should occur after removal of the pleural air. A 10F or 12F chest tube or an 8.5F pigtail catheter can then be placed.

HYPOGLYCEMIA

In the first four hours after birth, asymptomatic hypoglycemia requiring IV therapy is defined as <25 milligrams/dL. Levels of 25 to 44 milligrams/dL (1.38 to 2.4 mmol/L) require feeding and repeat evaluation in 1 hour. After 4 hours of age, serum glucose levels should be ≥45 milligrams/dL (2.4 mmol/L), with levels of 35 to 44 milligrams/dL (1.94 to 2.4 mmol/L) requiring feeding and 1-hour postprandial glucose checks. Risk factors for hypoglycemia in the newborn include prematurity, low birth weight (<2.5 kg), small for gestational age, large for gestational age (>4 kg), infant of a diabetic mother, hypothermia, sepsis, and intrapartum stress.

Clinical Features

Symptoms of hypoglycemia are quite varied and include tremors, irritability, lethargy, hypotonia, apnea, tachypnea, tachycardia, cyanosis, high-pitched cry, and seizures. Hypoglycemic newborns may be asymptomatic despite very low glucose levels.

Treatment

Treat mild hypoglycemia (25 to 44 milligrams/dL [1.38 to 2.4 mmol/L]) in an otherwise well newborn by feeding. Treat significant hypoglycemia (<25 milligrams/dL [1.38 mmol/L]) immediately with a bolus of dextrose (10% dextrose in water, 2 mL/kg IV/IO), and then administer continuous IV therapy with 10%

dextrose in water at 100 mL/kg/24 h and adjust based on serum glucose levels every 30 to 60 minutes until stable and ≥45 milligrams/dL (2.4 mmol/L).

CONGENITAL DIAPHRAGMATIC HERNIA

Congenital diaphragmatic hernias are frequently diagnosed prenatally with US, which expedites proper initial newborn resuscitation.

Anatomically, congenital diaphragmatic hernia is a diaphragmatic defect, either posterolaterally through the foramen of Bochdalek or, less commonly, through the retrosternal foramen of Morgagni. Most are left-sided.[16,17,18,19] The diaphragmatic defect allows intra-abdominal contents, including stomach, bowel, and, occasionally, liver, to enter the chest during the second trimester of gestation, leading to pulmonary hypoplasia.

The lung ipsilateral to the diaphragmatic defect is hypoplastic, although the degree of hypoplasia may vary. Ultimate morbidity and mortality are determined both by the extent of hypoplasia of the contralateral lung secondary to compression from the abdominal contents in the thoracic space and whether or not the liver is located in the thorax.[17] Total lung volumes >45% of normal are predictive of survival.[18] Significant associated malformations, especially cardiac defects, are seen in one fourth to one half of patients with congenital diaphragmatic hernia.

Clinical Features

The clinical hallmark is persistent respiratory distress at birth, often with a characteristic "seesaw" side-to-side respiratory pattern due to the severely hypoplastic ipsilateral lung. A halting, gasping type of respiratory pattern along with persistent cyanosis is frequently noted. The abdomen appears scaphoid, because abdominal contents are partially situated in the thoracic space. Auscultation of bowel sounds in the chest strongly suggests the presence of congenital diaphragmatic hernia. Radiographic examination is confirmatory.

Treatment

Rapid endotracheal intubation is the treatment of choice for respiratory distress. **Bag-mask ventilation will inflate the GI contents in the chest and will further compromise ventilation.** Endotracheal intubation followed by ventilation with a rate of 40 to 50 breaths/min and lowest peak inspiratory pressures that allow for normal chest rise will help avoid pneumothoraces due to barotrauma to the hypoplastic lungs. Gentle hyperventilation to a PCO_2 between 30 and 35 mm Hg may help lower pulmonary vasculature resistance and allow for an easier stabilization phase before surgical correction of the diaphragmatic defect. Place a large-bore (10F) orogastric tube set to low continuous suction to minimize further lung compression from overaerated GI contents.

Obtain chest and abdominal x-rays and blood gas analysis to confirm the initial diagnosis and guide stabilization and management. After initial stabilization, emergent referral to a pediatric specialty center is essential.

Despite the advent of antenatal diagnosis, alternate ventilatory strategies (high-frequency oscillatory ventilation), a larger armamentarium to treat pulmonary hypertension (inhaled nitric oxide therapy, sildenafil, and extracorporeal membrane oxygenation), and changing surgical strategies (delayed repair to allow more time for resolution of pulmonary hypertension), mortality from congenital diaphragmatic hernia remains quite high at 30% to 60%.[19,20,21]

GASTROSCHISIS AND OMPHALOCELE

A gastroschisis is a defect located to the right of the umbilicus from which uncovered intestine is extruded. An omphalocele is a large, centrally located defect of the abdominal wall containing stomach, intestine, and, frequently, liver that is covered by the mesentery. The umbilical cord inserts directly into the omphalocele sac. Rarely, an omphalocele may be ruptured either before delivery or during delivery. Associated anomalies are seen in 10% to 21% of patients with gastroschisis and 40% to 75% of patients with omphalocele.[22,23,24] Associated defects include cardiac defects (such as tetralogy of Fallot), syndromes (Beckwith-Wiedemann), and chromosomal abnormalities (trisomy 13, trisomy 18), in addition to associated intestinal atresias.

Treatment

The initial management of gastroschisis and omphalocele is similar: handle the covered sac and free intestine with care. Place the newborn on a radiant warmer to help prevent hypothermia due to increased heat loss from the exposed abdominal contents. The ABCs of stabilization should be performed as needed.

If an omphalocele is present, note its size and contents. Cover the sac with warmed saline gauze wrapped gently around the baby with Kerlix, and then place an additional cover with plastic wrap to help minimize evaporative losses. Start IV 10% dextrose in water at 1.5× maintenance (i.e., 5 to 6 mL/kg/h or 120 to 150 mL/kg/24 h) to compensate for the additional insensible water loss. Monitor urine output and electrolytes closely to determine ongoing fluid needs.

If a gastroschisis is present, immediately check for dusky or cyanotic intestine, which indicates reduced flow to the affected bowel due to torsion and vascular occlusion. Gently rotate the bowel to relieve torsion if necessary to prevent bowel infarction. Emergency pediatric surgical consultation is essential. If the intestine appears pink, cover with warmed saline gauze, wrap with Kerlix, and further cover with plastic wrap. **Be careful not to compress the intestine, which could result in obstruction of blood flow to the bowel.** Insensible water loss is much greater in the newborn with gastroschisis due to the open defect with large amounts of extruded bowel. Therefore, IV 10% dextrose in water should be started at 6 to 7 mL/kg/h (at least 150 mL/kg/24 h).

Check glucose periodically. Antibiotics, usually ampicillin, 50 to 100 milligrams/kg IV, and gentamicin, 4 to 5 milligrams/kg IV, should be given.

Immediate consultation with a neonatologist and pediatric surgeon is necessary. The overall outcome for gastroschisis is quite good, with survival exceeding 90%.[25,26] Survival with omphalocele is somewhat less at 73% to 88%, depending largely on the presence of associated anomalies.[25]

TRACHEOESOPHAGEAL FISTULA

Tracheoesophageal fistula develops secondary to a failure of separation of the developing foregut structures, the trachea and esophagus, during the embryologic stage of development. There are five types of tracheoesophageal fistula: (1) esophageal atresia with a distal tracheoesophageal fistula (88% of cases), (2) isolated esophageal atresia without tracheoesophageal fistula (7%), (3) esophageal atresia with proximal tracheoesophageal fistula (1%), (4) esophageal atresia with proximal and distal tracheoesophageal fistulas (1%), and (5) H-type fistula without esophageal atresia (3%). Tracheoesophageal fistula is highly associated with several other malformations, designated as the acronyms VATER or VACTERL association: *v*ertebral anomalies, *a*nal atresia, *c*ardiac anomalies, *t*racheo*e*sophageal fistula, *r*adial anomaly/*r*enal anomalies, and *l*imb anomalies.

Diagnosis

The diagnosis of tracheoesophageal fistula can be missed during prenatal US examination. When esophageal atresia is present, polyhydramnios is usually noted before or at delivery. Newborns with tracheoesophageal fistula/esophageal atresia will usually have excessive oral secretions noted shortly after birth. When attempting to pass a nasogastric tube, the tube coils in the esophageal pouch and often comes back out of the mouth. No air passage will be heard when a 5-mL air bolus is injected into the nasogastric tube. A chest x-ray with a nasogastric tube in place will demonstrate the esophageal pouch. Confirmatory contrast studies are not indicated and may actually be contraindicated because the esophageal contents can be aspirated into the lungs.

Treatment

Management of tracheoesophageal fistula includes placing the child in head-up (reverse Trendelenburg) positioning to help prevent passage of gastric contents through the tracheoesophageal fistula into the lungs, placing the nasogastric tube into the esophageal pouch on low intermittent suction to prevent buildup and possible aspiration of oral secretions, and giving the newborn nothing by mouth. Initially, standard 10% dextrose in water IV fluids are best. Immediate referral to a center with neonatologists and pediatric surgeons is essential.

REFERENCES

1. http://www.who.int/maternal_child_adolescent/documents/basic_newborn_resuscitation/en/ (World Health Organization: Guidelines on Basic Newborn Resuscitation, 2012). Accessed on July 7, 2014.

2. American Academy of Pediatrics/American College of Obstetricians and Gynecologists Policy Statement: The Apgar score. *Pediatrics* 117: 1444, 2006.
[PubMed: 16585348]

3. Perlman JM, Wyllie J, Kattwinkel J et al.: Part II: Neonatal Resuscitation: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Circulation* 122: S516, 2010.
[PubMed: 20956259]

4. Wyckoff MH: Neonatal resuscitation guidelines versus the reality of the delivery room. *J Pediatr* 163: 1542, 2013.
[PubMed: 24274198]

5. McCarthy LK, Morley CJ, Davis PG et al.: Timing of interventions in the delivery room: does reality compare with neonatal resuscitation guidelines. *J Pediatr* 163: 1553, 2013.
[PubMed: 23866717]

6. American Academy of Pediatrics: *AAP/AHA Neonatal Resuscitation Instructor Manual—Brief Summary of Changes*. Elk Grove Village, IL: American Academy of Pediatrics, 2011:xi–xxvi.

7. Kattwinkel J (ed): *Textbook of Neonatal Resuscitation* (6th ed). Elk Grove Village, IL: American Academy of Pediatrics, 2011.

8. Beveridge CJE, Wilkinson AR: Sodium bicarbonate infusion during resuscitation of infants at birth. *Cochrane Database Syst Rev* 1: CD004864, 2006.
[PubMed: 16437499]

9. Gibbs J, Newson T, Williams J, Davidson DC: Naloxone hazard in infant of opioid abuser. *Lancet* 2: 159, 1989.
[PubMed: 2567922]

10. Edwards AD, Brocklehurst P, Gunn AJ et al.: Neurological outcomes at 18 months of age after moderate hypothermia for perinatal hypoxic ischaemic encephalopathy: synthesis and meta-analysis of trial data. *BMJ* 340: c63, 2010.
[PubMed: 20219818]

11. Simbruner G, Mittal RA, Rohlmann F et al.: Systemic hypothermia after neonatal encephalopathy: outcomes of neon.nEURO.network RCT. *Pediatrics* 126: e771, 2010.
[PubMed: 20855387]

12. Shankaran S, Pappas A, McDonald SA et al.: Childhood outcomes after hypothermia for neonatal encephalopathy. *N Engl J Med* 366: 2085, 2012.
[PubMed: 22646631]

13. Sussman CB: While waiting: early recognition and initial management of neonatal hypoxic-ischemic encephalopathy. *Adv Neonatal Care* 13: 415, 2013.
[PubMed: 24300960]

14. Perinatal Continuing Education Program: *Neonatal Care—Book II*. Elk Grove Village, IL: American Academy of Pediatrics, 2007:209, 216.

15. Unpublished data from Vermont-Oxford Network, 2003–2005, reported in Neonatal Resuscitation Program online Web page. http://www2.aap.org/nrp/. Accessed September 15, 2009.

16. Slavotinek AM, Warmerdam B, Lin AE, Shaw GM: Population-based analysis of left- and right-sided diaphragmatic hernias demonstrates different frequencies of selected additional anomalies. *Am J Med Genet A* 143A: 3127, 2007.
[PubMed: 18008313]

17. Beck C, Alkasi O, Nikischin W et al.: Congenital diaphragmatic hernia, etiology and management—a 10-year analysis of a single center. *Arch Gynecol Obstet* 277: 55, 2008.
[PubMed: 17680260]

18. Thibeault DW, Haney B: Lung volume, pulmonary vasculature, and factors affecting survival in congenital diaphragmatic hernia. *Pediatrics* 101: 289, 1998.
[PubMed: 9445506]

19. Laye MR, Rehberg JF, Kosek MA et al.: Prenatal diagnoses and outcomes of congenital diaphragmatic hernia. *J Miss State Med Assoc* 48: 67, 2007.
[PubMed: 17941262]

20. Rocha GM, Bianchi RF, Severo M et al.: Congenital diaphragmatic hernia—the neonatal period (part 1). *Eur J Pediatr Surg* 18: 219, 2008.
[PubMed: 18704887]

21. Fisher JC, Jefferson RA, Arkovitz MS, Stolar CJ: Redefining outcomes in right congenital diaphragmatic hernia. *J Pediatr Surg* 43: 373, 2008.

[PubMed: 18280293]

22. Torfs C, Curry C, Roeper P: Gastroschisis. *J Pediatr* 116: 1, 1990.
[PubMed: 2136909]

23. Hunter AG, Stevenson RE: Gastroschisis: clinical presentation and associations. *Am J Med Genet Semin Med Genet* 148C: 219, 2008.
[PubMed: 18655099]

24. Stoll C, Alembik Y, Dott B, Roth MP: Omphalocele and gastroschisis and associated malformations. *Am J Med Genet A* 146A: 1280, 2008.
[PubMed: 18386803]

25. Aizenfisz S, Dauger S, Gondon E et al.: Gastroschisis and omphalocele: retrospective study of initial postoperative management in the ICU. *Eur J Pediatr Surg* 16: 84, 2006.
[PubMed: 16685612]

26. Jager LC, Heij HA: Factors determining outcome in gastroschisis: clinical experience over 18 years. *Pediatr Surg Int* 23: 731, 2007.
[PubMed: 17576575]

USEFUL WEB RESOURCES

American Academy of Pediatrics, APLS Online—http://www.aplsonline.com

American Academy of Pediatrics, Life Support Programs—http://www.aap.org/profed/nrp

American Academy of Pediatrics, Neonatal Resuscitation Program—http://www.aap.org/nrp/nrpmain.html

European Resuscitation Council, Pediatric Life Support—http://www.erc.edu, http://www.erc.edu/index.php/pls_overview/en