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Asphyxia neonatorum

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I

INTRODUCTION

The hungry cry of an infant in the early hours of the morning may be quite distressing to the parents; yet this lusty cry was music to the attending physician at the time the delivery was performed.

The problem of asphyxia of the newborn is one which concerns anyone assuming responsibility at the delivery, whether he be obstetrician, pediatrician, general practitioner, or anesthesiologist. In order to prevent the condition or treat it when it exists, one must have an understanding of the causes of asphyxia neonatorum, be able to recognize the condition, and understand the respiratory apparatus of the newborn. Then and only then can sound treatment of the condition be instituted.

What, then, is asphyxia? Campbell (1) gives the following definition: "Asphyxia neonatorum may be defined as failure to establish normal respiration after birth, and includes foetal anoxia, primary respiratory failure, persistent atelectasis and respiratory obstruction in the newborn."

The fact that some infants were born with respiratory distress has been recorded in the earliest writings of man. Major (2) quotes the following passage from

The Papyrus Ebers which contained the pediatric knowledge of the ancient Egyptians:

> The prognosis for the child on the day on which it is born may be determined by its cry. If it cries "nee" it will live. If it cries "ba" it will die.

The document, however, does not say what will happen if the baby does not cry at all.

It has only been within the last fifty years that the condition of asphyxia neonatorum has been extensively studied and remedial efforts instituted in prevention of its cause as far as possible. Treatment methods have been established which are scientifically sound. Consequently, as Potter (3) points out, the rate of fetal deaths, and deaths of infants in the first few days of life, has decreased greatly during the past fifty years. She further mentions the fact that fetal and infant death rates have been cut almost in half during the period from 1922 to 1950. She relates that in Fetal Death Statistics United States the fetal deaths in 1922 were 39.4 and neonatal deaths (under 28 days) were 39.7 per thousand live births, a total rate of 79.1 deaths per thousand live births. In 1950 the fetal deaths were 22.9 and the neonatal deaths 20.5 per thousand live births, a total rate of 43.4, and a reduction of 35.7 over the rate in 1922.

Litchfield (4) cites a generally accepted statement:

Two out of every hundred babies refuse to breathe promptly at birth (under ideal circumstances), of which one will never breathe because of permanent injury, and the remainder, that is, one in the hundred, will live or die according to the measures of resuscitation used by the attending physician.

II

ETIOLOGY OF ASPHYXIA NEONATORUM

Little and Tovell (5), in their review of asphyxia neonatorum, divide the etiologic factors into three categories:

1. Factors associated with the mother.
2. Factors associated with the products of conception.
3. Factors associated with labor and delivery.

Maternal causes include age, parity, and health. According to Russ and Strong (6), an arbitrary line may be drawn at the age of 35 years. After this age mothers are more likely to deliver asphyxiated infants.

Parity of the mother is quite important since generally primiparas produce more asphyxiated babies than do multiparas. The reason for this is more cerebral trauma is likely to occur to the baby of the primiparous mother. For some undetermined reason, however, the third, eighth, and eleventh babies seem to have a greater tendency toward asphyxia. Little and Tovell (5) state that after the eighth baby, the incidence of asphyxia increases with parity.

The health of the mother is important since, with diseased conditions of the cardiovascular system, genitourinary system, gastrointestinal system, and respiratory system, as well as in deficiency and metabolic diseases,

the incidence of asphyxia is increased.

Under fetal causes, perhaps the greatest cause of asphyxia is prematurity and immaturity. Congenital anomalies such as defects in the respiratory or circulatory systems, diaphragmatic hernia, hypoplasia of the mandible, abnormal mobility of the tongue, and lesions of the larynx (web, laryngomalacia, stenosis, laryngocoele) may exist. Chalkley (7) cites a case of neonatal asphyxia due to bilateral choanal atresia. Congenital syphilis and erythroblastosis fetalis may produce asphyxia.

The third cause of asphyxia neonatorum, namely, the events of labor and delivery, are of course mainly obstetrical considerations. Russ and Strong (6) report that prolongation of labor is by far the most outstanding single cause of asphyxia. In primiparous mothers, when the first stage of labor exceeds twelve hours, or the second stage has been six hours, the incidence of asphyxia rises sharply. They further report that, when the first stage of labor has passed sixteen hours or the second stage of labor has passed eight or nine hours, over 80-85 per cent of all babies may be expected to be born asphyxiated. In multiparous, the length of labor which may be endured without asphyxia is only eight hours in the first stage and four hours in the second stage.

The reason for this seems to be that in the

multiparous mother who has had relaxation of bone and ligaments, if delivery is prolonged, the effort to push the baby through the birth canal is so much greater than is normally expected that cerebral trauma occurs. Induction with quinine and pituitrin may also produce asphyxia.

The type of delivery influences the incidence of asphyxia. According to Russ and Strong (6), the asphyxial rate in decreasing incidence occurs with version and extraction, high forceps, breech extraction, midforceps, Caesarean section, low forceps, spontaneous delivery, and low forceps with episiotomy.

Other causes of anoxia may be those of placental origin, as gross infarction of the placenta, low insertion of the placenta, placenta previa, and premature separation of the placenta. Anoxia of cord origin may be due to prolapse, shortness, kinking or knotting, the cord about the neck, or rupture of velamentous insertion. Anoxia of uterine origin may be tetany, contraction ring, or hemorrhage.(5)

Further respiratory emergencies in the newborn due to structural or pathologic changes have been listed by Goff (8).

1. Atelectasis
   a. Unexpanded lung
   b. Obstructed bronchus
2. Pneumothorax
3. Atresia of esophagus with tracheo-esophageal fistula

4. Diaphragmatic hernia
5. Thymus gland enlargement
6. Lung cyst

Goff (8) states that atelectasis is by far the most common of these chest emergencies.

Also of extreme importance is consideration of analgesia and anesthesia as used in labor in their role as causes of asphyxia. Clifford (9) states that when babies were delivered naturally 90-95 per cent cried and breathed spontaneously at birth. Litchfield (4) points out that if drugs and anesthetics are improperly used, as many as 30-50 per cent of newborns may fail to breathe promptly at birth. There is general agreement that maternal analgesia and anesthesia result in a high incidence of sleepy babies. Cole (10) reports that with satisfactory analgesia 75 per cent of all newborn infants will possess an intact central nervous system and will spontaneously initiate extra uterine respiration within thirty seconds. He further states that 10 per cent of the babies showed delayed respiration but ultimately cried spontaneously, while 15 per cent required resuscitation. Cole feels that a delay in respirations for as long as thirty seconds after birth is definitely abnormal.

The type, duration, and depth of anesthesia and analgesia are important factors in the production of slow babies. Cole (10) reports that duration of anesthetic is

much less significant than degree of anesthesia. However, he thinks that degree and duration of anesthesia are probably more important than anesthetic agent in production of asphyxia.

Russ and Strong (6) report that usually more than one cause exists to initiate asphyxia in the newborn and that no single factor except cerebral hemorrhage usually produced a severe asphyxia in the newborn.

Taylor (11) states that when a delivery is complicated by major operative obstetrics or major complications of pregnancy, the dangers of analgesia and anesthesia are multiplied many times. He believes that in the delivery of premature infants, twins, breech, difficult forceps, or in patients whose labor has been complicated by hemorrhage or toxemia, no general anesthetic should be given.

Taylor further champions the use of Demerol, 100 mg. with scopalamine, 0.4 mg. combined with inhalation of trichlorethylene for analgesia. He reports that in his series no infant required resuscitation after regional anesthesia, as compared with 10-60 per cent who did after general anesthetics. In his report he also shows that the fetal mortality in Caesarean section delivered with general anesthetics was 19 per cent over a five-year period, and only 8 per cent in a five-year period using

regional or local anesthesia.

The scope of anesthetic agents and analgesic agents in obstetrics is too large for this discussion.

III

PHYSIOLOGY OF RESPIRATION OF THE NEWBORN

In order to plan a rational therapy for the asphyxiated newborn, a knowledge of the physiology of the onset of respiration would be useful. Numerous theories have been proposed to explain this mechanism. Preyer (12), in 1882, suggested that physical factors such as trauma of labor reflexly stimulated the respiratory center. This, however, is disproved by everyday obstetrical practice since trauma increases the incidence of asphyxia.

Theories of Becker (13) and Farber (14) stated that initiation of respiration was caused by insufficient oxygen, an excess of carbon dioxide, or a change in pH of the blood.

Henderson (15) believed that carbon dioxide accumulation acted as the stimulus of respiration, while Coryllos (16) contended that the pH of the blood and specifically the carbonic acid-bicarbonate ratio played a large part in stimulation of the respiratory center.

Barcroft (17) suggested that various sensations tend to initiate respiration and included pain, a rise in blood pressure, a flood of stimuli from the skin, muscles, and joints, and perhaps the strain of the newborn's own weight.

Snyder and Rosenfeld (18) have advanced the theory that from the third or fourth month of intrauterine life, respiratory movements are made by the fetus which become rhythmic and coordinated, and this activity continues after birth; thus the mechanism of respiration is established long before birth.

These movements, they have found, are suppressed by either anoxemia, acapnia, or anesthesia. Sudden respiratory movements have, however, been observed following circulatory disturbances in the human fetus as young as eleven weeks, as shown by Windle (19).

Yet, toward the end of pregnancy, many fetuses apparently not disturbed by circulatory disturbances make rhythmic movements as shown by Snyder and Rosenfeld (18).

Wilson and Farber (20) report:

> We know that these movements continue sometimes for an hour or so but are inconstant and whether or not they play a part in fetal life of every human baby is not yet known. Whether or not respiratory movements have occurred, the lung at birth is a wet and soggy tissue which must at birth begin the process of conversion to an air filled organ. This is a process which requires more effort on the part of the thorax than will be needed later to maintain expansion.

They found that the force needed to overcome the resistance of an atelectatic lung of premature infants to vary from 25 cm. to 30 cm. of water. Smith (21) found the resistance in the lungs of normal human beings

at birth to vary from 20 cm. to 30 cm. of water. Yet he noted that at this pressure there was some bleb formation indicating rupture of the alveoli.

Anoxia of the Fetus

The types of anoxemia are recognizable physiologically and have been classified as anoxic, anemic, and stagnant by Barcroft *et al*. (22). Smith (23) explains these as anoxic anoxia signifying a situation in which the tension of oxygen in the arterial blood is lower than normal and the hemoglobin is unable to become saturated to its usual extent. It is this condition that is most likely to inflict serious anoxemia upon the fetus since anything that disturbs the efficiency of respiration or circulation of the mother, or anything that alters the air she has to breathe--such as anesthetic gases--and anything that reduces the close apposition of the placenta to the uterine wall will lower the tension of oxygen in the arterial blood available to the fetus by means of the cord.

The anemic type of anoxia probably occurs in only such conditions as fetal hydrops where the blood becomes a poor oxygen carrier.

Stagnant anoxia may occur in situations which result in pressure on the cord or knotting it.

Histotoxic anoxemia (introduced by Peters and Van Slyke) probably occurs from any factor which lowers the functioning integrity of the cells of the respiratory center and the tissues of the body; but among such factors, the leading place is given to anoxic anoxemia. The latter form embodies the greatest difficulties for the tissues in both fetal and neonatal life.

Anoxia of the Newborn

Smith further cites a list of clinical mechanisms causing anoxic anoxemia prepared by Best and Taylor, which he modified to fit discussion of neonatal asphyxia:

1. Failure of central stimulation or regulation of breathing.
2. Low oxygen tension in inspired air.
3. Pulmonary disturbances; obstructed airway, atelectasis, pneumonia.
4. Unaerated right to left shunts through or around the heart.

In the above cited situations the blood distributed by the large arteries is insufficiently saturated with oxygen. The first mechanism, i.e., failure of central stimulation or regulation of breathing, does not apply to fetal life since the fetus does not get oxygen by breathing. The fetus may be the victim of the second mechanism: low oxygen tension in inspired air if the mother is breathing an anesthetic gas low in oxygen content or if she has any serious respiratory or circulatory disturbances. Also,

since the placenta serves as a lung, pathologic changes of the placenta and cord may be substituted for pulmonary disturbances (mechanism three). The fourth mechanism does not apply to the fetus since normally it exists by means of a right to left shunt in the circulatory system.

Once the fetus is born, however, as Smith further illustrates, it is dependent upon its own lungs and its respiratory center. Here mechanism one becomes of primary importance since anoxia will rapidly become total if there is no respiratory impulse. The infant is unlikely to be exposed to low oxygen tensions in inspired air so that mechanism two does not hold after birth.

Pulmonary disturbances (mechanism three) are frequent, however--particularly obstructed airways and atelectasis. The circulatory abnormalities of mechanism four occur in numerous instances of congenital anomalies of the heart and the great vessels.

Anoxic anoxemia is thus the most frequent form to occur in the fetus and the newborn; yet it is the form most apt to be alleviated by the administration of oxygen. Unless due to complete failure of the nervous impulse or to an anatomic circulatory shunt, the fundamental discrepancy in saturation of arterial blood can be mitigated if not completely removed by elevating the oxygen content in

the inspired air. By increasing the oxygen content of the air, one may make a proportionate improvement in the amount and tension of oxygen reaching the pulmonary circulation and thence to the left side of the heart--the aorta and the placenta in the case of the fetus in utero, or the tissues in case of the newborn infant.

Stagnant anoxia may occur in utero in situations of cord compression as mentioned previously, or in any condition causing slowing of the blood stream with an impaired circulation. It occurs most frequently in the newborn period with conditions causing local cooling. This causes reduced blood flow due to reflex capillary constriction and pooling of blood in the extremities. The more generalized, prolonged, and serious stagnations are, those resulting from cardiac weakness and states of shock which are often the results of anoxic anoxia affecting the cardiac muscles and other tissues essential to circulation. Purely stagnant anoxia cannot be relieved by increasing the oxygen content of the inspired air since the blood leaving the left side of the heart is already normally oxygenated.

Anemic anoxia does not occur in organisms normally as well supplied with hemoglobin and red cells as the fetus. Anemic states as erythroblastosis fetalis or instances of converted hemoglobin to methemoglobin may occur.

IV

RECOGNITION OF THE ASPHYXIATED INFANT

Judd (24) lists the usual indications of fetal distress as being a rapid heart rate, 170 to 180 per minute; a slow heart rate of less than 100 per minute (which may indicate a knot in the cord, a coil about the neck, or tension or prolapse of the cord); a fluctuation of the heart rate, slow and fast; irregular heart rate (significant between contractions); passage of meconium in other than breech presentations; and increased fetal activity. McCall and Fulsher (25), in reviewing fetal distress as indicated by listening to the fetal heart, found it to be an unreliable sign and their figures show how unpredictable the infant's condition may be with only these signs present. When signs of fetal distress are accompanied by or preceded by a recognized clinical entity known to affect the fetus, these signs can be given importance.

Clifford (9) reports that fetal anoxia may occur days or weeks prior to birth producing a characteristic clinical syndrome that can be recognized at birth by the golden yellow color of the amniotic fluid and the vernix caseosa along with the yellow staining of the infant's skin and nails. He has seen this approximately six times

per thousand births and thus states that yellow vernix is not pathegnemonic for erythroblastosis fetalis.

The time-honored terms of asphyxia pallida and livida have been abandoned for a more clinically sound classification of the degree of asphyxia as described by Flagg (26).

> 1. Mild Asphyxia (Stage of depression)
> Breathing does not take place within thirty seconds of birth of the head. The muscle tone is good. The infant resists movement of the head and limbs. Conjunctival and gag reflexes are present. The heart rate is normal or rapid. Amniotic fluid and mucous plugs may fill the airways. In this group, truly asphyxiated babies must be separated from those simply anesthetized or narcotized by maternal analgesia or anesthesia.
>
> 2. Moderate Asphyxia (Second stage or Stage of Spasticity)
> The muscle tone is poor or absent. There is no resistance to opening the mouth. No reflex irritation is induced by aspiration or by stimulation of the glottis. The heart rate is rapid and soon slows ominously.
>
> 3. Severe Asphyxia (Third stage or Stage of Flaccidity)
> The infant does not respond to attempts at resuscitation. He appears livid or pallid. There is absence of any respiratory movement. Only an occasional flicker of the cardiac impulse may be detected through the thoracic wall. The reflexes are absent.

Cole (10) states that a newborn infant should be considered asphyxiated when breathing does not take place within thirty seconds after birth. He further states that about four minutes is the maximum time brain cells can survive without some oxygen, and that in many cases

irreversible changes may occur in less than one minute.

Clifford (9) emphasizes that the condition of the asphyxiated newborn is never static but that in mild asphyxia, for example, the baby may either rapidly recover or proceed equally rapidly through the moderate and severe stages of asphyxia to death.

V

METHODS OF RESUSCITATION OF THE ASPHYXIATED NEWBORN

Russ and Strong (27) list methods of resuscitation that have been used up until 1941:

1. The use of alternating immersion in hot and cold water.
2. Swinging the child between the legs of the resuscitator with head down.
3. Folding the child in "accordion" fashion.
4. Manual manipulation of the chest by alternately compressing and releasing the thoracic cage.
5. Mouth to mouth insufflation of the lungs.
6. Stripping mucous from the throat by hanging the baby head downward and stroking the neck from chest to chin.
7. Spanking the child, snapping the soles, or rubbing the skin with towels.
8. Sprinkling cold water or ether on the skin.
9. Intratracheal catheterization with the use of a mechanical apparatus to insufflate the lungs with a mixture of carbon dioxide and oxygen under visual pressure.
10. The use of a baby laryngoscope and intratracheal catheterization under direct visualization.
11. Aspiration of the nose, mouth, and throat with a rubber bulb syringe.
12. Intravenous, subcutaneous, and intramuscular use of such drugs as alpha lobeline, caffeine with sodium benzoate, coramine, metrazol, epinephrine and strychnine.
13. The application of carbon dioxide and oxygen or plain oxygen by face mask.
14. The use of mechanical apparatus, such as the Drinker respirator and the Easton and Johnson resuscitator. (To this list several other forms of resuscitators have been more recently added. --Ed. note.)

For the main part, obstetricians realize the futility of the first eight methods. Some of these are

actually barbaric beside adding trauma to the already anoxically insulted newborn.

Russ and Strong (6) list four major principles which must be followed in resuscitation. Each is of equal importance and must be included in each resuscitation. These are:

1. A minimum of handling.
2. Immediate warmth.
3. A clear open airway.
4. Oxygenation of the blood stream within 30 seconds of severing the cord.

All authors are in accord with these main principles, but when one reviews the actual literature concerning methods, one finds many different opinions on the most effective method to be used.

The first procedure is to clear the baby's airway of blood, amniotic fluid, mucous, meconium, and cellular debris. Since the first movement of respiration of necessity must be one of inspiration, the first act of resuscitation should be the cleansing of the respiratory tract of the nares, the mouth, the pharynx, and, when necessary, even the trachea and upper bronchial tree.(28)

If the infant is in the stage of flaccidity with collapsed glottic structures and absent glottic reflexes, it is essential to intubate the trachea for the purposes of aspiration and establishment of an airway through which the lungs may be insufflated with oxygen. Bruns (29)

points out that "cleaning the air passages" enjoys the only universal popularity in methods of resuscitating a newborn since quite often, as the air passages are being cleaned, respirations begin.

There are several methods of cleansing the air passages.

Nasopharyngeal Aspiration.--The first most effective means of preventing a respiratory problem in the newborn is cleansing the nose, mouth, and pharynx when the infant's head first emerges over the perineum. The important factor is taking time to accomplish this procedure whether it be done with a nasopharyngeal catheter or with a rubber bulb. Morgan and Reyes (30) point out that care should be taken to introduce the catheter into the region of the glottis in order to aspirate any material that might be sucked into the larynx and trachea from this point. The nasopharyngeal catheter seems to be more effective in cleansing than the rubber bulb. Cappe and Pallin (31) recommend a method of preparing a pharyngeal suction catheter: The distal 1 inch of an ordinary No. 14 French rubber catheter is cut off, the new end is placed in a flame for 3-4 seconds and the scorched end is then brushed with ether. This imparts a spongy consistency to the suction tip which is not traumatic. An extra hole is then cut in the catheter 1/4 inch from

the tip and then the catheter is attached to a mucous trap mouthpiece.

Tracheobronchial Aspiration.--Morgan and Reyes (30) believe that direct intubation is not necessary. They believe it is sufficient to strip the mucous out of the trachea with the external finger and to aspirate it from the mouth, nose, and nasopharynx with a soft rubber catheter. They feel that direct laryngoscopic intubation is a highly technical procedure and should not be done by unskilled personnel. Bruns (29), however, points out that the dangers of direct intubation are overemphasized and in his series of 155 neonatal fatalities who had this procedure performed, no evidence of epithelial damage was seen at autopsy. Bloxsom(32) discourages tracheobronchial aspiration since he feels that the risk of trauma is so great that tracheal catheterization is associated with a considerable increase in mortality and that an occasional rupture of the lung or stomach occurs with its use.

Gastrointestinal Aspiration.--Bloxsom(33) relates an accepted fact by stating the purpose of labor is twofold: that of birth of the infant, and conditioning the fetus to start respirations promptly. When the head has been delivered, the chest is squeezed by the birth canal and this serves to expel fluid from the respiratory tree

as well as from the esophagus and stomach.

Gellis et al. (34) thus introduced the procedure of gastric suction to prevent the syndrome of delayed respiratory difficulty which they felt was due to the ingestion of large amounts of amniotic fluid, followed by regurgitation and aspiration. Their results compared with a control group were very encouraging. Russ and Strong (6) found that delayed respiratory difficulties were lessened after routine intratracheal catheterization of babies delivered by Caesarean section. They obtained an average of 3-7 cc. of mucoid material from these tracheas compared to 1-2 cc. from the tracheas of babies delivered by normal birth.

Gravity Aspiration.--Most authorities agree that a "head down" position facilitates drainage of amniotic fluid from the lungs. Gibberd (35) relates that inspiration of liquor amnii occurs before delivery if the fetus is asphyxiated and may occur at birth in delivery by Caesarean section or in vaginal delivery. He states, therefore, that from the moment the head is born the most useful measure one can adopt for prevention of asphyxia neonatorum or "atelectasis" is to make every effort to keep the head of the infant lower than the trunk. This act is to be performed until the infant is placed in the crib which also arranges for approximately 15-30°

Trendelenberg. He believes that in two to three cases out of a thousand this makes all the difference between life and death.

It has been pointed out that more of an incline than this may cause intracranial hemorrhage or increase it. Holding the infant by the heels and gyrating it carries with it the risk of cerebral hemorrhage and the hazard of aspirating mucous and amniotic fluid. Goff (8) places the infants on their backs rather than on their sides or face to allow far better expansion of the lungs.

Umbilical Cord Care.--In a study performed by Ballentine (36), he found that by delayed clamping of the umbilical cord in newborn infants pulsations had ceased; the infant was given 96 cc. of blood. DeMarsh et al. (37) states that immediate clamping of the cord in the newborn deprives the infant of an average of 107 cc. of blood. Bruns (29) recommends that, since cord pulsations usually stop after twenty minutes, one should obtain the maximum amount of blood for the fetus in the shortest possible time. Since the blood trapped in the placenta will be transported to an extent of three-fourths its amount to the fetus in three minutes, the combination of holding the infant below the level of the mother and gently stripping the umbilical cord

eight to ten times will promote a rather complete transfusion quickly and atraumatically.

Landau et al. (38) believe hematogenic shock is a contributing factor in the mortality of Caesarean section babies and advocate removing infant, placenta, and cord intact and then suspending the placenta above the infant for a period of six to ten minutes. They estimated they would save about 90 cc. of blood for the infant by this technique and considerably circumvent the picture of delayed shock seen in section babies.

Stimulation.--Gibberd (35) points out that in the treatment of an established condition of asphyxia neonatorum it is important to remember that medullary depression is due not to a deficiency in the stimuli which reach it, but to a diminished sensitivity due to anoxia, trauma, or narcosis. Thus attempts of stimulation by drugs or stimulation of the skin are misguided and may be harmful.

Stimulation of the skin in the past has been done by many ways: alternately immersing the newborn in hot and cold water, spanking the child, snapping the soles or rubbing the skin with towels, sprinkling cold water or ether on the skin, blowing air on the skin; all these procedures entail exposure and add an element of shock to an ill baby.

Morgan and Reyes (30), however, state that if the newborn is a nice rosy pink color, a little external stimulation such as rubbing the back or even a gentle spanking applied to the buttocks (not to the small of the back due to danger of adrenal hemorrhage) may initiate respiration. Cappe and Pallin also advise a little external stimulation by slapping the soles of the feet or rubbing the skin over the vertebral spinous processes. They cite Langstein and Ylppo who pointed out that when the child is stimulated to cry, the lungs are more completely expanded.

Analeptics, such as alpha lobeline, pituitrin, camphor, coramine, picrotoxin, caffeine sodiobenzoate, metrazol, adrenalin, and whiskey are to be condemned because they stimulate the medullary centers as part of the generalized cerebral stimulation. They increase metabolic rate, thus increasing tissue oxygen requirements. Following the use of most of these drugs, there is a short stimulant phase which is followed by a more severe depressive stage than that for which it was used to overcome. Recovery from the effects of cerebral depressants occurred distinctively more rapidly when no analeptics were given.(31)

Litchfield (4) reported that alpha lobeline hydrochloride is a safe and effective stimulant of respiration.

The drug lowers the carbon dioxide threshold of the medullary center and is not a cardiac stimulant. He uses grains 1/20 injected into the umbilical vein. Little and Tovell (5), however, found the drug to have respiratory stimulative properties only in doses at a convulsive level.

After respirations have been established, the use of coramine (4-6 minums at 30-minute intervals) may help the heart rate and possibly aid the rhythm of respiration. Russ and Strong (6).

Litchfield (4) reports having saved two infants, and Brunka (39) reports one case by the use of intracardiac injection of adrenalin. When no heart tones are palpable or present on auscultation, the use of adrenalin, 1 cc. of a 1:1000 solution, injected intracardially, may be justified.

The use of 10 mg. Nalorphine given prophylactically to the mother ten minutes prior to delivery or injected in doses of 0.1 mg. via the umbilical vein at delivery is justifiable if one is certain the depression is due to morphine or its derivatives.

Oxygen and Carbon Dioxide.--Anesthetists are in agreement that a flushing of the mother's lungs with pure oxygen before cutting the cord is an excellent stimulus to the newborn's respirations. Authors also seem to be

in accord on the use of 100 per cent oxygen in the resuscitation process. Bruns' (29) review of the literature on oxygen toxicity recommends that after the resuscitation process, the baby should be placed in an environment of oxygen less than 70 per cent for short periods to avoid possible toxic effects of high oxygen concentration.

The question of whether or not to use carbon dioxide in the resuscitation is controversial. Henderson (15) states that in the asphyxiated child there is more carbon dioxide in the blood than normally and that the respiratory center is so depressed that this stimulation is insufficient to excite natural breathing. He states further that when a sufficient percentage of carbon dioxide is administered, the center responds normally. He advocates an addition of 7 per cent carbon dioxide to the oxygen.

Cole (10) cites Eastman who found that carbon dioxide is a stimulant to the respiratory center up to a certain point beyond which the sensitivity of the center is reversed and carbon dioxide becomes a depressant. Cole (10) draws the following conclusions from his clinical experience: In mild cases of asphyxia a mixture of carbon dioxide and oxygen should be used and in severe cases of asphyxia, straight oxygen is indicated. It has been shown

(40) that when the concentration of carbon dioxide in the alveolar air rises above 7 per cent, it paralyzes the respiratory center and results in an initial blood pressure rise followed by an alarming blood pressure fall.

Russ and Strong (6) recommend the use of a mixture of 5 per cent carbon dioxide and 95 per cent oxygen given as inhalation for one or two minutes every thirty minutes for the first six hours to aid in deeper respiration and thus inflate more alveoli.

VI

RATIONAL OF RESUSCITATION

Gibberd (35) states that there are two essentials for adequate oxygenation of the newborn's blood:

1. The respiratory centers in the medulla must be capable of initiating respiratory movements.
2. The respiratory passages must be patent so that air may reach the alveoli of the lungs.

Thus one can see that the main crux of the problem of asphyxia neonatorum centers on a damaged or depressed respiratory center and the presence of atelectasis neonatorum. The treatment, therefore, is one designed to cope with these two situations.

It is established that if irreversible changes due to anoxia or trauma have not already occurred in the vital centers of the medulla, supplying oxygen will restore the depressed medullary centers since the reason for the depression was anoxia.

Many techniques aimed at aeration of the lungs have been described, each usually being described as being the most effective form of resuscitation accompanied with impressive statistics. Bruns (29), however, adequately summarizes the situation:

> Most of the methods of resuscitation, however, are not applicable to the newborn since they presuppose that expanded lung tissue is available and capable of function. The primary effort in the apneic

newborn consists in initially expanding a sufficient number of alveoli to provide an adequate exchange of gases. After the alveoli have been opened, the respiratory epithelium must be mature enough to function.

As has already been pointed out in the physiology section, a considerable amount of pressure is needed to expand the lungs initially--30 cm. of water or more. Smith (21) found that at 19 cm. of water pressure, some alveoli will rupture and produce emphysematous blebs. Most authors agree that if positive pressures are used initially to expand the alveoli, the pressure should be between 10 cm. to 20 cm. of water pressure. The margin of safety is small, however, since 25 cm. of water pressure may result in alveolar rupture.

The acceptable pressure for maintenance by positive pressure is 12 cm. of water pressure given at a rate of forty times a minute.

Clifford (9) makes no effort to expand the asphyxiated infant's lungs forcibly, but keeps him in an atmosphere of 50-60 per cent oxygen. Then if the infant is to survive the condition, X-ray and physical examination will reveal expansion of atelectatic areas by the fourth day of life. Morgan and Reyes (30) feel that the technique of cleaning the airway, supplying warmth, and a high oxygen environment by means of a face mask or tracheal intubation and "leaving the rest to the infant"

is inadequate. They feel that in actively inflating the lungs, one stimulates the Hering-Breuer reflex which they feel is probably the strongest reflex there is in initiating respiration. They remind that inflation of the lung reflexly initiates expiration and deflation of the lung stimulates inspiration. Further, they point out that when air is blowing down the trachea, the diaphragm reflexly relaxes producing expiration and air blowing up the trachea reflexly causes contraction of the diaphragm and inspiration.

VII

TECHNIQUES OF RESUSCITATION

With the aforementioned statements in mind, one may discuss the various methods of resuscitation.

Manual Resuscitation.--An early method of manual resuscitation designed to change the size of the thoracic cage is still in use but it is not physiologically sound. Methods used are compression of the chest and abdomen, or "jackknifing" the body. More recently the same principle of displacement of the abdominal viscera has been attempted with teetering or rocking the newborn, either manually or in specifically constructed incubators as the Eve Rocking Cradle. Goff (8) finds it difficult to understand how air can be expressed from airless lungs. The manual methods also have the risk of increasing intracranial hemorrhage and producing damage to the chest or to the viscera.

Mechanical Respiration.--Mouth to mouth artificial respiration is probably the oldest known method of insufflation. This may be modified by blowing down an intratracheal catheter. This procedure is condemned on several accounts: The exact dosage of the amount of air or the amount of pressure cannot be determined, and there is also the danger of transmitting pathogenic bacteria. If no

insufflation apparatus is available, the method of Russ and Strong (6) may be used. This is as follows: If the infant shows no attempt to breathe, an intratracheal catheter is introduced gently about one-half inch into the trachea and secretions are aspirated. Then, by means of the resuscitator's own breath through the catheter and bulb, the chest is gently inflated at a rate of 24-30 times a minute and allowed to deflate by its own elastic recoil. One should inflate the chest sufficiently to cause the chest wall to begin to expand. Further inflation than that may cause damage.

Intermittent Positive Pressure Resuscitation.--The Kreiselman infant respirator is an approved resuscitator which delivers controlled pressures by a face mask at rhythms controlled by the operator but cycled to the infant. The machine is also equipped with a heated bassinet, continuous flow mask, and an electric aspirator. This machine is championed by authors who favor resuscitation by positive pressure. It has been criticized due to expansion of the stomach, impediment of circulation, and rupture of alveoli.

Niggli (40) outlines the inexpensive construction of an apparatus similar to the Kreiselman machine: The oxygen is taken from one of the usual tanks with a manometer, reducing valve, moistener, and the desired number of

liters per minute is set by means of the flowmeter. The oxygen current flows through a rubber tube into a breathing bag of two liters capacity connected with a mask; a safeguard against excessive pressure is placed into this pathway in the form of a glass cylinder containing water into which a T tube is immersed to a variable depth, depending on how much maximal pressure should not be exceeded. By rhythmic pressure on the breathing bag with a frequency of 20-30 times a minute, artificial respiration is effected. In order that carbon dioxide does not accumulate, 2-3 liters of oxygen per minute is sufficient to rinse the bag repeatedly. Thus one prevents establishment of a closed system.

Judd (24) describes a similar procedure, but in his apparatus the oxygen bag is connected to an endotracheal tube by means of a cannula having a 2 mm. aperture. The size of the aperture is to regulate the amount of positive pressure.

Flagg (26) and Blaikley (41) have advocated the treatment of the asphyxiated infant by the administration of 100 per cent oxygen by an intratracheal catheter with intermittent pressure. It is felt that absorption of oxygen at some level of the respiratory tract with this technique is sufficient to keep the infant aerated.

Flagg (26) states that the actual condition of

the baby is measured by muscle tone and the reflexes that it presents. Each infant should have immediate suction of the mouth and pharynx on birth. If the muscle tone and reflexes permit easy laryngoscopy, this should be performed immediately and the pharynx and larynx inspected. If no resistance is offered to this procedure, the larynx should be intubated and aspirated. If the glottic reflex is reduced or absent, the intratracheal insufflation should be begun with oxygen at a pressure of 15-20 mm. of mercury. This should be continued until respiration is re-established or the heart has ceased to beat.

Intermittent Negative Pressure.--Mechanical resuscitation has also been attempted with intermittent negative pressure by the use of such machines as the Drinker-Infant respirator. The apparatus is like an "iron lung." The body of the infant is placed in the enclosed chamber with the head outside. Intermittent negative pressures are applied around the body and automatically cycled. Since the efficacy of this machine depends on an intrapleural negative pressure, it is useless in a child who has never breathed (intrapleural negative pressure is nonexistent in a non-breathing infant). Also the machine cannot be regulated to the infant's respiratory pattern and does not enhance cardiac filling. For these

reasons, the machine is no longer in accepted use.

Alternating Positive-Negative Pressure Machines.--(suck and blow machines) These machines, as, for example, the Emerson or E. & J. machines, deliver a negative pressure of 9 mm. and a positive pressure of 13 mm. of mercury. Treatment by this method is acceptable by most authors.

Recent and Experimental Methods of Resuscitation.--More recent methods of active artificial respiration include the Bloxsom Positive Pressure Oxygen-Air Lock (32, 42,43). The asphyxiated infant is placed wholly in the resuscitator. The pressure is cycled between one and three pounds at one-minute intervals, thirty to forty seconds being employed for the positive phase which increases the pressure from one to three pounds and fifteen seconds for the negative phase which decreases the pressure from one to three pounds.

The theory behind this mechanism is based upon the fact that the bronchial tree presents a resistance to the pressure wave of gases diffusing into and out of the alveolae and pulmonary sacs amounting to a differential of about 5 cm. of water. This differential is supposedly sufficient to cause gentle compression of the thoracic cage when the pressure is being raised in the lock, and gentle expansion of the thoracic cage when the pressure

is being lowered in the lock. When compression occurs, secretions in the upper respiratory tract are forced out of the mouth and nares (according to Bloxsom), thus eliminating the trauma of intubation and aspiration. The effectiveness of this machine is controversial.

Another experimental apparatus for the resuscitation of the apneic newborn is the Electrophrenic artificial respirator. External electrodes are used to stimulate the phrenic nerve in the neck and thus produce diaphragmatic activity and inspiration. Cross and Roberts (44) have demonstrated that this method is capable of lung expansion, but since normal muscles are utilized to expand the lung, the dangers of pulmonary emphysema, ruptured alveoli, and distention of the stomach--encountered with positive pressure--are not encountered. This method is still considered to be in the experimental stage.

The Use of Gastric Oxygen.--It has been demonstrated that the gastric mucosa is capable of absorbing oxygen and its use is a valuable method of oxygenating the apneic premature. This method is simple and atraumatic to the lungs. Lord et al. (45) use this method on prematures of under four pounds. They have found that this technique supplies sufficient oxygen to enable a depressed respiratory center to recover. They feel that endotracheal oxygen insufflation is too traumatic a

procedure for small prematures to tolerate. On mature infants this procedure is not used since they hesitate to deny them the benefits of endotracheal oxygen. Gastric oxygen is administered by the use of one or two catheters (one catheter for insufflation and one catheter for deflation), supplying 2-3 liters of oxygen per minute.

Immediate Warmth.--Warmth is important to the premature and to the infant who has had distress (immediate warmth, however, should be given the infant who has spontaneous respirations) because the heat regulating center may not be completely developed in the premature or may have been damaged in the baby who has undergone distress.

VIII

AFTERCARE OF THE RESUSCITATED NEWBORN

The aftercare of the resuscitated newborn is of equal importance to that of the resuscitation. A resuscitated infant must be treated as if he were ill and as if he were a premature baby. Russ and Strong (6) recommend the following instruction:

1. The baby should be placed in an incubator at approximately 90° F.
2. Oxygen should be given continuously for at least six to twelve hours.
3. Careful observation of the baby must be made every fifteen or twenty minutes as to color, respirations, and activity.
4. No feeding is offered for the first twelve to eighteen hours, or as long as forty-eight hours.
5. Coramine (dosage 4-6 minims) may be given at thirty minute intervals if the heart beat seems to be too slow or if respirations seem to be flagging.
6. Carbon dioxide, 5 per cent, and oxygen, 95 per cent, should be given as inhalation for one or two minutes every thirty minutes for the first six hours to aid in deeper respiration and to inflate more alveoli.

These authors, however, point out that every case must be individualized and the orders should be changed to meet the specific circumstances. Cappe and Pallin (31) further recommend frequent suctioning of the nasopharynx, turning the infant on the opposite side and stimulating him to cry every thirty minutes. The infant should be placed in an incubator where temperature,

humidity, and oxygen concentration can be controlled. There has been recent work with surface tension reducing agents to "unstick" collapsed alveoli.

IX

SUMMARY, CONCLUSION, AND AN OUTLINE OF THE TREATMENT OF ASPHYXIA NEONATORUM

With this paper an attempt has been made to point out the causes of asphyxia as to maternal causes, fetal causes, and obstetrical causes. The physiology of respiration of the newborn has been reviewed, as have been the methods available for resuscitation. A summary, then, of the author's opinion on the methods used for resuscitation as demonstrated in the literature of the last twenty-five years is in order.

In the author's opinion, Cappe and Pallin (31) and Beattie and Zwerling (46) outline a very rational and straightforward treatment of the asphyxiated infant. The following paragraphs will incorporate their plan of therapy, as well as the author's conclusions from previously mentioned sources.

Of course, in any plan of resuscitation, one must adhere to the cardinal principles of a minimum of handling, immediate warmth, a clear airway, and oxygenation of the blood stream.

As soon as the head has been delivered, the mouth, nasopharynx, and nares are suctioned with a No. 14 French rubber catheter. When the delivery is completed, the

infant is held in a "head down" position of 15°, and the mouth, nasopharynx, and nares are again suctioned. (Cappe and Pallin (31) state that when intracranial hemorrhage is suspected, a condition manifested by a high-pitched, weak cry, the infant is placed in a horizontal position.)

Body warmth is maintained by wrapping the newborn in warm blankets. The majority of infants will have cried or begun respirations by the time this is performed. The skin over the spine may be rubbed or the soles may be snapped gently to help initiate respirations. The cord is not clamped for three minutes or more unless special indications are present. After ligation of the cord, the infant is placed in a heated incubator in the Trendelenberg 15° position and allowed to remain there for ten minutes. If he shows no signs of distress during this period, he is removed to the nursery.

If the infant shows signs of obstructive breathing and respiration of the nasopharynx does not remove the obstruction, the larynx is intubated under direct vision, using an infant laryngoscope and a plastic endotracheal tube No. 0 or No. 1. The trachea is then aspirated, using a No. 8 or No. 9 French catheter or polyethylene tubing. On direct visualization of the larynx and trachea, congenital abnormalities may be

detected and treatment thereof instituted if possible.

If no respiratory movements are present at this time, a plastic oropharyngeal airway is inserted and positive pressure by means of a face mask or connection to the endotracheal tube is instituted. Oxygen, 100 per cent, at a pressure of 12 mm. of mercury, is given at approximately the infant's respiratory rate, i.e., 40 per minute.

Cappe and Pallin (31) recommend using the face mask initially and applying it loosely at first and then gradually increasing the pressure of contact. They feel that if the pressure within the respiratory tract is increased gradually, no damage will be incurred to the pulmonary tissue. If the infant remains flaccid under this treatment, the infant is intubated and the endotracheal tube is connected to the resuscitator. The infant is extubated or the airway is removed when reflex activity of the infant is established.

Beattie and Zwerling (46) recommend determination of the degree of asphyxia. If the infant is in the first stage of asphyxia (stage of depression), the treatment should be tracheal aspiration, insertion of an oropharyngeal airway, and administration of intermittent positive pressure at 12 mm. of mercury. After the respirations become regular, a continuous flow of oxygen at 4 liters

per minute replaces the use of intermittent positive pressure. If the respirations are regular for twenty minutes and the infant's color and condition appear good, the infant is placed in an incubator for the next twenty-four hours, being supplied with 8 liters of oxygen per minute. (Recent work shows that oxygen concentration should not exceed 40 per cent in order to avoid retrolental fibroplasia.--Ed. note) The infant should be frequently examined for recurrent asphyxia.

When the stage of spasticity or second stage of asphyxia exists, treatment should be the same as for stage one. The infant should be observed closely for symptoms indicating progression into the third stage of asphyxia.

If the infant is in the stage of flaccidity or stage three of asphyxia, Beattie and Zwerling (46) recommend passage of an infant bronchoscope and aspiration of the trachea and main stem bronchi. Intermittent positive pressure is now given, but at the first signs of spontaneous respiration, or when activity of the glottis reappears, the bronchoscope is removed and an oropharyngeal airway is inserted. Intermittent positive pressure is now given by the face mask. Again these authors recommend observation. If normal respirations are present for twenty minutes and color appears satisfactory, the infant

is removed to an incubator in which 8 liters of oxygen per minute are delivered. The infant should again be carefully observed for a period of twenty-four hours.

It must be remembered, however, that use of an infant bronchoscope should only be made by skilled operators.

Analeptics may be of some value once respirations have been established, but it is generally considered that they should not be used.

If the infant is less than four pounds in weight, the infant should not be subjected to the trauma of intubation. Rather, gastric oxygen should be given to maintain oxygenation.

To prevent regurgitation and aspiration of stomach contents, gastric aspiration should be performed on infants delivered by Caesarean section and prematures and newborns who have had a period of distress.

Aftercare is as important as resuscitation. The infant should be carefully observed as to color, respirations, and activity. Each half hour carbon dioxide inhalation should be given; the infant is also then turned to the opposite side and stimulated to cry. The temperature, humidity, and oxygen supply should be well regulated.

With good obstetrical care and logical systematic

treatment of asphyxia when it exists, the infant mortality rate can be significantly decreased.

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