Neonatal Resuscitation

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Following birth, numerous physiologic changes must rapidly transpire in order for the fetus to successfully make the transition to a neonate. Despite the complexity of this process, on average ten percent of newborns require some assistance to begin breathing and only one percent of newborns require full resuscitation in the delivery room.[1] This percentage rises quickly, however, among newborns who weigh less than 1500 grams. Given the relative rarity of the need for full resuscitation, it is optimal for all delivery room personnel to frequently review the neonatal adaptations to extrauterine life, make provision for resuscitation, understand the predictors of need for resuscitation and know how to respond appropriately.

Neonatal Adaptations to Extrauterine Life[2,3] The placenta is the organ for gas exchange in the fetus. A substantial right-to-left shunt exists. The shunt persists due to high pulmonary vascular resistance (PVR) coupled with low systemic vascular resistance (SVR). Ninety percent of right ventricular output shunts across the ductus arteriosus. Forty percent of cardiac output flows to the low resistance placenta. During vaginal delivery, compression of the infant thorax expels fluid from the mouth and upper airways. With crying, the lungs fill with air, surfactant is released and oxygenation increased. In animal models, the presence of increased oxygen tension and increased blood flow stimulate endothelial cells in the pulmonary vasculature to release nitric oxide ultimately resulting in pulmonary vasodilatation.[4] These changes significantly decrease PVR. Simultaneously, clamping of the umbilical cord removes the low resistance placental bed from the circulation, increasing SVR. Within minutes after birth the right-to-left shunt across the foramen ovale and ductus arteriosus is substantially reduced. Transient hypoxemia or acidosis is well tolerated by a normal newborn and prompt resuscitation usually prevents any permanent physiologic alteration. Prolonged neonatal hypoxemia or acidosis impedes the transition from fetal to neonatal physiology. The fetus/neonate initially responds to hypoxemia by redistributing blood flow to the heart, brain and adrenal glands. Tissue oxygen extraction increases to the maximum extent possible. Eventually, as additional oxygen extraction is unattainable, myocardial contractility and cardiac output decrease. Hypoxemia and acidosis promote patency of the ductus arteriosus, counteracting the normal neonatal increase in pulmonary artery blood flow. Blood flowing through a patent ductus arteriosus is not oxygenated, contributing to increasing hypoxemia. Spontaneous ventilatory drive is reduced by both indirect central nervous system depression and direct diaphragmatic depression. The net result of these physiological responses is a neonate with persistent pulmonary hypertension and little or no ventilatory drive. Ideally, prompt resuscitation prevents these physiologic perturbations.

Preparation for Resuscitation Preparation for neonatal resuscitation is an ongoing activity on all labor and delivery units. A number of tasks including acquisition and maintenance of the proper equipment, identification, education and training of responding personnel and development of contingency plans for additional personnel if needed must be constantly monitored for completeness. Equipment and medications should be organized together in one location in the delivery room, checked frequently for proper functioning and expiration date, and replenished immediately after use (Table 1).[1]

At least one person skilled in newborn resuscitation should attend every delivery. Additional personnel should be available when a high-risk delivery is anticipated. The importance of trained personnel following protocol driven maneuvers is critical. The need for anesthesia personal to participate in neonatal resuscitation is likely to increase as pediatric residents spend more time in primary care training and less in neonatology. A recent survey of the proficiency of third year pediatric residents at performing neonatal endotracheal intubation (n=131 observed intubation attempts) found that intubation was successful on the first or second attempt by only 62% of these residents.[5] The technical competency of pediatricians at

Table 1. Equipment & Medications for Neonatal Resuscitation

Suction Equipment	**Bag & Mask Equipment**
Bulb syringe	Neonatal resuscitation bag with pressure relief valve
Mechanical suction	Face masks - newborn & premature sizes
Suction catheters 5F - 10F	Oral airways
Meconium Aspirator	Oxygen with flowmeter & tubing
Intubation Equipment	**Medications**
Laryngoscope	Epinephrine 1:10,000
Straight blades #0 and #1	Naloxone hydrochloride 0.4 mg/mL or 1.0 mg/mL
Extra bulbs & batteries	Volume expander
Endotracheal tubes 2.5 - 4.0 mm	Sodium bicarbonate 4.2% (5 mEq/10 mL
Stylet	Dextrose 10%
Scissors & gloves	Sterile water & Normal saline
Miscellaneous	
Radiant warmer	Umbilical artery catheterization tray
Stethoscope	Umbilical tape
ECG	Umbilical catheters 3.5F, 5F
Adhesive tape	Three-way stopcocks
Syringes & needles	Feeding tube, 5F
Alcohol sponges	

performing neonatal endotracheal intubation should be assessed on an individual basis and not assumed to be present by the anesthesia team.

In determining need for personnel trained in neonatal resuscitation, anesthesiologists in the United States can refer to Guideline VII of the American Society of Anesthesiology, Guidelines for Regional Anesthesia in Obstetrics. The guideline states: "Qualified personnel, other than the anesthesiologist attending the mother, should be immediately available to assume responsibility for resuscitation of the newborn. The primary responsibility of the anesthesiologist is to provide care to the mother. If the anesthesiologist is also requested to provide brief assistance in the care of the newborn, the benefit to the child must be compared to the risk to the mother."

Assessment of Risk By using ante- and intrapartum fetal assessment, the need for neonatal resuscitation can be predicted in about 80% of cases. Antepartum assessment includes evaluation for major fetal anomalies and identification of maternal factors that may influence fetal well being (Table 2).[1,2] Intrapartum events often predict the need for neonatal resuscitation (Table 3).[1,6] Assessment must continue throughout labor as the clinical situation can change rapidly. Intrapartum evaluation includes fetal heart rate monitoring with, when indicated, fetal scalp stimulation or fetal scalp blood sampling for pH determination. Intrapartum fetal heart rate (FHR) monitoring is the first line of fetal assessment.[7] FHR monitoring is most reliable in confirming fetal well-being and is more than 90% accurate in predicting a 5 minute Apgar score greater than 7.[8,9] In predicting fetal compromise, however, FHR monitoring has a false positive rate of at least 35-50%.[9,10] Even though an abnormal fetal heart rate trace may not correlate well with a poor long-term prognosis, the presence of an abnormal tracing is highly correlated with the need for neonatal resuscitation in the delivery room.[6] Additionally, it is important to remember that even in the presence of a reassuring fetal heart rate trace nearly 50% of babies born by cesarean delivery will require some active form of resuscitation.[6] Fetal pulse oximetry has been tested as a method of intrapartum fetal assessment. Consensus from several large human trials is that normal fetal oxygen saturation is between 35% and 65% and that metabolic acidosis develops after the fetal oxygen saturation is less than 30% for 10 to 15 minutes.[11-13] One randomized study of 1010 patients found a >50% reduction in the number of cesarean deliveries performed for nonreassuring fetal status when fetal pulse oximetry data was used compared with FHR monitoring alone but, no difference in the overall cesarean delivery rate or neonatal outcome between the two groups.[14] A larger randomized trial of 5341 nulliparous women found no difference in the cesarean delivery rate when clinicians had access to fetal pulse oximetry data and no difference in neonatal outcome between the two groups.[15] Thus, there is no evidence to support the clinical use of fetal pulse oximetry.[16]

Table 2. Maternal & Fetal Factors Associated with Need for Resuscitation

Maternal diabetes	Post-term gestation
Pregnancy-induced hypertension	Pre-term gestation
Chronic hypertension	Multiple gestation
Previous Rh sensitization	Size-dates discrepancy
Previous stillbirth	Polyhydramnios
Bleeding in the second or third trimester	Oligohydramnios
Maternal infection	Maternal drug therapy including
Lack of prenatal care	Reserpine, lithium carbonate
Maternal substance abuse	Magnesium, adrenergic-blockers
Known fetal anomalies	

Table 3. Intrapartum Events Associated with Need for Resuscitation

Cesarean delivery	General anesthesia
Abnormal fetal presentation	Uterine tetany
Premature labor	Meconium-stained amniotic fluid
Rupture of membranes > 24 hours	Prolapsed cord
Chorioamnionitis	Abruptio placentae
Precipitous labor	Uterine rupture
Prolonged labor > 24 hours	Difficult instrumental delivery
Prolonged second stage > 3-4 hours	Maternal systemic narcotics within 4 hours of delivery
Nonreassuring fetal heart rate patterns	

In the presence of a non-reassuring fetal heart rate trace, the practitioner may wish confirmatory studies of fetal well-being or lack thereof. Digital stimulation of the fetal scalp will result in fetal heart rate accelerations in a healthy, nonacidotic fetus. Fetal scalp pH determination can confirm or exclude fetal acidosis. A pH of less than 7.2 is considered abnormal and if confirmed by a second measurement may indicate the need for delivery. Predictors specifically of the need for endotracheal intubation include administration of general anesthesia to the mother and low infant weight.[17,18] In growth-restricted infants, factors predicting low uterine artery pH and/or 5 minute Apgar score <7 include: preeclampsia, fetal distress, breech delivery, forceps use, older maternal age, need for amnioinfusion, general anesthesia and nalbuphine use during labor.[18]

Response: *Intrapartum* Intrapartum resuscitation is attempted once fetal compromise is identified. Maternal factors that may impair oxygen delivery to the fetus must be identified and corrected if possible. Considerations include maternal hypotension or decreased cardiac output secondary to aorto-caval compression, sympathectomy, hemorrhage or cardiac disease. Disease states that may interfere with maternal oxygenation such as asthma, pneumonia, or pulmonary edema should be considered and if present, treated appropriately. Attention must also be directed to the uterus where hyperstimulation, tetany, placental abruption or uterine rupture may interfere with blood flow to the fetus. Stopping an oxytocin infusion or administering a tocolytic agent will reduce uterine tone and potentially allow the fetus the opportunity to recover. Emergent delivery will be required if placental abruption or uterine rupture are severe. Umbilical cord prolapse should always be considered if fetal heart rate changes are sudden, severe and prolonged. Oligohydramnios is a risk factor for umbilical cord compression and variable decelerations. Obstetricians may administer a saline amnioinfusion to try to alleviate cord compression.[19] Saline amnioinfusion is performed by infusing warm saline into the uterus via an intrauterine pressure catheter. Saline amnioinfusion, once frequently utilized in cases of thick meconium in an attempt to dilute the meconium, has been shown to have no impact on the severity of meconium aspiration syndrome.[20]

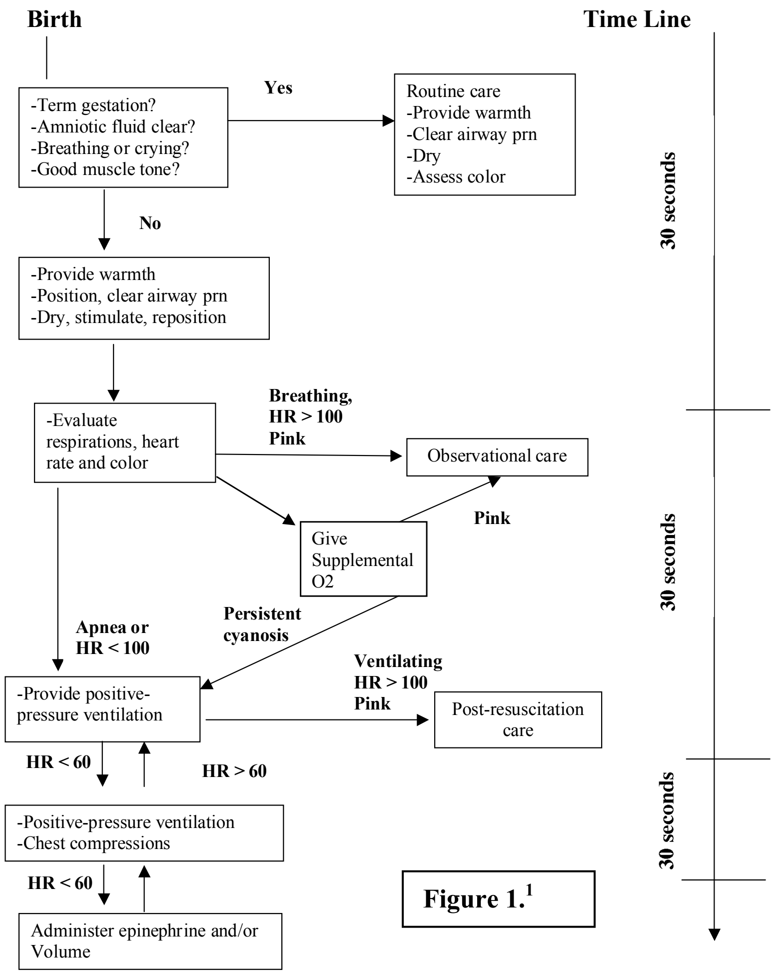

Figure 1.[1]

At Birth The American Heart Association/American Academy of Pediatrics recommends the neonatal resuscitation protocol that follows. Updated protocol recommendations were released in May 2006.[1] The first 30 seconds of neonatal resuscitation should include an assessment of the overall condition of the neonate and steps to minimize heat loss (Figure 1). Depressed, asphyxiated infants often have an unstable thermal regulatory system. Cold stress leads to hypoxemia, hypercarbia and metabolic acidosis, all of which will promote persistence of the fetal circulation and hinder resuscitation. Within the first 20 seconds of birth, the newborn should be dried, placed under a radiant warmer and undergo suctioning of mouth and nose.

***Management of Meconium**:* A major shift in thinking has occurred over the last five years concerning the management of meconium. In the presence of meconium, routine intrapartum oropharyngeal and nasopharyngeal suctioning is no longer recommended.[1,21] Additionally, routine endotracheal intubation and suctioning are no longer recommended. Tracheal suctioning is recommended only if there is meconium stained fluid AND the baby is NOT vigorous.[1] A vigorous infant has strong respiratory efforts, good muscle tone and a heart rate >100 beats per minute (bpm). The data to support this recommendation comes from several sources.[22-24]

Following the first steps of stabilization, further resuscitative efforts should be guided by repeated assessment of respirations, heart rate, and color.[1] If the neonate is gasping or apneic and/or has a heart rate <100 bpm, begin positive-pressure ventilation (PPV) at a rate of 40-60 breaths per minute. Peak inspiratory pressures of 30 to 40 cmH2O or higher are necessary for initial lung expansion.[25] Ventilation that is adequate should promptly restore the heart rate to >100 bpm. The majority of infants requiring any resuscitation will respond to these first two steps. Indications for endotracheal intubation include ineffective bag and mask ventilation, anticipated need for prolonged mechanical ventilation, or as a route for administration of medicine.[1] With prolonged bag and mask ventilation, a nasal or oro-gastric tube should be inserted to decompress the stomach.

Use of Oxygen The scientific basis for the use of 100% oxygen to resuscitate newborns has never been established. Evidence is growing from both human clinical trials and animal models that resuscitation with oxygen may not be optimal for all neonates.[26-35] Exposure to high concentrations of oxygen during resuscitation appears to promote the formation of excessive levels of reactive oxygen intermediates in neonatal tissue, producing injury.[33] A recent meta-analysis including randomized or pseudo-randomized trials of neonatal resuscitation with room air (n=881) versus 100% oxygen (n=856) found the following: overall neonatal mortality was 8.0 versus 13.0% in the 21 and 100% groups, respectively (OR 0.57, 95% CI 0.42-0.78); neonatal mortality in term infants was 5.9 versus 9.8% in the 21 and 100% groups, respectively (OR 0.59 95% CI 0.40-0.87); for infants with a 1 minute Apgar score less than 1, there was no difference between groups; Apgar score at 5 minutes and heart rate at 90 seconds were significantly higher in the room air group; time to first spontaneous breath was significantly shorter in the room air group.[32] Data is accumulating from normal newborns not requiring resuscitation that normal neonatal oxygen saturations are quite low during the first minute of age, ranging from 43% to 77%. At 3, 5, and 10 minutes after birth, preductal mean values were 82%, 89% and 94%, respectively.[36,37] This data must be balanced with concerns about tissue damage from prolonged asphyxia. The updated guidelines reflect these concerns and recommend but do not require supplemental oxygen whenever positive-pressure ventilation is indicated. They note that beginning resuscitation with room air or oxygen is reasonable. They recommend that oxygen be available to use if there is no improvement in the neonate within 90 seconds after birth.[1]

Chest compressions are indicated for a heart rate < 60 bpm despite adequate ventilation with supplemental oxygen for 30 seconds.[1] Neonatal cardiac arrest is generally secondary to respiratory failure producing hypoxemia and tissue acidosis. The result of these metabolic changes is bradycardia, decreased cardiac contractility and eventually cardiac arrest. Cardiac auscultation with a stethoscope should be used for the most accurate assessment of the neonatal heart rate.[38] Chest compressions should be instituted at a rate of 90 compressions per minute. The recommended ratio between chest compressions and ventilations is 3:1, producing 90 compressions and 30 ventilations each minute. In practice, this equals thirty, 2-second cycles/minute. A 2 second cycle consists of 3 chest compressions in 1.5 seconds, leaving 1/2 second for ventilation. Compressions should continue until the spontaneous heart rate is greater than 60 bpm.[1]

Medications are indicated if, after adequate ventilation with 100% oxygen and chest compressions for 30 seconds, heart rate remains below 60 beats per minute. Medications, doses and routes of administration are given in Table 4. Epinephrine is the vasopressor of choice and can be repeated every 3 to 5 minutes until the heart rate is greater than 60 beats per minute. Intravenous administration of epinephrine is strongly preferred due to lack of efficacy of the recommended dose given endotracheally.[1,39] While working to obtain IV access, epinephrine up to 0.1 mg/kg can be given via the endotracheal tube but the safety and efficacy of this practice has not been evaluated in neonates.[1] Epinephrine is not indicated before adequate ventilation has been established because it will increase myocardial oxygen consumption. In the absence of adequate oxygen, this will likely lead to myocardial damage. Establish the airway first. The use of blood volume expanders is rarely indicated and may be detrimental.[40,41] Their use should be restricted to situations in which there is evidence of acute blood loss, such as feto-maternal hemorrhage, accompanied by clear signs of shock.[40,41] Volume expansion should occur over 5 to 10 minutes. Rapid expansion has been associated with intracranial hemorrhage.[1] Naloxone hydrochloride is indicated specifically for neonatal respiratory depression due to maternal opioid administration during labor but should not be given to a neonate born of a narcotic addicted mother as this can precipitate acute withdrawal and seizures in the neonate.[42] The use of sodium bicarbonate is controversial during neonatal resuscitation. Sodium bicarbonate should be given only if ventilation is adequate (or respiratory acidosis will replace metabolic acidosis) and metabolic acidosis is documented or presumed, or all other measures have been unsuccessful.[25,43] Recent data from a small randomized

Table 4. Medications for Neonatal Resuscitation

Medication	Concentration	Dosage / Route	Rate
Epinephrine	1:10,000	0.01 – 0.03 mg/kg (0.1 – 0.3 mL/kg) IV preferred ET dose up to 0.1 mg/kg	Give rapidly Flush catheter/ET tube with saline
Volume expanders	Normal saline O negative blood	10 mL/kg IV (umbilical vein)	Give over 5 - 10 minutes
Naloxone hydrochloride	0.4 mg/mL	0.1 mg/kg IV	Give rapidly
Sodium Bicarbonate	0.5 mEq/mL (4.2% solution)	2 mEq/kg (4 mL/kg) IV (umbilical vein)	Give slowly, over at least 2 minutes **Give only if neonate is effectively ventilated
Dopamine		2 – 20 µg/kg/min IV	

ET = endotracheal, IV = intravenous

trial suggests that sodium bicarbonate administered to neonates still requiring positive pressure ventilation at 5 minutes of age had no impact on morbidity or mortality.[44]

Apgar Score (Table 5) Although 1 and 5 minute Apgar scores are recorded as one way of assessing neonatal response to resuscitation, the practitioner should not wait for the 1 minute score to begin resuscitation. If the 5 minute score is less than 7, additional scores should be obtained every 5 minutes until 20 minutes have passed or until 2 successive scores are greater than or equal to 7.[45] In a study of stillborn infants, 66.6% were resuscitated and left the delivery room alive.[46] Of these, 39% survived beyond the neonatal period. Survival is unlikely if the Apgar score is 0 at ≥ 10 minutes of age.[46] Current guidelines now suggest that after 10 minutes of continuous and adequate resuscitative efforts, discontinuation of resuscitation may be justified if there are no signs of life.[1] A recent retrospective cohort analysis of 151,891 live-born singleton infants without malformations found that an Apgar score between 0 and 3 at 5 minutes was a better predictor of neonatal mortality than the umbilical-artery blood pH value.[47]

Table 5. APGAR Score

Sign	0	1	2
Heart Rate	Absent	< 100 bpm	> 100 bpm
Respiratory Effort	Absent	Slow, irregular	Crying
Muscle Tone	Flaccid	Some flexion of extremities	Active motion
Reflex Irritabililty	No response	Grimace	Vigorous cry
Color	Blue, pale	Blue extremities	Completely pink

Laryngeal Mask Airway (LMA) The size-1 LMA has been used successfully to resuscitate newborns of both normal and low birth weight requiring PPV at birth.[48-51] The size-1 LMA can be life-saving in neonates with Pierre-Robin Syndrome or other conditions associated with a hypoplastic mandible in whom both bag and mask ventilation and endotracheal intubation have failed.[52,53]

End-tidal CO_2 Detection Both infrared absorption and pediatric size colorimetric disposable devices are readily available for the detection of expired carbon dioxide. Clinical trials have shown that both devices are reliable and significantly more rapid than clinical exam in both confirming endotracheal intubation and detecting esophageal intubation.[54,55] End-tidal CO_2 confirmation is the recommended technique to confirm intubation if the heart rate does not increase quickly after intubation.[1]

Neonatal Hypoglycemia Approximately 10% of healthy term neonates have transient hypoglycemia. Other neonates at risk include those born of diabetic mothers or mothers who received a large amount of intravenous dextrose during labor. If a dextrose strip glucose level is <40 to 45 mg/dl, the neonate should be treated either with oral feedings (2-3 cc/kg D10% in water) or by intravenous infusion (8 mg/kg/min).

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