

Part 13: Neonatal Resuscitation Guidelines

The following guidelines are intended for practitioners responsible for resuscitating neonates. They apply primarily to neonates undergoing transition from intrauterine to extrauterine life. The recommendations are also applicable to neonates who have completed perinatal transition and require resuscitation during the first few weeks to months following birth. Practitioners who resuscitate infants at birth or at any time during the initial hospital admission should consider following these guidelines. The terms *newborn* and *neonate* are intended to apply to any infant during the initial hospitalization. The term *newly born* is intended to apply specifically to an infant at the time of birth.

Approximately 10% of newborns require some assistance to begin breathing at birth. About 1% require extensive resuscitative measures. Although the vast majority of newly born infants do not require intervention to make the transition from intrauterine to extrauterine life, because of the large number of births, a sizable number will require some degree of resuscitation.

Those newly born infants who do not require resuscitation can generally be identified by a rapid assessment of the following 4 characteristics:

- Was the baby born after a full-term gestation?
- Is the amniotic fluid clear of meconium and evidence of infection?
- Is the baby breathing or crying?
- Does the baby have good muscle tone?

If the answer to all 4 of these questions is “yes,” the baby does not need resuscitation and should not be separated from the mother. The baby can be dried, placed directly on the mother’s chest, and covered with dry linen to maintain temperature. Observation of breathing, activity, and color should be ongoing.

If the answer to any of these assessment questions is “no,” there is general agreement that the infant should receive one or more of the following 4 categories of action in sequence:

- A. Initial steps in stabilization (provide warmth, position, clear airway, dry, stimulate, reposition)
- B. Ventilation
- C. Chest compressions
- D. Administration of epinephrine and/or volume expansion

The decision to progress from one category to the next is determined by the simultaneous assessment of 3 vital signs: respirations, heart rate, and color. Approximately 30 seconds

is allotted to complete each step, reevaluate, and decide whether to progress to the next step (see the Figure).

Anticipation of Resuscitation Need

Anticipation, adequate preparation, accurate evaluation, and prompt initiation of support are critical for successful neonatal resuscitation. At every delivery there should be at least one person whose primary responsibility is the newly born. This person must be capable of initiating resuscitation, including administration of positive-pressure ventilation and chest compressions. Either that person or someone else who is immediately available should have the skills required to perform a complete resuscitation, including endotracheal intubation and administration of medications.¹

With careful consideration of risk factors, the majority of newborns who will need resuscitation can be identified before birth. If the possible need for resuscitation is anticipated, additional skilled personnel should be recruited and the necessary equipment prepared. Identifiable risk factors and the necessary equipment for resuscitation are listed on the Neonatal Resuscitation Program website: www.aap.org/NRP. If a preterm delivery (<37 weeks of gestation) is expected, special preparations will be required. Preterm babies have immature lungs that may be more difficult to ventilate and are also more vulnerable to injury by positive-pressure ventilation. Preterm babies also have immature blood vessels in the brain that are prone to hemorrhage; thin skin and a large surface area, which contribute to rapid heat loss; increased susceptibility to infection; and increased risk of hypovolemic shock caused by small blood volume.

Initial Steps

The initial steps of resuscitation are to provide warmth by placing the baby under a radiant heat source, position the head in a “sniffing” position to open the airway, clear the airway with a bulb syringe or suction catheter, and dry the baby and stimulate breathing. Recent studies have examined several aspects of these initial steps. These studies are summarized below.

Temperature Control

Very low birth weight (<1500 g) preterm babies are likely to become hypothermic despite the use of traditional techniques for decreasing heat loss (LOE 5).² For this reason it is recommended that additional warming techniques be used, such as covering the baby in plastic wrapping (food-grade, heat-resistant plastic) and placing him or her under radiant heat (Class IIa; LOE 2^{3,4}; LOE 4^{5,6}; LOE 5⁷). Temperature must be monitored closely because of the slight but described (LOE 2)⁴ risk of hyperthermia with this technique. Other techniques to maintain temperature during stabilization of the baby in the delivery room (eg, drying and swaddling, warming pads, increased environmental temperature, placing the baby skin-to-skin with the mother and covering both with a blanket) have been used (LOE 8),^{8,9} but they have not been

(*Circulation*. 2005;112:IV-188-IV-195.)

© 2005 American Heart Association.

This special supplement to *Circulation* is freely available at <http://www.circulationaha.org>

DOI: 10.1161/CIRCULATIONAHA.105.166574

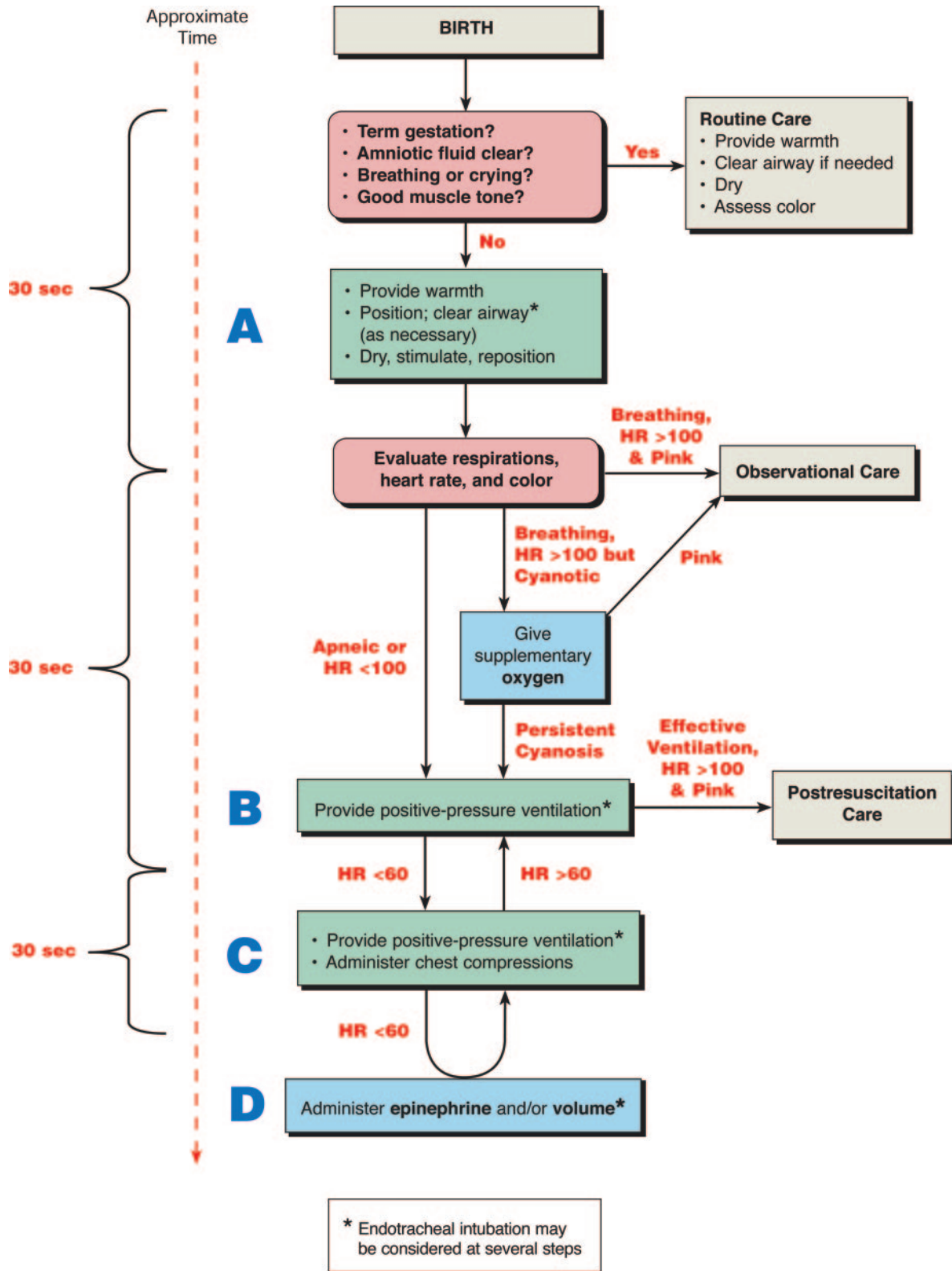


Figure. Neonatal Flow Algorithm.

evaluated in controlled trials nor compared with the plastic wrap technique for premature babies. All resuscitation procedures, including endotracheal intubation, chest compression, and insertion of lines, can be performed with these temperature-controlling interventions in place.

Infants born to febrile mothers have been reported (LOE 4)¹⁰⁻¹² to have a higher incidence of perinatal respiratory depression, neonatal seizures, and cerebral palsy and increased risk of mortality. Animal studies (LOE 6)^{13,14} indicate that hyperthermia during or after ischemia is associated with

progression of cerebral injury. Hyperthermia should be avoided (Class IIb). The goal is to achieve normothermia and avoid iatrogenic hyperthermia.

Clearing the Airway of Meconium

Aspiration of meconium before delivery, during birth, or during resuscitation can cause severe aspiration pneumonia. One obstetrical technique to try to decrease aspiration has been to suction meconium from the infant's airway after delivery of the head but before delivery of the shoulders (intrapartum suctioning). Although some studies (LOE 3¹⁵; 4^{16,17}) suggested that intrapartum suctioning might be effective for decreasing the risk of aspiration syndrome, subsequent evidence from a large multicenter randomized trial (LOE 1)¹⁸ did not show such an effect. Therefore, current recommendations no longer advise routine intrapartum oropharyngeal and nasopharyngeal suctioning for infants born to mothers with meconium staining of amniotic fluid (Class I).

Traditional teaching (LOE 5)^{19–21} recommended that meconium-stained infants have endotracheal intubation immediately following birth and that suction be applied to the endotracheal tube as it is withdrawn. Randomized controlled trials (LOE 1)^{15,22} have shown that this practice offers no benefit if the infant is vigorous (Class I). A vigorous infant is defined as one who has strong respiratory efforts, good muscle tone, and a heart rate >100 beats per minute (bpm). Endotracheal suctioning for infants who are not vigorous should be performed immediately after birth (Class Indeterminate).

Periodic Evaluation at 30-Second Intervals

After the immediate postbirth assessment and administration of initial steps, further resuscitative efforts should be guided by simultaneous assessment of respirations, heart rate, and color. After initial respiratory efforts the newly born infant should be able to establish regular respirations that are sufficient to improve color and maintain a heart rate >100 bpm. Gasping and apnea indicate the need for assisted ventilation.²³ Increasing or decreasing heart rate can also provide evidence of improvement or deterioration.

A newly born infant who is uncompromised will achieve and maintain pink mucous membranes without administration of supplementary oxygen. Evidence obtained with continuous oximetry, however, has shown that neonatal transition is a gradual process. Healthy babies born at term may take >10 minutes to achieve a preductal oxygen saturation >95% and nearly 1 hour to achieve postductal saturation >95% (LOE 5).^{24–26} Central cyanosis is determined by examining the face, trunk, and mucous membranes. Acrocyanosis (blue color of hands and feet alone) is usually a normal finding at birth and is not a reliable indicator of hypoxemia but may indicate other conditions, such as cold stress. Pallor or mottling may be a sign of decreased cardiac output, severe anemia, hypovolemia, hypothermia, or acidosis.

Administration of Oxygen

There are concerns about the potential adverse effects of 100% oxygen on respiratory physiology and cerebral circulation and the potential tissue damage from oxygen free

radicals. Conversely there are also concerns about tissue damage from oxygen deprivation during and after asphyxia. Studies (LOE 6)^{27–31} examining blood pressure, cerebral perfusion, and various biochemical measures of cell damage in asphyxiated animals resuscitated with 100% oxygen versus 21% oxygen (room air) have shown conflicting results. One (LOE 2)³² study of preterm infants (<33 weeks of gestation) exposed to 80% oxygen found lower cerebral blood flow when compared with those stabilized using 21% oxygen. Some animal data (LOE 6)²⁷ indicated the opposite effect, ie, reduced blood pressure and cerebral perfusion with 21% oxygen (room air) versus 100% oxygen. Meta-analysis of 4 human studies (LOE 1)^{33,34} showed a reduction in mortality rate and no evidence of harm in infants resuscitated with room air versus those resuscitated with 100% oxygen, although these results should be viewed with caution because of significant methodological concerns.

Supplementary oxygen is recommended whenever positive-pressure ventilation is indicated for resuscitation; free-flow oxygen should be administered to babies who are breathing but have central cyanosis (Class Indeterminate). The standard approach to resuscitation is to use 100% oxygen. Some clinicians may begin resuscitation with an oxygen concentration of less than 100%, and some may start with no supplementary oxygen (ie, room air). There is evidence that employing either of these practices during resuscitation of neonates is reasonable. If the clinician begins resuscitation with room air, it is recommended that supplementary oxygen be available to use if there is no appreciable improvement within 90 seconds after birth. In situations where supplementary oxygen is not readily available, positive-pressure ventilation should be administered with room air (Class Indeterminate).

Administration of a variable concentration of oxygen guided by pulse oximetry may improve the ability to achieve normoxia more quickly. Concerns about potential oxidant injury should caution the clinician about the use of excessive oxygen, especially in the premature infant.

Positive-Pressure Ventilation

If the infant remains apneic or gasping, if the heart rate remains <100 bpm 30 seconds after administering the initial steps, or if the infant continues to have persistent central cyanosis despite administration of supplementary oxygen, start positive-pressure ventilation.

Initial Breaths and Assisted Ventilation

In term infants, initial inflations—either spontaneous or assisted—create a functional residual capacity (LOE 5).^{35–41} The optimum pressure, inflation time, and flow rate required to establish an effective functional residual capacity have not been determined. Average initial peak inflating pressures of 30 to 40 cm H₂O (inflation time undefined) usually successfully ventilate unresponsive term infants (LOE 5).^{36,38,40–43} Assisted ventilation rates of 40 to 60 breaths per minute are commonly used, but the relative efficacy of various rates has not been investigated.

The primary measure of adequate initial ventilation is prompt improvement in heart rate. Chest wall movement

should be assessed if heart rate does not improve. The initial peak inflating pressures needed are variable and unpredictable and should be individualized to achieve an increase in heart rate and/or movement of the chest with each breath. If inflation pressure is being monitored, an initial inflation pressure of 20 cm H₂O may be effective, but ≥ 30 to 40 cm H₂O may be required in some term babies without spontaneous ventilation (Class IIb). If pressure is not monitored, the minimum inflation required to achieve an increase in heart rate should be used. There is insufficient evidence to recommend an optimum inflation time. In summary, assisted ventilation should be delivered at a rate of 40 to 60 breaths per minute (Class Indeterminate; LOE 8) to promptly achieve or maintain a heart rate >100 bpm.

Devices

Effective ventilation can be achieved with a flow-inflating bag, a self-inflating bag, or with a T-piece (LoE 4^{44,45}; LOE 5⁴⁶). A T-piece is a valved mechanical device designed to control flow and limit pressure. The pop-off valves of self-inflating bags are flow-dependent, and pressures generated may exceed the value specified by the manufacturer (LOE 6).⁴⁷ Target inflation pressures and long inspiratory times are more consistently achieved in mechanical models when T-piece devices are used rather than bags (LOE 6),⁴⁸ although the clinical implications are not clear. To provide the desired pressure, healthcare providers need more training in the use of flow-inflating bags than with self-inflating bags (LOE 6).⁴⁹ A self-inflating bag, a flow-inflating bag, or a T-piece can be used to ventilate a newborn (Class IIb).

Laryngeal mask airways (LMAs) that fit over the laryngeal inlet have been shown to be effective for ventilating newly born near-term and full-term infants (LOE 2⁵⁰ and LOE 5⁵¹). There is limited (LOE 5)^{52,53} data on the use of these devices in small preterm infants. Data from 3 case series (LOE 5)^{51,54,55} shows that the use of the LMA can provide effective ventilation in a time frame consistent with current resuscitation guidelines, although the babies being studied were not being resuscitated. A randomized controlled trial (LOE 2)⁵⁰ found no clinically significant difference between the use of the LMA and endotracheal intubation when bag-mask ventilation was unsuccessful. It is unclear whether this study can be generalized because the LMA was inserted by experienced providers. Case reports (LOE 5)^{56–58} suggest that when bag-mask ventilation has been unsuccessful and endotracheal intubation is not feasible or is unsuccessful, the LMA may provide effective ventilation. There is insufficient evidence to support the routine use of the LMA as the primary airway device during neonatal resuscitation, in the setting of meconium-stained amniotic fluid, when chest compressions are required, in very low birth weight babies, or for delivery of emergency intratracheal medications (Class Indeterminate).

Assisted Ventilation of Preterm Infants

Evidence from animal studies (LOE 6)⁵⁹ indicates that preterm lungs are easily injured by large-volume inflations immediately after birth. Additional animal studies (LOE 6)^{60,61} indicate that when positive-pressure ventilation is applied immediately after birth, the inclusion of positive end-expiratory pressure (PEEP) protects against lung injury

and improves lung compliance and gas exchange (LOE 6).^{60,61} Evidence from case series in human infants indicates that most apneic preterm infants can be ventilated with an initial inflation pressure of 20 to 25 cm H₂O, although some infants who do not respond require a higher pressure (LOE 5).^{62,63}

When ventilating preterm infants after birth, excessive chest wall movement may indicate large-volume lung inflations, which should be avoided. Monitoring of pressure may help to provide consistent inflations and avoid unnecessary high pressures (Class IIb). If positive-pressure ventilation is required, an initial inflation pressure of 20 to 25 cm H₂O is adequate for most preterm infants (Class Indeterminate). If prompt improvement in heart rate or chest movement is not obtained, higher pressures may be needed. If it is necessary to continue positive-pressure ventilation, application of PEEP may be beneficial (Class Indeterminate). Continuous positive airway pressure in spontaneously breathing preterm infants after resuscitation may also be beneficial⁶³ (Class Indeterminate).

Endotracheal Tube Placement

Endotracheal intubation may be indicated at several points during neonatal resuscitation:

- When tracheal suctioning for meconium is required
- If bag-mask ventilation is ineffective or prolonged
- When chest compressions are performed
- When endotracheal administration of medications is desired
- For special resuscitation circumstances, such as congenital diaphragmatic hernia or extremely low birth weight (<1000 g)

The timing of endotracheal intubation may also depend on the skill and experience of the available providers.

After endotracheal intubation and administration of intermittent positive pressure, a prompt increase in heart rate is the best indicator that the tube is in the tracheobronchial tree and providing effective ventilation (LOE 5).⁶⁴ Exhaled CO₂ detection is effective for confirmation of endotracheal tube placement in infants, including very low birth weight infants (LOE 5).^{65–68} A positive test result (detection of exhaled CO₂) in patients with adequate cardiac output confirms placement of the endotracheal tube within the trachea, whereas a negative test result (ie, no CO₂ detected) strongly suggests esophageal intubation (LOE 5).^{65,67} Poor or absent pulmonary blood flow may give false-negative results (ie, no CO₂ detected despite tube placement in the trachea), but endotracheal tube placement is correctly identified in nearly all patients who are not in cardiac arrest (LOE 7).⁶⁹ A false-negative result may also lead to unnecessary extubation in critically ill infants with poor cardiac output.

Other clinical indicators of correct endotracheal tube placement are evaluation of condensed humidified gas during exhalation and the presence or absence of chest movement, but these have not been systematically evaluated in neonates. Endotracheal tube placement must be assessed visually during intubation and by confirmatory methods after intubation if the heart rate remains low and is not rising. Except for

intubation to remove meconium, exhaled CO₂ detection is the recommended method of confirmation (Class IIa).

Chest Compressions

Chest compressions are indicated for a heart rate that is <60 bpm despite adequate ventilation with supplementary oxygen for 30 seconds. Because ventilation is the most effective action in neonatal resuscitation and because chest compressions are likely to compete with effective ventilation, rescuers should ensure that assisted ventilation is being delivered optimally before starting chest compressions.

Compressions should be delivered on the lower third of the sternum^{70,71} to a depth of approximately one third of the anterior-posterior diameter of the chest. Two techniques have been described: compression with 2 thumbs with fingers encircling the chest and supporting the back^{72–74} (the 2 thumb–encircling hands technique) or compression with 2 fingers with a second hand supporting the back. Because the 2 thumb–encircling hands technique may generate higher peak systolic and coronary perfusion pressure than the 2-finger technique (LOE 5⁷⁵; LOE 6⁷⁶), the 2 thumb–encircling hands technique is recommended for performing chest compressions in newly born infants. However, the 2-finger technique may be preferable when access to the umbilicus is required during insertion of an umbilical catheter.

A compression-relaxation ratio with a slightly shorter compression than relaxation phase offers theoretical advantages for blood flow in the very young infant.⁷⁷ Also, compressions and ventilations should be coordinated to avoid simultaneous delivery (LOE 6).⁷⁸ The chest should be permitted to fully reexpand during relaxation, but the rescuer's thumbs should not leave the chest. There should be a 3:1 ratio of compressions to ventilations with 90 compressions and 30 breaths to achieve approximately 120 events per minute to maximize ventilation at an achievable rate (Class Indeterminate). Thus, each event will be allotted approximately ½ second, with exhalation occurring during the first compression after each ventilation.

Respirations, heart rate, and color should be reassessed about every 30 seconds, and coordinated chest compressions and ventilations should continue until the spontaneous heart rate is ≥60 bpm (Class IIa; LOE 8).

Medications

Drugs are rarely indicated in resuscitation of the newly born infant.⁷⁹ Bradycardia in the newborn infant is usually the result of inadequate lung inflation or profound hypoxemia, and establishing adequate ventilation is the most important step to correct it. But if the heart rate remains <60 bpm despite adequate ventilation with 100% oxygen and chest compressions, administration of epinephrine or volume expansion, or both, may be indicated. Rarely, buffers, a narcotic antagonist, or vasopressors may be useful after resuscitation.

Route and Dose of Epinephrine Administration

Past guidelines recommended that initial doses of epinephrine be given through an endotracheal tube because the dose can be administered more quickly than when an intravenous route must be established. But animal studies (LOE 6)^{80–82} that

showed a positive effect of endotracheal epinephrine used considerably higher doses than are currently recommended, and the one animal study (LOE 6)⁸³ that used currently recommended doses given endotracheally showed no effect. Given the lack of data on endotracheal epinephrine, the IV route should be used as soon as venous access is established.

The recommended IV dose is 0.01 to 0.03 mg/kg per dose. Higher IV doses are not recommended (Class III) because animal (LOE 6)^{84,85} and pediatric (LOE 7)⁸⁶ studies show exaggerated hypertension, decreased myocardial function, and worse neurologic function after administration of IV doses in the range of 0.1 mg/kg. If the endotracheal route is used, doses of 0.01 or 0.03 mg/kg will likely be ineffective. Therefore, IV administration of 0.01 to 0.03 mg/kg per dose is the preferred route (Class IIa). While access is being obtained, administration of a higher dose (up to 0.1 mg/kg) through the endotracheal tube may be considered (Class Indeterminate), but the safety and efficacy of this practice have not been evaluated. The concentration of epinephrine for either route should be 1:10 000 (0.1 mg/mL).

Volume Expansion

Consider volume expansion when blood loss is suspected or the infant appears to be in shock (pale skin, poor perfusion, weak pulse) and has not responded adequately to other resuscitative measures. An isotonic crystalloid rather than albumin is the solution of choice for volume expansion in the delivery room (Class IIb; LOE 7).^{87–89} The recommended dose is 10 mL/kg, which may need to be repeated. When resuscitating premature infants, care should be taken to avoid giving volume expanders too rapidly, because rapid infusions of large volumes have been associated with intraventricular hemorrhage.

Naloxone

Administration of naloxone is not recommended as part of initial resuscitative efforts in the delivery room for newborns with respiratory depression. If administration of naloxone is considered, heart rate and color must first be restored by supporting ventilation. The preferred route is IV or intramuscular. Given the lack of clinical data in newborns, endotracheal administration of naloxone is not recommended (Class Indeterminate). The recommended dose is 0.1 mg/kg, but no studies have examined the efficacy of this dose in newborns. In one case report, naloxone given to a baby born to an opioid-addicted mother was associated with seizures (LOE 8).⁹⁰ Therefore, naloxone should be avoided in babies whose mothers are suspected of having had long-term exposure to opioids (Class Indeterminate). Naloxone may have a shorter half-life than the original maternal opioid; therefore the neonate should be monitored closely for recurrent apnea or hypoventilation, and subsequent doses of naloxone may be required.

Postresuscitation Care

Babies who require resuscitation are at risk for deterioration after their vital signs have returned to normal. Once adequate ventilation and circulation have been established, the infant should be maintained in or transferred to an environment in which close monitoring and anticipatory care can be provided.

Glucose

Low blood glucose has been associated with adverse neurologic outcome in a neonatal animal model of asphyxia and resuscitation (LOE 6).⁹¹ Neonatal animals (LOE 6)^{92,93} that were hypoglycemic at the time of an anoxic or hypoxic-ischemic insult had larger areas of cerebral infarction or decreased survival, or both, when compared with controls. One clinical study (LOE 4)⁹⁴ showed an association between hypoglycemia and poor neurologic outcome after perinatal asphyxia.

No clinical neonatal studies have investigated the relation between hyperglycemia and neurologic outcome, although hyperglycemia in adults (LOE 7 [extrapolated]⁹⁵) is associated with worse outcome. The range of blood glucose concentration associated with the least brain injury after asphyxia and resuscitation cannot be defined based on available evidence. Infants who require significant resuscitation should be monitored and treated to maintain glucose in the normal range (Class Indeterminate).

Induced Hypothermia

In a multicenter trial (LOE 2)⁹⁶ involving newborns with suspected asphyxia (indicated by need for resuscitation at birth, metabolic acidosis, and early encephalopathy), selective head cooling (34°C to 35°C) was associated with a nonsignificant reduction in the overall number of survivors with severe disability at 18 months but a significant benefit in the subgroup with moderate encephalopathy. Infants with severe electrographic suppression and seizures did not benefit from treatment with modest hypothermia (LOE 2).⁹⁶ A second large multicenter trial (LOE 2)⁹⁷ of asphyxiated newborns (indicated by need for resuscitation at birth or presence of metabolic encephalopathy) involved treatment with systemic hypothermia to 33.5°C (92.3°F) following moderate to severe encephalopathy. Hypothermia was associated with a significant (18%) decrease in death or moderate disability at 18 months.⁹⁷ A third small controlled pilot study (LOE 2)^{98,99} in asphyxiated infants with early induced systemic hypothermia found fewer deaths and disability at 12 months.

Modest hypothermia is associated with bradycardia and elevated blood pressure that do not usually require treatment, but a rapid increase in body temperature may cause hypotension (LOE 5).¹⁰⁰ Cooling to a core temperature <33°C may cause arrhythmia, bleeding, thrombosis, and sepsis, but studies so far have not reported these complications in infants treated with modest (eg, 33°C to 34.5°C [91.4°F to 94.1°F]) hypothermia (LOE 2).^{96,101}

There is insufficient data to recommend routine use of modest systemic or selective cerebral hypothermia after resuscitation of infants with suspected asphyxia (Class Indeterminate). Further clinical trials are needed to determine which infants benefit most and which method of cooling is most effective. Avoidance of hyperthermia (elevated body temperature) is particularly important in babies who may have had a hypoxic-ischemic event.

Guidelines for Withholding and Discontinuing Resuscitation

Morbidity and mortality for newborns varies according to region and availability of resources (LOE 5).¹⁰² Social science studies¹⁰³ indicate that parents desire a larger role in decisions to initiate resuscitation and continue life support of

severely compromised newborns. Opinions among neonatal providers vary widely regarding the benefits and disadvantages of aggressive therapies in such newborns (LOE 5).¹⁰⁴

Withholding Resuscitation

It is possible to identify conditions associated with high mortality and poor outcome in which withholding resuscitative efforts may be considered reasonable, particularly when there has been the opportunity for parental agreement (LOE 5).^{2,105}

A consistent and coordinated approach to individual cases by the obstetric and neonatal teams and the parents is an important goal. Noninitiation of resuscitation and discontinuation of life-sustaining treatment during or after resuscitation are ethically equivalent, and clinicians should not hesitate to withdraw support when functional survival is highly unlikely. The following guidelines must be interpreted according to current regional outcomes:

- When gestation, birth weight, or congenital anomalies are associated with almost certain early death and when unacceptably high morbidity is likely among the rare survivors, resuscitation is not indicated (Class IIa). Examples may include extreme prematurity (gestational age <23 weeks or birth weight <400 g), anencephaly, and chromosomal abnormalities incompatible with life, such as trisomy 13.
- In conditions associated with a high rate of survival and acceptable morbidity, resuscitation is nearly always indicated (Class IIa). This will generally include babies with gestational age ≥25 weeks (unless there is evidence of fetal compromise such as intrauterine infection or hypoxia-ischemia) and those with most congenital malformations.
- In conditions associated with uncertain prognosis in which survival is borderline, the morbidity rate is relatively high, and the anticipated burden to the child is high, parental desires concerning initiation of resuscitation should be supported (Class Indeterminate).

Discontinuing Resuscitative Efforts

Infants without signs of life (no heart beat and no respiratory effort) after 10 minutes of resuscitation show either a high mortality or severe neurodevelopmental disability (LOE 5).^{106,107} After 10 minutes of continuous and adequate resuscitative efforts, discontinuation of resuscitation may be justified if there are no signs of life (Class IIb).

References

1. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. In: Gilstrap LC, Oh W, eds. *Guidelines for Perinatal Care*. 5th ed. Elk Grove Village, Ill: American Academy of Pediatrics. 2002:187.
2. Costeloe K, Hennessy E, Gibson AT, Marlow N, Wilkinson AR. The EPICure study: outcomes to discharge from hospital for infants born at the threshold of viability. *Pediatrics*. 2000;106:659–671.
3. Vohra S, Frent G, Campbell V, Abbott M, Whyte R. Effect of polyethylene occlusive skin wrapping on heat loss in very low birth weight infants at delivery: a randomized trial. *J Pediatr*. 1999;134:547–551.
4. Vohra S, Roberts RS, Zhang B, Janes M, Schmidt B. Heat Loss Prevention (HeLP) in the delivery room: a randomized controlled trial of polyethylene occlusive skin wrapping in very preterm infants. *J Pediatr*. 2004;145:750–753.
5. Lyon AJ, Stenson B. Cold comfort for babies. *Arch Dis Child Fetal Neonatal Ed*. 2004;89:F93–F94.

6. Lenclen R, Mazraani M, Jugie M, Couderc S, Hoenn E, Carbajal R, Blanc P, Paupe A. Use of a polyethylene bag: a way to improve the thermal environment of the premature newborn at the delivery room. *Arch Pediatr*. 2002;9:238–244.
7. Bjorklund LJ, Hellstrom-Westas L. Reducing heat loss at birth in very preterm infants. *J Pediatr*. 2000;137:739–740.
8. Baum JD, Scopes JW. The silver swaddler: device for preventing hypothermia in the newborn. *Lancet*. 1968;1:672–673.
9. Besch NJ, Perlstein PH, Edwards NK, Keenan WJ, Sutherland JM. The transparent baby bag: a shield against heat loss. *N Engl J Med*. 1971;284:121–124.
10. Petrova A, Demissie K, Rhoads GG, Smulian JC, Marcella S, Ananth CV. Association of maternal fever during labor with neonatal and infant morbidity and mortality. *Obstet Gynecol*. 2001;98:20–27.
11. Lieberman E, Lang J, Richardson DK, Frigoletto FD, Heffner LJ, Cohen A. Intrapartum maternal fever and neonatal outcome. *Pediatrics*. 2000;105:8–13.
12. Grether JK, Nelson KB. Maternal infection and cerebral palsy in infants of normal birth weight. *JAMA*. 1997;278:207–211.
13. Coimbra C, Boris-Moller F, Drake M, Wieloch T. Diminished neuronal damage in the rat brain by late treatment with the antipyretic drug dipyron or cooling following cerebral ischemia. *Acta Neuropathol (Berl)*. 1996;92:447–453.
14. Dietrich WD, Alonso O, Halley M, Busto R. Delayed posttraumatic brain hyperthermia worsens outcome after fluid percussion brain injury: a light and electron microscopic study in rats. *Neurosurgery*. 1996;38:533–541; discussion 541.
15. Wiswell TE, Gannon CM, Jacob J, Goldsmith L, Szyld E, Weiss K, Schutzman D, Cleary GM, Filipov P, Kurlat I, Caballero CL, Abassi S, Sprague D, Oltorf C, Padula M. Delivery room management of the apparently vigorous meconium-stained neonate: results of the multicenter, international collaborative trial. *Pediatrics*. 2000;105:1–7.
16. Falciglia HS, Henderschott C, Potter P, Helmchen R. Does DeLee suction at the perineum prevent meconium aspiration syndrome? *Am J Obstet Gynecol*. 1992;167:1243–1249.
17. Carson BS, Losey RW, Bowes WA Jr, Simmons MA. Combined obstetric and pediatric approach to prevent meconium aspiration syndrome. *Am J Obstet Gynecol*. 1976;126:712–715.
18. Vain NE, Szyld EG, Prudent LM, Wiswell TE, Aguilar AM, Vivas NI. Oropharyngeal and nasopharyngeal suctioning of meconium-stained neonates before delivery of their shoulders: multicentre, randomised controlled trial. *Lancet*. 2004;364:597–602.
19. Gregory GA, Gooding CA, Phibbs RH, Tooley WH. Meconium aspiration in infants: a prospective study. *J Pediatr*. 1974;85:848–852.
20. Rossi EM, Philipson EH, Williams TG, Kalhan SC. Meconium aspiration syndrome: intrapartum and neonatal attributes. *Am J Obstet Gynecol*. 1989;161:1106–1110.
21. Davis RO, Philips JB III, Harris BA Jr, Wilson ER, Huddleston JF. Fatal meconium aspiration syndrome occurring despite airway management considered appropriate. *Am J Obstet Gynecol*. 1985;151:731–736.
22. Halliday HL. Endotracheal intubation at birth for preventing morbidity and mortality in vigorous, meconium-stained infants born at term. *Cochrane Database Syst Rev*. 2001;CD000500.
23. Dawes GS. *Foetal and Neonatal Physiology. A Comparative Study of the Changes at Birth*. Chicago, Ill: Year Book Medical Publishers Inc; 1968.
24. Harris AP, Sendak MJ, Donham RT. Changes in arterial oxygen saturation immediately after birth in the human neonate. *J Pediatr*. 1986;109:117–119.
25. Reddy VK, Holzman IR, Wedgwood JF. Pulse oximetry saturations in the first 6 hours of life in normal term infants. *Clin Pediatr (Phila)*. 1999;38:87–92.
26. Toth B, Becker A, Seelbach-Gobel B. Oxygen saturation in healthy newborn infants immediately after birth measured by pulse oximetry. *Arch Gynecol Obstet*. 2002;266:105–107.
27. Solas AB, Kutzsche S, Vinje M, Saugstad OD. Cerebral hypoxemia-ischemia and reoxygenation with 21% or 100% oxygen in newborn piglets: effects on extracellular levels of excitatory amino acids and microcirculation. *Pediatr Crit Care Med*. 2001;2:340–345.
28. Solas AB, Munkeby BH, Saugstad OD. Comparison of short- and long-duration oxygen treatment after cerebral asphyxia in newborn piglets. *Pediatr Res*. 2004;56:125–131.
29. Solas AB, Kalous P, Saugstad OD. Reoxygenation with 100 or 21% oxygen after cerebral hypoxemia-ischemia-hypercapnia in newborn piglets. *Biol Neonate*. 2004;85:105–111.
30. Huang CC, Yonetani M, Lajevardi N, Delivoria-Papadopoulos M, Wilson DF, Pastuszko A. Comparison of postasphyxial resuscitation with 100% and 21% oxygen on cortical oxygen pressure and striatal dopamine metabolism in newborn piglets. *J Neurochem*. 1995;64:292–298.
31. Kutzsche S, Kirkeby OJ, Rise IR, Saugstad OD. Effects of hypoxia and reoxygenation with 21% and 100%-oxygen on cerebral nitric oxide concentration and microcirculation in newborn piglets. *Biol Neonate*. 1999;76:153–167.
32. Lundstrom KE, Pryds O, Greisen G. Oxygen at birth and prolonged cerebral vasoconstriction in preterm infants. *Arch Dis Child Fetal Neonatal Ed*. 1995;73:F81–F86.
33. Tan A, Schulze A, O'Donnell CP, Davis PG. Air versus oxygen for resuscitation of infants at birth. *Cochrane Database Syst Rev*. 2005;CD002273.
34. Davis PG, Tan A, O'Donnell CP, Schulze A. Resuscitation of newborn infants with 100% oxygen or air: a systematic review and meta-analysis. *Lancet*. 2004;364:1329–1333.
35. Karlberg P, Koch G. Respiratory studies in newborn infants, III: development of mechanics of breathing during the first week of life. A longitudinal study. *Acta Paediatr*. 1962;(suppl 135):121–129.
36. Vyas H, Milner AD, Hopkin IE, Boon AW. Physiologic responses to prolonged and slow-rise inflation in the resuscitation of the asphyxiated newborn infant. *J Pediatr*. 1981;99:635–639.
37. Vyas H, Field D, Milner AD, Hopkin IE. Determinants of the first inspiratory volume and functional residual capacity at birth. *Pediatr Pulmonol*. 1986;2:189–193.
38. Boon AW, Milner AD, Hopkin IE. Lung expansion, tidal exchange, and formation of the functional residual capacity during resuscitation of asphyxiated neonates. *J Pediatr*. 1979;95:1031–1036.
39. Mortola JP, Fisher JT, Smith JB, Fox GS, Weeks S, Willis D. Onset of respiration in infants delivered by cesarean section. *J Appl Physiol*. 1982;52:716–724.
40. Hull D. Lung expansion and ventilation during resuscitation of asphyxiated newborn infants. *J Pediatr*. 1969;75:47–58.
41. Upton CJ, Milner AD. Endotracheal resuscitation of neonates using a rebreathing bag. *Arch Dis Child*. 1991;66:39–42.
42. Boon AW, Milner AD, Hopkin IE. Physiological responses of the newborn infant to resuscitation. *Arch Dis Child*. 1979;54:492–498.
43. Milner AD, Vyas H, Hopkin IE. Efficacy of facemask resuscitation at birth. *BMJ*. 1984;289:1563–1565.
44. Allwood AC, Madar RJ, Baumer JH, Readdy L, Wright D. Changes in resuscitation practice at birth. *Arch Dis Child Fetal Neonatal Ed*. 2003;88:F375–F379.
45. Hoskyns EW, Milner AD, Hopkin IE. A simple method of face mask resuscitation at birth. *Arch Dis Child*. 1987;62:376–378.
46. Cole AF, Rolbin SH, Hew EM, Pynn S. An improved ventilator system for delivery-room management of the newborn. *Anesthesiology*. 1979;51:356–358.
47. Ganga-Zandzou PS, Diependaele JF, Storme L, Riou Y, Klosowski S, Rakza T, Logier R, Lequien P. Is Ambu ventilation of newborn infants a simple question of finger-touch? *Arch Pediatr*. 1996;3:1270–1272.
48. Finer NN, Rich W, Craft A, Henderson C. Comparison of methods of bag and mask ventilation for neonatal resuscitation. *Resuscitation*. 2001;49:299–305.
49. Kanter RK. Evaluation of mask-bag ventilation in resuscitation of infants. *Am J Dis Child*. 1987;141:761–763.
50. Esmail N, Saleh M, Ali A. Laryngeal mask airway versus endotracheal intubation for Apgar score improvement in neonatal resuscitation. *Egyptian J Anesthesiol*. 2002;18:115–121.
51. Gandini D, Brimacombe JR. Neonatal resuscitation with the laryngeal mask airway in normal and low birth weight infants. *Anesth Analg*. 1999;89:642–643.
52. Brimacombe J, Gandini D. Airway rescue and drug delivery in an 800 g neonate with the laryngeal mask airway. *Paediatr Anaesth*. 1999;9:178.
53. Lonnqvist PA. Successful use of laryngeal mask airway in low-weight preterm infants with bronchopulmonary dysplasia undergoing cryotherapy for retinopathy of the premature. *Anesthesiology*. 1995;83:422–424.
54. Paterson SJ, Byrne PJ, Moleky MG, Seal RF, Finucane BT. Neonatal resuscitation using the laryngeal mask airway. *Anesthesiology*. 1994;80:1248–1253.
55. Trevisanuto D, Ferrarese P, Zanardo V, Chiandetti L. Laryngeal mask airway in neonatal resuscitation: a survey of current practice and perceived role by anaesthesiologists and paediatricians. *Resuscitation*. 2004;60:291–296.
56. Hansen TG, Joensen H, Henneberg SW, Hole P. Laryngeal mask airway guided tracheal intubation in a neonate with the Pierre Robin syndrome. *Acta Anaesthesiol Scand*. 1995;39:129–131.
57. Osses H, Poblete M, Asenjo F. Laryngeal mask for difficult intubation in children. *Paediatr Anaesth*. 1999;9:399–401.

58. Stocks RM, Egerman R, Thompson JW, Peery M. Airway management of the severely retrognathic child: use of the laryngeal mask airway. *Ear Nose Throat J*. 2002;81:223–226.
59. Ingimarsson J, Bjorklund LJ, Curstedt T, Gudmundsson S, Larsson A, Robertson B, Werner O. Incomplete protection by prophylactic surfactant against the adverse effects of large lung inflations at birth in immature lambs. *Intensive Care Med*. 2004;30:1446–1453.
60. Nilsson R, Grossmann G, Robertson B. Bronchiolar epithelial lesions induced in the premature rabbit neonate by short periods of artificial ventilation. *Acta Pathol Microbiol Scand [A]*. 1980;88:359–367.
61. Probyn ME, Hooper SB, Dargaville PA, McCallion N, Crossley K, Harding R, Morley CJ. Positive end expiratory pressure during resuscitation of premature lambs rapidly improves blood gases without adversely affecting arterial pressure. *Pediatr Res*. 2004;56:198–204.
62. Hird MF, Greenough A, Gamsu HR. Inflating pressures for effective resuscitation of preterm infants. *Early Hum Dev*. 1991;26:69–72.
63. Lindner W, Vossbeck S, Hummler H, Pohlandt F. Delivery room management of extremely low birth weight infants: spontaneous breathing or intubation? *Pediatrics*. 1999;103:961–967.
64. Palme-Kilander C, Tunell R. Pulmonary gas exchange during facemask ventilation immediately after birth. *Arch Dis Child*. 1993;68:11–16.
65. Aziz HF, Martin JB, Moore JJ. The pediatric disposable end-tidal carbon dioxide detector role in endotracheal intubation in newborns. *J Perinatol*. 1999;19:110–113.
66. Bhende MS, Thompson AE. Evaluation of an end-tidal CO₂ detector during pediatric cardiopulmonary resuscitation. *Pediatrics*. 1995;95:395–399.
67. Repetto JE, Donohue P-CP, Baker SF, Kelly L, Noguee LM. Use of capnography in the delivery room for assessment of endotracheal tube placement. *J Perinatol*. 2001;21:284–287.
68. Roberts WA, Maniscalco WM, Cohen AR, Litman RS, Chhibber A. The use of capnography for recognition of esophageal intubation in the neonatal intensive care unit. *Pediatr Pulmonol*. 1995;19:262–268.
69. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. *Am J Emerg Med*. 1996;14:349–350.
70. Orłowski JP. Optimum position for external cardiac compression in infants and young children. *Ann Emerg Med*. 1986;15:667–673.
71. Phillips GW, Zideman DA. Relation of infant heart to sternum: its significance in cardiopulmonary resuscitation. *Lancet*. 1986;1:1024–1025.
72. Thaler MM, Stobie GH. An improved technique of external cardiac compression in infants and young children. *N Engl J Med*. 1963;269:606–610.
73. David R. Closed chest cardiac massage in the newborn infant. *Pediatrics*. 1988;81:552–554.
74. Todres ID, Rogers MC. Methods of external cardiac massage in the newborn infant. *J Pediatr*. 1975;86:781–782.
75. Menegazzi JJ, Auble TE, Nicklas KA, Hosack GM, Rack L, Goode JS. Two-thumb versus two-finger chest compression during CRP in a swine infant model of cardiac arrest. *Ann Emerg Med*. 1993;22:240–243.
76. Houry PK, Frank LR, Menegazzi JJ, Taylor R. A randomized, controlled trial of two-thumb vs two-finger chest compression in a swine infant model of cardiac arrest. *Prehosp Emerg Care*. 1997;1:65–67.
77. Dean JM, Koehler RC, Schlieen CL, Berkowitz I, Michael JR, Atchison D, Rogers MC, Traystman RJ. Age-related effects of compression rate and duration in cardiopulmonary resuscitation. *J Appl Physiol*. 1990;68:554–560.
78. Berkowitz ID, Chantarojanasiri T, Koehler RC, Schlieen CL, Dean JM, Michael JR, Rogers MC, Traystman RJ. Blood flow during cardiopulmonary resuscitation with simultaneous compression and ventilation in infant pigs. *Pediatr Res*. 1989;26:558–564.
79. Perlman JM, Risser R. Cardiopulmonary resuscitation in the delivery room: associated clinical events. *Arch Pediatr Adolesc Med*. 1995;149:20–25.
80. Ralston SH, Voorhees WD, Babbs CF. Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: improved regional blood flow and resuscitation in dogs. *Ann Emerg Med*. 1984;13:79–86.
81. Ralston SH, Tacker WA, Showen L, Carter A, Babbs CF. Endotracheal versus intravenous epinephrine during electromechanical dissociation with CPR in dogs. *Ann Emerg Med*. 1985;14:1044–1048.
82. Redding JS, Pearson JW. Metabolic acidosis: a factor in cardiac resuscitation. *South Med J*. 1967;60:926–932.
83. Kleinman ME, Oh W, Stonestreet BS. Comparison of intravenous and endotracheal epinephrine during cardiopulmonary resuscitation in newborn piglets. *Crit Care Med*. 1999;27:2748–2754.
84. Berg RA, Otto CW, Kern KB, Hilwig RW, Sanders AB, Henry CP, Ewy GA. A randomized, blinded trial of high-dose epinephrine versus standard-dose epinephrine in a swine model of pediatric asphyxial cardiac arrest. *Crit Care Med*. 1996;24:1695–1700.
85. Burchfield DJ, Preziosi MP, Lucas VW, Fan J. Effects of graded doses of epinephrine during asphyxia-induced bradycardia in newborn lambs. *Resuscitation*. 1993;25:235–244.
86. Perondi MB, Reis AG, Paiva EF, Nadkarni VM, Berg RA. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. *N Engl J Med*. 2004;350:1722–1730.
87. So KW, Fok TF, Ng PC, Wong WW, Cheung KL. Randomised controlled trial of colloid or crystalloid in hypotensive preterm infants. *Arch Dis Child Fetal Neonatal Ed*. 1997;76:F43–F46.
88. Emery EF, Greenough A, Gamsu HR. Randomised controlled trial of colloid infusions in hypotensive preterm infants. *Arch Dis Child*. 1992;67:1185–1188.
89. Oca MJ, Nelson M, Donn SM. Randomized trial of normal saline versus 5% albumin for the treatment of neonatal hypotension. *J Perinatol*. 2003;23:473–476.
90. Gibbs J, Newson T, Williams J, Davidson DC. Naloxone hazard in infant of opioid abuser. *Lancet*. 1989;2:159–160.
91. Brambrink AM, Ichord RN, Martin LJ, Koehler RC, Traystman RJ. Poor outcome after hypoxia-ischemia in newborns is associated with physiological abnormalities during early recovery: possible relevance to secondary brain injury after head trauma in infants. *Exp Toxicol Pathol*. 1999;51:151–162.
92. Vannucci RC, Vannucci SJ. Cerebral carbohydrate metabolism during hypoglycemia and anoxia in newborn rats. *Ann Neurol*. 1978;4:73–79.
93. Yager JY, Heitjan DF, Towfighi J, Vannucci RC. Effect of insulin-induced and fasting hypoglycemia on perinatal hypoxic-ischemic brain damage. *Pediatr Res*. 1992;31:138–142.
94. Salhab WA, Wyckoff MH, Laptook AR, Perlman JM. Initial hypoglycemia and neonatal brain injury in term infants with severe fetal acidemia. *Pediatrics*. 2004;114:361–366.
95. Kent TA, Soukup VM, Fabian RH. Heterogeneity affecting outcome from acute stroke therapy: making reperfusion worse. *Stroke*. 2001;32:2318–2327.
96. Gluckman PD, Wyatt JS, Azzopardi D, Ballard R, Edwards AD, Ferriero DM, Polin RA, Robertson CM, Thoresen M, Whitelaw A, Gunn AJ. Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomised trial. *Lancet*. 2005;365:663–670.
97. Donovan EF, Fanaroff AA, Poole WK, Wright LL, Higgins RD, Finer NN, Carlo WA, Duara S, Oh W, Cotten CM, Stevenson DK, Stoll BJ, Lemons JA, Guillet R, Jobe AH. Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy. *N Engl J Med*. 2005;353:1574–1584.
98. Eicher DJ, Wagner CL, Katikaneni LP, Hulsey TC, Bass WT, Kaufman DA, Horgan MJ, Languani S, Bhatia JJ, Givelichian LM, Sankaran K, Yager JY. Moderate hypothermia in neonatal encephalopathy: safety outcomes. *Pediatr Neurol*. 2005;32:18–24.
99. Eicher DJ, Wagner CL, Katikaneni LP, Hulsey TC, Bass WT, Kaufman DA, Horgan MJ, Languani S, Bhatia JJ, Givelichian LM, Sankaran K, Yager JY. Moderate hypothermia in neonatal encephalopathy: efficacy outcomes. *Pediatr Neurol*. 2005;32:11–17.
100. Thoresen M, Whitelaw A. Cardiovascular changes during mild therapeutic hypothermia and rewarming in infants with hypoxic-ischemic encephalopathy. *Pediatrics*. 2000;106:92–99.
101. Shankaran S, Laptook A, Wright LL, Ehrenkranz RA, Donovan EF, Fanaroff AA, Stark AR, Tyson JE, Poole K, Carlo WA, Lemons JA, Oh W, Stoll BJ, Papile LA, Bauer CR, Stevenson DK, Korones SB, McDonald S. Whole-body hypothermia for neonatal encephalopathy: animal observations as a basis for a randomized, controlled pilot study in term infants. *Pediatrics*. 2002;110:377–385.
102. De Leeuw R, Cuttini M, Nadai M, Berik I, Hansen G, Kucinkas A, Lenoir S, Levin A, Persson J, Rebagliato M, Reid M, Schroell M, de Vonderweid U. Treatment choices for extremely preterm infants: an international perspective. *J Pediatr*. 2000;137:608–616.
103. Lee SK, Penner PL, Cox M. Comparison of the attitudes of health care professionals and parents toward active treatment of very low birth weight infants. *Pediatrics*. 1991;88:110–114.
104. Kopelman LM, Irons TG, Kopelman AE. Neonatologists judge the “Baby Doe” regulations. *N Engl J Med*. 1988;318:677–683.
105. Draper ES, Manktelow B, Field DJ, James D. Tables for predicting survival for preterm births are updated. *BMJ*. 2003;327:872.
106. Jain L, Ferre C, Vidyasagar D, Nath S, Sheftel D. Cardiopulmonary resuscitation of apparently stillborn infants: survival and long-term outcome. *J Pediatr*. 1991;118:778–782.
107. Haddad B, Mercer BM, Livingston JC, Talati A, Sibai BM. Outcome after successful resuscitation of babies born with apgar scores of 0 at both 1 and 5 minutes. *Am J Obstet Gynecol*. 2000;182:1210–1214.

Part 13: Neonatal Resuscitation Guidelines

Circulation. 2005;112:IV-188-IV-195; originally published online November 28, 2005;

doi: 10.1161/CIRCULATIONAHA.105.166574

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2005 American Heart Association, Inc. All rights reserved.

Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:

http://circ.ahajournals.org/content/112/24_suppl/IV-188

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Circulation* is online at:
<http://circ.ahajournals.org/subscriptions/>