Resuscitation of the Depressed Newborn

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TECHNIQUES for resuscitation of newborn infants with cardiorespiratory depression are undoubtedly as old as mankind. Mouth to mouth resuscitation of an infant was described in the Bible,[1] and mouth to intratracheal cannula ventilation was described as early as 1780 by Scheele.[2] John Snow reported the use of Reid's efficient intermittent positive pressure device for infant resuscitation in 1841.[3] Yet despite the early availability of methods similar to those to be outlined in this report, equally sincere proposals continue to be made for the use of ineffectual and potentially dangerous techniques such as "intragastric respiration"[4]; "cutaneous respiration"[5]; and convulsant analeptic drugs.[6, 82]

Unfortunately, the relative effectiveness of these resuscitative techniques is still highly controversial. Because of the urgent nature of the problem and the patient involved, it is difficult to make well-controlled comparisons of the clinical value of different methods. Results of studies in newborn animals can be misleading when applied to the human. In addition, the multiple etiologies of human newborn depression, and the lack of uniform methods for observing and reporting results are partly responsible for the controversy surrounding the proper method of therapy.

Education regarding effective techniques for newborn resuscitation is, however, a most urgent and important public health need. Nearly 35 per cent of all deaths during the first year of life occur in the first 24 hours after birth[7] and from 44[8] to 59 per cent of these deaths are related to anoxia and pulmonary pathology.[9] Prolonged depression at birth is a factor in the respiratory distress syndrome of

the newborn.[10] In addition, a great deal of evidence has accumulated that gross neurologic defects,[11, 12] as well as more subtle behavioral and intelligence deficiencies may result from asphyxia neonatorum.[13] Therefore, the prevention and reversal of newborn cardiorespiratory depression should clearly be considered as a delivery room procedure of utmost importance. Moreover, long before birth adequate provision must be made for the education of all personnel in sound techniques of resuscitation, in the delineation of responsibility for resuscitation, and for the maintenance of modern resuscitative equipment.

This report will briefly review the pertinent physiological factors which have lead us to adopt the procedures we recommend, the actual techniques of resuscitation, the necessary equipment, and finally, other currently proposed methods of infant resuscitation.

Regulation of Breathing in the Newborn

In 1882, Preyer[14] suggested that the "trauma of labor" reflexly prepares the infant for its first breath. We have reached sufficient sophistication to realize that a combination of asphyxial, thermal, tactile, and enteroceptive stimuli are usually responsible for the initiation of breathing. The asphyxial stimulus is undoubtedly the strongest[10] and yet, curiously enough, respiration can begin with an apparently adequate umbilical circulation.[16] Moreover, it has been pointed out that the respiratory center of the newborn is less resistant to hypoxia than the adult[17] and that once asphyxia is experienced the infant respiratory center fails to respond to increased P_{CO_2} thereby aggravating acidosis and fetal depression.[18] This was confirmed by Miller[19] who made the important observation that the overall reaction of the infant to hypoxia and hypercarbia is a further depression of the respiratory mechanism in the newborn, up to 6 hours of age. Therefore, it would seem that although mild asphyxia may stimulate the respiratory

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center, increasing hypoxia and hypercarbia would serve as a further depressant to respiration at birth. More recent opinion regards the medullary center as more adult in its response to CO_2, but agrees that at high partial pressures of CO_2, the center becomes depressed.[20]

Upon breathing 100 per cent oxygen the healthy nonasphyxiated infant has a mild decrease in ventilation [21] similar to the adult. On the other hand, unlike the adult, with 10 to 12 per cent oxygen there is little or no increase in ventilation in the very young newborn.[19] This suggests a decreased sensitivity of the carotid body receptors in the newborn infant, which is confirmed by the lack of action potentials found in the carotid sinus nerve of several animal species during the first few hours after birth.[23]

Little work exists regarding the independent function of pH in the respiratory control of the newborn. However, it has recently been shown that in monkeys asphyxiated to the point of no respiratory effort, correction of pH by infusion of buffers or artificial ventilation will reinstitute gasping.[24]

Minor stimuli in initiation of respiration include short exposures to cold [25] and increased endobronchial pressure. As little as 2.5 cm. positive pressure applied to the tracheobroncheal tree of the newborn is a much stronger reflex stimulant to gasping than in the adult,[26] and is undoubtedly a factor in the effectiveness of positive pressure breathing in resuscitative procedures.

Expansion of the Lungs of the Newborn

Early studies utilizing lungs at autopsy indicated that 25 to 30 cm. of water pressure generated in the newborn lung frequently lead to rupture.[27] More recent observations in the healthy intact newborn by Karlberg [28, 29] indicate that the normal newborn frequently develops as much as 70 cm. of negative intrathoracic pressure during initial attempts at respiration, without damage to his lungs. This event is immediately followed in normal infants by a cry which in itself produces up to 40 cm. of positive pressure within the thorax. External pressures in excess of 25 cm. of water and even up to 50 cm. have been found to be safe for brief periods up to 0.3 seconds.[27] Although the normal tidal volume of the new-

born is approximately 15 ml. the initial efforts to breathe may draw in as much as 80 ml. The excess presumably establishes the residual volume which is estimated at 75 to 90 ml. in the newborn.[30]

With the decreasing pulmonary resistance found soon after birth these high pressures diminish rapidly and there is a rapid decrease in the effort of respiration.[29] Surface tension at the air fluid interface requires twice as much pressure for expansion of the lung with air, as with fluid.[31] In the normal lung, it appears that these pressures would be even higher except that opening pressures are markedly reduced by a specific lipoprotein, or "surfactant," which reduces the alveolar surface tension and thereby lowers the resistance to alveolar opening.[32] While it is apparent that this substance is of great significance, its exact role in pathologic states is not yet clear. The substance is under investigation at present as possible therapeutic measure for the respiratory distress syndrome of the newborn infant.[33]

Cardiovascular Changes at Birth

The initiation of effective respiration is equally dependent upon the cardiovascular as well as the ventilatory system. There must be a reduction in pulmonary vascular resistance in order that an adequate flow of blood can be diverted from the shunting mechanisms of the foramen ovale and the ductus arteriosis, to flow through the pulmonary vascular bed. During fetal life only about 10 to 20 per cent of the right ventricular output flows through the pulmonary circulation, and for several hours after birth the critical shunt through the foramen ovale and ductus arteriosus is reversible.[34] Therefore, any stimulus to increased pulmonary vascular resistance such as acute asphyxia, which causes intense pulmonary vasoconstriction, tends to shunt blood past the pulmonary vessels, to open a previously constricted ductus,[35] and thereby, to re-establish the fetal type of shunt. In contrast, high oxygen content decreases pulmonary vascular resistance in the newborn, fostering proper flow through the lungs. It should also be remembered that this reversal is even more likely to occur owing to the persistent high pressures maintained in the pulmonary circulation for several hours postpartum.[36]

Aside from the obvious interference with oxygenation, abnormal shunting is the major feature of the respiratory distress syndrome.[37] In the respiratory distress syndrome it has been estimated that as much as 60 per cent of the alveoli are under-perfused: even in the normal infant 25 per cent of the cardiac output bypasses the lungs via a right to left shunt.[38] Therefore, adequate expansion of the lungs with oxygen decreases the pulmonary vascular resistance and decreases intrapulmonary shunting of blood in the pulmonary vascular tree, thereby increasing fetal respiratory exchange. In addition to the beneficial effects upon the exchange of gases between the pulmonary blood and alveoli, adequate expansion of the lung contributes a reflex stimulus from bronchial receptors that further enhances respiratory effort.

Etiology of Depression in the Newborn

Anesthesia may contribute to newborn depression either through direct drug depression of the newborn respiratory regulatory mechanisms,[39] or by adverse effects on the mother such as hypotension, hypoxia, hypercapnia; or, as has been recently demonstrated, by inadvertent hypocapnia during vigorous ventilation.[40] In addition to anesthetic agents and drugs, certain maternal and fetal complications contribute to the depressed state. These include maternal infection, anemia, diabetes, toxemia, tetanic uterine contractions, abruptio placentae and placenta previa. Furthermore, there are fetal factors such as nuchal or prolapsed cord, superimposed traumatic forceps delivery, prematurity, congenital defects, and respiratory disease due to aspirated meconium or prepartum amnionitis. Many of these complications contribute to depression by causing asphyxia in the infant.

Birth Asphyxia

James[16] described four phases in the response of the newborn monkey to asphyxia; initial hyperpnea, primary apnea, gasping, and secondary apnea. The latter three phases correspond well to the observations of Flagg made 34 years ago in the human being.[41] The end result of inadequate respiratory function is hypercarbia, hypoxemia, decreased pH, decrease in buffer base, and increase in blood lactic acid.

Although for many years depression of the newborn was gauged by oxygen saturation in the umbilical arterial blood at birth, it is now widely accepted that healthy infants may be born with an oxygen saturation ranging from nearly zero to above 70 per cent.[15] This marked variation in oxygen saturation at birth is due to the rapidity with which oxygen saturation falls with occlusion of the umbilical or the placental circulation, for even brief periods. Umbilical cord oxygen saturation can fall to zero within two and one-half minutes of occlusion. On the other hand, carbon dioxide rises at a rate of approximately 10 mm. of mercury per minute in the apneic newborn and pH falls at approximately 0.1 pH unit per minute.[42]

It should be emphasized that acidosis leads to decreased cardiac efficiency. Burnard[43] demonstrated roentgenographic evidence of cardiac dilatation in depressed infants. Moreover, as found in the adult, myocardial contractility and conduction are depressed by a lowered pH. In the case of the newborn, such cardiac depression can contribute to a cycle of inadequate circulation and increased metabolic acidosis which further aggravates the cardiac depression.

Late in pregnancy asphyxia can also initiate strong respiratory movements which move amniotic fluid into the fetal lungs.[16] Ordinarily, however, the well-known fetal respiratory movements *in utero* are incapable of moving fluid into the trachea.[44]

Both hypercapnia[45] and hypoxia[46] increase the passage of depressant drugs from the blood stream across the blood-brain barrier. Therefore, in the presence of depressant drugs their effects are intensified not only by the synergism of hypoxic depression, but also by augmented concentrations in the central nervous system.

In addition, the control of respiration in the newborn is far more easily affected by depressant drugs than is the adult brain.[47, 48, 49]

In view of the pathological significance of neonatal asphyxia it is unfortunate that the problem should have been confused by reference to the ability of fetal brain to engage in anaerobic metabolism.[50] Since there is an in-

creased tolerance to anoxia found in some species of animals which are still immature at birth,[51, 52] it has been suggested that resuscitation should not be an emergency measure in the newborn human. Evidence for this tolerance in the human infant has never been strong, and rests largely on isolated reports of infants who have survived prolonged periods of apparent apnea. Not only does this concept disregard the reported effects of hypercarbia and acidosis but, even more important, in clinical practice it is impossible to tell to what extent hypoxia has progressed before the moment of birth. In addition, the supposed protection conferred by the shift to the left of the fetal hemoglobin oxygen saturation curve is of little practical protective effect against hypoxia, measuring only a 6 mm. of mercury difference from the maternal curve at 50 per cent saturation.[53]

Prevention of Depression in the Newborn

Resuscitation of the newborn begins with measures to prevent severe depression. Establishment of a satisfactory doctor-patient rapport produces a relaxed patient who requires less medication for comfort during labor. Since the maternal and fetal etiologic factors contributing to asphyxia at birth are to some extent unpredictable it is advantageous to choose a form of analgesia which will not augment this depression. The use of systemic anesthesia may significantly retard recovery of the infant from the effects of birth asphyxia.[42] Therefore, greater emphasis should be placed on techniques such as natural childbirth or medical hypnosis,[54] regional anesthesia[55] or various types of inhalation analgesia utilizing intermittent low concentrations of nitrous oxide, trichloroethylene, chloroform[56] or cyclopropane.[57]

Recently experiments have been conducted in an effort to learn whether pulmonary hyperventilation and alkalosis produced in the mother will influence favorably the acid-base status of the neonate, since this may prove to be a means of avoiding or minimizing the acidosis and asphyxia which are frequently found in the newborn.[40] It appears that moderate shifts in maternal acid-base balance toward alkalosis are reflected in the infant circulation at birth. However, above a maternal

pH of approximately 7.55, the infant acid-base values cease to follow those of the mother, and at a pH level of about 7.68 adverse changes were observed in the biochemical status as well as in the clinical condition of the infants.[40]

The vital signs of mother and infant should be followed closely throughout labor. Electrocardiographic studies show that persistent fetal bradycardia is a reliable sign of fetal distress.[59, 58] This is particularly true when meconium stained amniotic fluid is found.[60] When intrauterine distress is recognized, administration of oxygen to the mother may improve fetal oxygenation slightly[61] but should never replace more definitive management, particularly prompt delivery.

Diagnosis of Depression in the Newborn

As in any other pathological condition, treatment of newborn depression rests upon accurate diagnosis. The various physical signs are conveniently summarized by the Apgar Score[62] which can be used as a general guide to resuscitative measures. The Score should be determined initially 60 seconds after the birth of the entire baby, and subsequently at 3, 5, and 10 minutes. Determination of the Score requires only an instant, and provides valuable additional information upon which to base resuscitative therapy at each time interval. A score from 7 to 10 generally applies to a "vigorous," 4–6 to a "depressed" and 0–3 to "markedly depressed" infant.

Valuable as it is, the Apgar Scoring System has certain limitations. It is immediately obvious that not all the scoring components are equally important in any one infant. For example, the majority of healthy infants have a degree of cyanosis that persists for some time after birth. Furthermore, respiratory effort is at times difficult to evaluate; mild flaring of the alae nasal is present in many healthy newborns, as are fine rales and rhonchi at least for the first 15 minutes of life[63]; whereas grunting and retraction usually are signs of respiratory difficulty. In the past, these considerations have led to some resistance to the use of the scoring system. One group, after evaluating only 829 infants concluded, in essence, that since some infants who scored 10 eventually died, and some with very low scores lived, the system was of little value.[64] However, it has

been conclusively shown in a Collaborative Study of Cerebral Palsy of over 32,000 infants in 13 different university centers [65] that the Score was remarkably consistent; the distribution was reproducible and the Score was equally effective in each center as a prognostic criterion for any one group of depressed babies.

Recommended Procedure for Resuscitation

Position. Once the cord is clamped, the infant is immediately wrapped in a warm receiving blanket [66] and placed in the semilateral recumbent position, with head lower than feet. This position aids in drainage of fluid from the nasopharynx and does not aggravate the tendency of the tongue to occlude the airway.[67] There should be a pad under the shoulders allowing the neck, with it the airway, to extend and straighten. An assistant should immediately place a stethoscope on the chest and visually or vocally indicate each beat of the infant's heart during the early minutes of resuscitation. In this manner the resuscitator is constantly aware of the effects of his treatment.

Establishment of an Airway. During fetal life, an acidic fluid, low in protein but iso-osmotic with plasma is produced by the lung [68] and makes up over half of the total weight of the organ at birth.[69] This thin mucus, observed in 57 per cent of babies with high scores in the first 15 minutes of life, is rapidly reabsorbed.[63] Amniotic fluid is less acidic (pH 6.4 versus pH 7.1) and only slightly more proteinaceous (327 versus 600 mg./100 ml.) than the normally present intratracheal fluid.[68] Nonetheless aspiration of amniotic fluid should be prevented by gentle, conservative measures.

Immediately after birth the head should be placed lowermost and fluid allowed to drain from the nasopharynx. "Milking the trachea" by finger manipulation is traumatic, ineffective, and may lead to laryngeal fracture. Suctioning with the bulb syringe should be thorough, but brief at each attempt. It is not generally realized that the newborn exhibits unusually strong vagotonia and will hold his breath reflexly, develop laryngeal spasm, and often extreme bradycardia during pharyngeal stimulation.[94]

Management of Ventilation in the Depressed Newborn Infant. A baby with an Apgar Score of 7 to 10 usually requires little help except prevention of airway obstruction and non-interference with normal crying and breathing. However, depressed infants with Scores of 4 to 6 often need help. Brief efficient pharyngeal suction, and gentle tactile stimulation often are sufficient to increase respiration and tone in these infants. Anal dilatation, back slapping, squeezing the thorax and other such measures should be condemned as traumatic and ineffective. A brisk slap across the soles of the feet, however, is stimulating but not dangerous.

Occasionally, a baby will continue to hypoventilate for 1 to 2 minutes after delivery. If this occurs, oxygen blown over the face or intermittent positive pressure breathing (IPPB) with the face mask should be initiated. Frequently, this is advisable when respirations and muscle tone are poor even if the heart rate remains above 100 beats per minute. Certainly if the heart rate falls below 100, IPPB should always be used. Oxygen, even in the premature, is not a cause of retrolental fibroplasia unless the exposure lasts longer than a few hours.[70]

In using a face mask, care must be exercised not to occlude the trachea with the finger supporting the mandible. One method of preventing this is to hold the mask against the face with the thumb while the index and middle fingers press along either side of the mandible. If the infant is significantly depressed, a number "zero" oral airway will be required in most cases to keep the tongue from obstructing the airway.

Pressures in excess of 25 cm. of water can rupture the lungs unless the duration of application is short. The briefest and lowest pressure which expands the lungs should always be used. This takes the form of short puffs of oxygen enriched air at 25 to 35 cm. of water pressure, for one or two seconds. At this range of inflating pressure there is a wide margin of safety. After initial expansion has been produced with three or four short puffs, pressures of 5 to 10 cm. of water for 0.5 second's duration can be used to provide continued artificial ventilation.[71]

In severely depressed infants scoring 0–3 at one minute, it is usually necessary to intubate the trachea if there is not an immediate re-

sponse to IPPB oxygen by mask. The number of infants requiring this treatment is never large. An incidence of 18 per cent of tracheal intubation has been reported after operative obstetrics in England,[72] but at the Sloane Hospital For Women in New York, the incidence has been no more than 2 per cent of all deliveries.[73]

The tip of the laryngoscope blade should be placed in the vallecula, and rarely does the epiglottis interfere. For the term newborn, a no. 16 Cole endotracheal tube will usually pass with ease. A stylet may be necessary in order to direct the tube around the curve of the base of the tongue. If meconium or thick mucus is observed after exposure of the cords, it should be aspirated via the Cole tube. Care should be exercised not to suction too forcefully.

Following intubation a few sharp puffs of oxygen through the tube will usually initiate breathing and aid in lung expansion. Excessive inflation will frequently cause rupture of the lungs—a force just sufficient to observe movements of the chest is enough. Although 30–40 ml. of air are required in the first few breaths, the normal tidal volume is only 15 ml. during quiet breathing. There is no need to expedite the removal of the tube after spontaneous breathing has begun, since a properly placed tube is of little danger to the newborn. It should be left in place until respiratory force and muscle tone have returned nearly to normal.

Treatment of Cardiac Arrest. When the heart beat has disappeared within 5 to 10 minutes before delivery or when it ceases shortly after delivery, external cardiac massage may be life-saving. Awareness of the possibility of neurological damage in such infants is important, but the likelihood of this sequela is directly proportional to the duration of arrest.[74]

The infant must be quickly laryngoscoped, the pharynx suctioned if necessary, the trachea intubated, and the lungs expanded with oxygen. With oxygenation alone cardiac action often returns in the form of a faintly beating heart. If not, the index and middle fingers of the operator are pressed sharply against the middle third of the baby's sternum, about 100 times per minute, with the infant resting on a hard surface. Effective pressures above 70 mm. of mercury systolic have been measured during external cardiac compression of the newborn.[74, 75, 76] Pressure applied to the lower third of the sternum is not only less effective but also limits the movement of the liver and predisposes to laceration.[77] We favor the method of two fingers rather than overlapping thumbs [77] because the pressure can be successfully carried out with one hand, leaving the other free to palpate the femoral pulse. Ventilation must be maintained during compression of the heart. When adequate compression is established standard measures similar to those in the adult may be carried out if necessary. These include electrocardiography, injection of epinephrine and sodium bicarbonate (see below).

Drug Therapy in Resuscitation of the Newborn. Hypoventilation, hypoxemia and hypercarbia are both a result and a source of depression in the newborn: *ventilation is the most important resuscitative measure.* Drug therapy should be conservative and directed toward temporary support of the failing cardio-respiratory system, counteracting acidosis, or in a very few instances providing specific drug antagonists.

Only the narcotics can be antagonized specifically. Levallorphan (Lorfan) 0.05 mg. or nalorphine (Nalline) 0.2 mg. injected intravenously into the umbilical vein, will often induce prompt recovery from narcotic-induced respiratory depression.[78] These drugs are of no value if the depression is due to asphyxia, barbiturates, or general anesthesia, and may cause further respiratory depression if not due to narcotics.[79] Despite early enthusiastic clinical reports, it has not been adequately shown that these drugs are effective in the newborn when given prophylactically to the mother, in combination with narcotics.[80]

Several drugs have been advocated for use in instances of barbiturate or other drug depression. Among these are caffeine, 30–50 mg. intravenously,[7] methylphenidate (Ritalin) 15–30 mg. intravenously prepartum to the mother or 1–1.5 mg. intramuscularly to the baby,[81] and ethamivan (Emivan) [82] 4–6 mg. intravenously to the baby. However, none of these has proved conclusively to be safe as well as effective.

Picrotoxin, metrazol, coramine and alpha-

lobeline are mentioned only to condemn their use as analeptics. Their basic action is to increase the metabolic rate of the already asphyxiated neural cells, resulting in convulsions due to cellular hypoxia.[83, 84] These drugs should never be used in resuscitation of the newborn.

Although it is not yet confirmed in the human, it has been shown in fetal monkeys that glucose in 50 per cent solution and infusion of buffers are helpful [85] in restoring respiration and counteracting cardiovascular depression. A dose of 4.4 ml. per kilogram per minute of a 0.5 M solution of THAM (trishydroxymethylaminomethane) has been assayed,[86] but respiratory depression, hypoglycemia, and hyperbilirubinemia are the complications that may result.[87] Sodium carbonate appears to cause liver damage, and sodium bicarbonate (7.7 ml./kg./minute of a 0.6 M solution) is not only ineffective in reducing P_{CO_2} of blood during asphyxia but also causes a rapid overswing of pH to the alkalotic side, as CO_2 is excreted.[86] However, it does restore breathing in apneic acidotic animals.

Epinephrine does cause constriction of the patent ductus [16] but, in practice, it is useful only as a direct cardiac stimulant after cardiac arrest, and then only in conjunction with cardiac compression. Extreme care should be exercised in preparing the minute dose of 0.05–0.1 mg. from the concentrated standard solutions.

It is curious, in view of the marked cardiovascular depression found in severe asphyxia, that there is only one report of the use of the commonly employed vasopressors. In one case, 0.4 μg. per kilogram per minute of norepinephrine was reported to have been successful in elevating the systemic pressure in order to prevent right-to-left shunting of blood.[88] It would seem logical that one of the β-activating sympathomimetics which provide a positive inotropic effect and decrease pulmonary vascular resistance without raising systemic pressure might be more valuable.

Responsibility for Resuscitation

During an emergency resuscitation the delivery room is not the place for jurisdictional dispute.[89] The Manual of the American College of Obstetricians and Gynecologists [90] states that the Obstetrician should be responsible for resuscitation. However, we believe that resuscitation is not the exclusive province of any one specialist and that, as a general rule, it is wisest to delegate this responsibility to the most experienced resuscitator present during the emergency regardless of his specialty.

Equipment for Resuscitation

The best of equipment is useless unless it functions properly and its operation is perfectly understood by the operator. Each device should be checked routinely before birth of the infant for such things as full gas cylinders, correct flow settings, proper connectors, functioning lights, and proper size. This is as much the responsibility of the resuscitator. as is skill in laryngeal intubation.

Positive Pressure Devices

1. *Mouth to nose and mouth to mouth* breathing were first described by Smellie in 1752.[91] They are the simplest methods of positive pressure breathing, but in practice they are somewhat difficult to apply to the newborn. If needed, it is best to cover both the nose and mouth of the infant with the resuscitator's own mouth and to extend the neck while elevating the mandible of the infant anteriorly with the finger tips placed behind the rami of the mandible. Positive airway pressure should be just sufficient to move the chest wall; excessive pressure will further embarrass respiration by distending the stomach.

2. *Mouth to tube* respiration was first reported in 1780 by Scheele [2] but was popularized in this country by DeLee [2] in 1924, who advocated placement of the catheter into the trachea, by tactile sense rather than laryngoscopy. Use of the mouth as a means of IPPB is a useful technique in the intubated infant because the source is always instantly available and, after skill has been achieved, is more delicately controllable than mechanical devices. A little practice with a manometer will demonstrate that dangerously high pressures can be generated by the cheek muscles alone, therefore, great care must be exercised in inflating the lungs.

3. *Gas-Activated Intermittent Positive Pressure Devices.* In 1928 Kreiselman presented a remarkably clear concept of the requirements

for successful infant resuscitation.[92] His device provided for maintenance of body temperature, correct head-down posture, clearing the airway by gentle suction, and intermittent positive pressure to the airway if required. The Kreiselman apparatus [92] found its way into almost every hospital, and every potential resuscitator should become familiar with its idiosyncracies. In older models with the water trap, it has been shown [93] that, owing to inadequate size of the gas escape ports in the water trap, which were designed to limit the pressure to 16 mm. of mercury, pressure in excess of 50 cm. of water can be generated briefly on opening the valve. This can be averted by manual adjustment of the spring-loaded "automat" while checking with an external manometer. Newer models provide mechanically operated safety valves; in addition, a slip ring on the head can be moved for initial inflating pressures up to 33 cm. of water. With these models care must be exercised not to ventilate with the highest available pressures for long inspiratory periods, because alveolar rupture may result.[27]

Extreme caution must be exercised with the accessory electric suction on these machines. They are designed for brief use and can withdraw several liters of air in a few seconds, thereby actually robbing the infant's lungs of air and causing arterial desaturation.[94]

A number of similar machines of different manufacture are presently in use in delivery rooms. Their individual characteristics should be completely understood before use. A description of these machines can be found elsewhere.[95] A new device, the Dann Resuscitator, appears to be promisingly versatile in the hands of the expert, but awaits further evaluation.

4. *Positive-Negative Ventilators.* The Emerson, E. & J., Handy,[96] and Mann-Toronto ventilators [97] are examples of resuscitators capable of developing positive pressure as high as 22 cm. of water and negative pressure as low as −12 cm. of water in closed systems. However, with the mask method of resuscitation, a sufficient seal to develop negative pressure in the lungs is difficult to achieve. With a tight fitting endotracheal tube in place negative intrapleural pressures can be developed by these machines and there is a possibility of

precipitating pulmonary edema when cardiac function is already borderline.[71]

5. *Manual Positive Pressure Devices.* As early as 1842, John Snow [3] reported on the use of an efficient hand-operated pressure device for resuscitation of the newborn. Goddard [98] has described a device known as the GBL Hand Resuscitator, which can develop very high pressures (60 cm. of water). Even though the designers cautioned that the duration of the impulse at these high available pressures should be from 0.2 to 0.3 seconds, we have found that safety depends upon the knowledge and experience of the operator in compressing the bulb properly to limit the duration of inflation.

The Ambient Air Breathing Unit, or AMBU, and several modifications recently available, can develop a relatively safe pressure of 40 cm. of water.[99] As with the GBL, an inexperienced resuscitator can easily deliver a dangerously long inspiratory phase to the infant. However, the device is reliable, durable, and uncomplicated.

6. *Direct Connections to Gas Sources.* A simple Ayre's tube (Y-piece) and similar tubes with inflow reservoir bags [100] have been suggested as sources of positive pressure. However, with only a 2-liter flow, pressure of over 280 mm. of mercury can be built up within 3 seconds in these systems. Even a water trap cannot prevent inadvertent high pressures due to temporary occlusion of the outflow tubing. Several deaths have been reported due to rupture of alveoli, mediastinal emphysema, and pneumopericardium.[101]

7. *Miscellaneous Devices*

Tank respirators,[102, 103] *body shells* and *plastic envelopes* attached to positive-negative machines have all been advocated but are patently unsuitable for emergency resuscitation, where seconds count and simplicity is the most important factor. *Rocker beds* [104] are not effective in the presence of unexpanded or atelectatic lungs, and have been of questionable success in maintaining oxygenation of tested laboratory animals.[105] Phrenic nerve stimulators [106] and respiratory muscle stimulators [107] have had a brief trial but were found to require much too much expertness in use.

Airway Equipment Required for Infant Re-

suscitation. A small handled laryngoscope with a straight blade is best. The no. 1 Miller blade is slightly too large for many term infants. The more versatile premature (or 0) size is preferred. Since the rima glottidis is located more superiorly in the infants than in adults, lying opposite the second to third [108] cervical bodies, a curved blade is not satisfactory. Cole endotracheal tubes, sizes 10 through 18, should be available with a simple malleable wire stylet for each tube. Connectors to fit the available positive pressure devices must be available but should not be preinserted into the tubes. Size 00 and 0 oropharyngeal airways, a bulb aspirator, a De-Lee trap with number 10 red rubber catheter, and a stethoscope complete the contents of the resuscitation tray. If a tray is not available for each delivery room, it should be moved from room to room prior to each delivery, regardless of the anticipated fetal condition.

One of the positive pressure devices should be available and examined before the delivery for proper function by the physician responsible for resuscitation, not merely left to an attendant.

Other Currently Proposed Methods of Resuscitation

Hypothermia. Miller [109] has shown in carefully controlled studies that the time of appearance of the last gasp in guinea pigs is greatly prolonged if the animals are subjected to asphyxia during hypothermia, and that piglets asphyxiated at normal temperatures, then cooled, have a higher survival rate and less neurological damage than controls.[110] Clinical reports of 130 human cases treated with hypothermia in Scandinavia have been favorable; in addition, tests of 4,000 c.p.s. auditory acuity in the treated babies seemed to indicate an intact cerebral function in almost all cases.[111, 109, 112]

These results are puzzling when compared to other well-documented reports that hypothermia in the human newborn is deleterious [113] and causes respiratory depression.[66] An increased oxygen requirement under hypothermia in the newborn rabbit,[114] which is initially, at least, in excess of the depression of metabolic requirement has even been reported.

Therefore at the present time hypothermia in resuscitation of the human newborn must be considered a measure still under investigation. In clinical practice the infant's temperature should be maintained at 98.6° F. However, in cases where all standard measures have failed to restore spontaneous breathing, it appears that total body hypothermia to 25° C. initiated by immersion in tap water may certainly be a worthwhile final measure.

Hyperbaric Oxygen. Early enthusiastic claims for resuscitation by continuous total body high pressure oxygenation in a tank [115] have not been confirmed in the human being [116] or in animal experiments.[117] As pointed out by the original authors, the method as currently proposed does not eliminate the danger of carbon dioxide accumulation or remedy the acidosis of asphyxia. Previous experiments with high pressure oxygen to 15 and 20 atmospheres, and total body immersion in physiologic solutions have shown interesting results in animals and in 15 previable human fetuses.[118] One nonviable human fetus was maintained for 31 hours under these conditions. However, even in the animal survivors there was a discouraging incidence of hyaline membrane disease.[118]

It should be recalled that the now discarded Bloxsom air lock was a true hyperbaric machine giving steady pressures of oxygen up to 5 p.s.i. or pulsatile pressures from one to three p.s.i.[119, 120] The lack of effectiveness of this treatment [121] has been recognized for over a decade.[122, 123] In addition, a recent carefully controlled study has shown no benefit from exposure to hyperbaric oxygen in the treatment of respiratory distress syndrome.[124]

Intragastric Oxygen. First clinically reported in 1950 [4] this unfortunate technique was widely acclaimed in Europe. It is practically the only technique now actively proposed which can be proved to be dangerous and of no theoretical or practical value in resuscitation of the asphyxiated newborn.[125] Not only is there little absorption of oxygen from the stomach, but the resultant distension of the stomach mechanically embarrasses whatever sponstaneous respiratory efforts may be present.

Summary

A physiologically sound technique for resuscitation of the newborn infant must rest

upon recognition of the immense importance of the depressive biochemical effects of asphyxia. Every effort should be directed toward the immediate establishment of a patent airway and expansion of the lungs with oxygen. Following this emergency phase, cardiovascular depression, drug or anesthetic-induced central respiratory depression, and metabolic acidosis can be promptly treated with appropriate measures. Resuscitation is a team effort requiring planning, cooperation and anticipation of emergencies before they arise.

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