The author reviews some of the causes of infant depression and explores some of the resuscitative measures available to the anesthetist.

The role of the nurse anesthetist as a member of the obstetrical team can be manifold. Not only are we available to assist the obstetrician in providing anesthesia to the mother for the delivery, but often we also are asked to assist in resuscitative procedures for an infant who is expectedly or unexpectedly depressed at birth. The purpose of this article is to review some causes of infant depression and to provide some basic concepts on resuscitation of the depressed newborn.

"The transition from intrauterine to extrauterine life is the most dangerous time of our existence."¹ Unless this transition is smooth, serious and permanent disabilities may result.

Several important changes must occur at birth. First, the lung must expand. Expansion, however, may not be a good word, since the lung is not collapsed in utero but is filled with 20-30 ml of fluid per kg of body weight. This fluid, which is made in the lungs and secreted into the oropharynx, has a high protein content and a pH of approximately 6.8. At birth, this fluid must be removed from the lung and replaced with gas.

Second, the fetal circulation must be converted to that of an adult. The cardiac output must be redistributed so that the lung receives 90-100% of the right ventricular output instead of the 5-10% that it received in utero. Third, the lung, rather than the placenta, must oxygenate and remove carbon dioxide from the blood.¹

Fluids may be removed from the lungs during and after birth by several means. Changes in esophageal pressure are related to tidal volumes during the birth process. At the time that the infant’s head is delivered and the shoulders are still in the vagina, the esophageal pressure is greater than 60 cm of water.¹

The large pressure gradient that exists between the lung and mouth forces fluid from the lung into the larynx and out the mouth. As much as 60 ml of fluid may be ejected. The intrathoracic pressure drops to atmospheric level as the thorax is expelled from the vagina. During the first breath, a negative pressure of 60 cm of water is generated; air is slowly taken into the lungs; the breath is held for approximately two seconds; and the gas is exhaled. "Approximately 75% of the first breath is retained within the lung as part of the developing functional residual capacity."¹

The next few breaths are very similar to the first, with lesser amounts of gas retained each time. This pattern of ventilation aids not only in removing fluid from the lungs, but also ventilates all areas of the lungs. After birth, the fluid that remains in the lungs is either...
removed by absorption into capillaries and lymphatics or coughed up and swallowed.

**Dawes' three phases of circulation**

The changes that occur in circulation at birth are very complex. Dawes divides these circulatory changes into three phases.

The fetal or parallel circulation is the phase in which blood returning from the placenta is shunted through the foramen ovale to the systemic circulation, and pulmonary artery blood is shunted across the ductus arteriosus to the placenta. The transitional circulation is the phase in which there is decreased flow through the foramen ovale but persistence of some flow through the ductus arteriosus. The adult or series circulation is that in which the blood flows from the right heart to the lungs, back to the left heart, and then to the body.

The parallel circulation of the fetus normally should convert to the series circulation of the adult soon after birth; and at this time, the right-to-left ductal and foraminal shunts are eliminated. Pulmonary blood flow, in addition, increases from 10% to almost 100% of right-sided cardiac output. "Clinically, depression of the newborn is failure to establish normal ventilation and circulation at birth."1

**Depression of infants**

Infant depression may result from many causes; some of the most common are prematurity, obstetrical trauma, fetal malformations, anesthesia, maternal medications, and asphyxia.

Some of the major problems associated with the premature infant may be deficient alveolar surfactant and a poorly developed musculoskeletal system. Obstetrical trauma may produce both circulatory and respiratory dysfunction and should be considered if an infant deteriorates rapidly, despite good cardiovascular and respiratory support. Birth injuries, such as encephalopinal injuries, are more prevalent in premature, breech, or difficult deliveries.2

Infants born with malformations that are incompatible with extrauterine life sometimes appear normal for the first few minutes after birth, but their condition may deteriorate rapidly thereafter. Some fetal malformations, such as bilateral diaphragmatic hernias, may represent exceptions, in that emergency surgical correction could be life-saving.3

Drug-depressed infants hypoventilate and are sluggish and lethargic; but, initially, they may present with a good color and have a heart rate which reflects their intrauterine well being. The infant who is narcotized usually will breathe and cry in response to a sharply painful stimulus. If the infant is placed on its side to maintain airway patency and is stimulated frequently, he will ultimately do well.

Infants that are delivered after prolonged inhalational anesthesia may be quite depressed at birth; but most are essentially anesthetized and merely require standard anesthesiological techniques, such as ventilatory and circulatory support for their emergence from anesthesia. Depression of the infant from drugs should be completely reversible when the drug is metabolized; and the infant should do well, provided he has expert ventilatory care. The greatest danger of drug depression is that it places the infant in jeopardy, unless expert intensive care is available from the minute of delivery. Normal acid-base balance and benign heart rate patterns in the fetus usually rule out asphyxia as the cause of depression.8

Asphyxia neonatorum or birth asphyxia has been defined as a condition of hypoxia, hypercarbia, and acidosis that may develop prior to birth or between birth and the establishment of normal breathing, or at any subsequent time in the early neonatal period.4 It is the most serious cause of infant depression at birth; and unlike drug depression, asphyxia may produce irreversibly damaged infants. "It has been said that some degree of asphyxia is present in every infant as a result of either vaginal or abdominal delivery."6
Intrauterine asphyxia may be caused by premature placental separation, excessive uterine contractility with compromise of placental circulation, or reduced uterine blood flow from either regional anesthesia hypotension or from mechanical compression of the umbilical cord during labor or delivery. Asphyxia is always present for at least a brief period at delivery when the umbilical cord is compressed during fetal expulsion.

Fetal depression may certainly occur from mixed etiologies. As a result, it may be difficult to precisely identify the exact etiology in any given case.

A preliminary evaluation is mandatory upon delivery of every newborn. Regardless of what method is used, it should be done quickly and efficiently. In 1953, Dr. Virginia Apgar introduced a scoring system that has since become almost universally accepted as a means of evaluating the status of the neonate. Many use Apgar scoring in the belief that it improves the qualities of observation and helps in the retrospective evaluation of the severity of asphyxia. The Apgar score will often predict which infants will develop the respiratory distress syndrome, and with some assurance, predict those who will have neurologic problems. Although perfection has never been claimed, the Apgar scoring system has provided stimulus for prompt and active resuscitation, better communication between medical centers, and caused intensive interest among those in the entire perinatal sphere.  

Criteria for resuscitation

Though careful evaluation of every infant at birth is essential before criteria for resuscitation can be established, the Apgar score is a simple and convenient method of evaluating the baby clinically. Each variable of the score should be evaluated individually, and the sum of the points should be computed and recorded at one and five minutes after birth. "There are five criteria: heart rate, respiratory effort, muscle tone, reflex irritability, and color."  

According to the absence/presence and vigor of each sign, a score of 0, 1, or 2 is assigned. Schnider breaks down his classification of depressed newborns into the following: mildly depressed infants, those with an Apgar 5-7; moderately depressed, those with an Apgar of 2-5; and severely depressed, those with an Apgar of 0-2.

Once it has been ascertained that resuscitation is necessary, time is of the utmost importance. Resuscitation should produce in the depressed newborn the same changes as do the vigorous cries of the normal newborn infant, that is, expansion of the lungs and speedy oxygenation of the blood.  

The first consideration is the establishment and maintenance of a clear airway so that oxygenation can take place. As the head is being delivered, the mouth and nose should be suctioned with a bulb syringe to remove mucous and blood. The infant should be placed on his side, with his head down to allow gravity drainage of any mucous or fluids.  

It should be mentioned here that the infant should be placed in a heated resuscitator immediately, as significant heat loss occurs during the first few minutes of life. Every minute of delay causes considerable loss of heat by conduction, convection, radiation, and most significantly, by evaporation of moisture from the skin. This rapid cooling may result in increased oxygen consumption, metabolic acidosis, and respiratory difficulties. Overhead heating and drying of the infant may also prevent further heat loss.  

Nasopharyngeal suction with a small catheter should be brief and gentle. Prolonged suction may cause laryngospasm, bradycardia, or other arrhythmias. These are caused by vagal reflex stimulation and loss of oxygen from excessive suctioning. The passage of the catheter through both nostrils may help to rule out choanal atresia.  

Some authors advocate, at this time,
the passage of the catheter into the stomach to rule out the possibility of esophageal atresia and to allow removal of gastric fluid. Jejunal atresia and other types of bowel obstruction should be suspected if more than 25 ml of fluid are obtained. This should only be done after respirations are well established.¹

As soon as a clear airway has been established, the primary objective becomes the initiation of expansion and the maintenance of adequate ventilation in the lungs. Oxygenation may be achieved through the stimulation of spontaneous efforts by the baby or, if necessary, through artificial ventilation. The precise method of producing this response is dependent upon the condition of the infant and, especially, upon that of his heart. Avery and Fletcher state that the most sensitive indicator of severe hypoxia is the heart rate.⁸ If the heart rate is less than 100 beats per minute and becomes slower in time, prompt resuscitation is indicated. As long as the rate is over 100 beats per minute, the chance of spontaneous onset of respiration is excellent.

The largest group requiring some form of resuscitation are the mildly depressed infants, those with Apgar scores of 5, 6, or 7. If the heart rate is more than 100 beats per minute but the infant is cyanotic, apneic, or hypventilating, one can usually obtain a good breathing and crying response with a few gentle slaps across the feet. This type of stimulation should not be prolonged. Severe traumatic types of stimulation should always be avoided. Hypoxia in this group of infants can usually be prevented by blowing oxygen gently over the face and with occasional stimulation of the feet.²

Artificial ventilation

If a moderately depressed infant cannot be stimulated to breathe adequately and the heart rate falls below 100 beats per minute, immediate artificial ventilation should be administered. Although mouth-to-mouth or mouth-to-nose resuscitation has been performed with success, most delivery rooms contain mechanical means of administering oxygen by intermittent positive pressure. In using a face mask, care must be taken not to occlude the trachea. The head should be in a neutral position, for in the newly born infant, the larynx is about four cervical vertebrae further cephalad than in adults and extending the head may obstruct the airway.

The lungs can rupture with pressures in excess of 25 cm of water, unless the duration is short. The briefest and lowest pressure which will expand the lungs should always be used. There is a wide margin of safety in the administration of short puffs of 100% oxygen at 25-35 cm of water pressure for only one to two seconds. After initial expansion, pressures of 5-15 cm may be effective for artificial ventilation.

These pressures are not sufficient to expand the alveoli; however, they may stimulate sensitive stretch receptors in the pulmonary tree and initiate a gasp in many cases. Endotracheal intubation may be indicated if there is no improvement. A small pharyngeal airway is recommended to prevent obstruction from the tongue whenever intermittent positive pressure breathing is required.⁸

The birth of a severely depressed infant requires urgent attention. If the infant is bradycardic, apneic or has periodic gasps, is pale, cyanotic, and limp, and if there is no response to intermittent positive pressure breathing of oxygen by mask, immediate laryngoscopy and intubation of the trachea must be performed. Oxygen should be administered through the endotracheal tube, but again, only in the least amount of pressure necessary to facilitate good expansion and exchange.

Often, the infant will initiate spontaneous respirations after intubation. If the clinical condition of the infant improves, the endotracheal tube may be removed. In the event that the clinical condition does not improve, the tube should be left in place and the infant

April/1976 177
should be ventilated as long as necessary. Thick meconium staining of the amniotic fluid at birth or the finding of blood and debris in the newborn infant's mouth are indications for immediate endotracheal intubation.

Proper oxygenation alone may sometimes return cardiac action. However, if no heart beat is detectable or if after 30 seconds of good ventilation, the rate is less than 100 beats per minute, external cardiac massage should be undertaken. The first two fingers should be placed immediately over the middle third of the sternum and rhythmic compression begun. A "thumb pressure" technique for cardiac massage on infants has been demonstrated in which the hand encircles the thorax, with the thumb on the sternum and the fingers supporting the back. Regardless of the method used, it should be gentle but effective.

Chemical resuscitation

Chemical resuscitation may be indicated in the depressed newborn. If possible, an umbilical vessel should be cannulated for drug administration. Umbilical venous catheter placement is usually chosen for an emergency situation, since it is technically simpler than umbilical artery catheterization.

Treatment of the severe metabolic acidosis which accompanies cardiac arrest should be instituted as soon as possible. Sodium bicarbonate, 3-10 milliequivalents per kg of body weight should be given over a period of two to five minutes. Evans advocates the use of an equal amount of glucose to dilute the bicarbonate for the additional therapeutic goal of minimizing hypoglycemia, which often accompanies severe asphyxia and may itself be a cause of infant depression.

If the preceding therapy fails to produce cardiovascular response, epinephrine, 1/20-1/10 mg may be injected into the umbilical vein or by direct intracardiac injection. The latter should be avoided if at all possible because of the danger of transecting a coronary artery.

In some instances, 10-20 mg of calcium chloride given over a three to five minute period in the umbilical artery or vein improves myocardial contractility and output. Moya and Schnider have indicated the infusion of isoproterenol, 3 mg in 250 ml of saline, for rates less than 80 per minute despite massage and ventilation.

Narcotic antagonists may be used with a depressed infant if the drug depression is due to maternal narcotic administration. These must be used cautiously, as they do not reverse barbiturate or other drug overdosage and when used alone (not in the presence of narcotic overdosage) act as depressants. Narcan® is the exception, in that it does not act as a depressant if narcotic overdosage is not present. The recommended dosage for Narcan® is 0.02 mg.

Summary

In conclusion, each newborn should be considered as a serious problem in acute care until it has proved itself healthy. If it is determined that the infant is depressed, treatment should be prompt and effective. Time is of the utmost importance.

The cardinal principles of infant resuscitation are: (1) establishment and maintenance of a patent airway from birth, (2) early but gentle expansion and ventilation of the lungs either by stimulation of spontaneous respiration or by artificial means, and (3) continuous evaluation of the condition and the response of the infant to resuscitation.

During an emergency resuscitation, the delivery room is not the place for jurisdictional dispute. It should be established that resuscitation is not the exclusive problem of any one specialist. And, as a general rule, it is wisest to delegate this responsibility to the most available and experienced resuscitator present during the emergency, regardless of his or her specialty.
REFERENCES

Additional references

AUTHOR
Gwen Thompson, CRNA, is a 1968 graduate of the Watts Hospital School of Nursing in Durham, North Carolina, and a 1975 graduate of the North Carolina Baptist Hospital Anesthesia Program for Nurses in Winston-Salem, North Carolina. At the time of this study, she was a student of nurse anesthesia, under the direction of Helen P. Vos, CRNA, Ms. Thompson presently serves as an anesthetist with both Wake Anesthesiology Associates and the Wake Memorial Hospital in Raleigh, North Carolina.