

KS tại chỗ làm chậm rụng rốn

Cần làm mau khô nhưng ko giảm NT rốn

Rửa vs nước muối sly và để khô cũng an toàn và hiệu quả như dùng kháng sinh

4. When should a parent begin to worry if an umbilical cord has not fallen off?

The umbilical cord generally dries up and sloughs by **2 weeks** of life. Delayed separation can be normal up to 45 days. However, because neutrophilic and monocytic infiltration appear to play a major role in autodigestion, persistence of the cord **beyond 30 days** should prompt consideration of an underlying **functional abnormality of neutrophils** (leukocyte adhesion deficiency) or neutropenia.

Roos D, Laws SK: Hematologically important mutations: leukocyte adhesion deficiency, *Blood Cell Mol Dis* 6:1000–1004, 2001.

Kemp AS, Lubitz L: Delayed cord separation in alloimmune neutropenia, *Arch Dis Child* 68:52–53, 1993.

59. When does the newborn infant's stomach begin to secrete acid?

The pH of gastric fluid in newborns is usually neutral or slightly acidic and decreases shortly after birth. pH values are less than 3 by 6 to 8 hours of age and then increase again during the second week of life. **Preterm infants** frequently demonstrate gastric pH values **greater than 7** for many days depending on the degree of prematurity.

60. When is meconium usually passed after birth?

Most infants pass some meconium during the first **12 hours of life**. Overall, 99% of term infants and 95% of premature infants pass meconium by **48 hours of life**. However, the smallest of premature infants may have a delayed passage of meconium as a result of the relative immaturity of rectal sphincter reflexes.

65. What is necrotizing enterocolitis (NEC)?

NEC is a necrotizing inflammatory intestinal disorder that is the most common acquired gastrointestinal emergency in newborns. Signs and symptoms include abdominal distention, increasing gastric residuals, stool with blood, erythema of the abdominal wall, and lethargy. Positive blood culture is found in about 25% of cases at the time of diagnosis.

66. What are the most important risk factors for NEC in preterm infants?

In an analysis of 15,072 neonates born at 98 centers over a 2-year period, the most important variables associated with NEC were **gestational age** and **birthweight**. Apgar score was not related. Other variables associated with an increased risk for NEC included **the use of a ventilator on the first day of life**, exposure to both **glucocorticoids** and **indomethacin** during **the first week of life**, and **symptomatic patent ductus arteriosus** requiring surgery. Cesarean delivery and the use of breast milk were associated with a lower risk for surgical NEC. The impact of antenatal steroids has varied between studies.

The exact cause is unclear

In preterm infants beyond 32 weeks of gestation, hematologic values differ only minimally from those of full-term infants, and therefore the same values may be used.

81. When and at what dose should iron supplementation be initiated and for how long should it be maintained?

The timing for initiation of iron supplementation in preterm infants has been a subject of controversy for decades. Recommendations of the AAP, Canadian Pediatric Society, and European Society of Pediatric Gastroenterology and Nutrition suggest that doses of 2 to 4 mg/kg per day of iron be initiated at 4 to 8 weeks of age and maintained for 12 to 15 months.

Rao R, Georgieff MK: Iron therapy for preterm infants, *Clin Perinatol* 36:27–42, 2009.

84. Why is the direct Coombs test frequently negative or weakly positive in infants with ABO incompatibility?

There are fewer A or B antigenic sites on the newborn RBC, and there is also a greater distance between antigenic sites compared with adult RBCs. Absorption of serum antibody by ABO antigens located on tissues throughout the body.

85. If fetomaternal hemorrhage is suspected as a cause of neonatal anemia, how is this diagnosed?

The Kleihauer-Betke test detects the presence of fetal cells in the maternal circulation. Because fetal hemoglobin is resistant to elution with acid, the treatment of a maternal blood smear with acid will result in darkly stained fetal cells among the maternal “ghost” cells. From the percentage of fetal RBCs and the estimated maternal blood volume, the size of the hemorrhage can be determined. One percent fetal cells in the maternal circulation indicates a bleed of about 50 mL.

87. How is polycythemia defined?

Polycythemia is defined by a venous hematocrit of 65% because this exceeds the mean hematocrit found in normal newborns by two standard deviations. As the central venous hematocrit rises above 65%, there is an increase in viscosity. In neonates, some of the increase in viscosity with polycythemia is ameliorated by the lower viscosity of plasma. Because direct measurements of blood viscosity are not readily available in most laboratories, a high hematocrit level is thought to be the best indirect indicator of hyperviscosity.

88. What are the clinical manifestations of polycythemia?

In symptomatic infants, the most common presentations relate to CNS abnormalities, including lethargy, hypotonia, tremulousness, and irritability. With severe CNS involvement, seizures can result. Hypoglycemia is common. Other organ systems can be involved, including the gastrointestinal tract (vomiting, distension, NEC), the kidneys (renal vein thrombosis, acute renal failure), and the cardiopulmonary system (respiratory distress, congestive heart failure). However, infants with polycythemia are often asymptomatic.

TABLE 12-10. GUIDELINES FOR PLATELET TRANSFUSION

Platelet Count $\times 10^9/L$	Nonbleeding Neonate (1st Week of Life)	Nonbleeding Neonate (2nd Week and Onward)	Neonate with Major Bleeding
<30	Transfuse	Transfuse	Transfuse
30-49	Transfuse if <1000 g, clinically unstable, evidence of previous bleed, coagulopathy, and/or undergoing surgery	Do not transfuse	Transfuse
50-99	Do not transfuse	Do not transfuse	Transfuse

Adapted from Roberts I, Stanworth S, Murray NA: Thrombocytopenia in the neonate. Blood Rev 22:173-186, 2008.

Nói chung < 30.000 là truyền, có major bleeding là truyền

93. What features on physical examination suggest a specific cause of thrombocytopenia?

- "Blueberry-muffin rash" (toxoplasmosis rubella cytomegalovirus herpes [TORCH] or viral infection)
- Absence of radii (thrombocytopenia absent radii [TAR] syndrome)
- Palpable flank mass and hematuria (renal vein thrombosis)
- Hemangioma, large, often with bruit (Kasabach-Merritt syndrome)
- Abnormal thumbs (Fanconi syndrome, albeit thrombocytopenia is less likely in newborns)
- Markedly dysmorphic features (chromosomal abnormalities, particularly trisomy 13 or 18)

Blueberry-muffin rash: ko phải là xuất huyết, mà là tạo máu ngoài tử

Causes [\[edit \]](#)

The condition was originally considered characteristic of rubella, but is now considered to be potentially associated with many other conditions,^[4] such as cytomegalovirus^[5], metastatic neuroblastoma, and Congenital Leukemia.

7. Blue berry muffin babies

- Blueberry muffin babies present with widespread rash of blue red papules & nodules at birth. The rash is due to congenital infection in utero



94. What are the two main types of neonatal thrombocytopenia caused by maternal antibody?

Transplacental passage of antibody from the mother to infant can be due to maternal idiopathic thrombocytopenic purpura (ITP), with the newborn a **secondary target**, and **isoimmune thrombocytopenia**, with the newborn a **primary target**. The diseases can have a **similar clinical appearance**. Babies generally appear **well**, do not have hepatosplenomegaly, and have thrombocytopenia that persists for 3 to 12 weeks postnatally.

95. In the mother with new-onset thrombocytopenia during pregnancy, how can one determine the risk to the fetus?

Because **only a small percentage** of infants born to mothers with ITP have severe thrombocytopenia, prediction of this outcome is **highly challenging**. There are conflicting

results regarding the predictive ability of platelet counts, platelet-associated immunoglobulin G (IgG), and circulating platelet autoantibodies. However, a **previous history of an affected infant** has been shown to be predictive in several studies. Although cordocentesis could provide direct data on the fetal platelet count, the risks of the procedure outweigh the risks of associated neonatal disease and thus cannot be justified.

103. What is believed to be the fraction of bilirubin that is toxic to the CNS?

Routine clinical laboratory tests measure the **total bilirubin** and the **conjugated bilirubin**. Of the total unconjugated bilirubin, most is bound to albumin and thus cannot cross the blood-brain barrier. Although the free bilirubin is believed to cause neurotoxicity, routine measurement in clinical practice is not available. Measurement of the **bilirubin (mg/dL)-to-albumin (g/dL) (B/A) ratio** may be helpful in managing jaundiced infants by acting as a surrogate for free bilirubin. Patients are thought to be at increased risk for bilirubin toxicity if the ratio is 8.0 or higher in infants born at 38 weeks' gestation or later, at least 7.2 in infants 35 to 36 weeks' gestation and well or at least 38 weeks' gestation and classified as high risk (e.g., sepsis, acidosis, hemolysis, glucose-6-phosphate dehydrogenase deficiency), or at least 6.8 in infants 35 to 36 weeks' gestation and classified as high risk.

TABLE 12-6. EXTERNAL GESTATIONAL AGE CHARACTERISTICS

External Characteristics	Gestational Age			
	28 Weeks	32 Weeks	36 Weeks	40 Weeks
Ear cartilage	Pinna soft, remains folded	Pinna slightly harder but remains folded	Pinna harder, springs back	Pinna firm, stands erect from head
Breast tissue	None	None	1-2 mm nodule	6-7 mm nodule
Male genitalia	Testes undescended, smooth scrotum	Testes in inguinal canal, few scrotal rugae	Testes high in scrotum, more scrotal rugae	Testes descended, pendulous scrotum covered with rugae
Female genitalia	Prominent clitoris, small widely separated labia	Prominent clitoris, larger separated labia	Clitoris less prominent, labia majora covers labia minora	Clitoris covered by labia majora
Plantar surface	Smooth	1-2 anterior creases	2-3 anterior creases	Creases cover sole

From Volpe JJ Neurology of the Newborn, 5th ed. Philadelphia, WB Saunders, 2008, p 122.

106. Which infants are “set-ups” for ABO incompatibility?

Infants who are type A or B and whose mothers are type O. In individuals with type A or B blood, naturally occurring anti-A and anti-B isoantibodies are primarily IgM and do not

cross the placenta. However, in type O individuals, isoantibodies are frequently IgG. These antibodies can cross the placenta and cause hemolysis. Although about 12% of maternal-infant pairs qualify as set-ups for ABO incompatibility, less than 1% of infants have significant hemolysis.

110. What distinguishes breastfeeding jaundice from breast-milk jaundice?

Hyperbilirubinemia in breastfed infants during the first week of life is called **breastfeeding jaundice** and is thought to be the result of poor caloric intake and/or dehydration.

Hyperbilirubinemia in breastfed infants after the first week of life is known as **breast-milk**

jaundice. The cause of breast-milk jaundice is uncertain; however, possible etiologies include an increased enterohepatic circulation of bilirubin as a result of the presence of β -glucuronidase in human milk and/or the inhibition of the hepatic glucuronosyl transferase by a factor such as free fatty acids in some human milk samples. The incidence and duration compared with physiologic jaundice are noted in Table 12-12.

TABLE 12-12. COMPARISON OF PHYSIOLOGIC, BREASTFEEDING, AND BREAST-MILK JAUNDICE

	Physiologic Jaundice	Breastfeeding Jaundice	Breast-Milk Jaundice
Time of onset (TSB >7 mg/dL)	After 36 hr	2-4 days	4-7 days
Usual time of peak bilirubin	3-4 days	3-6 days	5-15 days
Peak TSB	5-12 mg/dL	>12 mg/dL	>10 mg/dL
Age when total bilirubin <3 mg/dL	1-2 wk	>3 wk	9 wk
Incidence in full-term neonates	56%	12%-13%	2%-4%

TSB = total serum bilirubin.
 From Gourley G: Pathophysiology of breast milk jaundice. In Polin RA, Fox W (eds): Fetal and Neonatal Physiology. Philadelphia, WB Saunders, 1992, p 1174.

111. Why should infants at risk for breastfeeding jaundice be fed more frequently?

Breastfed infants exhibit their maximal weight loss by day 3 of life and lose on average 6.1% \pm 2.5% of their birthweight. Infants breastfed an average of more than 8 times per day during the first 3 days of life have significantly lower serum bilirubin concentrations than

those who are less frequently breastfed. This practice accelerates and enhances the acquisition of milk supply. With increased milk available, dehydration is less likely to occur, and the excretion of bilirubin by the gastrointestinal tract is more rapid. Infants with adequate intake should have four to six wet diapers per day.

113. Where does bilirubin go when you turn on the lights?

It becomes lumirubin (through a "cyclicization" reaction) and is rapidly excreted in bile, with a half-life of about 2 hours. In addition to the aforementioned principal pathway of bilirubin elimination, photoisomers are also formed, and because of their water solubility, they can be excreted in the urine.

115. What are the contraindications to phototherapy?

Infants with a family history of light-sensitive porphyria should not receive phototherapy. The presence of direct hyperbilirubinemia is not considered a contraindication, but it will decrease the effectiveness of phototherapy may result in bronze baby syndrome.

117. A newborn develops dark skin discoloration and dark urine after beginning phototherapy. What is the diagnosis?

Bronze baby syndrome. Infants who develop the syndrome typically have an **elevated direct serum bilirubin** concentration. The bronze baby syndrome results from the **retention of photoproducts** (e.g., lumirubin) that cannot be excreted in the bile. Most infants appear to recover without complications. Direct hyperbilirubinemia is not a contraindication to phototherapy.

119. What is the relationship between delayed neonatal jaundice and urinary tract infection (UTI)?

Unexplained jaundice developing between 10 and 60 days of age can be associated with a UTI in infants. The typical patient is usually afebrile (in two thirds of cases) with hepatomegaly and minimal systemic symptoms. Hyperbilirubinemia is usually conjugated, and liver transaminases may be normal or mildly elevated. Treatment of the UTI (usually caused by *Escherichia coli*) results in reversal of the liver dysfunction, which is believed to be the result of endotoxins.

120. Can transcutaneous bilirubin measurements be used in place of serum levels?

Numerous devices have been developed that accurately measure bilirubin levels that are highly correlated with serum bilirubins. However, most studies show that the deviation of transcutaneous measurements is greatest (about 3 mg/dL) at the highest levels (>13 to 15 mg/dL). Therefore, many authorities recommend serum confirmation if the transcutaneous bilirubin is greater than the 75th percentile, more than 13 mg/dL, or if a level that is 3 mg/dL higher would be clinically meaningful. In any case, this methodology should lead to a sharp reduction in the need for blood measurements.

Grohmann K, Roser M, Rolinski B, et al: Bilirubin measurement for neonates: comparison of 9 frequently used methods, *Pediatrics* 117:1174–1183, 2006.

122. Who was Sister Ward?

In the early 1950s, Sister Ward was the nurse in charge of the unit for premature infants at Rochford General Hospital in Essex, England. On warm summer days, Sister Ward would take her infants to the courtyard to give them a little fresh air and sunshine. It was after such an afternoon of sunshine that Sister Ward observed that sunlight was able to “bleach” the skin of jaundiced neonates. The account of her discovery, as recorded by R.H. Dobbs, follows:

One particularly fine summer's day in 1956, during a ward routine, Sister Ward diffidently showed us a premature baby, carefully undressed and with fully exposed abdomen. The infant was pale yellow except for a strongly demarcated triangle of skin very much yellower than the rest of the body. I asked her, "Sister, what did you paint it with—iodine or flavine—and why?" But she replied that she thought it must have been the sun. "What do you mean Sister? Suntan takes days to develop after the erythema has faded." Sister Ward looked increasingly uncomfortable, and explained that she thought it was a jaundiced baby, much darker where a corner of the sheet had covered the area. "It's the rest of the body that seems to have faded." We left it at that, and as the infant did well and went home, fresh air treatment of prematurity continued.

126. What is the definition of neonatal hypoglycemia?

Based on statistical definition, most authorities accept 40 mg/dL as the lower limit of normal during the first 24 hours of life. However, a normal glucose is that level which is needed to meet the requirements of cerebral energy metabolism, which cannot be readily measured.

127. When is hypoglycemia most likely to occur in a neonate?

During gestation, glucose is freely transferred across the placenta by the process of facilitated diffusion. However, after birth, the infant must adjust to the sudden withdrawal of this transplacental supply. In all infants, there is a nadir in blood sugar between 1 and 3 hours of life. During the first 12 to 24 hours of life, newborns are at increased risk for hypoglycemia because gluconeogenesis and especially ketogenesis are incompletely developed. These factors are accentuated in preterm infants, infants of diabetic mothers, infants with erythroblastosis fetalis, asphyxiated infants, and infants who are small or large for gestational age.

Sperling MA, Menon RK: Differential diagnosis and management of neonatal hypoglycemia, *Pediatr Clin North Am* 51:703-723, 2004.

129. What features on physical examination suggest the etiology of hypoglycemia?

- **Macrosomia:** This occurs in infants of diabetic mothers, infants with severe congenital hyperinsulinism, and infants with Beckwith-Wiedemann syndrome; recall that insulin is a growth factor and that hyperinsulinism leads to macrosomia.
- **Midline defects:** Congenital pituitary deficiency can be associated with midline defects such as cleft lip, cleft palate, single central incisor, and microphthalmia.
- **Micropenis:** Congenital gonadotropin deficiency and possible pituitary abnormalities cause this condition.
- **Hepatomegaly:** This is associated with glycogen storage diseases and fatty acid oxidation disorders.

131. What are the manifestations of hypocalcemia in the neonate?

The major manifestations are jitteriness and seizures. Additional signs such as high-pitched cry, laryngospasm, Chvostek sign (facial muscle twitching on tapping), and Trousseau sign (carpopedal spasm) may be present, but more commonly these are absent during the neonatal period.

NEONATAL SEPSIS

136. Can sepsis be distinguished from other causes of respiratory distress in the neonate?

Not reliably. Diagnosis is confirmed only by a positive blood, urine, or CSF culture.

137. What laboratory tests can rule out sepsis on admission?

None. Total white blood cell (WBC) counts, immature-to-total (I:T) ratios of neutrophils, and C-reactive protein are of limited value as single tests for the diagnosis of bacterial sepsis in the newborn. In one third of infants with proven bacterial disease, total WBC counts are normal, particularly early during the course of infection. The most sensitive neutrophil index for identifying septic infants is the I:T neutrophil ratio. An I:T ratio of more than 0.2 has been considered abnormal, although some studies have suggested that a ratio as high as 0.27 may be seen in healthy term newborns. Neutropenia (total WBC $<5000/\text{mm}^3$ or absolute neutrophil count $<1750/\text{mm}^3$) is the most specific indicator. The least sensitive neutrophil index is the absolute band count (normal, $<2000/\text{mm}^3$). Generally, abnormal neutrophil indices have low PPVs and therefore are not helpful as sole tests for clearly identifying which infants are infected. However, they have a much higher negative-predictive value, particularly if repeated 12 hours after birth, and thus they can be very helpful for determining which infants do not have infection.

141. Should an LP be performed on all newborns as part of the sepsis evaluation?

The need for LP as part of the sepsis evaluation of a newborn is controversial, with some authors suggesting its omission in asymptomatic infants. However, in symptomatic infants, an LP should be strongly considered because of the following: (1) bacterial meningitis can be present in newborns without CNS symptoms; (2) a significant number of infants (15% to 30%) can have meningitis without bacteremia, especially after the first week of life, and (3) meningitis can coexist in premature infants with suspected respiratory distress syndrome. The procedure should be postponed in an infant with cardiorespiratory instability or significant thrombocytopenia.

Stoll BJ, Hansen N, Fanaroff AA, et al: To tap or not to tap: high likelihood of meningitis without sepsis among VLBW infants, *Pediatrics* 113:1181–1186, 2004.

Wiswell TE, Baumgart S, Gannon CM, Spitzer AR: No lumbar puncture in the evaluation for early neonatal sepsis: will meningitis be missed? *Pediatrics* 95:803–806, 1995.

KEY POINTS: SEPSIS



1. Because there are no reliable screening tests for sepsis, clinical judgment is paramount.
2. Screening cultures for group B streptococcus should be performed for all pregnant women at 35 to 37 weeks of gestation.
3. Coagulase-negative staphylococci are the most common bacterial pathogens responsible for nosocomial infections.
4. Neonatal meningitis can occur in the absence of a positive blood culture.
5. Fungal infection must be considered in sick preterm infants who are evaluated for sepsis.

147. What are the most common pathogens that are responsible for late-onset sepsis in the newborn infant?

- Coagulase-negative staphylococci (48%)
- *Staphylococcus aureus* (8%)
- *Enterococcus* species (3%)
- Gram-negative enterics (18%)
- *Candida* species (10%)

Stoll BJ, Hansen N, Fanaroff AA, et al: Late-onset sepsis in very low birth weight neonates: the experience of the NICHD Neonatal Research Network, *Pediatrics* 110:285–291, 2002.

167. What is the most common brachial plexus palsy?

Erb palsy (Fig. 12-11).

Neonatal brachial plexus injuries occur in less than 0.5% of deliveries and are often associated with shoulder dystocia and breech or forceps delivery.

- Involves upper plexus (C5, C6)
- In 50% of cases, C7 is affected
- Arm held limply adducted, internally rotated, and pronated with wrist flexed and fingers flexed ("waiter's tip" position)
- Biceps reflex absent, Moro reflex with hand movement but no shoulder abduction, palmar grasp present
- Ipsilateral diaphragmatic involvement in 5%



Figure 12-11. Erb palsy. Newborn demonstrating characteristic posture with the right arm limply adducted and internally rotated. (From Zitelli BJ, Davis HW: *Atlas of Pediatric Physical Diagnosis*, 5th ed. Philadelphia, Mosby, 2007, p 45.)

168. What is Klumpke paralysis?

A brachial plexus palsy involving injury to the lower plexus (C8, T1). It is associated with weakness of the flexor muscles of the wrist and the small muscles of the hand ("claw hand"). Up to one third of these patients have an associated Horner syndrome.

173. Do newborns prefer to turn their heads to the right or to the left?

Healthy neonates prefer to turn their heads to the right, which may reflect the normal asymmetry of cerebral function at this age. This preference has been observed as early as 28 weeks of gestation. By 39 weeks of gestation, 90% of newborn infants spend 80% of the time with their heads turned to the right side.

174. Does hypothermia improve the outcomes of term infants following perinatal asphyxia?

Timely use of mild therapeutic hypothermia following intrapartum asphyxia results in reduction in the combined incidence of mortality and major neurodevelopmental disability at 18 months of age as well as in the incidence of these outcomes individually. Short-term adverse effects

were few (e.g., small increase in need for inotropic support, thrombocytopenia, and insignificant sinus bradycardia) and were outweighed by the benefits. The reduction in both of these outcomes is extremely reassuring in demonstrating that the increased survival does not result in more impaired infants.

Agostoni C: Role of long chain polyunsaturated fatty acids in the first year of life. *J Pediatr Gastroenterol Nutr* 47:S41–S44, 2008.

Barks JD: Current concepts in hypothermic neuroprotection, *Semin Fetal Neonatal Med* 13:30–34, 2008.

Jacobs S: Cooling for newborns with hypoxic-ischemic encephalopathy, *Cochrane Database Syst Rev* 4:CD003311, 2007.

183. What advice should be given to a mother who plans to express and save breast milk for later feedings?

Ideally, she should collect the milk as cleanly as possible and then store it rapidly at 3° to 4°C or colder; the milk should then be used within 5 days. Alternatively, breast milk can be stored in the freezer compartment of a refrigerator for about 6 months. If more prolonged storage is necessary (12 months), the milk should be kept frozen at a temperature of –20°C or lower (usually in a separate freezer). After the milk has thawed, it should not be refrozen.

188. Is vitamin supplementation necessary for exclusively breastfed term infants?

As a result of the growing concerns about the relationship of sunlight exposure and skin cancer, the low concentration of vitamin D in breast milk, and the inability to predict adequate exposure as a result of diverse lifestyle and cultural practices, cases of rickets in breastfed infants have been reported. The following recommendations have therefore been made:

- Beginning in the first 2 months of life, all breastfed infants should be supplemented with 400 IU/day of vitamin D to prevent the occurrence of rickets.
- Malnourished mothers may need to supplement their breastfed babies with multivitamins.
- Mothers who are strict vegetarians may have low concentrations of B vitamins in their breast milk, and infants may need supplementation with vitamin B₁₂.

American Academy of Pediatrics: *Pediatric Nutrition Handbook*, ed 6, Elk Grove Village IL, 2009.

197. What causes infants to grunt?

Infants with respiratory disease tend to expire through closed or partially closed vocal cords to elevate transpulmonary pressure and to therefore increase lung volume. The latter effect results in an improved ventilation-to-perfusion ratio with better gas exchange. It is during the last part of expiration, when gas is expelled through the partially closed vocal cords, that the audible grunt is produced.

198. What do hyperpnea and tachypnea signify in the neonate?

■ **Hyperpnea** refers to deep, relatively unlabored respirations at mildly increased rates. It is typical of situations in which there is reduced pulmonary blood flow (e.g., pulmonary atresia), and it results from the ventilation of underperfused alveoli.

■ **Tachypnea** refers to shallow, rapid, and somewhat labored respirations, and it is seen in the setting of low lung compliance (e.g., primary lung disease, pulmonary edema).

206. Has nasal prong continuous positive airway pressure (CPAP) been proved to decrease the risk for BPD?

In the largest trial to date, early administration of CPAP compared with intubation in infants born at 25 to 28 weeks' gestation does not reduce the incidence of death or chronic lung disease. The question still remains about whether significant reduction will be achieved by combining this treatment with early administration of surfactant.

Morley CJ, Davis PG, Doyle LW, et al: COIN trial investigators: nasal CPAP or intubation at birth for very preterm infants, *N Engl J Med* 358:700–708, 2008.