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of
PEDIATRICS

GELLIS

THE YEAR BOOK of PEDIATRICS

(1955-1956 YEAR BOOK Series)

EDITED BY

SYDNEY S. GELLIS, M.D.

Assistant Professor of Pediatrics, Harvard Medical School;
Senior Physician, Children's Medical Center, Boston;
Pediatrician-in-Chief, Beth Israel Hospital, Boston

ISAAC A. ABT, M.D.

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INTRODUCTION

This introduction appears to be a fitting place for remarks concerning the editorial comments which follow many of the abstracts included in the YEAR BOOK OF PEDIATRICS. In this edition we have continued our practice, as in the past, of asking physicians with special interests to comment on various papers or to clarify some controversial points. It has been argued that authors of articles should be permitted a rebuttal when objections to their conclusions are raised. Such an argument may be a sound one; rebuttals would prove stimulating but are unfortunately impractical since they could go on ad infinitum. When we are aware that the problem is not entirely clear, we are interested in offering another viewpoint. Two opposing opinions may leave the reader up in the air, but the position seems warranted when studies are not definitive.

We wish again to thank all those who have contributed comments and express our appreciation for their patience and helpfulness.

—SYDNEY S. GELLIS.

Resuscitation of Newborn Infant. R. M. Cherniack and A. Boyd[1] (Winnipeg) state that, to initiate respiration, the newborn infant must overcome a resistance to lung expansion which is often as high as 30 cm. H_2O. The lungs will remain atelectatic until an adequate respiratory effort is made. The infant may make one or two feeble attempts, then cease breathing because the efforts are of insufficient magnitude. If initial atelectasis is a significant factor, effort should be exerted to alleviate it.

Since the lungs offer a resistance to expansion of about 30 cm. H_2O, any resuscitator should exert pressures great enough

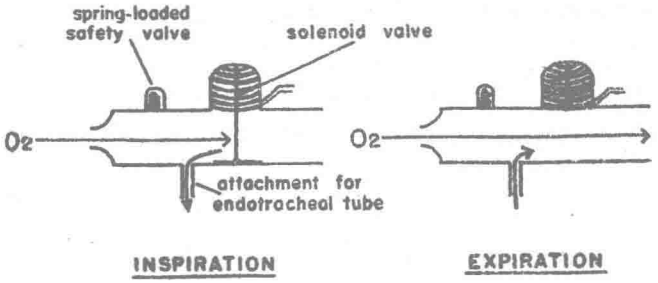


Fig. 1. (Courtesy of Cherniack, R. M., and Boyd, A.: Pediatrics 14:49-52, July, 1954.)

to overcome it. However, Wilson and co-workers found that although pressure of 24.4 cm. H_2O did not cause the lung of the stillborn infant to expand, it did cause gross pulmonary damage. Others have warned of the danger of alveolar rupture at even lower pressures. Present day resuscitators therefore operate at low pressures. The importance of time-pressure relationships has been pointed out by Day *et al.*, who demonstrated that a pressure of 40 cm. H_2O effectively overcame atelectasis of animal fetal lungs without producing lung damage if pressure was applied for less than 0.25 second.

The authors describe the use of a resuscitator (Fig. 1) which diverts oxygen into the endotracheal tube for 0.10 second 10 times a minute at 50 cm. H_2O. A preliminary investigation on

(1) Pediatrics 14:49-52, July, 1954.

three infants who had been dead for about one hour showed no detectable damage or alveolar rupture. The resuscitator was then used successfully on six of seven newborn infants. One died of pneumonia five days after birth. There was no evidence of pneumothorax in any.

Premature infant of 32 weeks' gestation, weighing 3½ lb., was delivered to a mother, 23, with forceps under trilene® anesthesia. The child took one breath, but breathing became markedly irregular, then stopped. Her condition deteriorated despite intubation, oxygen, manual artificial respiration and use of the Kreiselman resuscitator. The new resuscitator was applied 17 minutes after birth. Spontaneous respiration began after the second burst, and the child was placed in an incubator. A chest film the following day was normal, and the course was uneventful.

In the other successful cases, the history was much the same. Gestation varied from 36 to 42 weeks. One infant made no attempt to breathe. Ordinary methods were unsuccessful. The resuscitator was applied 4-17 minutes after birth and its use was continued for 5 minutes in one case.

It is not surprising that use of high pressures did not produce pulmonary damage since the newborn infant can exert inflating pressures of more than 40 cm. H_2O. The method of resuscitation used is based on the concept that persistence of atelectasis is in itself an obstacle to initiation of respiration in the newborn. Once the lungs have been adequately expanded, respiration requires much less respiratory work and the infant can carry on by his own efforts.

[Dr. Richard L. Day commented as follows:

"The authors have shown with reasonable certainty that inflation of infant lungs with higher pressures than customarily recommended does not injure the lungs provided duration of inflation is controlled. The evidence that this method of resuscitation saves lives is on less secure grounds, since the number of cases is small. In order to prove the life-saving capacities of any method of infant resuscitation it would be necessary to carry out a more extensive study and in addition to use controls. No method of resuscitation has been studied in this way.

"An important feature of the short sharp pressure method which is advocated is that the amount of air entering the lungs is in part related to the size of the intratracheal tube. Anyone attempting this method should therefore study his apparatus on cadavers first, to be sure of its characteristics. Use of such pressures with a face mask instead of an intratracheal tube needs to be studied also."—Ed.]

Simple Device for Oxygen Therapy of Infants, Particularly in Asphyxia Neonatorum is reported by Y. Åkerrén[2] (Götenborg, Sweden). Barcroft pointed out that afferent nerve impulses from the region of the trigeminal nerve first release respiratory movements in fetal lambs. To stimulate this region

(2) J. Obst. & Gynaec. Brit. Emp. 61:477-479, August, 1954.

intensely and supply oxygen to the asphyxiated fetus, he directed a stream of oxygen via a rubber tube toward the nose and mouth. To stimulate as much of the trigeminal nerve region as possible, the stream of oxygen should be as powerful as allowable without producing injury. If respiratory movements are released, the oxygen content in the inspired air rises.

For two years the author has used a similar device in the labor and premature wards for treatment of asphyxia. It consists of a plastic or glass funnel, the shaft of which is completely filled by a stiff rubber tube. The funnel is placed so that its lower edge encircles the infant's chin and the end of the rubber tube is directed at the infant's upper lip at a distance of 0.5-2 cm. With a flow of 3-4 L./minute of oxygen through the rubber tube, such a strong jet is usually produced that phenomena of stimulation, as respiratory and/or defensive movements, are produced.

The device is used most often for asphyxia neonatorum and attacks of apnea in prematures. In the latter, risk of retrolental fibroplasia is not increased because the stimulation usually lasts only short periods. In prolonged or repeated use of the funnel, the oxygen content must be controlled. The method has also been used with gastrointestinal administration of oxygen in asphyxia, in severe respiratory tract conditions and in two cases of severe diarrhea with coma and cessation of or inadequate respiration.

Effects of Hypoxia on Respiration of Newborn Infants. Herbert C. Miller and Franklin C. Behrle[3] (Univ. of Kansas) administered atmospheres containing 10 and 12% oxygen to healthy newborn infants of different ages. Infants under 24 hours old tended to hypoventilate throughout the hypoxia. Those 6-11 days old hyperventilated for the first two or three minutes and then showed a decrease, although not so marked as in those under 24 hours. Infants aged 16-48 days showed the greatest increase in ventilation, but even in them it was poorly maintained compared with responses in adults.

Hypoxia produced slowing of the respiratory rate in all infants except the oldest. Tidal air was increased at first and then decreased. Increases in tidal air accounted for most of the increase in minute volume seen in the older infants. Hypoxia increased the incidence of periodic breathing in the two older groups of infants but had little effect on those under 24 hours

(3) Pediatrics 14:93-103, August, 1954.

old. Respiratory patterns appeared unaltered by the hypoxic conditions.

The comparatively weak response to hypoxia of infants immediately after birth possibly is dependent on relatively poor chemoreceptor reflexes which gradually increase in strength during the first weeks of postnatal life. It is also suggested that the relative inability to induce periodic breathing in infants under 24 hours of age, compared with those several weeks old, is further indication that metabolism of the medullary respiratory centers is to a larger extent anaerobic than at later periods of postnatal life.

Effect of High Concentrations of Carbon Dioxide and Oxygen on Respiration of Full Term Infants. Herbert C. Miller[4] (Univ. of Kansas) reports that 5% carbon dioxide added to 20% oxygen is a more powerful stimulus to respiration of healthy full term infants than 100% oxygen. In full term infants hypoventilating because of exposure to low concentrations of oxygen, minute volumes were restored more rapidly when 5% carbon dioxide was added to oxygen concentrations of 12-95% than when 100% oxygen was used. Age appeared to have but slight effect on response to breathing 5% carbon dioxide or 100% oxygen. Infants under two or three hours old were not tested.

Too little is known concerning the chemical, physiologic and anatomic changes occurring in asphyxia neonatorum or even in healthy newborn infants to warrant a pessimistic attitude toward use of 5% carbon dioxide added to air or oxygen in treating newborn infants with respiratory insufficiency.

Hyaline Membranes and Atelectasis in Lungs of Newborn Infants. Fritz Reutter[5] (Univ. of Zurich) found hyaline membranes in 62 lungs during a study of microscopic lung specimens of 378 stillborn babies and infants up to age 1 year. Autopsy protocols and case histories were reviewed.

Hyaline membranes develop in newborn infants who have breathed for some time. In present series, the minimal life span necessary for development of these membranes was 1½ hours. The appearance of some of the specimens indicated that the membranes developed from a thick liquid material in the alveoli and alveolar passages. Respiratory movements pressed the substance against the alveolar wall and formed it into membranes.

(4) Pediatrics 14:104-113, August, 1954.
(5) Gynaecologia 137:367-384, June, 1954.

The characteristics of the liquid material are unknown. Hyaline membranes are found mostly in premature infants. In this series, 44% of premature infants, who died within a few days after birth, had membranes.

Patients with hyaline membranes can be divided into two groups. The first group consists of premature infants weighing 1,000-2,500 Gm. and, rarely, full term newborn infants. The infants do not live longer than four days and clinically present typical symptoms of asphyxia. In the second group are older infants, children and adults in whom the membranes accompany other lung changes, mostly exudative-hemorrhagic, without uniform symptomatology.

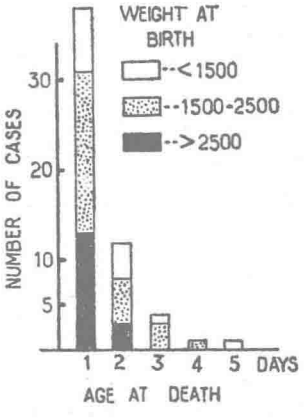
Reutter considers anoxia the etiologic factor in development of hyaline membranes. On the one hand, it leads to increased capillary permeability with exudation into the alveoli and on the other to aspiration of meconium-containing amniotic fluid, which by itself increase capillary exudation from alveolar septa. The amniotic fluid and the protein-containing exudate form the substance for the membranes. The hyaline membranes themselves may cause secondary atelectasis.

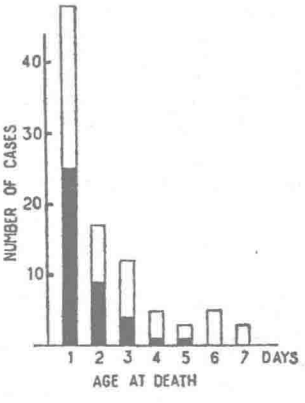
In newborn infants, hyaline membranes produce a vicious circle. Asphyxia causes increased exudation and forced respiration, which lead to hyaline membrane formation. The membranes increase the asphyxia either by decreasing directly the number of functioning alveoli or by causing secondary atelectasis or by increasing exudation into the alveoli. Ultimately, the active respiratory surface becomes insufficient and death by asphyxia ensues. In present series, this vicious circle was the cause of death in 44 infants.

Diagnosis is not difficult clinically, especially in an immature baby of 1,000-2,500 Gm. who presents no clinical signs other than those of increasing asphyxia. Usually the auscultatory findings are not typical. On fluoroscopy, the lungs generally appear less translucent. Congenital heart disease, pneumonia or cerebral hemorrhage must be considered in differential diagnosis.

Pulmonary Hyaline Membranes in Newborn are seen more often in premature than in full term infants and only in infants who have breathed. During the first few hours of life there may be no pathologic physical findings, but later in the day cyanosis appears. At the same time, respirations are heavy and breath sounds diminish in intensity.

Kr. Harnaes and K. H. Torp[6] (Univ. of Oslo) report statistics on 56 cases of hyaline membranes, detected at 216 consecutive autopsies on infants who died during the first week of life. Of the 56 infants, 70% died within the first 24 hours and 70% were premature (Fig. 2). Roughly half of all premature





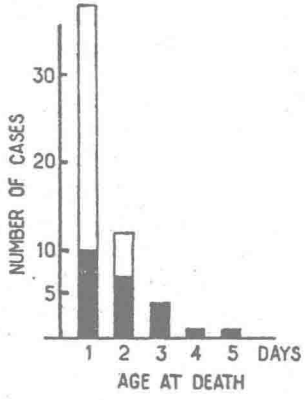


Fig. 2 (above left).—Distribution of 56 cases of pulmonary hyaline membranes in relation to birth weight (in grams) and age at death.

Fig. 3 (above).—Incidence of hyaline membranes (black column) among all premature infants dying within first week of life.

Fig. 4 (left).—Incidence of other major complications (black column) in infants with hyaline membranes.

(Courtesy of Harnaes, Kr., and Torp, K. H.: Arch. Dis. Childhood 29:199-200, June, 1954.)

infants who died within the first three days had hyaline membranes in the lungs (Fig. 3). Other pathologic conditions considered capable of explaining death were found in 23 of the 56 infants (Fig. 4). These included pneumonia, malformations, erythroblastosis or intracranial hemorrhage, all of severe grade, and were found in 10 of 38 infants (26%) who died within the

(6) Arch. Dis. Childhood 29:199-200, June, 1954.

first 24 hours of life and in 13 of 18 (72%) who died during the second to fifth day.

Potter states that the respiratory difficulty seen in this condition often causes death within 24 hours and that she never found an uncomplicated hyaline membrane in infants surviving more than 48 hours. The authors' findings agree with this. They believe that in suspected cases of hyaline membranes therapeutic measures should be instituted even in the first hours of life, to minimize the risk of death.

[We agree that the majority of prematures who apparently die of uncomplicated pulmonary hyaline membranes succumb during the first 24 hours of life; however, the second and third days of life appear to be the critical ones for term infants with this condition.—Ed.]

Pulmonary Hyaline Membranes in Infants of Diabetic Mothers. W. D. Winter, Jr., and Sydney S. Gellis[7] (Harvard Med. School) believe the frequent signs of respiratory distress in infants of diabetic mothers, including rapid labored respirations, cyanosis and decreased breath sounds are due to pulmonary hyaline membranes. They reviewed autopsy findings in 40 infants born of diabetic mothers and dying in the neonatal period, to determine the presence of a major cause of death, presence of pulmonary hyaline membranes and the relation of the latter to cesarean section. Only 13 infants had a demonstrable major cause of death. Hyaline membranes were present in 23% of these infants and in 78% of the remaining 27. They were present in 68% of infants delivered by cesarean section, but in only 53% of pelvic deliveries. Among the infants dying in 1-72 hours, 63% showed hyaline membranes, compared with 37% in the general series of Bylstad *et al.* and 39% in the series of Miller and Jennison. This indicates a definitely higher incidence of membranes among infants of diabetic mothers.

The significance of hyaline membranes as a cause of death must be determined. The authors' material usually showed atelectasis of at least three fourths of the alveoli, with hyaline material lining most of the aerated spaces. However, the theory of obstruction by hyaline membranes can only be tested critically by correlation of the clinical picture with multiple sections, from both dependent and superior portions of the lungs of a large series of early neonatal deaths.

The higher incidence of hyaline membranes following cesarean section is comparable to findings of other workers and can be related to the increased amounts of aspirated amniotic fluid.

(7) A.M.A. Am. J. Dis. Child. 88:702-707, June, 1954.

However, elective cesarean section appears justified in diabetic mothers, because of high fetal mortality. No correlation was found between the presence of cardiomegaly, congestive heart failure and pulmonary hyaline membranes in deaths of infants of diabetic mothers.

Clinical Pathologic Study of Newborn Lung with Hyaline-Like Membranes in 407 infants weighing over 1,000 Gm. is reported by Ernest F. Latham, Robert E. L. Nesbitt, Jr., and George W. Anderson.[8]

Hyaline-like membranes were present in 124 infants (30.5%), an incidence per 1,000 live births of 3.7 in the 1,000-2,499 Gm. group and 0.99 in the 2,500 Gm. and over group. This lesion was not found in the lungs of 563 stillborn infants. When estimated percentage of associated atelectasis was considered the total incidence of the lesion per 1,000 live births (over 1,000 Gm.) of 4.7 fell to 2.4 when it was combined with at least 70% atelectasis. Incidence of multiple factors was higher in infants with hyaline-like membranes than it is generally. These factors included prematurity (6.9 times higher), multiple pregnancy and cesarean section (27% of total cases). Also, incidence of these membranes was higher in the lungs of infants of diabetic mothers, especially those delivered by cesarean section. Previously, 900 cases of neonatal death associated with pulmonary hyaline-like membranes were reported.

Infants with hyaline-like membranes lived an average of 30 hours, with a definite inverse proportion between length of life and extent of membrane. Those surviving longest had a higher incidence of associated pneumonitis; the latter was found oftenest when there were maternal complications of intrapartum fever and membrane rupture for three to four days before delivery.

Cases in which hyaline-like membranes were considered the sole cause of death were associated with 70% or more pulmonary atelectasis. They represented 30% of the total group and only 8.6% of the total neonatal deaths. However, the authors believe that infants with hyaline-like membranes, in conjunction with other pulmonary disease, make up a large group with "abnormal pulmonary ventilation," which ranks high as a causative factor in neonatal deaths, especially in prematures. Also, the important role of atelectasis must be considered in expressing the total cases of "abnormal pulmonary ventilation."

(8) Bull. Johns Hopkins Hosp. 96:173-198, May, 1955.