Round Table Discussion

THE PREMATURE INFANT

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Chairman Sanford: Dr. Herman N. Bundesen, Commissioner of Health of Chicago, organized 12 years ago the "Chicago Premature Plan." This consists in registering all premature infants with the City Health Department within a few hours after birth. The premature infant who is born at home, or in a hospital that does not have adequate premature care, is transported in an oxygenated incubator ambulance to a hospital which specializes in such care.

From 1936 to 1947 premature infant deaths in Chicago have been lowered 6½%. The full term infant death rate during the same period has been lowered about 3%. Inasmuch as the premature death rate has been lowered about double that of the full term infant rate, we believe this procedure has been the cause of reduction. In 1936 there were 47,000 live births in Chicago. In 1947 there were 82,000, or an increase of 80%. In this number the full term infants increased from 45% to 60%, whereas the premature infants increased from 2000 to over 5000, or about 140% increase of premature infants born in Chicago during the last 10 years.

This adds a considerable increase to the number of infants for our available premature infants beds. Where formerly we planned 5 premature births to each 100 full term births, we now find that prematures have increased to 8 per 100 full term infants.

Causes of prematurity are multiple births, toxemia, heart disease, syphilis, tuberculosis, infections, accidents, premature separation of the placenta and abnormalities of the reproduction tract. It is generally understood that there is a tendency for more premature births among the Negro race than the white race. Our Negro population in Chicago has increased in the last 10 years but our increase in premature infants has not been racial. I have no explanation. Premature births certainly are increasing in this area.

I have been impressed with another phase and that is many premature infants that we see are from a cesarian birth. Obstetrics has produced more live births but at a premature level. We also find that abnormal pregnancies account for a much higher premature mortality less noticed in the lower weight group.

Any good feeding in the hands of a competent pediatrician is entirely satisfactory. In Presbyterian Hospital, Chicago, we use breast milk entirely as a food for our premature infants. This is obtained from our maternity mothers. We encourage breast feeding and most of our mothers have an excess which we pump and bank. The breast milk is frozen in 4 oz. bottles and stored in a deep freeze. This can be used as long as a year later with safety. The City Health Department operates a very efficient breast milk station and will furnish breast milk gratis to any premature infant born in Chicago. If we happen to have an excess, we give it to the City Health Department and they give us credit for it, and then if we run short, we borrow from them. In this way every premature infant is sure of obtaining breast milk. It is not necessary to use pure breast milk for all premature infants. You can use any good type of artificial food and dilute the breast milk with it up to about equal parts, and obtain almost as good results as with pure breast milk, except in very small infants or those who are sick. When we have such a baby, it is taken off our standard formula of equal parts breast milk and lactic acid and given pure breast milk. This formula is a great saving of pure breast milk.

How often should a premature infant be fed and how soon should they be fed? I think the usual tendency is to wait at least 24 to 48 hours, or even longer. They should be fed in the way that is easiest for the baby and easiest for the personnel. We gavage them at 4 hour intervals until the weight is about 4 pounds, then the nurse begins bottle feedings. In some institutions the premature infants

From the Annual Meeting of the American Academy of Pediatrics, Chicago, Oct. 16, 1950.

are discharged at 4½ pounds. We like to keep them until at least 5½ pounds because we find that we gain bed space in the long run since fewer return with gastrointestinal upsets.

Dr. Charles E. Snelling, Toronto: I would be interested to know how many men in this group are using breast feeding for prematures?

Chairman Sanford: There are about 6 in this group of 33.

Dr. Hearn Bradley, Nashville: I would like to know how many men are using skimmed boiled milk with 10% added sugar.

Chairman Sanford: One or 2 of this group seem to be using this formula.

Dr. Harold J. Freedman, Boston: I worked in Chicago with Dr. Hess and was quite impressed with the method of using breast feeding, but when I went to Boston they weren't very much concerned with this system, perhaps because they did not have the set-up. We used 2% skimmed milk with highly concentrated carbohydrates. We generally fed them after 48 hours, starting with very small amounts and increasing very slowly. I wonder if you do the same thing?

Chairman Sanford: We allow the nurse to decide the increase as she can observe the baby's capacity better than anyone else.

As to accessory fluid, a great many will give dextrose or saline between feedings. In studies we made over a 10 year period, we found that 5% carbohydrate suppressed the amount of feeding that these children would take by suppressing hunger. We give only water by dropper between feedings, as much as the baby will take, and do not have to use intravenous or subcutaneous injection.

Lund and Kimbel in 1943 found that the premature infant received very little vitamin A from the mother and very little reserve was stored in the liver. The premature infant might, therefore, be deficient in vitamin A. Warkany found that rats deficient in vitamin A produced offsprings with eye defects. Clifford found that there was much better absorption of water soluble A to reach an optimum blood level of 29 units. We should, therefore, give about 20,000 units of vitamin A in a water soluble form.

Vitamin B, I believe, is unnecessary. Vitamin C is necessary for oxidation of amino acids, as well as against scurvy. There has been a tendency to increase vitamin C intake over the years and we find 150 to 200 mg. frequently given. Actually 50 mg. is usually sufficient and 100 mg. maximum. It makes no difference how it is given. It can be orange juice or combined as a multivitamin. Vitamin D is usually given in 1000 unit amounts.

It has been thought for many years that the premature infant had a lower prothrombin content of the blood than the full term infant. Actually this idea is based on a few studies of a small number of premature infants. We thought we would correlate the prothrombin estimations that we had made on the full term infants with similar studies on 100 premature infants. These studies showed that there is a decrease in prothrombin content of the blood in the premature baby during the first 3 days of life which corresponds to the decrease observed in the full term baby except that it is not as great and it rises faster. Like the full term baby, the prothrombin content of the blood can be increased by the administration of vitamin K.

In this study there were 83 premature infants who were given no vitamin K at all and 17 who were given vitamin K. While the administration of vitamin K increased the prothrombin content of the blood, it had no effect on the hemorrhagic manifestations. Those premature infants not given vitamin K had 6% of hemorrhagic manifestations, and those given vitamin K had 24%. However, this increase was due to the fact that this group was brought in from outside the hospital and had considerably more manipulation. These figures simply demonstrate that capillary fragility is an important factor in the premature infant and that the administration of vitamin K does not materially affect the hemorrhage after it has already begun. It was also found that the percentage of hemorrhagic manifestations was no greater in the premature infant, 9%, than in the full term infant, 6%.

There were no deaths from hemorrhage in the group not given vitamin K and one death from hemorrhage (cerebral) in the group given vitamin K. I have never thought that cerebral hemorrhage was influenced by the speed of blood coagulation. This study shows that even when vitamin K is given in ample amounts, cerebral hemorrhage occurs just the same.

Dr. J. H. Root, Jr., Waterbury, Conn.: Do you have any statistics on mothers who were given vitamin K?

Chairman Sanford: None of the mothers of the premature infants born in the hospital were given vitamin K. Almost all the mothers of those born at home received vitamin K before delivery.

Dr. Snelling: What about the prenatal diet of those in the home who later come to the hospital?

Chairman Sanford: It was not as adequate as those delivered in the hospital. The question was asked "Do I give vitamin K in hemorrhagic disease?" I have not given premature infants any vitamin K routinely in 10 years. I do give vitamin K in hemorrhagic disease if the condition is due to hypoprothrombenemia.

As to the administration of minerals, the only one you need consider is iron. We estimated the red cells and hemoglobin over a period of 5 years on 500 premature infants and found exactly the same results as were found by Blackfan several years ago, namely, that the premature infant, irrespective of birth weight, suffers a reduction of hemoglobin until about 10 weeks of age. There was then a static state for several weeks followed by a slow increase. The fall in hemoglobin does not depend upon the weight of the child but on the age of the child in weeks. A large premature infant leaves the hospital before this decrease is observed, whereas a small premature infant will stay in the hospital for weeks and become anemic because you are observing it at the time maximum decrease occurs. There are many clinics that give an iron preparation after the second week of life. We estimate the child's hemoglobin and erythrocytes beginning at the third week of life and if the decrease is below 10 gm. of hemoglobin, we administer iron. If it falls below 9 gm. of hemoglobin, we give a transfusion and then iron. I would say that about 50% will need iron therapy. Someone asked me about vitamin B_{12}. When the soluble preparation became available, we began to use it. All I can say is that thus far we have not observed any hematonic effect.

Dr. J. E. Brown, Columbus: How well do the premature infants assimilate the iron in the first few weeks?

Chairman Sanford: I think they assimilate it well. The preparation we use is U.S.P. iron and ammonium citrate, 50% solution. You can add 1 or 2 drops to each feeding.

Estrogen therapy was recommended by Shelton and Varden in 1946. They gave 2.5 mg. of methyl testosterone in the feedings. They later increased this to 5 mg. Confirmatory articles appeared saying that the resulting gain in weight shortened the hospital stay 50%. We used testosterone for a year with absolutely no effect. A recent study by Seitchek and Agesty on 57 premature infants reported no evidence of superior gain in weight.

Asphyxia is one of the most important things we have to contend with. Formerly there was no means of attacking this except clearing the child's air passages and giving unlimited oxygen. We found that the infant bronchosope, as used by House and Owen, is of great value in atelectasis if confined to 1 or 2 lobes.

If atelectasis is due to cohesion of moist surfaces of the lung sacs and you drain out a main bronchi you can eliminate cohesion in a single lobe. We now routinely take x-rays of all premature infants' chests if there is asphyxia as soon after birth as possible.

Dr. John J. Slavens, Toronto: I am very much interested to find out if you have ever discovered any relationship between asphyxia and the type of anesthesia used on the mother, intensity and the degree of anesthesia used at the time of delivery. Also, I would like to know if you have any cerebral trauma in the asphyxia.

Chairman Sanford: Yes, in the 2 deaths from cerebral hemorrhage quoted, there was asphyxia in both, which I think was more the cause of death than cerebral hemorrhage. The longer and heavier the anesthesia, the more the asphyxia.

Dr. Frank van Schoick, Jackson, Mich.: I have had some interesting experiences that might shed some light on these processes of resuscitation. For the past 18 years I have attempted resuscitation under direct visual control with the Flagg laryngoscope and a rubber-tipped tracheal insufflation tube which is long enough to reach the primary bronchi. There are 3 or 4 things I should like to mention. One, inasmuch as the fetus has respiratory movements in utero, the question is not "why does not the child breathe?" but why does it stop? This may be a little less true of the premature than the full term baby. Two, the larynx of the new born apneic baby is so poorly differentiated and possibly edematous that it looks very much like the external genitalia of the newborn female. When the insufflation tube is introduced into the larynx, occasionally gentle force must be used to get by the vocal cords. This force is not traumatizing as judged by the fact that I have never had a show of blood following the procedure. I question the possibility of passing a soft tracheal catheter by the usual digital methods in such a case. Three, with the insufflation tube in place and following the first good inspiratory effort, thin fluid comes cascading out of the larynx in a volume sufficient to soil the operator's gown. Four, on more than one occasion, with a tracheal tube in place, and after removing any mucus and fluid, CO_2, O_2 in a 5-95 mixture failed to establish respiration. When a

mixture of 30-70 was subsequently used, prompt and vigorous respiratory effort took place. To re-establish the normal respiratory mechanism after it has been depressed from any cause may require more than casual stimulation.

Chairman Sanford: At the present time we give as few fluids as possible except by mouth as plain water. In case of vomiting or diarrhea, fluids are given as conditions demand. After Branning's report in 1942 that the carbon dioxide content of the blood of the premature infant was less than that of the normal infant and that the blood organic acid content of the plasma was 2 or 3 times the amount considered normal for infancy, sodium lactate was given almost routinely to premature infants. Gamble in 1948 questioned whether an attempt to correct this acidosis was rational. A situation that is pathologic for older babies may be physiologic for the premature infant.

Clement Smith found in 1948 that disappearance of edema was accomplished by the loss of Na and K in the urine. Physiologically and clinically, therefore, it would not be indicated to flood the premature organism with fluids and minerals.

In regard to weight gain in premature infants, in a 5 year study of 500 the following was observed: We know that a full term baby can lose between 5 and 10% of its birth weight. The premature infant of 5 pounds will lose 12% of its birth weight and take 2 weeks to regain it. The premature infant that weighs 4½ pounds will lose about 15% of its birth weight and will regain it in 2 weeks. It will take another 2 weeks to gain to 5½ pounds, or about a month before the baby is ready for discharge. A premature that weighs 3½ pounds will lose almost 20% of its birth weight. It will continue to lose for about 2 weeks and will return to birth weight in 4 weeks. It will take 2 months before it will weigh 5½ pounds. With smaller prematures the loss will be 25% of their birth weight, or more than a pound. It takes a good month or more to regain this and they will remain in the hospital for 3 months before they are ready for discharge. The idea of getting them out of the hospital as soon as possible is laudable but it is important to remember that the average premature infant gains slowly and it should remain in the hospital until it can carry on its own existence.

Congenital defects represent 10% of premature mortality. The only one we wish to discuss today is retrolental fibroplasia. This was first described by Terry in 1942 as a congenital persistence of the primary vitreous. Owen later described instances where it developed after birth as an increased dilatation of the vessels. Krause believes that these 2 forms of disease are different. The first type, or congenital, is more often associated with cerebral and somatic defects. The second consists of eye defects alone. The increase in incidence varies in different parts of the country and even in the same city. Krause in Chicago reports 6.8% in 1937 and 30.4% in 1948. Our incidence is 6% and has remained unchanged throughout the years.

All agree that 75% are under 1600 gm. in weight. The increase is not due to saving more small premature infants for the proportion is the same over the past 10 years. Other causes given have been increased bleeding in the mother, fetal anoxia, virus disease and toxic agents. Ingals was unable to find any correlation from the mother's prenatal history. Vitamin deficiency has been thought to be a cause. Vitamin A has not decreased the incidence and vitamin E is not conclusive. Krause believes it may be the emulsifying agent used in water soluble vitamins. Incubator lights, drugs, iron and oxygen have all been considered as a cause. None have been proved.

The only difference in care that might account for the lower incidence in our premature group (6%) and others is that we give breast milk as a feeding. Whether this contains some protective substance we do not know.

Infections are difficult to diagnose in the premature infant as there are no characteristic physical signs. In 25 infants with various infections there was no one definite diagnostic point except jaundice which occurred in half of them. The same was true of laboratory tests. There was no characteristic finding except half the above infants showed anemia and a white cell count of over 12,000. It is best to remember that if the premature infant fails to gain, vomits or has any diarrhea, it may have an infection. Our treatment is to give such an infant pure breast milk and 100,000 units of crysticillin and streptomycin 0.1 gm./kg. until a definite organism is found. Then the antibiotics may be changed to suit the individual organism.

Premature mortality, as reported from various clinics in the country, varies from 12.7% to 22%. The mortality of premature infants born outside a hospital is from 10% to 15% higher than those born inside a hospital. Until uniform standards of prematurity are prevalent, it is useless to make any comparisons of mortality.

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