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Technic for Perfusion of Previable Human Fetus with oxygenated blood is described by Björn Westin, Rune Nyberg and Göran Enhörning[1] (Karolinska Inst., Stockholm). The apparatus (Fig. 1) consists of a perfusion chamber and an oxygenator. The perfusion chamber encloses the fetus and insures a constant temperature and blood volume. The oxygenator oxygenates the blood and elevates it to a hydrostatic level suitable for perfusion of the fetus.

TECHNIC.—After catheters have been introduced into the umbilical vessels, the fetus is placed in the chamber, which then is filled with isotonic glucose and closed with the cover. Because all air is evacuated and the system closed, the volume of the fetus cannot change. Any volume of oxygenated blood injected into the umbilical vein will appear quantitatively in the outflow. The ECG, blood pressure and blood flow are recorded simultaneously on the same film.

The period of survival of 7 previable fetuses. as shown by recordings of ECG, blood pressure, blood flow and determinations of oxygen consumption of the fetus, varied from 5 to 12 hours. During perfusion the fetuses moved the head, body and limbs. Swallowing and respiratory movements were not observed. In fetuses weighing 200-375 Gm. and recorded for several hours at 37 C., the heart rate was 120-180/minute, the P-Q time was about 0.1 second and arterial pressure 25-50 mm. Hg. In a fetus weighing 275 Gm. and perfused at 37 C., the heart rate 6 hours after birth was 120, P-Q time 0.1 second, blood flow via both umbilical arteries 14.2 ml./minute and arterial pressure 30 mm Hg. The oxygen tension in the umbilical vein and one artery was 95 and 44 mm. Hg. The experimentally obtained oxygen consumption was 7.6 ml. O_2/kg./minute.

This perfusion technic offers new possibilities for extending knowledge on the physiology of the human fetus. The technic may possibly be used in treatment of premature and asphyxiated newborns.

► [These are basic studies and technics which undoubtedly hold great

(1) Acta paediat. 47:339-349, July, 1958.

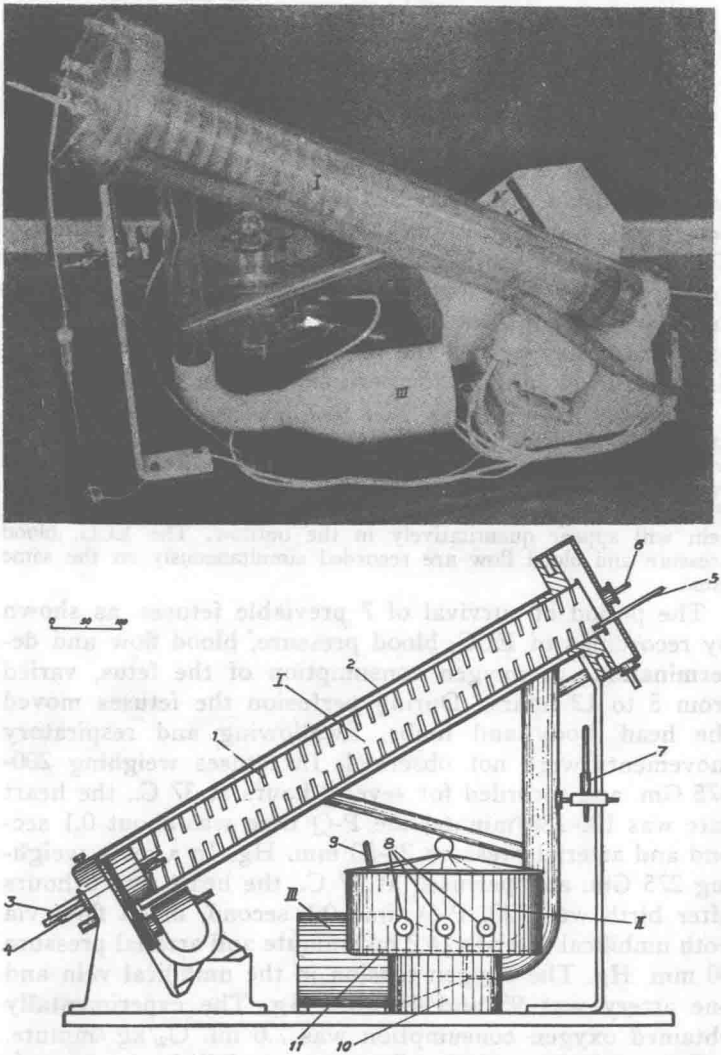

Fig. 1.—Perfusion apparatus. *I*, oxygenator; *1*, spiral; *2*, encasing cylinder; *3* and *4*, inlets for artery catheters; *5*, thermometer; *6*, oxygen inflow; *7*, oxygen flow meter. *II*, perfusion chamber; *8*, inlets with rubber stoppers; *9*, thermometer; *10*, thermoregulated heating equipment; *11*, perforated disk. *III*, thermocontrolled heating equipment for oxygenator. This apparatus is manufactured by KIFA AB of Stockholm and is distributed in the United States by Schick X-Ray Co., Inc., Chicago. (Courtesy of Westin, B., *et al.*: Acta paediat. 47:339-349, July, 1958.)

promise for the future. By perfusion of the previable human fetus and maintaining life, answers should come for many of the problems which hitherto have been studied only in the fetus of animal species, leaving the investigator to wonder if his findings possibly can be applied to the human.—Ed.]

Evaluation of Newborn Infant. The method proposed by Apgar was tested in 1,947 infants by E. K. Ahvenainen and Tapio Veistola[2] (Jyväskylä, Finland). Assessment was made 60 seconds after birth, in most cases by the midwife in charge of delivery. Heart rate, respiratory effort, reflex irritability, muscle tone and color were each given a rating of 0, 1 or 2, depending on whether the sign was absent, weak or normal. A score of 10 indicated that the infant was in the best possible condition.

Of 57 infants with a neonatal score of 1-6, 18 (32%) died. Among 144 infants with a score of 7-8, mortality was 4.8%, and among 1,746 with a score of 9-10, mortality was 0.62%. Thus, mortality and neonatal score were closely correlated. Where midwives are in charge of deliveries, the method is considered useful in providing the pediatrician with information concerning the infant's condition at birth.

No correlation was found between presence of discolored amniotic fluid and the infant's condition at birth. The average score of infants with fetal distress, as estimated on the basis of fetal heart sounds, did not differ significantly from the score of other infants. Mortality was higher, however, in infants with this sign of fetal distress (5.2%) than in those without this sign (1.6%).

Evaluation of Newborn Infant—Second Report is presented by Virginia Apgar, Duncan A. Holaday, L. Stanley James, Irvin M. Weisbrot and Cornelia Berrien[3] (New York). The condition of each newborn was expressed by a score, the sum of 5 numbers obtained within 60 seconds after complete birth. The numbers were obtained by observations of heart rate, promptness and vigor of first respiratory efforts, reflex response to certain stimuli, muscle tone and color. The highest possible score was 10, representing optimum condition of the infant.

The predictive value of such scores was established by a study of scores of 15,348 infants. The death rate among infants scoring 0, 1 or 2 was 15%, whereas that for infants

(2) Ann. paediat. Fenniae 5:27-32, 1959.
(3) J.A.M.A. 168:1985-1988, Dec. 13, 1958.

scoring 10 was 0.13%. Over 20% of infants delivered by breech presentation had scores of 0, 1 or 2, whereas 12% of those delivered by cesarean section and 3% of those delivered by vaginal vertex presentation had such scores. The death rate was 1.4% for vaginal vertex, 3.8% for cesarean section and 13.3% for breech delivery. Incidence and death rates of the various scores among 1,642 premature infants followed a pattern similar to that of the entire group of 15,348 infants.

The scoring system is a useful guide to need for resuscitation. Since death rates dropped sharply after a score of 4 in the preliminary study, a score of 4 or less has been used as indication of need for resuscitative measures. Biochemical studies have corroborated this clinical impression, as chemical findings characteristic of asphyxia have generally been found in blood obtained by umbilical catheterization in infants with low scores.

The scoring method is simple to teach to any personnel regularly in the delivery room. Whoever has the infant directly in his care at 1 minute after birth can best assign the score. A brisk tangential slap of the soles of the feet is the simplest and most effective method of testing reflex irritability. A cry is given a value of 2, a grimace or movement a value of 1 and no reaction a value of 0.

Serial scoring is of value. Apparently, the longer a low score stays low, the worse the prognosis for survival. If a score of 0, 1 or 2 does not improve by 15 minutes, prognosis is extremely grave. Only occasionally does a good score, such as 8, 9 or 10, drop after 1 minute. The usual cause is reflex laryngospasm from the catheter tip touching the vocal cords during aspiration of the pharynx. Other causes are respiratory obstruction or hypoventilation from drug depression. These complications are completely preventable.

►[In our experience, it is much easier to accurately score a baby at 5 minutes than at 1 minute after birth. In addition, the babies who are in apparent poor condition at 1 minute and are well at 5 minutes get a truer score; similarly, the infants who seem well at 1 minute and soon after are in difficulty are given a score more in keeping with the situation.—Ed.]

Fluid Shift from Vascular Compartment Immediately after Birth. Douglas Gairdner, John Marks, Janet D. Roscoe and R. O. Brettell[4] (Cambridge Maternity Hosp.) found the mean hemoglobin level of the cord blood of 92 infants

(4) Arch. Dis. Childhood 33:489-498, December, 1958.

to be 16.6 Gm./100 ml. and that of samples taken 1-8 hours after birth, 19.1 Gm./100 ml., giving a postnatal rise of 16%. In both vaginal (73) and cesarean (19) deliveries, the hemoglobin level often rose significantly a few minutes after birth, reaching a maximum in 3 hours or less. In addition, there was a rise in plasma protein concentration of up to 20% and of mean corpuscular hemoglobin concentration averaging 4% but no change in plasma sodium concentration.

The cord of 17 infants delivered by elective cesarean section before onset of labor was clamped as soon as possible, so that no appreciable amount of placental blood could have been transferred to the infant. Nevertheless, the postnatal hemoglobin rise was comparable to that observed in infants delivered vaginally. These findings indicate that the postnatal rise occurs independently of placental blood transfer.

By exclusion of other possible mechanisms, the authors conclude that the rise must be mainly due to hemoconcentration from a shift of fluid from the vascular compartment. The fluid was largely whole plasma, and its volume may amount to a large fraction (25% or more) of the circulating plasma at birth. Although the exact mechanism of the postnatal shift of plasma is not known, it is suggested that the loss of protein-rich fluid takes place from capillaries subjected to an increased pressure after circulatory rearrangements at birth. The pulmonary circulation is one site where such alterations would occur.

The results of this study imply that the fetus in utero is hydremic, i.e., has a large plasma volume. Hemodilution is thus partly responsible for the remarkably wide range of the normal cord hemoglobin level.

The authors suggest that in the premature infant the normal mechanism by which the lymphatic system recovers protein leaving the circulation is overwhelmed and that pulmonary edema results, with some of the protein-rich fluid exuding into the alveolar space. This edema may be the primary disturbance in the pulmonary failure which so often occurs in the premature infant shortly after birth.

► [Dr. Clement Smith commented:

"This paper is particularly interesting for the quantity and quality of

reasoning its authors have applied to a moderate amount of data obtained by relatively simple procedures from normal infants. The data are in keeping with those of other workers. There is no apparent reason to doubt them. The deduction as to an early postnatal shift of plasma fluid and protein from the vascular compartment seems unassailable. We share the authors' satisfaction over their discovery of a clue to the puzzle of wide variation in hemoglobin and erythrocyte content of cord blood—a condition more easily understood on the basis of varying hydremia than of a varying hemopoiesis. The other deduction, that the volume of the red cells normally contracts by about 3% postnatally, rests on a little less firm footing, but this explanation seems at least as well supported as the earlier theory of change caused by release of smaller cells into the circulation.

"The further proposal that fluid shifts from cells and plasma may explain accumulation of visible edema and onset of respiratory distress with hyaline membranes will require more evidence. Both these common clinical manifestations—often appearing together—need investigation by any new tool, and a fresh insight, though unproved, can be a very good tool, indeed.

"The plethysmographic demonstration of accumulation and release of edema fluid in the legs of premature infants with respiratory distress, presented by Dr. James Sutherland at a recent Ross Laboratories Conference, is in keeping with this approach. There is less evidence of pulmonary edema (as usually conceived) from roentgenologic and pathologic studies." —Ed.]

Effect of Position on Respiratory Rate of Premature and Mature Newborn Infants. Harvey Kravitz, Lawrence Elegant, Bernard Block, Mary Babakitis and Evelyn Lundeen[5] (Chicago) studied the respiratory rates of 119 premature and 49 full-term infants in the supine and prone positions. Prematures had a significant increase in respiratory rate in the prone position compared to the supine. This difference decreased with increasing weight and age. Mature infants showed a slight increase in respiratory rate in the prone position compared to the supine. The reason for the differences in the respiratory rates of the premature infants is not clear. Further studies of the relation of position to tidal volume, amplitude of respiration and respiratory rate are needed. An important consideration is that in the supine position the intercostal and abdominal muscles can more freely assist the diaphragm with each respiratory excursion, whereas in the prone position the abdominal and intercostal muscles are compressed against the surface of the incubator or bed and cannot assist the diaphragm in the respiratory activity. The weaker the abdominal muscles and the weaker and softer the anterior thorax, the less are the resulting respiratory excursions.

Respirations of regular rate and amplitude were charac-

(5) Pediatrics 22:432-435, September, 1958.

teristic of the premature infant in the prone position, whereas irregularity of rate and amplitude was noted in the supine position. The amplitude of respiration was greater in the supine position than in the prone. The reason for the greater irregularity in rate and amplitude in the supine position is unknown. No difference was noted in the respiratory rates of premature infants lying supine at 30-degree elevation as compared with respiratory rates in the horizontal supine position.

► [This article points up the relative lack of information we have in the simpler aspects of medicine. We continue for years to use many procedures without definite reasons for the establishment of such procedures. It has not been clear to us why the supine position was used for newborns; this study indicates that it is superior to the prone position, at least from the viewpoint of respiratory rate, which appears as physiologic a method as any for assaying the merit of one position as compared with others.

Dr. William Silverman commented:

"Kravitz *et al.* observed that the respiratory rates of premature infants whose birth weight was more than 1,750 Gm. were not influenced (significantly) by position (prone vs. supine). These observations are in agreement with those made by Cortner, Dower and me in infants whose birth weight was more than 1,900 Gm. Since we have not studied the influence of position on the respiratory rates of smaller infants, I cannot make any comments based on data of our own. It is unlikely that Cortner will publish our observations since they merely confirm the Chicago group's findings—the absence of an effect in large infants.

"The lower incidence of periodic breathing in the group that displayed the more rapid respiratory rate (the prone group) is interesting. Kenneth Cross once made a comment that is appropriate to this observation: 'It is my feeling that what cuts down periodic breathing is anything that increases the minute volume of respiration.'

"Now that the Chicago group has demonstrated an influence of position on the respiratory rate of smaller prematures, we await their further observations on the influence of position on mortality among these infants."—Ed.]

Severe Apnea and Irregular Respiratory Rhythms among Premature Infants: Clinical and Laboratory Study of 100 consecutively born infants with birth weights of less than 1,751 Gm. was made by Herbert C. Miller, Franklin C. Behrle and Ned W. Smull[6] (Univ. of Kansas). Pneumographic tracings were obtained from 47 of the infants during the first few days of life. All infants were classified according to the trend of respiratory rates as described in a previous publication. Infants in group III had significant increases in respiratory rates after birth and infants in groups I and II did not.

Severe apnea was observed in only 1 of 32 infants with

(6) Pediatrics 23:676-685, April, 1959.

no or only mild respiratory insufficiency (groups I and II), but occurred in 29 of 68 with severe respiratory insufficiency (group III). Birth weight did not appear to be a factor in the occurrence of severe apnea unless severe respiratory insufficiency was present.

Prognosis for survival was significantly poorer among infants with severe apnea. Death occurred in 10 of 70 infants who did not have severe apnea and in 18 of 30 who did. Severe apnea was exceptional among surviving infants during the first 3 days of life but occurred often in that period in infants who died. Between the 4th and 7th day severe apnea occurred frequently in both surviving and dying infants but was uncommon thereafter.

Severe apnea seemed to be an extreme form of irregular breathing in premature infants. There was a direct relation between degree of irregularity of respiratory rhythms and occurrence of long single periods of apnea.

The total amount of apnea was slightly less when infants breathed 40% oxygen than when they breathed room air, but there was a marked increase in the number of long single periods of apnea during oxygen administration. The color of the infants was better during long periods of apnea in 40% oxygen than during shorter periods in room air.

These findings strengthen a growing impression that the respiratory centers of infants in group III tend to be more feeble than those of infants in groups I and II.

Respiratory Activity and Function in Newborn Infants Dying with Pulmonary Hyaline Membranes was compared by Herbert C. Miller, Franklin C. Behrle and Ned W. Smull[7] (Univ. of Kansas) with the respiratory activity of surviving infants who had severe respiratory insufficiency. Both groups compensated by increasing respiratory rates during the period of respiratory insufficiency, but the respiratory rates of surviving infants rose to higher levels than did those of the 10 dying with hyaline membranes. Dyspnea usually decreased rapidly and often disappeared entirely in the surviving infants during the periods of increased respiration, whereas dyspnea persisted from birth onward in the other group. Thus, prognosis appears more favorable if the respiratory rate increases and dyspnea lessens or disappears, than if the rate rises and dyspnea

(7) Pediatrics 22:665-674, October, 1958.

does not decrease or shows an increase. Very high rates of 100/minute or more accompanied by a synchronous pattern of breathing are not to be feared as much as lesser rates of 60-80/minute with marked dyspnea.

The infants with hyaline membranes received 40% oxygen, but this concentration was not sufficient to keep them constantly free from cyanosis. The authors do not consider that higher concentrations were more effective, but they have not hesitated to raise the oxygen concentration for larger infants.

Hyaline membranes have not been observed in infants dying within 1 hour of birth. This suggests some delay in formation after birth, but there is little evidence that the delay is accompanied by a symptom-free interval. Of the 10 infants, 8 had marked dyspnea from birth and 9 had either apnea lasting 2 minutes or more at birth or bradypnea and cyanosis during the 1st hour. Previous reports which have emphasized the so-called symptom-free period immediately after birth have not included detailed observations, nor have they critically defined normal respiration during this period.

Studies of respiratory activity and function have not enabled a positive clinical distinction to be made between infants with hyaline membranes and those with distress produced by other factors. From a clinical point of view, inability to diagnose pulmonary hyaline membranes during life has not been a serious handicap. Until more specific and proved therapeutic procedures are available, accurate evaluation of the degree of respiratory insufficiency is more important.

► [The authors' statement that infants with hyaline membranes do not have a "quiet" interval after birth is far from new (see report of the Fifth M & R Pediatric Research Conference on Pulmonary Hyaline Membranes, 1953). The quiet interval was described by pathologists who drew their clinical observations from data recorded by physicians who did not observe infants immediately after delivery.

We agree that respiratory studies do not of themselves permit a diagnosis of hyaline membranes. To be certain of the diagnosis, one must have a chest film which shows the diffuse finely granular "infiltrate" which has at this point been repeatedly described in this condition. To diagnose hyaline membranes without confirmatory x-ray is to invite disaster; e.g., pneumonia may exactly simulate the clinical picture of hyaline membranes.

We take issue with the authors' statement that "From a clinical point of view, inability to diagnose pulmonary hyaline membranes during life has not been a serious handicap." This statement is made largely, we as-

sume, because there is no specific therapy available. We maintain that this diagnosis must be made with a reasonable degree of certainty on the basis of the clinical findings plus the x-ray; lacking a definitive diagnosis, and with the possibility of pulmonary infection always in mind, treatment should then be instituted.—Ed.]

Obstructive Factors in Pulmonary Hyaline Membrane Syndrome in Asphyxia of the Newborn. By injection of gelatin-India ink and latex masses under controlled conditions into the lungs of infants with asphyxia associated with hyaline membrane formation, John M. Craig, Kevin Fenton and David Gitlin[8] (Boston) demonstrated that such membranes prevent filling of the distal alveoli. The membranes line all the expanded air-containing spaces beyond the epithelium-lined bronchi and apparently interfere with diffusion of oxygen and carbon dioxide within these alveoli, besides blocking effective passage of air into the more distal alveoli.

Infants with the hyaline membrane syndrome have increased respiratory rate and normal tidal volume but remain cyanotic. Cook explains this by the fact that only a small portion of the respired volume reaches the alveoli where gaseous exchange occurs. This hypothesis is consistent with the authors' interpretation that the membranes block passage of air to the more peripheral spaces.

Plasmin introduced into the lung caused dissolution of hyaline membranes in vitro. Plasmin is a fibrinolysin obtained by activation of plasminogen by streptokinase. It has been shown to be effective in vitro and in vivo in lysing fibrin clots. The authors have demonstrated previously that the mass of material in hyaline membranes is fibrin. Dissolution of such membranes by plasmin is consistent with these observations.

Introduction of plasmin in high concentration into the trachea of an infant with hyaline membrane syndrome may prove to be an effective treatment. If introduced in a volume as small as 3 ml., it should not cause much distress, since volumes of fluid many times this size may be introduced into the trachea with only minor respiratory embarrassment.

Chest Radiography of Prematures: Planned Study of 104 Patients Including Clinicopathologic Correlation of Respiratory Distress Syndrome is presented by William Allen

(8) Pediatrics 22:847-856, November, 1958.

Bauman and Jerome Nadelhaft[9] (Babies Hosp., New York). The chests of 104 unselected newborn prematures were studied by x-ray in the first 72 hours of life in a routine manner which yielded satisfactory x-rays and involved minimal disturbance of the patients. The technic involved use of a modification of Silver's plastic platform within the incubator, with accurate positioning of the infant with aid of arm, leg and head restraints.

Clinical signs of dyspnea sufficient to suggest some degree of the respiratory distress syndrome were present in 19 infants. X-rays compatible with hyaline membrane syndrome were obtained in 35. Of 25 who died during the first 8 days of life, 23 came to autopsy; 10 of these had hyaline membranes, and 8 of the 10 while alive had shown respiratory distress and pulmonary roentgenographic appearance of bilateral diffuse increased reticulogranularity. In statistical analyses, this association is highly significant ($P<0.006$).

That several infants with both of these abnormalities survived indicates that prematures may recover from the respiratory distress syndrome. The hypothesis that prematures with pulmonary hyaline membranes may survive is supported by findings in 2 of the present cases and others studied by the authors, in which microscopic appearance compatible with resorption of the membrane was noted. Until diagnosis can be established with greater certainty during life, the possibility of spontaneous recovery must be considered in the evaluation of therapeutic measures. In the individual case, a premature with a high retraction score, according to the standards of Silverman and Andersen, and pulmonary x-rays demonstrating increased reticulogranularity has a greater chance of dying because of hyaline membrane formation than does the infant with absence of these symptoms and signs during life.

► [In many newborn nurseries at present physicians are finding close correlation between the clinical diagnosis of pulmonary hyaline membranes—supported, of course, by x-rays showing increased reticulogranularity—and subsequent autopsy findings. Thus, the diagnosis has gradually reached an acceptable status despite the efforts of those who have objected to the use of the term outside an autopsy protocol. It is often tempting to avoid the use of x-ray and make the diagnosis on the basis of clinical findings and course. One is always reluctant to subject premature infants to procedures which are not absolutely necessary. In this instance, however, x-ray should be considered unavoidable.—Ed.]

(9) Pediatrics 21:813-824, May, 1958.

Possibility of Clinical Diagnosis of Hyaline Membranes in the Newborn: Preliminary Report on results of cultures of throat swabs is presented by K. Österlund and L. Hjelt[1] (Univ. of Helsinki).

METHOD.—Material for culture was obtained by throat swab from the pharynx of infants suspected of having pulmonary hyaline membranes. A smear was made on a glass slide, fixed in a solution of

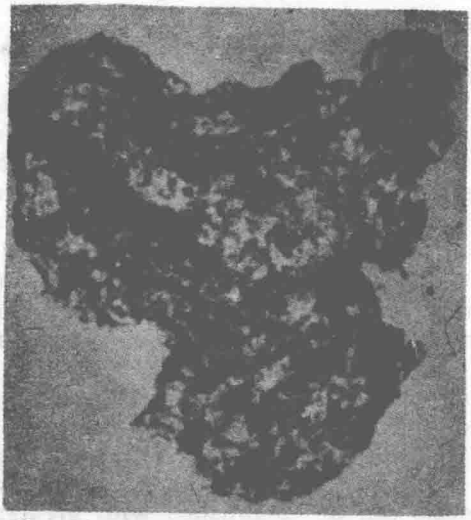

Fig. 2.—Lepehne-Pickworth "positive" membrane-like structure found in throat swab of infant with neonatal asphyxia. (Courtesy of Österlund. K., and Hjelt, L.: Ann. paediat. Fenniae 5:33-38, 1959.)

equal parts of ethyl ether and 96% ethyl alcohol and stained by the Lepehne-Pickworth technic, which produces an intense blue tint in hyaline membranes.

Studies were done on 2 newborn infants with no respiratory difficulties, on 3 with clinical findings typical of pulmonary hyaline membrane disease, on 2 with a diagnosis of neonatal asphyxia and on 1 premature infant with a rapid respiratory rate during the first few hours of life only. Lepehne-Pickworth "positive" membrane-like structures were observed in 2 of the infants with clinical evidence of hyaline membrane disease and in 1 with a clinical diagnosis of neonatal asphyxia (Fig. 2). None of the infants with findings not clinically compatible with a diagnosis of hyaline

(1) Ann. paediat. Fenniae 5:33-38, 1959.