Neonatal Resuscitation

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Introduction

Neonatal resuscitation skills are essential for all health care providers who are involved in the delivery of newborns. The transition from fetus to newborn requires intervention by a skilled individual or team in approximately 10% of all deliveries. This figure is concerning because 81% of all babies in the United States are born in nonteaching nonaffiliated level I or II hospitals. In such hospitals, the volume of delivery service may not be perceived to economically justify the continuous inhospital presence of personnel with high-risk delivery room experience as recommended by the American Academy of Pediatrics (AAP) and the American College of Obstetricians and Gynecologists (ACOG) in Guidelines for Perinatal Care.

Perinatal asphyxia and extreme prematurity are the 2 complications of pregnancy that most frequently require a complex resuscitation by skilled personnel. However, only 60% of asphyxiated newborns can be predicted antepartum. The remaining newborns are not identified until the time of birth. Additionally, approximately 80% of low birth weight infants require resuscitation and stabilization at delivery. Nearly one half of newborn deaths (many of which are extremely premature infants) occur during the first 24 hours following birth. A number of these early deaths also have a component of asphyxia and/or respiratory depression as an etiology. For the surviving infants, effective management of asphyxia in the first few minutes of life may influence long-term outcome.

Even though prenatal care is able to identify many potential fetal difficulties antepartum, allowing maternal transfer of care to the referral center, many women who experience preterm labor are not identified prospectively, therefore not allowing the appropriate maternal transfer to a tertiary perinatal center. Consequently, many deliveries of extremely premature infants occur in smaller hospitals. For this reason, all personnel involved in delivery room care of the newborn should be trained adequately in all aspects of neonatal resuscitation.

This chapter reviews the adaptation to extrauterine life and the steps necessary to optimally resuscitate neonates. Along with the necessary skills, the practitioner should approach any resuscitation with a good comprehension of transitional physiology and adaptation, as well as an understanding of the infant's

response to resuscitation. Resuscitation involves knowing much more than an ordered list of skills and having a resuscitation team; it requires excellent assessment skills and a grounded understanding of physiology.

To decrease neonatal morbidity and mortality, the practitioner must be able to rapidly identify infants whose transition from an intrauterine to extrauterine physiology is delayed. Neonatal transition requires spontaneous breathing and successful cardiopulmonary changes, as well as other changes to independent organ system functions. A thorough understanding of normal transitional physiology leads to a better understanding of the needs of the infant who is experiencing difficulties and, therefore, should result in a more effective resuscitative effort.

The Physiology of Transition

Respiratory adaptation

Following birth, for the lungs to operate as a functional respiratory unit providing adequate gas exchange, the airways and the alveoli must be cleared of fetal lung fluid; an increase in pulmonary blood flow also must occur. In utero, most of the blood flow is shunted away from the lungs and directed to the placenta where fetoplacental gas exchange occurs. Fetal pulmonary vascular resistance is high, and the fetal systemic vascular resistance is low. Within minutes of delivery, the newborn's pulmonary vascular resistance may decrease by 8- to 10-fold, causing a corresponding increase in neonatal pulmonary blood flow. At birth, the lungs must transition rapidly to become the site for gas exchange, or cyanosis and hypoxia rapidly develop.

An understanding of the structure and function of the fetal pulmonary vascularity and the subsequent transition to neonatal physiology is important to assist with adaptation effectively during resuscitation. In utero, the lungs develop steadily from early in gestation. Respiratory development is classified into 4 stages . Based on this information, it is easy to see why infants neonates born before approximately 23-24 weeks' gestational age often do not have sufficient lung development for survival because of the absence of a capillary network adjacent to the immature ventilatory units.

Fetal pulmonary physiology

The fetal lung is filled with approximately 20 mL fluid at term. Fetal airways, alveoli, and terminal saccules are open and stable at normal fetal lung volumes, distended by lung fluid. A constant flow of this fluid is secreted into the alveolar spaces throughout development, which contributes to the fetal amniotic fluid. Pulmonary and bronchial circulation also develops as the alveoli appear. Because of the compressive effect of the fetal lung fluid and the low partial pressure alveolar oxygen (paO 2) in utero, the pulmonary capillary bed and pulmonary blood vessels remain constricted. High vascular resistance and low pulmonary blood flow results.

The placenta provides the respiratory function for the fetus. Two major characteristics of placental circulation enable the placenta to maintain adequate oxygenation of the fetus. First, the placenta has a multivillous circulation that allows for maximum exposure of maternal and fetal blood. Second, several factors result in the lowering of maternal pH and increasing of fetal pH, which results in increased transfer of oxygen by the maternal and fetal hemoglobins.

Maternal blood, carrying oxygen on adult hemoglobin, releases oxygen to the fetal circulation and accepts both carbon dioxide and various byproducts of metabolism from the fetal circulation. These transfers result in a decrease in the maternal placental blood pH and a corresponding shift of the maternal oxygen-dissociation curve to the right, which results in a lower affinity of the hemoglobin for oxygen and the release of additional oxygen to the fetal hemoglobin. The corresponding shift in the fetal oxygen-dissociation curve to the left allows the fetal hemoglobin to bind more oxygen.

Fetal "breathing" begins at approximately 11 weeks and increases in strength and frequency throughout gestation. Fetal respirations are controlled by chemoreceptors located in the aorta and at the bifurcation of the common carotid. These areas sense both pH and partial pressure of carbon dioxide (pCO 2). A reflex response to altered pH and pCO 2 is present at approximately 18 weeks' gestation; however, the fetus is not able to regulate this response until approximately 24 weeks of gestation. Recent studies have indicated that this response cannot be elicited in utero even when the pH and pCO 2 are altered, leading researchers to believe that this response is suppressed in utero and is not activated until birth. Studies also suggest that the low paO 2 in utero may be the mechanism that inhibits continuous breathing, and when paO 2 is increased, continuous breathing is stimulated.

Neonatal pulmonary physiology

As discussed above, the fetal airways and alveoli are filled with lung fluid that needs to be removed before respiration. Only a portion of this fetal lung fluid is removed physically during delivery. During the thoracic squeeze, 25-33% of the fluid may be expressed from the oropharynx and upper airways, although this amount may be markedly less. Thoracic recoil allows for passive inspiration of air into the larger bronchioles. Effective transition requires that any remaining liquid be quickly absorbed to allow effective gas exchange.

A recent study showed that the decrease in lung fluid begins during labor. Using lamb fetuses, the researchers were able to show that the production of lung fluid is decreased on onset of labor. The subsequent reduction in lung fluid was associated with improved gas exchange and acid-base balance. In addition, labor is associated with an increase in catecholamine levels that stimulate lymphatic drainage of the lung fluid. These findings could account for the increased incidence of transient tachypnea of the newborn after a repeat cesarean section without labor. After birth, lung fluid is removed by several mechanisms, including evaporation, active ion transport, passive movement from Starling forces, and lymphatic drainage. Active sodium transport by energy-requiring sodium transporters, located at the basilar layer of the pulmonary epithelial cells, drive liquid from the lung lumen into the pulmonary interstitium where it is absorbed by the pulmonary circulation and lymphatics.

The first breath must overcome the viscosity of the lung fluid and the intraalveolar surface tension. This first breath must generate high transpulmonary pressure, which also helps drive the alveoli fluid across the alveolar epithelium. With subsequent lung aeration, the intraparenchymal structures stretch and gasses enter the alveoli, resulting in increased paO 2 and pH. The increased paO 2 and pH result in pulmonary vasodilation and constriction of the ductus arteriosus.

Lung expansion and aeration also is a stimulus for surfactant release with the resultant establishment of an air-fluid interface and development of functional residual capacity (FRC). Normally, 80-90% of FRC is established within the first hour of birth in the term neonate with spontaneous respirations. The pulmonary vascularity is stimulated to dilate by chemical mediators, nitric oxide, and prostaglandins. Nitric oxide is released when pulmonary blood flow and oxygenation increases. The formation of certain prostaglandins, such as prostacyclin, is induced by the presence of increased oxygen tension. Prostacyclin acts on the pulmonary vascular smooth muscle bed to induce pulmonary vasodilation. Prostacyclin has a short half-life in the bloodstream and, therefore, does not affect the systemic circulation.

Two major physiologic responses have been described for the initial lung inflation in the neonate.

The first response is the "rejection response," in which the neonate responds to positive pressure lung inflation with a positive intraesophageal pressure to resist the inflation. That is to say, the infant actively resists attempts to inflate the lungs by generating an active exhalation. This response acts to not only reduce lung inflation, but also may cause high transient inflation pressures.

The second response is “Head's paradoxical response” in which the neonate responds to positive pressure lung inflation with an inspiratory effort, causing a negative intraesophageal pressure. This inspiratory

effort, with the resultant negative, pressure produces a fall in inflation pressures but results in a transient increase in tidal volume.

Of course, the neonate may demonstrate no response to the inflation attempt, not generating any change in intraesophageal pressure during the positive pressure inflation, and passive inflation subsequently results. It is important to recognize that these physiologic responses to positive pressure inflation in the delivery room may cause large variability in the tidal volume and intrapulmonary pressures, despite constant delivery of inflation pressure.

Stimuli for the first breath may be multifactorial. The environmental changes that occur with birth (eg, tactile and thermal changes, increased noise and light) activate a number of sensory receptors that may help initiate and maintain breathing. Clamping of the cord removes the low resistance placenta, causing an increase in systemic vascular resistance and consequently causing an increase in both systemic blood pressure and pulmonary blood flow. Certain evidence also suggests that the increased arterial paO 2 following the initial breaths may be responsible for the development of continuous breathing via hormonal or chemical mediators that are still undefined.

When the newborn lungs fill with air, the paO 2 should rise gradually. In term infants with a persistent hypoxia, an initial increase in ventilation occurs, followed by a decrease in ventilation occurs. This effect is even more profound in premature infants whose CNS is not as mature. The carotid bodies and peripheral chemoreceptors located at the bifurcation of the common carotids are stimulated during hypoxia to increase minute ventilation. In asphyxiated infants who cannot increase minute ventilation (eg, because of extreme prematurity or sedation), profound bradycardia may result.

Cardiovascular adaptation

Fetal circulation

To understand the cardiovascular changes that occur in the neonate at birth, it is essential to have an understanding of normal fetal circulation. The umbilical vein carries the oxygenated blood from the placenta to the fetus. Blood flow in the umbilical vein divides at the porta hepatis, with 50-60% of the blood passing directly to the inferior vena cava and the remainder of the blood passing into the portal circulation. This portal blood flow perfuses the liver and then passes into the inferior vena cava.

Flow studies have revealed that relatively little mixing of the blood occurs in the inferior vena cava from these 2 sites. The more highly oxygenated blood, which has bypassed the liver, streams into the inferior vena cava to pass preferentially through the patent foramen ovale into the left atrium. The desaturated blood returning from the liver and lower body streams into the inferior vena cava to the right atrium. In the right atrium, it mixes with blood returning from the coronary sinus and superior vena cava and flows into the right ventricle. The more highly oxygenated blood that crosses the foramen ovale mixes with the small amount of pulmonary venous return and then crosses the mitral valve into the left ventricle.

The output from the left ventricle passes into the ascending aorta to the heart, brain, head, and upper torso. The less saturated blood from the right ventricle passes into the pulmonary arteries. Because the pulmonary vessels are constricted and highly resistant to flow, only about 12% of the blood enters the pulmonary veins. The remainder of the blood takes the path of least resistance through the patent ductus arteriosus into the descending aorta. Approximately one third of this blood is carried to the trunk, abdomen, and lower extremities, with the remainder entering the umbilical artery where it is returned to the placenta for reoxygenation.

Neonatal circulation

The aeration of the lung results in an increase in arterial oxygenation and pH, with a resulting dilation of the pulmonary vessels. Decompression of the capillary lung bed further decreases the pulmonary vascular

resistance. There is also a corresponding decrease in right ventricular and pulmonary artery pressures. The decrease in pulmonary vascular resistance leads to an increase in blood flow to the lungs and in pulmonary venous return. Clamping of the umbilical cord removes the low resistance placental vascular circuit and causes a resultant increase in the total systemic vascular resistance with a resultant increase in left ventricular and aortic pressures. The increased systemic vascular resistance, combined with the decreased pulmonary vascular resistance, reverses the shunt through the ductus arteriosus (from right-to-left shunting to left-to-right shunting) until the ductus completely closes.

All of these peripartum events result in closure of the other fetal shunts. With the decrease in right atrial pressure and the increase in left atrial pressure, the “flap-valve” foramen ovale is pushed closed against the atrial septum. This functional closure at birth is followed by anatomical closure that usually occurs at several months of age. The ductus venosus closes because of the clamping of the umbilical cord, which terminates umbilical venous return. Functional mechanical closure of the ductus venosus is accomplished by the collapse of the thin-walled vessels. Anatomical closure subsequently occurs at approximately 1-2 weeks. The constriction and closure of the patent ductus arteriosus is accomplished by contractile tissue within the walls of the ductus arteriosus. The contraction of this tissue is dependent on both the increase in arterial oxygen related to the onset of spontaneous respirations and a fall in circulating prostaglandin E 2 (PGE2).

The placenta is a major site of fetal PGE2 production, thus the removal of the placenta from the circulation causes circulating PGE2 concentration to decrease markedly. Further reduction occurs in the concentration of PGE2 because of increased blood flow to the lungs (the site of PGE2 metabolism). Functional closure of the ductus generally occurs within 72 hours of life, with anatomical closure by age 1-2 weeks. In summary, functional postnatal circulation generally is established within 60 seconds; however, completion of the transformation can take up to 6 weeks.

Response to asphyxia

The fetus or newborn that is subjected to asphyxia begins a “diving” reflex (so termed because of certain similarities to the physiology of diving seals) in an attempt maintain perfusion and oxygen delivery to vital organs. Pulmonary vascular resistance increases, leading to a decreased pulmonary blood flow and increased blood flow directly to the left atrium. Systemic cardiac output is redistributed, with increased flow to the heart, brain, and adrenal gland and decreased flow to the rest of the body. Early in asphyxia systemic blood pressure increases. However, with ongoing hypoxia and acidosis, the myocardium fails and the blood pressure begins to decrease, leading to tissue ischemia and hypoxia.

Infants who are undergoing asphyxia have an altered respiratory pattern. Initially, they have rapid respirations. These respiratory efforts eventually cease with continued asphyxia (termed primary apnea). During primary apnea, the infant responds to stimulation with reinstitution of breathing. However, if the asphyxia continues, the infant then begins irregular gasping efforts, which slowly decrease in frequency and eventually cease (termed secondary apnea).

Infants who experience secondary apnea do not respond to stimulation and require positive pressure ventilation to restore ventilation. Primary and secondary apnea cannot be clinically distinguished. Therefore, if an infant does not readily respond to stimulation, positive pressure ventilation should be instituted as outlined in the Neonatal Resuscitation Program guidelines. If an infant is experiencing primary apnea, the stimulation of the ventilatory efforts cause the infant to resume breathing. If the infant is in secondary apnea, positive pressure ventilation is required for a period. The longer the infant is asphyxiated, the longer the onset of spontaneous respirations is delayed following the initiation of effective ventilation through the use of positive pressure ventilation.

Preparation for Resuscitation

A number of sources of information concerning the training of skills and procedures that are needed for the delivery room resuscitation of the newborn are available. One highly respected source of information concerning the preparation and practice of neonatal resuscitation is the Neonatal Resuscitation Program, which has been codeveloped by the AAP and the American Heart Association. The following sections contain a review of resuscitation procedures in a format that is similar to the format used by the Neonatal Resuscitation Program. Completion of the Neonatal Resuscitation Program should be considered for all hospital personnel who may be involved in the stabilization and resuscitation of neonates in the delivery room. To develop true expertise, additional supervised time with skilled personnel is essential.

Although the current program for neonatal resuscitation is considered a highly respected reference, it is important that more research continue to evaluate the effectiveness of the techniques of neonatal resuscitation. The Neonatal Resuscitation Program has evolved and will continue to evolve with new data from clinical studies and basic physiologic research.

Anticipation

The goals of resuscitation are to assist with the initiation and maintenance of adequate ventilation and oxygenation, adequate cardiac output and tissue perfusion, and normal core temperature and serum glucose. These goals may be attained more readily when risk factors are identified early, neonatal problems are anticipated, equipment is available, personnel are qualified and available, and a care plan is formulated.

Causes of depression and asphyxia

A large number of antepartum and intrapartum maternal conditions carry an increased risk for intrapartum asphyxia. A number of excellent texts review the extensive medical and surgical problems of the obstetrical patient. It is not within the purview of this article to review this topic.

Equipment

The delivery room should be equipped with all the necessary tools to successfully resuscitate any gestational age newborn. The equipment should include a radiant warmer, warmed blankets, a source of oxygen, instruments for visualizing and establishing an airway, a source of regulated suction, instruments and supplies for establishing intravenous access, trays equipped for emergency procedures, and drugs that may be useful in resuscitation.

Trained personnel

For all deliveries, at least one person should be present who is skilled in neonatal resuscitation and has responsibility for only the infant. Personnel should be available who are capable of performing a complete resuscitation, including intubation, medication administration, and emergency procedures. If the delivery is identified as high risk, 2 or more skilled individuals should be assigned for the infant at delivery. Remember that staff trained in neonatal resuscitation need to apprentice with experienced personnel for some time before they can be independently responsible for an infant at a delivery.

Neonatal Resuscitation

Thermoregulation

It is essential to prevent heat loss during the resuscitation. Intrauterine thermoregulation is passive, with no use of calories or oxygen by the fetus. This intrauterine thermoregulation allows for maximal

intrauterine growth without fetal energy expenditure for thermal homeostasis. Brown fat storage begins during the third trimester. It is the brown fat that is used for heat production in the newborn period.

Several factors lead to increased heat losses in the newborn infant. The neonate has a large skin surface area–to–body weight ratio, which increases heat and fluid evaporative loss. The fluid loss from the skin (not due to sweating but caused by direct transdermal water loss) results in massive heat loss. The thin skin with blood vessels that are near the surface provides poor insulation, leading to further heat loss. Additionally, the newborn infant (especially if premature) has a limited capacity to change body position for heat conservation. Animals ordinarily attempt to decrease heat loss by decreasing exposed surface area. This reduction in exposed surface area is accomplished by assuming a flexed position; however, premature, critically ill, and depressed infants are unable to accomplish flexed positioning.

Neonates have a very limited capacity for metabolic heat production. The newborn infant has limited energy stores, largely because of decreased subcutaneous fat and brown fat stores. This paucity of fat stores is more pronounced in premature and growth-retarded infants. Additionally, infants do not shiver effectively, which is a major source of heat production in the adult. The main source of heat production in the newborn is nonshivering thermogenesis.

Thermoreceptors in the face have a marked sensitivity to heat and cold. Stimulation by cold leads to norepinephrine production and thyroid hormone release causing brown fat to be metabolized. Brown fat is highly vascularized and stored in pockets around the neonate's body. When brown fat is metabolized, triglycerides are hydrolyzed to fatty acids and glycerol. Additionally, glycolysis is initiated and glycogen stores are used; both processes resulting in glucose production. Heat is produced as a byproduct of the increased metabolic rate and oxygen consumption.

Infants who experience heat loss have an increased metabolic rate and use more oxygen. Increased oxygen consumption can be dangerous in infants who are experiencing respiratory compromise. The addition of cold stress in infants who are poorly oxygenated potentially can lead to a change from aerobic to anaerobic metabolism. This change in metabolism may lead to tissue hypoxia and acidosis because of the buildup of metabolic byproducts such as lactate. Because of the inefficiency of anaerobic metabolism, the infant uses up glucose and glycogen reserves rapidly and still produces only a limited amount of energy for heat production. Therefore, cold stress can lead to both metabolic acidosis and hypoglycemia.

Based on this information, it is essential to prevent excessive heat loss in the delivery room. Newborns should be dried with prewarmed blankets or towels and placed on a prewarmed heat source. Open bed warmers, which use radiant heat, are used in most delivery rooms. They provide warmth during resuscitation and for any subsequent invasive procedures. It is important for the practitioner to keep in mind that this source of heat does not protect the infant from evaporative heat loss but, instead, encourages evaporative heat losses.

It is also necessary to consider the environmental temperature in relationship to controlling heat loss in the newborn. As a fetus, the thermal environment is regulated precisely by the mother's core temperature, and heat losses are nonexistent. Following delivery, even when drying and a radiant heat source are used, infants continue to lose large amounts of heat. This occurs by convective and evaporative heat loss. When the environmental air is less than the neutral thermal environment for the infant being resuscitated, this cooler air causes further thermal losses.

Heat losses are related to the differences both in water concentrations between the skin and the air as well as the absolute temperature gradient. The primary goal in neonatal thermoregulation is to prevent heat loss, compared to later rewarming a cold infant. Ideally, an area (eg, a stabilization room) should be separate from the operating room (OR) or labor room that allows special attention to the unusual thermal and environmental needs of the newborn high-risk infant. This stabilization area should be kept as warm as possible, balancing the requirements of the high-risk infant with the comfort of the adult staff in that area. Centers with such stabilization areas generally quote temperature goals in the range of 80-88°F.

Another common source of heat loss in the newborn infant undergoing resuscitation is the use of unheated nonhumidified oxygen sources for the bag-valve-mask device. Inspired gasses that are sent to the lungs are subsequently heated and humidified by the infant, thus resulting in massive heat exchange and insensible water loss. Therefore, whenever possible, warmed and humidified gasses should be provided in the resuscitation area. Alternatively, the intubated and ventilated infant should be placed on a heated ventilator circuit as soon as is feasible.

Airway management

Once the infant is placed in a heated environment, the infant should be positioned to open the airway, and the mouth and nose should be suctioned. A bulb syringe should be used for the initial suctioning. Infants have a vagal reflex response to sensory stimulation of the larynx, which may induce hypotension, bradycardia, swallowing, and apnea. Therefore, the act of suctioning the airway with a catheter because of extremely thick or meconium-stained fluids may cause profound central apnea, bradycardia, and laryngospasm. This reflex bradycardia may be profound. Therefore, deep suctioning of the trachea should be limited to infants who have thick mucous that cannot be removed by bulb syringe or used for the aspiration of aspirate stomach contents, when necessary.

The instillation of saline into the trachea also has been shown to stimulate the afferent sensory neurons leading to these sequelae and has no place in the immediate resuscitation period. Lung inflation has been shown to reverse the effects of vagal stimulation. Vigorous suctioning of the nares with a catheter can lead to edema with resulting respiratory distress after the infant leaves the delivery room. Wall suction should be set so that pressures do not exceed 100 mm Hg.

Stimulation

Drying and suctioning often is enough stimulation to initiate breathing; however, if more vigorous stimulation is necessary, slapping the soles of the feet or rubbing the back may be effective. The back should be visualized briefly for any obvious defect in the spine before beginning these maneuvers. If there is no response to stimulation, it may be assumed the infant is in secondary apnea, and positive pressure ventilation should be initiated. At this point, the infant's respiratory rate, heart rate, and color should be evaluated. Most infants do not require further intervention.

Supplemental oxygen

Infants who have a sustained heart rate more than 100 beats per minute (BPM) and adequate respiratory effort but who remain cyanotic should receive blow-by oxygen via oxygen tubing or a mask. It is arguably advantageous to provide heated, humidified oxygen if possible, but this is rarely available in the delivery room environment. Supplemental oxygen should be initially provided with a FiO 2 of 1 at a flow rate of 8-10 L/min. If supplemental oxygen is to be provided for a prolonged period, then heated humidified oxygen should be supplied via an oxy-hood with the FiO 2 adjusted to result in pulse-oximetry saturations of 92-96% in the term infant and 88-92% in the preterm infant.

Positive pressure ventilation

For a number of reasons (discussed above), it can be difficult for the infant to clear fluid from the airways and establish air-filled lungs. Initial respiratory efforts may need to be augmented by the addition of either continuous positive airway pressure (CPAP) or positive pressure ventilation. The addition of positive pressure aids in the development of functional residual capacity and is needed more commonly in premature infants. Mechanical lung inflation also is important to reverse persistent bradycardia in an apneic asphyxiated infant.

Infants with adequate respirations who are having respiratory distress manifested by tachypnea, grunting, flaring, retracting, or persistent central cyanosis may benefit from CPAP. If the infant is apneic, has

inadequate respiratory efforts, or a heart rate less than 100 BPM, then positive pressure ventilation should be initiated immediately. The bag must be equipped to deliver positive end-expiratory pressure and the appropriately sized mask should be applied firmly to the face.

Some infants respond to brief mechanical ventilation and subsequently begin independent ventilation; others need continued ventilatory support. It is essential that sufficient, but not excessive, initial pressure be used to adequately inflate the lungs, or bradycardia and apnea will persist. A pressure manometer always should be used with a pressure release valve limiting the positive pressure to 30-35 cm H 2 O. To provide adequate distending pressure, the infant must be positioned properly and the upper airway cleared of secretions; the mask must be the correct size and form a tight seal on the face.

While providing assisted breaths, look for a rise and fall in the chest and an immediate increase in heart rate. If no chest rise occurs, either the airway is blocked or insufficient pressure is being generated by the squeezing of the bag. Ventilatory rates of 40-60 breaths per minute should be provided initially, with proportionally fewer assisted breaths provided if the infant's spontaneous respiratory efforts increase. Although not studied extensively, it has been reported that the initial inflation of the newborn's lungs with either slow-rise or square wave inflation to 30 cm H 2 O pressure for approximately 5 seconds results in more rapid formation of a functional residual capacity.

It should be remembered that, at the moment of delivery and first, breath the neonatal lung is converting from a fetal, nonaerated status to a neonatal status. The neonatal lung has a requirement for gas exchange, and this requires the development of a functional residual capacity because of the resorption of lung fluid and the resolution of most of the atelectasis. Therefore, it is logical to conclude that initial slow ventilation with more prolonged inspiratory times may be useful to assist in this task, balanced with the avoidance of inappropriate inspiratory pressures.

However, these observations are counterbalanced with data showing that an increased risk of chronic lung disease in infants with very low birth weight is associated with centers who initiate mechanical ventilation more frequently. Therefore, prospective, randomized clinical trials are urgently needed to resolve several issues related to the timing of surfactant administration, use of various forms of positive end-pressure (CPAP), and/or initiation of mechanical ventilation.

Intubation

Infants may require tracheal intubation if direct tracheal suctioning is required, effective bag-mask ventilation cannot be provided, congenital diaphragmatic hernia is suspected, or a prolonged need for assisted ventilation exists. An appropriate blade (Miller size 0-1) should be chosen depending on the size of the infant. Premature infants may be more easily intubated with a size 0 blade, and term infants require a size 1 blade. An appropriate size of endotracheal tube (ETT) should be chosen based on the weight of the infant.

Upon insertion of the ETT, the tube should be advanced until the vocal cord guide mark near the distal tip of the tube is visualized to be slightly past the vocal cords. This guide mark is positioned a variable distance from the distal tip (depending on the ETT size) and is designed to result in the placement of the tube tip between the vocal cords and the carina at the bifurcation of the right and left mainstem bronchi. The ETT should then be secured and cut to an appropriate length to minimize dead space and flow resistance.

Another estimate of correct placement of the ETT is to use the weight of the infant in kilograms plus 6 to arrive at the number of centimeters at which the tube should be secured at the lip. Before securing the ETT, the infant should be assessed for equal bilateral breath sounds with maintenance of oxygenation. ETT position is confirmed with a chest x-ray. Free flow oxygen should be provided throughout the procedure, and then effective ventilation via the bag or ventilator after the infant is intubated.

Cardiovascular support and chest compressions

Most infants who present at delivery with a heart rate less than 100 BPM respond to effective ventilatory assistance with a rapid increase in heart rate to normal rates. In contrast, if an effective airway and effective ventilation is not established, further support is not effective. Chest compressions should be initiated following only 30 seconds of effective positive pressure ventilations if the heart rate remains less than 60 BPM.

An assessment of the heart rate can be obtained by palpating the umbilical stump at the level of insertion of the infant's abdomen or by direct auscultation of the precordium. Chest compressions should be discontinued as soon as the heart rate is higher than 60 BPM. Chest compressions may be performed either by circling the chest with both hands and using a thumb to compress the sternum or by supporting the infant's back with one hand and using the tips of the middle and index finger to compress the sternum. The thumb technique is preferred because of improved depth control during compressions, however the 2-finger technique can be used.

Pressure should be applied to the lower portion of the sternum depressing it 0.5-0.75 inches at a rate of 90 per minute. One ventilation should be interposed after every 3 chest compressions allowing for 30 breaths per minute. The recommended ratio of chest compressions to ventilations is 3:1. Evaluate the heart rate and color every 30 seconds. Infants who fail to respond may not be receiving effective ventilatory support, thus it is imperative to be evaluating the ventilation of the infant constantly.

Medications

Neonatal resuscitation drugs should be stocked in any area in which neonates may be resuscitated, including each delivery and stabilization area, as well as the emergency room. Personnel should be familiar with neonatal medications, concentrations, dosages, and routes of administration. Drugs currently recommended include epinephrine (1:10,000), sodium bicarbonate (0.5 mEq/mL), and isotonic sodium chloride solution (0.9%) as a volume expansion agent.

Epinephrine use should be considered only when ventilation has been established and provided for at least 30 seconds. The only exception to this rule may be in infants who are born without a detectable pulse or heart rate. The current recommended dose for epinephrine is 0.01-0.03 mg/kg (0.1-0.3 mL of the 1:10,000 solution) via IV or intratracheal route.

No studies are currently available that assess the use of higher dosages or repeated dosing in neonates. Epinephrine administered via the ETT either should be diluted into 1 mL of saline or should be followed immediately by 1-2 mL of saline to ensure the distribution and absorption of the small volume of drug. If an umbilical venous catheter is used for medication administration, the catheter should be inserted only until blood flow is obtained, usually 3-5 cm. Because the dosing recommendations for epinephrine have included the endotracheal route of administration, the need for emergent placement of umbilical venous catheters has been reduced markedly in the delivery room.

In an editor's note commenting on an article entitled Cardiopulmonary Resuscitation in the Delivery Room, Catherine DeAngelis writes, "....check the airway (optimize respiratory support) one more time before compressing the chest. More often than not, you and the infant can then take deep breaths, and you can beat your own chest instead of the infant's."

The article reported that approximately one third of infants in their study with neonatal depression at birth had associated fetal acidemia. However, in the remaining infants without fetal acidemia, chest compressions were initiated as a consequence of improper or inadequate ventilatory support at birth. In this population of infants without the initial acidemia, chest compressions and/or epinephrine therapy was ineffective. The heart rate only improved after either effective tracheal intubation established a patent airway and/or after incremental increases in positive-pressure ventilation exceeded the opening pressure

of the lungs, establishing ventilation. This study and others continue to reinforce the primary importance of the establishment of effective ventilation—for without ventilation, other therapies, including medications, will not be effective in establishing adequate heart rate and perfusion.

Sodium bicarbonate has been recommended in the delivery room to reverse the effects of metabolic acidosis related to hypoxia and asphyxia. However, recent studies show that 0.9% saline provides better cardiac and blood pressure support to correct both the acidosis and the underlying etiology of the metabolic acidosis. Sodium bicarbonate should not be used until adequate ventilation is obtained because of the concomitant production of carbon dioxide following the use of this drug. If sodium bicarbonate is used in the face of a persistent respiratory acidosis and elevated pCO 2 , the acidosis will not be corrected.

To correct a documented or presumed metabolic acidosis following the establishment of adequate ventilation, a dose of 2 mEq/kg IV may be administered. If the base deficit is known, then a more precise dose can be administered. Use of sodium bicarbonate in the delivery room has been associated with an increased incidence of intraventricular hemorrhage in very low birthweight infants; thus, caution is advised.

Volume expansion may be used in neonates with evidence of acute blood loss or with evidence of shock of any etiology. In general, the neonatal heart responds well to the increase in preload at the atrial level caused by the volume expansion. Hypovolemia may be masked in a newborn infant because of the significant peripheral vasoconstriction caused by the elevated catecholamines following delivery. Systolic blood pressure also may be elevated falsely with pain.

The current recommendations for volume expansion during resuscitation include isotonic sodium chloride solution or lactated ringers, 5% albumin, Plasmanate, or O-negative blood that has been cross matched with the mother. However, because of the advantages of long shelf life, low cost, ready availability, and the lack of evidence of the superiority of other agents, isotonic sodium chloride solution is the most frequently used agent for volume expansion. The currently recommended dosage for volume expansion is 10 mL/kg IV over 5-10 minutes, and it may be infused more cautiously in extremely preterm infants.

Naloxone should be used only in neonates exhibiting respiratory depression in the setting of a laboring mother who has received iatrogenically administered narcotics within 4 hours of delivery. Naloxone should not be administered to neonates who are not in distress or to neonates with another predisposing perinatal complication to explain the distress.

The great majority of neonates born to mothers who receive narcotics do not receive and do not require naloxone administration. The intramuscular administration of naloxone should not be used as a substitute for other forms of vigorous stimulation. Additionally, the administration of naloxone to a neonate who is born to a mother with long-term narcotic use (eg, heroin, methadone) may induce seizures secondary to acute withdrawal. Remember that some narcotic agents have a longer half-life in the neonate than naloxone, which may lead to a later recurrence of respiratory depression in the nursery. Therefore, any infant who has received naloxone should be monitored for the recurrence of respiratory depression for 12 hours. Naloxone dosage is 0.1 mg/kg of the 0.4-mg/mL solution given intravenously, endotracheally, or intramuscularly.

The immediate postresuscitation period

Maintenance of airway and ventilation

The goal of delivery room management is to stabilize the airway and assure effective oxygenation and ventilation. Once initial lung recruitment is obtained, it is essential to avoid overdistension. Breaths delivered by bag-mask ventilation may be difficult to control and may result in overdistension and consequent pneumothorax or pneumomediastinum. Additionally, the unheated nonhumidified oxygen can

quickly cool the infant via the large surface area of the lungs, resulting in hypothermia. Therefore, mechanical ventilation should be initiated as soon as possible once the infant is stabilized.

Although the ideal mode of assisted ventilation is controversial, it is essential to provide adequate positive end-expiratory pressure to prevent atelectasis, while at the same time preventing overinflation. Once the appropriate functional residual capacity is obtained, it is essential to use the lowest support possible to allow for adequate oxygenation and ventilation. Oxygen saturations should be monitored continually and arterial blood gas analyses performed as needed during the initial stabilization period. Saturations should be maintained in the 90-96% range for the term infant and 88-92% in the preterm infant after the initial stabilization.

Fluid and electrolyte management

In utero, nutrients are provided in their basic form. Glucose is the major energy substrate of the fetus. Fetal glucose uptake parallels maternal blood glucose concentration. The liver, heart, and brain receive the greatest cardiac output and, therefore, the greatest amount of glucose. The fetus uses glucose, lactate, and amino acids to store fuels that are used during transition. Neonates must develop a homeostatic balance between energy requirements and the supply of substrate as they move from the constant glucose supply of fetal life to the normal intermittent variations in the availability of glucose and other fuels. With the clamping of the cord, the maternal glucose supply is cut off. A fall in blood glucose during the first 2-6 hours of life occurs in healthy newborns. The blood glucose usually reaches a nadir and stabilizes at 50-60 mg/dL.

The immediate goal of fluid and electrolyte support following resuscitation is to maintain an appropriate intravascular volume and to provide glucose homeostasis and electrolyte balance. The neonatal cardiovascular system is very sensitive to preload, requiring adequate intravascular volume to maintain adequate cardiac output. Therefore, expansion of intravascular volume with appropriate solutions (eg, isotonic sodium chloride solution) often is considered in the neonate with inadequate blood pressure or perfusion.

Additionally, as discussed in previous sections, hypoglycemia may occur rapidly in critically ill or premature infants. Blood glucose determinations should be performed as soon as possible and a continuous infusion of glucose should be started at 4-6 mg/kg/min for those infants who are not able to tolerate enteral feedings. Dextrose boluses should be limited to symptomatic infants because they may result in transient hyperosmolarity and rebound hypoglycemia. Electrolytes, such as sodium, potassium, and chloride, should not be added initially because the fluid shifts from other body compartments allow for adequate electrolyte supply until adequate renal function is documented.

The practitioner should monitor the weight, clinical hydration status, urine output, and serum sodium concentrations closely because inappropriate fluid overload or restriction can lead to increased mortality and morbidity. Taking the infant's environment into account when calculating fluid requirements is very important. Fluid rates may be started at 60-80 mL/kg/d for the infant in a humidified incubator, while fluid rates may be much higher for the infant in a dry radiant warmer environment.

Preparation for transport

Preparation of the infant for transfer to a remote nursery for subsequent care requires several considerations. First, it is important to complete all the routine care that is required of newborn infants. These basics of care may be neglected in the rush to prepare the infant for transport, with potentially disastrous results. Following resuscitation, care must be taken to secure all lines, tubes, catheters, and leads for transport. Monitoring in the transport environment is only possible with functioning leads in place, which is frequently difficult. Rapid and complete documentation of the resuscitation and subsequent therapies also is required for future caretakers.

Special problem during resuscitation

This section is devoted to congenital neonatal conditions that may present in the delivery room and that may alter the resuscitation. The presentation of the disease and the immediate resuscitative efforts are discussed. Please refer to other specific chapters for further information on these disease processes.

Extreme prematurity

Premature infants have special needs that must be considered during the critical period immediately following delivery if mortality and morbidity are to be decreased in this group. This population of infants is at increased risk for respiratory failure, insensible water losses, hypoglycemia, and intraventricular hemorrhages. It is impossible to adequately review the many difficulties of extreme prematurity in this section, but special concerns regarding the care of these infants during the resuscitation period is discussed.

Insensible water loss in the premature infant is increased secondary to the infant's poorly cornified epidermis and an immature stratum corneum, which presents little barrier to evaporative heat loss. The stratum corneum is not functionally mature until 32-34 weeks' gestation. Differences in skin maturity, prenatal nutritional status, ventilation requirements, and environmental conditions all may influence the magnitude of insensible water loss that occurs following birth.

The skin is the most important route for water depletion after delivery of the extremely immature infant. Transepidermal water loss (TEWL) is highest at birth in infants who are born before 28 weeks' gestation and decreases slowly with advancing gestational age. Despite declines in TEWL with advancing age, infants born before 28 weeks' gestation continue to have increased TEWL for 4-5 weeks following birth, compared to infants born at term. Because of high evaporative loss with the accompanying heat loss, the ability to achieve and maintain thermoregulation is compromised further. The skin barrier dysfunction increases the risk for infection, especially because of organisms that colonize the skin surface (eg, staphylococcal species). This thin skin barrier also places the extremely immature infant at risk for toxicities from topically applied substances. Additionally, skin integrity is disrupted easily by the use of adhesives, which should be limited in premature infants.

Premature infants need increased fluid administration rates initially if they are on radiant warmers for a prolonged period. With increased parenteral fluid administration using dextrose-containing fluids, the dextrose needs to be monitored closely to ensure euglycemia. Placing infants in a humidified environment decreases transepidermal water loss, improves the maintenance of body temperature, and does not delay skin maturation. Measures to decrease insensible water loss should be initiated at delivery. Because radiant warmers are used routinely at deliveries because of a need for maximal patient access, infants less than 1000 g should have a plastic blanket or other barrier applied to decrease evaporative water loss until they can be placed in a humidified environment. However, care should be taken to ensure that the barrier does not block the transmission of the radiant heat source.

Premature infants are at risk for intraventricular hemorrhages and periventricular leukomalacia (PVL) secondary to their immature cerebral vascular regulation and the persistence of the germinal matrix. Ventricular hemorrhage and periventricular leukomalacia often lead to serious permanent neurodevelopmental disabilities. Prevention or reduction of the severity of these disorders may begin in the delivery room. Mechanical ventilation and fluid administration must be managed cautiously in this group of infants. Volume expansion should only be administered in the face of true hypotension. It is essential to know normal blood pressure values for infants of various gestational ages. Volume expansion in the face of normal blood pressure increases the risk of IVH.

Additionally, when administering hyperosmolar medications (eg, sodium bicarbonate), slow administration is important. Mechanical ventilation may lead to harmful fluctuations in cerebral blood flow, especially when pCO 2 and pH are altered rapidly. Rapid alterations in pCO 2 and pH result in

acute fluctuations in the cerebral blood flow of the premature infant with immature cerebral vascular autoregulation.

Premature infants are also at high risk for volutrauma caused by poor lung compliance and overventilation following the administration of exogenous surfactants if changes in lung compliance are not monitored carefully. Overventilation with excessive tidal volumes and hypocarbia are associated with chronic lung disease. Stabilization of the infant using the lowest possible peak inspiratory pressures that are required to oxygenate and ventilate adequately is essential. Hand ventilation of an intubated infant, especially by inexperienced personnel, often leads to inconsistent tidal volumes and pressures. Use of a mechanical ventilator designed for infants offers the advantages of more consistent tidal volumes and a reduction of the heat losses because of the use of unheated nonhumidified air with hand bagging.

Although artificial surfactant administration is associated with a reduction of adverse sequelae in infants, its administration may lead to hyperventilation and overdistension when not administered by experienced attentive personnel. Following the instillation of artificial surfactant, rapid reaction to changes in pulmonary compliance to prevent the onset of hypocarbia and alkalosis is essential. Following the institution of mechanical ventilation, care should be taken with airway suctioning because vigorous or frequent airway suctioning is associated with hypoxia, intraventricular hemorrhage, and periventricular leukomalacia. Prematurity with respiratory distress syndrome (RDS) is not associated with mucous production in the first 24 hours of life, thus suctioning protocols should be altered to provide minimal suctioning during this time.

Airway problems

Choanal atresia

Choanal atresia is caused by a failure of embryologic regression of nasal airway tissue, thereby resulting in a partial or complete occlusion of the nasal airway. These choanal defects may be bony or membranous, with most having a boney component. Complete bilateral stenosis usually results in a neonatal respiratory emergency at birth because infants generally are obligate nasal breathers during the first 6-8 weeks of life. At rest, these infants usually manifest severe apnea, retractions, and respiratory distress that may be relieved with crying.

Wheezing or stridor may be audible with inspiration, and collapse of the small airways with vigorous inspiratory effort can occur. The infant in respiratory distress should be stimulated to cry and an artificial oral airway may be used to avoid intubation. The clinical diagnosis is achieved by the inability to pass a small caliber catheter through the nasal passages. However, the act of passing catheters, especially with repeated attempts, causes nasal passage swelling in any infant with the subsequent iatrogenic occlusion mimicking the congenital condition.

An alternative noninvasive method of excluding the diagnosis of complete atresia is to place a glass slide under the nasal orifices and look for fogging with expiration. Supplemental oxygen should be administered to infants with choanal atresia, and an oral airway may be of assistance. If the infant remains in significant respiratory distress, intubation is necessary. Intubation relieves the obstruction so that minimal ventilation (if any) is required.

Pierre Robin syndrome

This syndrome presents with micrognathia and with a resultant displacement of the tongue into the posterior pharynx, which may occlude the upper airway. A central cleft of the soft palate usually is present. Respiratory distress and cyanosis are caused by the obstruction of the upper airway. In the delivery room, the infant should be given supplemental oxygen and placed in a prone position in an attempt to have the tongue move forward in a dependent fashion from the posterior pharynx, relieving the airway obstruction. If the infant continues to have persistent respiratory distress, an oral airway may be

placed. Alternatively, an appropriately sized endotracheal tube may be passed through the nose into the hypopharynx. Tracheotomies are generally not necessary and should be avoided. Intubation of these infants often is not easy because visualization of the larynx is difficult.

Tracheal webbing

The pathogenesis of tracheal webbing originates in the tenth week of gestation when an arrest in the development of the larynx near the vocal cords results in a residual web of tissue persisting in the airway. Approximately 75% of tracheal webs occur at the level of the vocal cords. These lesions are critical if more than 50% of the airway diameter is occluded, but this is rare. These disorders may be relatively asymptomatic at birth, with the development of distress later when activity increases and the need for airway flow increases.

When attempting to intubate these infants, an obstructive covering may be observed over the larynx and may occlude the airway completely. If the web consists of a thin membrane, the ETT may be pushed beyond the obstruction. However, if the membrane is thick, the infant requires an emergency tracheotomy. If the infant is manifesting severe distress, a large bore needle or catheter may be placed into the trachea to allow for gas exchange while arranging for emergency treatment. Caution must be used because inexperienced personnel may confuse this rare disorder with simple inability to visualize the vocal cords.

Esophageal atresia with and without tracheoesophageal fistulae

This condition rarely is considered a life-threatening emergency; however, early diagnosis is essential to prevent further complication.

he most common clinical symptoms in types of esophageal atresia with an esophageal-tracheal fistula are coughing, choking, and cyanosis. Infants with isolated esophageal atresia usually do not demonstrate respiratory distress immediately in the delivery room but may have excess secretions. The atretic air-filled esophageal pouch occasionally may be observed on a chest radiograph, manifested by a midthoracic rounded lucency. This pouch is visualized more readily by the passage of a radioopaque catheter into the esophagus before the chest radiograph.

Because secretions or oral feedings are not capable of passage into the stomach, the contents of the esophageal pouch readily reflux, placing these infants at high risk for aspiration. A Replogle suction catheter should be inserted to reach the esophageal pouch and placed on low continuous suction as soon as possible. Infants with an associated distal fistula to the trachea are also at high risk for aspiration of gastric contents into the lungs via the gastrobronchial fistula, which most often empties into the airway near the carina.

If at all possible, positive pressure ventilation should be avoided in these infants. Any positive pressure applied to the airway results in inflation of the fistula, stomach, and bowel, which then results in abdominal distention. This distending pressure cannot be relieved by esophageal reflux through the atretic esophagus. Relief of the distending pressure occurs with reflux of gastric contents into the lungs via the fistula. The continued application of positive pressure ventilation also may lead to massive gastric distention and possible rupture. In rare emergency situations, percutaneous gastrotomy may be required to decompress the stomach; however, controlled surgical placement of a gastrostomy tube is preferable.

Cystic adenomatoid malformation

Cystic adenomatoid malformations of the lung are masses that may cause a spectrum of symptoms, from massive mediastinal shifts in the fetus (resulting in pulmonary hypoplasia) to isolated subsegmental lobar masses in the newborn (or adult) with minimal associated symptoms. Severe lesions also may cause fetal cardiac compromise and result in hydrops. If the infant requires positive pressure ventilation, extreme

caution must be used because the distending pressure may inflate the cystic malformation. An inflated cystic malformation is capable of massive expansion, causing respiratory embarrassment because of the prevention of ventilation of other normal lung tissue.

Cystic hygromas

This condition is the result of a congenital deformity of the lymphatic channels. Lymph accumulates and may compress the airway, depending on the size and location of the lymph accumulation. Approximately 80% of these lymphatic cystic accumulations occur in the neck and may compress the trachea. These infants may present with significant respiratory distress and require immediate intubation with deep positioning of the ETT to relieve the obstruction by stinting open the airway. However, most of these lesions expand outward from the neck and do not cause significant airway compromise in the delivery room.

Pulmonary compression

Congenital diaphragmatic hernia

The pathogenesis of this disorder is caused by the incomplete formation of the diaphragm in the fetus, resulting in a migration of the abdominal viscera into the chest during development. If the defect is large and the abdominal viscera have caused long-standing compression of the developing lungs, pulmonary hypoplasia may develop.

The diagnosis of diaphragmatic hernia is established frequently by prenatal ultrasound, which allows the management to be transferred to a perinatal referral center where pediatric surgery and appropriate medical support are available, including extracorporeal bypass. In the delivery room, the infant often presents with respiratory distress. Physical signs may include a scaphoid abdomen and a shift in heart sounds to the right hemithorax.

Respiratory distress in the delivery room may be caused by either a pulmonary hypoplasia or may be secondary to an expansion of the bowel caused by swallowed air. The expansion of the bowel results in compression of the lung. Delivery room management includes immediate intubation and passage of a large catheter for gastric decompression. Intubation prevents distention of the stomach and bowel contents because of crying or bag-valve-mask ventilation. The gastric decompression should be achieved with a Replogle or Salem pump suction catheter connected to a low continuous drain. Constant maintenance of the gastric suction during the preoperative and immediate postoperative period is essential.

New modes of ventilation such as high frequency oscillatory ventilation has decreased the use of extracorporeal membrane oxygenation (ECMO) in this population. However, the survival rate for infants with this anomaly has not changed over the past decade.

Pneumothorax

Air leak syndromes are disorders produced when a rupture of pulmonary tissue occurs with the resultant escape of air into spaces in which air would not be present normally. The incidence of pneumothorax varies with gestational age, severity of pulmonary disease, need for assisted ventilation, mode of ventilation, and expertise of delivery room personnel. Following the initial rupture of a small airway or an alveolus, air may enter the perivascular and peribronchial spaces and track along the lymphatic channels. Air that dissects into the hilum results in a pneumomediastinum. Air that tracks into the pleural space manifests as a pneumothorax. Spontaneous rupture of the lung directly into the pleural space is thought to occur rarely but may be caused iatrogenically with the percutaneous insertion of a chest tube. Caution is required.

Pneumomediastinum frequently is an isolated disorder that occurs spontaneously in infants with minimal pulmonary disease. These infants usually are asymptomatic or minimally symptomatic because air in the mediastinum is capable of escaping to the tissues of the neck. Intrathoracic tension is relieved and circulation is not compromised. Infants with a pneumomediastinum should be observed. Intervention usually is unnecessary.

Pneumothorax may occur immediately in the delivery room or later when significant pulmonary disease has developed. The occurrence of a pneumothorax often is associated with positive pressure ventilation, but it also may occur in infants who are not receiving assisted ventilation. Following the initial air leak, the subsequent expansion of intrathoracic spaces often rapidly results in an increase of intrathoracic pressure such that there is an inability to ventilate the lungs and an inability to return venous blood to the heart. This is termed a “tension pneumothorax.”

The rapid clinical deterioration of such infants is caused by circulatory collapse and an inability to ventilate. Any infant who has a sudden precipitous change in ventilatory status associated with an abrupt fall in blood pressure should be evaluated immediately for a pneumothorax. Transillumination of the chest may be used for the rapid diagnosis of severe tension pneumothorax. In cases where the clinical situation allows, an x-ray should be used to make or confirm the diagnosis. Infants in acute distress should have a needle aspiration performed to relieve the tension while preparation is made to place a chest tube. Symptomatic pneumothorax is managed with the insertion of a chest tube until the pulmonary leak is resolved. A chest tube may not be required if the pneumothorax is small and does not involve an infant who is not receiving positive pressure ventilation. Supplemental oxygen (FiO 2 = 1) often is administered for 6-12 hours to hasten reabsorption of the trapped intrapleural air.

Miscellaneous

Multiple gestation

The delivery and subsequent resuscitation of multiple infants presents a considerable challenge to the labor and delivery team. The first consideration to be addressed with the initial prenatal diagnosis of multiple gestations is to ensure that the care of such a pregnancy is at an institution capable of providing such support for the mother and multiple infants at delivery. As for singleton births, a minimum of 2 experienced personnel should be available for each infant. Because multiple gestation infants often are born prematurely (especially with higher order gestation) more personnel may be required for each infant. Therefore, for higher order gestation involving triplets or more, preparation to ensure the presence of appropriate personnel and equipment must be planned well in advance of the delivery.

The team should be led and organized by a designated experienced leader, and the planning phase should involve a number of disciplines, including neonatologists, perinatologists, nurse practitioners, pediatricians, nursery and obstetrics nurses, respiratory therapists, and pharmacists. The delivery team should consist of other individuals who are prepared to make complex decisions, perform technical skills, and respond quickly to changing circumstances. Organization and teamwork is essential with adequate personnel prospectively identified to respond to each infant. These preparations are becoming more commonplace because an increasing number of multiple birth pregnancies are resulting from assisted conception.

Hydrops

When preparing for the resuscitation of a hydropic infant, it is essential to have a sufficient number of skilled personnel in the delivery room to ensure that the multiple needs of this significantly compromised neonate can be met. Equipment should be prepared before the delivery, and all personnel in the room should be assigned specific procedures, such as a paracentesis or thoracentesis, if required. These procedures may need to be performed immediately if the fluid accumulation is causing difficulties in ventilation. If the hydrops is caused by anemia, blood for transfusion should be available in the delivery

room. Because of the excess fluid in the lungs, often using high pressures and oxygen are necessary initially. Artificial surfactant administration also has been attempted in the delivery room to treat any surfactant deficiency in an attempt to improve pulmonary function. Umbilical venous and arterial lines should be placed and central venous pressures monitored.

Omphalocele and gastroschisis

Gastroschisis is an abdominal wall defect lateral to the umbilicus that does not have a sac or membrane covering the bowel. In contrast, an omphalocele involves the bowel herniating through the umbilical opening, with the bowel covered by a thin membrane, unless the membrane has been ruptured intrapartum. For both omphalocele and gastroschisis, it is necessary to maintain adequate intravascular fluid volume, to maintain thermoregulation, and to prevent bowel ischemia. Preoperatively, these infants have increased fluid requirements unless the bowel is appropriately wrapped with an airtight material.

The bowel may be first wrapped with warmed saline-soaked gauze. Care should be taken to support the bowel and not compromise blood flow. Observe the bowel closely to ensure no areas are compromised from the bowel twisting. A 10F Replogle or Salem pump suction catheter should be placed at low continuous suction to decompress the bowel and prevent further ischemic injury. If the infant is diagnosed with an omphalocele, the blood glucose should be assessed because this defect may be associated with Beckwith-Wiedemann syndrome. Trisomy 18 is associated with this anomaly. Therefore, if the condition is recognized prenatally, amniocentesis for chromosomal analysis should be offered to the family.

If chromosomal information is not available at the time of delivery and there are other anomalies are consistent with Trisomy 18, surgery should be delayed until a complete genetic evaluation is complete.

Congenital anomalies

Severe malformations observed in the delivery room should not change the resuscitative management unless skilled and experienced care providers are able to determine that the condition is incompatible with life. The family should be involved in any decision in which no resuscitation is to occur. Infants with severe malformations should be resuscitated and stabilized until an accurate diagnosis can be made.

Controversies in resuscitation

Neonatal resuscitation has been standardized with the development of a certification program. Evaluation and recommendations for changes in the current standards is an ongoing process. As new research is published, it is essential to evaluate the quality of the studies and make changes in practice based on evidence. This section outlines some of the current controversies and concerns in resuscitation.

Room air versus 100% oxygen

Oxygen is a drug with the potential for serious adverse effects that must be considered. Oxygen free radicals are capable of tissue injury and have been implicated in several disease states in the neonate. The use of lower oxygen concentrations when resuscitating the neonate may decrease the number of oxygen free radicals and their damaging adverse effects. In one study, resuscitation in room air was shown to be as effective as 100% oxygen at lowering pulmonary vascular resistance. Other investigations have shown that there are no benefits in raising the pO 2 higher than 50 mm Hg.

Although large controlled multicentered trials have been performed indicating room air (FiO 2 = 0.21) is just as effective as 100% oxygen when resuscitating infants, long-term outcomes are still pending. The results of these studies do highlight that, in situations in which 100% oxygen is not available, the resuscitation should proceed with the use of room air and a self-inflating bag. However, recent animal model studies have shown that rats exposed to high oxygen concentrations have increased cellular sodium channel activity that assists in limiting pulmonary edema. Hypoxia has been shown to inhibit cellular

sodium channel activity and increase lung fluid. Therefore, there may be a significant rationale for using high oxygen concentrations during resuscitation of the asphyxiated or hypoxic infant.

Timing of artificial surfactant administration

Surfactant deficiency, which leads to RDS, is the most likely cause for persistent and progressive respiratory distress in premature infants. Controlled randomized clinical studies have shown that the prophylactic use of exogenous surfactant administered to premature infants effectively reduces death secondary to RDS. Controlled randomized clinical studies also have shown that treatment of only those infants who develop RDS symptoms has a significant reduction of death secondary to RDS. Prophylactic dosing of artificial surfactant is performed in the delivery room before the first breath or within 30 minutes following birth.

Controversies related to the prophylactic treatment regiment are related to the interruption of the standard resuscitation paradigm for the administration, treatment, and attendant risks of a population of infants who would not develop RDS, as well as the additional costs related to this dosing scheme. The argument for prophylactic surfactant dosing is that treated infants who require surfactant replacement should have more uniform and effective drug distribution when the lungs are fluid-filled without air-fluid interfaces. Obviously, the treatment of only those infants with the diagnosis of RDS-established results in a smaller proportion of infants being treated. The proportion of infants given prophylactic artificial surfactant therapy who would not develop RDS depends on the entry criteria for prophylactic treatment and population characteristics.

Recent studies have demonstrated that early prophylactic dosing of surfactant is efficacious and associated with better outcomes in extremely premature infants. Researchers have recommended that, whenever possible, infants with surfactant deficiency be identified before delivery using lecithin-sphingomyelin ratio or testing for the presence of phosphatidylglycerol. Researchers also suggest that all infants delivered earlier than 28 weeks of gestation receive their first dose of surfactant in the delivery room within a few minutes of life, following cardiopulmonary stabilization. Infants born later than 28 weeks of gestation should receive rescue therapy as soon as they show clinical signs of RDS.

Intubation and suctioning for meconium

Meconium staining of amniotic fluid occurs in 5-12.5% of all deliveries and rarely is seen before 34 weeks of gestation. Of newborns born with meconium stained fluid, 60% require stabilization and/or resuscitation. Of these infants that require stabilization and/or resuscitation, 4-6% are diagnosed with meconium aspiration syndrome. Meconium aspiration in a newborn can lead to atelectasis, overdistension, pneumothorax, pneumonitis, surfactant deficiency, and persistent pulmonary hypertension. Trained personnel should be at all meconium stained deliveries. Suctioning of the oropharynx and nasal pharynx once the head is delivered is the current standard in obstetrics. Intubation and suctioning of infants beyond this point is still controversial.

In a recent, multicentered, prospective, randomized, controlled trial, it was shown that regardless of the type of meconium, vigorous infants do not have any increased risk for meconium aspiration syndrome if they are not intubated and suctioned. The study also indicated that depressed infants, regardless of the type of meconium, do benefit from intubation and suctioning before the initiation of positive pressure ventilation (PPV).

Depressed infants should be placed on a radiant heat source and no drying or stimulation provided until they are intubated and direct tracheal suctioning is performed. A meconium aspirator should be applied directly to the ETT, and continuous pressure should be applied using 120-150 mm Hg as the tube is removed. If meconium is obtained, it is necessary to evaluate the heart rate before a second intubation is performed. With the second intubation, the practitioner may want to consider providing PPV through the ETT once suctioning is performed. Once an infant has been stabilized, intubation and suctioning can be

performed again. Researchers have stated that meconium can be suctioned from the trachea up to an hour or longer following birth. Note that infants who are vigorous at delivery and then develop respiratory distress or become depressed also should be intubated and suctioned before initiation of PPV, if meconium was present.

Preliminary studies show potential benefits in using dilute surfactant lavage in infants with meconium aspiration syndrome. New research has shown that infants who receive surfactant replacement therapy within 6 hours of delivery have improved oxygenation and a reduced incidence of air leaks, pulmonary morbidity, and length of stay; however, further studies still are necessary before this can be recommended as standard care.

Bibliografy

- American Academy of Pediatrics/American Heart Association: Textbook of Neonatal Resuscitation. American Heart Association 2000.
- Berger PJ, Smolich JJ, Ramsden CA, Walker AM: Effect of lung liquid volume on respiratory performance after caesarean delivery in the lamb. J Physiol (Lond) 1996 May 1; 492 (Pt 3): 905-12 [Medline] .
- Cleary GM, Wiswell TE: Meconium-stained amniotic fluid and the meconium aspiration syndrome. An update. Pediatr Clin North Am 1998 Jun; 45(3): 511-29 [Medline] .
- Daly MD: Carotid chemoreceptor reflex cardioinhibitory responses: comparison of their modulation by central inspiratory neuronal activity and activity of pulmonary stretch afferents. Adv Exp Med Biol 1993; 337: 333-43 [Medline] .
- Dreyfuss D, Saumon G: Role of tidal volume, FRC, and end-inspiratory volume in the development of pulmonary edema following mechanical ventilation. Am Rev Respir Dis 1993 Nov; 148(5): 1194-203 [Medline] .
- Findlay RD, Taeusch HW, Walther FJ: Surfactant replacement therapy for meconium aspiration syndrome. Pediatrics 1996 Jan; 97(1): 48-52 [Medline] .
- Graf H, Leach W, Arieff AI: Evidence for a detrimental effect of bicarbonate therapy in hypoxic lactic acidosis. Science 1985 Feb 15; 227(4688): 754-6 [Medline] .
- Graziani LJ, Spitzer AR, Mitchell DG, et al: Mechanical ventilation in preterm infants: neurosonographic and developmental studies. Pediatrics 1992 Oct; 90(4): 515-22 [Medline] .
- Hudak ML, Martin DJ, Egan EA, et al: A multicenter randomized masked comparison trial of synthetic surfactant versus calf lung surfactant extract in the prevention of neonatal respiratory distress syndrome. Pediatrics 1997 Jul; 100(1): 39-50 [Medline] .
- Lucky J, William K: Resuscitation of the fetus and newborn . Clinics in Perinatology Sept 1999; 26(3): 585-627; 641-67; 683-715.
- Wiswell TE, Gannon CM, Jacob J, et al: Delivery room management of the apparently vigorous meconium-stained neonate: results of the multicenter, international collaborative trial. Pediatrics 2000 Jan; 105(1 Pt 1): 1-7 [Medline] .
- Wung JT, Sahni R, Moffitt ST, et al: Congenital diaphragmatic hernia: survival treated with very delayed surgery, spontaneous respiration, and no chest tube. J Pediatr Surg 1995 Mar; 30(3): 406-9 [Medline] .
- Yue G, Russell WJ, Benos DJ, et al: Increased expression and activity of sodium channels in alveolar type II cells of hyperoxic rats. Proc Natl Acad Sci U S A 1995 Aug 29; 92(18): 8418-22 [Medline] .