THE PREMATURE INFANT

PART I

MORRIS GLEICH, M.D., L.P.

Director, Pediatric Department Harlem Hospital; Associate Pediatrician, Lutheran and Jewish Memorial Hospitals; Member, Committee on Prematurity, New York.

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This work is essentially a handbook for doctors, nurses and hospital superintendents. It is intended to supply practical information on the care of premature infants. No attempt is made to deal exhaustively with any one phase of prematurity. For details the references will be found helpful. It is hoped that sufficient interest will be aroused to establish premature infant units in needed districts; to train special nurses for premature infant care; to set aside rooms in obstetrical departments for the premature infant. Thus do we hope to reduce premature infant mortality.

GENERAL CONSIDERATIONS

Many premature infants die within 48 hours after birth because of some congenital maldevelopment or extreme immaturity which is of a sufficiently serious nature to interfere with vital processes concerned in extra-uterine life. At present, very little can be offered to help such infants.

The majority of premature infants who survive the first 48 hours can be saved. For these infants our air-conditioned nurseries, carefully selected formulas, good nursing methods and prophylaxis against infections prove of great value in reducing the shock incident to the new environment for which they are not fully prepared.

Many infants are immature. Since we now stress weight at birth rather than period of uterine gestation as our criterion for prematurity, we may eventually discard the word "premature" and for it substitute "immature".

Infants weighing 5½ pounds (2,500 grams) or less at birth, regardless of their period of uterine gestation, shall be considered immature and treated as such. While, scientifically speaking, all premature infants, especially the heavier ones, are not necessarily immature, the word immaturity will emphasize the need for special care.

In 1900 the infant mortality rate in New York City was 135 per 1,000 live births.[141] In 1938 the newborn death rate was 38.3 per 1,000 live births.[141] This saving of lives occurred prin-

*From the Pediatric Department of Harlem Hospital, New York City.
This is the first of a series of articles by Dr. Gleich on "The Premature Infant" which will appear in consecutive issues of ARCHIVES OF PEDIATRICS.

cipally in the three large disease groups, namely, digestive (mainly diarrhea), the pneumonias and the infectious diseases including tuberculosis. Deaths due to birth injury, congenital malformations, atelectasis, asphyxia and premature births have not declined in the same proportion.

Prematurity is now responsible for 12 deaths per 1,000 live births or about one-third of the total infant mortality.[141] If we are to further reduce infant mortality in New York City our efforts must be concentrated on the problems of prematurity.

Approximately 100,000 babies are born in New York City yearly. About 5,500-6,000 of these infants are classified as premature.

The mortality rate for New York City public hospitals in 1938 was 44.7 per cent. (see Table 1-A); and 33.3 per cent. for 1940 (see Table 1-B); for private hospitals 23-27 per cent. In other words, one-quarter to one-half of all the premature infants die.

The mortality rate at Harlem Hospital before the installation of the premature infant unit was 45.2 per cent. (see Table 3). After the premature infant unit was in operation we noted a mortality rate of 20.5 per cent. (see Table 5).

It has been said that merely thinking about the problem of prematurity results in a reduced morbidity and mortality. This is true. An aroused public consciousness, both lay and medical, can do much to save the lives of these infants. In New York City I am certain we can save at least five-sixths of the premature infants born each year. The excellent work of Dr. Julius Hess can be repeated in New York City.

PREMATURITY AS A PUBLIC HEALTH PROBLEM

The mortality rate for prematurity in our large New York City public hospitals is rather high. Thirteen institutions report an average mortality rate of 44.7 per cent. for 1938 and 33.3 per cent. for 1940 (see Tables 1-A and 1-B). In 1938 five of the institutions lost more than 60 per cent. of the infants. A glance at the per cent. mortality rate (Tables 1-A and 1-B) shows a distinct need for concentration on the part of lay and public health officials in behalf of the premature infant if we are to reduce this mortality.

The Board of Health in the City of New York has already begun special work in the care of the premature infant. A premature infant committee, consisting of leading pediatricians and

TABLE 1-A. *Newborn and Premature Infant Service*—1938 (*furnished by Dr. C. Martin, Records Division, Department of Hospitals, New York City*)

	Newborn			*Premature Infants Born in 1938**		
Hospitals	Cases	Died	% Mort. Rate	Cases	Died	% Mort. Rate
Bellevue	1,384	58	4.2	75	46	61.3
Morrisania	1,143	30	2.6	56	25	44.6
Fordham	1,279	40	3.1	58	24	41.4
Harlem	2,542	64	2.5	179	46	25.7
Lincoln	1,592	48	3.0	54	34	62.9
Greenpoint	1,210	34	2.8	40	25	62.5
Coney Island	916	17	1.9	14	11	78.6
Gouverneur	302	19	6.3	22	15	68.2
Cumberland	1,023	25	2.4	64	11	17.2
Queens	1,570	42	2.7	50	23	46.0
Kings County	3,232	131	4.1	196	95	48.5
Metropolitan	777	26	3.3	28	16	57.1
City	526	20	3.8	28	15	53.6
Total	17,496	554	3.2	864	386	44.7

*Included in Newborn figures.

TABLE 1-B. *Prematures Born in* 1940 (*furnished by Central Statistical Research Dept. of the Dept. of Hospitals*)

Hospitals	Cases	Deaths	% Mort. Rate
Bellevue	108	48	44.4
Morrisania	41	27	65.9
Fordham	41	16	39.0
Harlem	315	54	17.1
Lincoln	116	30	25.9
Greenpoint	52	20	38.5
Coney Island	37	14	37.8
Gouverneur	13	6	46.2
Cumberland	74	18	24.3
Queens	142	50	35.2
Kings County	215	90	41.9
Metropolitan	69	19	27.5
City	40	28	70.0
Total	1,263	420	33.3

obstetricians and Board of Health representatives, is busily engaged in formulating plans to reduce morbidity and mortality of prematurity. Premature infant units will eventually be set up in

various hospitals throughout the city, and regulations concerning prematurity care for institutions and doctors will be embodied in the sanitary code. The fine work in Chicago[42] will soon be a reality in New York City.

It has been noted[141] that the incidence of prematurity is in direct proportion to the number of previous infant death losses. Thus a mother who has had an infant death loss is more likely to bear a premature infant than one who has not had a previous death loss. It has also been shown that premature births are more likely to be followed by more premature births. From July 1937 to November 1939, forty mothers gave a history of giving birth to more than one premature infant while fifteen mothers who gave birth to premature infants had previously been delivered of dead babies.[19] Twenty-two women gave a history of miscarriage prior to the birth of their premature infants.[19]

A glance at the section to be published later, "The Visiting Nurse Service" (special study of thirty-three records), shows the abnormalities which the visiting nurses find among pregnant women. These abnormalities resulted in the birth of premature infants. With proper antenatal care, many of these women could be carried to full term. The value of antenatal care is thus seen in these ten instances. Proper antenatal care can easily destroy the concept that a woman who has a premature infant would necessarily give birth to another premature infant since we have eliminated the cause of the original premature birth, e.g., the elimination of abnormalities such as hypertension, tuberculosis placenta praevia.

The morbidity and mortality of adults, in any community, determines the health of its children. With a general mortality rate of 20.65 and 14.12 per 1,000 population for the Central and East Harlem Districts, respectively, (New York City average 10.23) (see Table 2); with the mortality rate per 1,000 population 458 and 301, respectively, for these same districts (New York City average 136); with the maternal mortality rate per 1,000 live births 10.9 and 8.4, respectively, for these districts, (New York City average 6.43); with the venereal diseases, new cases per 100,000 population 3,809 and 1,477, respectively, (New York City average 727); small wonder that the infant mortality rate per 1,000 live births is 91 and 85, respectively, for Central

and East Harlem districts (New York City average 53) (see Table 2) and the incidence for prematurity in the last two years. 1937 and 1938, 8.2 per cent. and 10.3 per cent., respectively, (New York City average 5.6 per cent.)

TABLE 2. *Comparative Rates Between Morrisania Hospital and Harlem Hospital Health Center Districts*—1933

		Maternity Mortality Rate per 1,000 Live Births	Birth Rate per 1,000 Population	Infant Mortality Rate per 1,000 Live Births	T.B. Mort. Rate per 100,000 Population	Pulmonary T.B. New Cases per 100,000 Population	Venereal Disease New Cases Rate per 100,000 Population	Other Infectious Diseases Rates per 100,000 Population	General Mortality Rate per 1,000 Population
Morrisania Hospital	Principally from Morrisania	4.0	12	53	49	97	231	1260	8.5
	Tremont	4.2	11	47	33	85	209	982	7.5
Harlem Hospital	Principally from Central Harlem	10.9	16	91	249	458	3809	1127	20.65
	East Harlem	8.4	22	85	139	301	1477	2127	14.12
New York City Average		6.43	14	53	62	136	727	987	10.23

The following tables clearly demonstrate the effectiveness of a premature infant unit in a large New York City hospital (Harlem Hospital) where the incidence for prematurity in 1938 was twice that of the City of New York.

TABLE 3. *Premature Infant Nursery Records July* 1, 1936-*July* 1, 1937 *Previous to Installation of the New Premature Infant Unit*

Birth Weight Grams	Total	Lived	% Lived	Mort. 1-24 hrs.	Mort. 24-48 hrs.	Mort. after 48 hrs.	Total Mort.	% Mort.
Under 1,000.....	0	0	0	0	0	0	0	0
1,001 to 1,500.....	6	1	16.7	2	1	2	5	83.3
1,501 to 2,000.....	45	19	42.2	6	3	17	26	57.8
2,001 to 2,500.....	42	31	73.8	1	0	10	11	26.2
Total	93	51	54.8	9	4	29	42	45.2

TABLE 4. *Premature Infant Nursery Records July* 28, 1937-*July* 1, 1938 *Since the Installation of the New Premature Infant Unit*

Birth Weight Grams	Total	Lived	% Lived	Mort. 1-24 hrs.	Mort. 24-48 hrs.	Mort. after 48 hrs.	Total Mort.	% Mort.
Under 1,000.....	11	0	0	9	2	0	11	100
1,001 to 1,500.....	32	10	31.3	17	1	4	22	68.7
1,501 to 2,000.....	50	36	72.0	4	3	7	14	28.0
2,001 to 2,500.....	88	86	97.5	1	0	1	2	2.27
Total	181	132	72.9	31	6	12	49	27.1

Premature infant mortality before installation of unit (Table 3) was 45.2 per cent, while 54.8 per cent. survived. Premature infant mortality after installation of unit (Table 4) was 27.1 while 72.9 per cent. survived.

TABLE 5. *Premature Infant Nursery Records July 9, 1938-July 1, 1939 Since the Installation of the New Premature Infant Unit*

Birth Weight Grams	Total	Lived	% Lived	Mort. 1-24 hrs.	Mort. 24-48 hrs.	Mort. after 48 hrs.	Total Mort.	% Mort.
Under 1,000.....	12	0	0	5	5	2	12	100
1,001 to 1,500.....	29	14	48.0	8	3	4	15	52
1,501 to 2,000.....	69	58	58.0	1	5	5	11	15
2,001 to 2,500.....	154	138	90.0	5	5	6	16	10
Total	264	210	79.5	19	18	17	54	20.5

TABLE 6. *Premature Infant Nursery Records for* 1939

Birth Weight Grams	Total	Lived	% Lived	Mort. 1-24 hrs.	Mort. 24-48 hrs.	Mort. after 48 hrs.	Total Mort.	% Mort.
Under 1,000.....	24	0	0	18	3	3	24	100
1,001 to 1,500.....	34	12	35.2	15	5	2	22	64.8
1,501 to 2,000.....	73	61	83.5	6	3	3	12	16.5
2,001 to 2,500.....	163	152	93.2	6	1	2	9	5.8
Total	294	225	76.5	45	12	10	67	23.5

Mortality of 23.5 per cent. for the year 1939. Note also that the majority of these infants died in the first 24 hours.

TABLE 7. *Premature Infant Nursery Record,* 1939, *Infants Born Outside of the Hospital (Harlem Hospital)*

Birth Weight Grams	Cases	Lived	% Lived	Mort. 1-24 hrs.	Mort. 24-48 hrs.	Mort. after 48 hrs.	Total Mort.	% Mort.
Under 1,000.....	2	0	0	1	1	..	2	100
1,001 to 1,500.....	5	3	60	2	..	..	2	40
1,501 to 2,000.....	14	11	79	..	1	2	3	21
2,001 to 2,500.....	20	16	80	2	..	2	4	20
Total	41	30	73	5	2	4	11	27

A little more than a year ago we set aside a room for premature infants born outside the hospital. The room adjoins the general pediatric ward. Three registered nurses are in charge. The first nurse reports at 7:00 A.M. and stays until 3:30 P.M. She is followed by another nurse who reports at 2:30 P.M. and stays until 11:00 P.M. A third nurse works from 11:00 P.M. to 7:00 A.M. Each nurse is relieved by a student nurse once a week when the former has her day off.

The equipment in this room is similar to that in the premature infant unit adjoining the maternity ward and used for premature infants born in the hospital. The procedures and feedings are similar to those used in the latter unit.

Out of a total of 41 premature infants born outside of the hospital, 11 died. Autopsies performed on these infants revealed

prematurity as the cause of death. Almost half of these infants died in the first 24 hours.

The Committee on Prematurity of the City of New York reports a mortality rate of 50 per cent. for infants born outside of the hospital. This represents a total of 22 premature infants admitted to 11 New York City hospitals from June to December 1938. It will be seen that our mortality figures represent about half of the above results. This is just another instance where good results are obtained merely by setting aside a clean warm room with special nurses. It appears that our separating the premature infant from the normal newborn and our keeping them out of the general ward is sufficient to keep the death rate down to 27 per cent.

In former years we lost 50 to 75 per cent. of all premature infants admitted to the hospital. We placed these infants in rooms adjoining the general ward and allowed general ward nurses to care for them.

Conclusion: The East and Central Harlem Health Centre districts show a prematurity incidence of 8 to 10 per cent. for the last five years. By a little effort we have reduced the premature infant mortality from 45.2 per cent. in 1937 to 23.5 per cent. in 1939. The above districts would be suitable places to establish a premature infant unit designed to nurse premature infants born in and outside of the hospital.

It hardly matters that I have employed statistics concerning the City of New York only. Other cities unquestionably have similar figures and results.

Unless we give more attention to the problems of prematurity we shall not save these infants nor shall we lower the mortality rate of all infants under one month of age.

Premature infants need special nurses and separate rooms. They require attentions far different from those accorded the normal newborns.

Premature infants need not be sent to a hospital if conditions at home are favorable. The general practitioner should and can raise a premature infant as well as the specialist in a special premature infant unit.

ANATOMICAL AND PHYSIOLOGICAL CHARACTERISTICS OF PREMATURITY

The premature infant often resembles a wizened old man. Commonly found are large fontanels with prominent sutures, shiny, almost transparent skin well covered with lanugo hairs, incompletely developed nails which do not reach to the ends of the fingers and scant subcutaneous fat. A rather large tongue and prominent exophthalmos are often noted and take about a year to disappear. A nasal discharge is a common occurrence.

Premature infants suck and swallow with difficulty. Their digestive systems are partly developed. However, in spite of these shortcomings, one notes the presence of enzymes rather early, thus making the use of predigested foods unnecessary.

Another evidence of immaturity is the poor development of the elastic lung structure of the premature infant[115]. The lower the birth weight of the premature infant, the weaker the elastic lung tissue. Deficient respiratory function is partly explained on this basis. Fortunately, within a period of two months, sufficient elastic fibers develop to promote normal respirations.

Some premature infants have a feeble cry and are often roused with difficulty. In fact, a vigorous cry from a premature infant connotes an excellent prognosis.

The low state of respiratory center development of many of these premature infants is often an indication of the degree of their immaturity. Their respirations may be slow or rapid, regular or irregular. Often, they appear to be in a stupor and one can hardly discern any evidence of respiratory movement. At times, excessive accumulations of carbon dioxide do not appear sufficient to stimulate the respiratory center.

A thin skin with its extensive blood vascular network, a scant layer of subcutaneous fat, the occasional presence of intracranial injury together with a low basal metabolic rate[110] are all evidences of a need of added heat for the premature infant. While the normal newborn responds to changes in its environmental temperature by perspiring or restlessness, the premature infant presents either a hyperpyrexia or a subnormal temperature. This maladjusted, heat regulating mechanism explains the need for incubators or nurseries kept at a constant temperature of 80° F.

At birth, the average premature infant shows a hemoglobin of 120-115 per cent., an erythrocyte count of 7½-5½ million, 5-2 per cent. reticulocytes, 35,000-15,000 white blood cells, with a differential count of polymorphonuclear leukocytes 60 per cent., lymphocytes 30 per cent., mononuclears 10 per cent., platelets 350,000. By the tenth day the hemoglobin is usually 100 per cent. (14 Gm. per 100 cc. blood), R.B.C. 5 million, reticulocytes 1 per cent., W.B.C. 12,000, polys 30 per cent., lymphs 62 per cent., monos 8 per cent., platelets 300,000.

In the premature infant, hemoglobin retains its fetal characteristics for a short period. In the normal newborn such characteristics are absent or short-lived.[119]

The protein level of the blood is usually constant in a normal neonate. It is low in a premature infant[81]. Studies have shown that low blood protein levels are frequent in premature infants even when they are fed adequate amounts of breast milk. On the addition of calcium caseinate to the apparently adequate daily supply of breast milk, the blood protein level rises to normal values (6-8 Gms. per cent.)

During the first three months of life there is normally a definite reduction in the number of red blood cells and hemoglobin. This process represents one of the changes from fetal to extra-uterine life. This rate of destruction is greater in the premature infant than in the normal newborn[25]. The greater the degree of prematurity the greater and faster is the drop in the hemoglobin level. Hemoglobin values as low as 7 Gms. per 100 cc. of blood are reported.

In the second and third months the hemoglobin of the premature infant is often as low as 40 to 50 per cent. while the erythrocyte count drops to 2½ to 3 million. At six to eight months of age, however, the hemoglobin and red blood count are similar to those of the normal neonate.

The rapid fall in hemoglobin and red blood cells seen in the first trimester of the premature infant's life appears to be due to the absorption of certain blood islands in the liver and spleen, a hypoplasia of bone marrow with decreased blood formation and a deficient antenatal iron storage. Which of these is to be considered the primary factor is still a moot question. Reticulosis

noted in the premature infant bespeaks, in a measure, the theory of decreased blood formation with bone marrow hypoplasia.[124]

Erythroblasts are found in the normal newborn infant's blood during the first day of life. In the premature infant, however, we find nucleated red cells persisting for several weeks. In fact, this persistence of erythroblasts beyond the first day definitely signifies prematurity and may have medicolegal significance.

Studies of the electrocardiograms of normal newborn and premature infants prove them to be essentially the same. Both full term and premature infants show a right axis deviation for approximately the first three months of life. About four months after birth R-1 becomes larger than S-1 and slowly the ventricular complex resembles the adult type of curve. Right ventricular preponderance found in these infants is due to the fact that the right ventricle is as large and as strong as the left ventricle. With the closing of the foramen ovale and ductus arteriosus, there is shifting of the circulatory system from "right" to "left". The fetal type of circulation gives way, over a period of three months, to the normal childhood type. This shunting of blood from the right to left side puts more work on the left ventricle and by approximately the fourth month left axis deviation is the rule. Right ventricular preponderance persists longer in the more premature infants. Cicatrization of the ductus arteriosus and closing of the foramen ovale occur sooner in the heavier premature infants.[126]

The blood calcium level during the first four days of life shows lower values both in the premature infant as well as in the normal newborn[81]. It takes about a week before it reaches a value slightly higher (11.5 mg.) than the usual 10 mg. per 100 cc. of blood found in the older infants[81].

A prolonged prothrombin and blood clotting time is also found in many premature infants. The prothrombin clotting time seems to be most prolonged in the second and third days of the infant's life and is normal five days after birth. The blood clotting time, also, is most prolonged in the second and third days of life and is normal at about the end of a week[79].

Prothrombin clotting and blood clotting time appear to increase or decrease together. For this reason, neonates often require therapeutic measures to hasten blood coagulation. Especially is this true in cases of prolonged labor and intracranial hemorrhage of the oozing type.

Premature infants under 1,500 Gms. and many weighing less than 1,250 Gms. present most of the above anatomical and physiological characteristics. Those over 1,500 Gms. are often as vigorous in crying, moving and swallowing as many normal newborn infants. It goes without saying that the former require incubators as well as all the attention we can give them if they are to survive. The latter or heavier infants, especially those of five pounds, will thrive in a room at 74° F., require no incubators and will gain on the usual normal newborn formulas used routinely in most hospital nurseries. Since our highest mortality rates are found in the "under 1,000 grams" and "1,001 to 1,500 grams" groups, it is here that our greatest efforts are needed.

DEFINITION

A premature infant is one who weighs 5½ pounds (2,500 grams) or less at birth, regardless of the period of uterine gestation. Some have complained about this definition, insisting that most 5½ pound infants are as well developed as the normal newborn. Hence to include such babies was to reduce the premature infant mortality. It must remembered, however, that any newborn baby weighing less than 1,000 grams, which breathes once or has but one audible heart beat, is considered in the same class. In the final analysis, this latter group accounts for most of the mortalities.

While 2,500 grams or less (e.g., weight) are taken as the criterion for prematurity, race and sex must be given due consideration. The latter are bound up closely with the degree of growth and development. Thus it is interesting to note that an x-ray study of the cuboid of 498 premature infants, taken within 72 hours after birth, revealed the following facts, viz., that the cuboid was present in 37 per cent. of the colored girls, 17 per cent. of the colored boys, and 15 per cent. of the white girls. The cuboid was not seen in the x-rays of the white boys.

This study of growth and development, in which race and sex are compared, needs careful consideration in the final analysis of mortality statistics. While the heavier premature infants have a better chance to survive, many premature infants weighing less but being more mature than the heavier infant will also survive.

INCIDENCE

About five to six per cent. of all newborns are premature

infants[141]. Of approximately 100,000 newborns delivered in New York City each year, according to the Board of Health 5,500 to 6,000 are premature infants (6.0 per cent. white, 8.5 to 10 per cent. colored). In a study[11] of 23,000 live-born infants from eight cities, the prematurity incidence was 5 per cent. In 1936, the incidence for premature infants born in New York State, exclusive of New York City, was 4.29 per cent.

At one of the large New York City Municipal Hospitals, (see section "Mortality") the prematurity incidence was 8.2 per cent. in 1937, and 10.3 per cent. in 1938. Fully 95 per cent of the mothers from this institution were Negroes.

The prematurity incidence of our large private New York City hospitals is about the same as we find in some of our municipal hospitals. From October 1936 through September 1937, 2,579 newborns were delivered at two large Brooklyn, New York City, private institutions. Twenty-eight were premature infants, an incidence of 3.3 per cent.[85] In another institution in New York City, where all the women were white, the incidence of prematurity was 8½ per cent.

There are certain factors which determine the incidence of prematurity. Some are previous infant death losses, previous prematurity[141], congenital maldevelopments, obstetrical abnormalities, such as placenta praevia and premature separation of the placenta, and the toxemias of pregnancy. Other causative agents for prematurity are acute and chronic diseases, especially those in the cardiac, nephritic and diabetic groups, straining, heavy lifting, tedious auto rides and emotional upsets. Pregnant women in industry also present a problem in prematurity.

The responsibility for proper care of diabetic, cardiac and nephritic pregnant women must be placed in a well managed antenatal clinic. Experience has shown the need for a diabetic diet, insulin, rest, frequent urine examinations and blood pressure readings. To help reduce the incidence of prematurity where the above chronic diseases are concerned, we must insist upon periodic visits to the homes. Especially is this important when women fail to keep their clinic appointments.

Pregnant women in industry are now in a better position to give birth to a normal newborn than their sisters in the sweatshop days. Our government, with its fine social program, is certainly cognizant of the problem and its solution is nearer than ever. In

addition, better antenatal care will certainly help reduce the incidence of prematurity found among women engaged in industry.

Better antenatal care will reduce the incidence of prematurity resulting from syphilis in pregnancy. Of a total 554 pregnant women who gave birth to premature infants, 10.2 per cent. had syphilis. The antiluetic treatment in our prenatal clinics, the premarital Wassermann requirements and our attempts to emulate the work in the Baltic States, as well as our governmental interest in the prevention and treatment, will certainly result in fewer premature infants due to syphilis.

The toxemias of pregnancy accounted for 10.2 per cent. of all the premature infants. Careful urine examinations, repeated blood pressure readings, hormone studies, refresher courses in obstetrics, a Visiting Nurse Service bringing pregnant women to the clinics, all of these efforts will undoubtedly result in less prematurity due to the toxemias.

CAUSES OF PREMATURITY

From July 28, 1937 to November 10, 1939, we studied the histories of 554 mothers and their premature infants—84.8 per cent. were colored and 16.2 per cent. were white.

A. *Obstetrical.* There were 57 cases of toxemia of pregnancy (10.2 per cent.); 5 placenta praevias (0.9 per cent.); 3 contracted pelves (0.5 per cent.); 15 premature separations of the placenta (2.7 per cent.); 27 multiple births (4.7 per cent.). I believe that there are more multiple births among colored than among white women.

B. *Congenital Maldevelopments.* This group accounts for the majority of premature infant deaths. They usually die in the first 48 hours. Very little can be done for these infants.

C. *Maternal.* (1) *Acute Diseases:* There were two cases of sepsis (0.3 per cent). While pneumonia and influenza are frequently reported as a cause for premature labor our series showed none.

(2) *Chronic Diseases:* There was 1 case of malaria (0.1 per cent.); 4 cases of pulmonary tuberculosis (0.7 per cent.); 1 diabetes mellitus (0.3 per cent.); 1 adenoma of the thyroid (0.1 per cent.); 2 cardiacs (0.3 per cent.); 1 uterine fibroid (0.1 per cent.). Of 57 mothers who gave birth to premature infants, nine had a 1

plus Kahn (1.6 per cent.); 18 had a two plus Kahn (3.2 per cent.); 10 had a three plus Kahn (1.8 per cent.); 20 had a four plus Kahn (3.6 per cent.). In other words, of a total of 554 premature infants (born from July 28, 1937 to November 10, 1939) only 57 were born of syphilitic mothers. It will be noted that 4.8 per cent. had a 1 plus or two plus Kahn and that 5.4 per cent. had a three plus or four plus Kahn. There were 10 known treated cases of syphilis among these 57 women.

The above figures do not tell us whether the premature infants themselves were syphilitic. Quantitative Wassermann tests often proved otherwise in several of these infants. In short, unless we actually find some organic lesion, such as osteochondritis, spirochetes in the nasal discharge or in the condylomatous secretions, or positive autopsy findings, (e.g., spirochetes in liver smears, etc.), we are unable to state just how many of the 57 infants were syphilitic.

Prematurity in the case of these 57 premature infants might still be due to factors other than syphilis (or perhaps in addition to syphilis). It is thus difficult to evaluate the relationship of syphilis to prematurity. Certainly, if there are such things as syphilitic pregnancy tragedies, (abortions, miscarriages and prematurity) they were not particularly evident in our series. Again we have a strong argument for autopsies on many of these dead premature infants in order to learn more of the relationship between syphilis and prematurity. We must also repeat the need for the quantitative Wassermann test, done at weekly intervals in order to diagnose congenital syphilis.

I have already indicated how difficult it is to consider syphilis as the only cause for prematurity. In so many of these pregnant women additional pathological entities, such as multiple pregnancies, gonorrheal vaginitis, myoma, were found. These conditions in themselves are ample cause for prematurity.

In one hospital,[9] where 146 complications of pregnancy were encountered, only four cases of syphilis were regarded as the sole cause of prematurity.[9] Syphilis was found associated with multiple pregnancy in two cases, with gonorrheal vaginitis and myomas in one case.

D. *Trauma—Falls—Blows.* Eight women who were delivered of premature infants gave a history of trauma.

E. *Miscellaneous.* The following table shows (a) *parity* as a factor in prematurity.

TABLE 8

Para	Cases	%
0	275	46.0
I	117	20.0
II	66	11.0
III	33	5.8
IV	29	4.9
V	7	1.2
VI	9	1.5
VII	1	0.1
VIII	1	0.1
X	2	0.3
XII	2	0.3
XIII	1	0.18
XIV	1	0.18
	544	

Forty-six per cent. of the women who gave birth to premature infants were primipara while 20 per cent. had given birth to one baby.

(b) *Youth.* Women between the ages of 13 and 17 years, inclusive, gave birth to six premature infants (11.0 per cent. of the total). In 1938, seven per cent. of the unwed mothers of New York City were under 16 years of age. From April 1937 to May 1939, 42 girls, whose ages were 18 years or less, were delivered of premature infants. Twenty-five of these girls were unwed and two were still in school. Seventeen were married.

In the last 15 months, the Visiting Nurse Service reported few mothers of premature infants who were under 18 years. This finding is similar to ours in a study of 554 women from July 28, 1937 to November 10, 1939.

As one views the causes of prematurity, the toxemias, syphilis, youth and parity stand out. It is against these causes that we must plan our attack. I have already discussed the problems of syphilis in pregnancy and the toxemias.

Youth and parity still remain important considerations in reducing prematurity incidence. If 46 per cent. of the women giving birth to premature infants were primipara, the question of antenatal care looms very largely as a factor. We must encourage these pregnant women to visit a physician just as soon as they become pregnant. Here is a fine opportunity for both the Social Service Department and the Visiting Nurse Service.

MORTALITY

A. *General Discussion.* Many a premature infant dies as a result of immaturity, atelectasis, cerebral hemorrhage, malformation or infection (upper or lower respiratory). Let us consider these causes in detail.

Prematurity, alone, is a frequent cause of death.[9] More than half of the infant deaths at one large hospital in Connecticut were due to prematurity.[9] In these instances, a state of immaturity exists which is incompatible with life. However, we should like to discourage the diagnosis of prematurity as a cause of death, since autopsy has often proved otherwise. We would like to encourage autopsies in these cases before accepting prematurity as the cause of death.

Infection is a common cause for the death of premature infants. About one-fourth of the premature infants in one institution died from infection.[9] Unless carefully sought for, sepsis, pneumonia, influenza, otitis media and meningitis are easily overlooked. Such infants are frequently signed out as "cause of death prematurity."

Atelectasis causes many deaths of premature infants. There are varying degrees of atelectasis and since infants under 1,500 grams show more of it than the heavier infants, we are at a loss to know when atelectasis is really the major cause of death. In some cases autopsy revealed other conditions, such as intracranial hemorrhage, bronchopneumonia and sepsis. Certainly, a clinical diagnosis of atelectasis is not always sufficient as the cause of death. It may be found at autopsy and not noted clinically.[9]

Too many premature infants, who die, are signed out with "causes of death unknown".[10] The death certificate reads atelectasis, congenital defects, immaturity, premature birth.[9] Unless extensive, atelectasis in itself is hardly a cause for death. It is rather an evidence of immaturity and as such only a factor in the causation of death.

In some instances autopsy does not help us determine the cause of death. We have already mentioned the findings of a group of Chicago workers, who, in 1936, noted no demonstrable pathological lesion in 12.3 per cent. of their cases[42]. It has been suggested that a lack of certain hormones in these infants, due to a failure of placental transmission, may be a factor.

Among other causes are poor or no antepartum care, failure to rule out syphilis, the use of anesthetics, oxytocics and analgesics in the hands of the inexperienced, toxemias of pregnancy, and placenta praevia. Improper methods of resuscitating the newborn premature infant and acute infections in the pregnant woman add to this high mortality.[42]

From July 1937 to November 1939, 554 premature infants were born in Harlem Hospital, New York City. A total of 112 autopsies were performed. The following are our findings: Immaturity 34 cases; atelectasis 45 cases; infection 7; otitis media 1; bronchopneumonia 4; tracheobronchitis 1; meningitis 1; abnormalities 3 (congenital heart, diaphragmatic hernia, congenital multiple intestinal atresias); intracranial hemorrhage 10; adrenal hemorrhage 1; enlarged thymus 1; petechial hemorrhages, all organs, 10; gastric hemorrhage (hemorrhagic disease newborn?) 1; icterus gravis 1.

In 1939 a detailed study of our mortality among premature infants was made. Sixty-seven infants died; we autopsied 32. The results are: Prematurity 24; atelectasis 18; intracranial hemorrhage 2; congenital heart 2; monstrosity 1; bilateral adrenal hemorrhage 1; icterus gravis 1; hemorrhagic disease newborn 1. A diagnosis of atelectasis was not made without an autopsy. Thirty-two cases of prematurity were diagnosed without autopsy. Fifty per cent. of the latter weighed less than 1,000 grams.

In a study done in New York State, 54 per cent. of the neonatal deaths were found among the premature infants. The mortality of premature births was 389.2 per 1,000 live births while the neonatal mortality for full term infants was 15.2 per 1,000 live births. The mortality rate for prematurely born infants was 25 times as high as for full term infants.

In 1938, 13 New York City municipal hospitals reported the birth of 864 premature infants, with a total mortality of 44.7 per cent. One of these hospitals with 64 premature infants had a death rate of 17.2 per cent. Another hospital (Harlem Hospital—85 per cent. Negro) reported the birth of 179 premature infants with a mortality rate of 25.7 per cent. One of the largest municipal institutions reported the birth of 196 premature infants with a mortality rate of 48.5 per cent. Another very large municipal hospital noted 75 premature infants with a death rate of 61.3

per cent. (See Table 1-A.). Two large Brooklyn private hospitals[85] reported a premature infant mortality rate of 25 per cent.

More premature infants die in the first 48 hours than thereafter[19]. From 1931 to 1939, 1,320 premature infants were born in one large New York City municipal hospital (Harlem Hospital.) Twenty-seven per cent. died in the first 48 hours and 15.6 per cent. after 48 hours (see Table 9). In 1939, 85 per cent. died in first 48 hours.

In 1935 Chicago reported a mortality rate of 69.7 per cent. for premature infants in the first 24 hours[42]. A New Haven hospital[9], in a ten year period, reported a death rate of 27 per cent. More than half of these deaths occurred in the first 24 hours of life.

The following table gives one an idea of the mortality rates of normal newborn and premature infants. Here, it will be noted, that, with but one exception (1934), the mortality rate in the first 24 hours was greater than after 48 hours. It will also be seen that the mortality rate in the first 48 hours far exceeded that of the period after 48 hours (exception 1934).

TABLE 9. *Newborn and Premature Infant Service, Harlem Hospital*

Year	No. N'born	% Mort. N'born	No. Prem. Inf.	% Incid. Prem. Inf.	% Mort. Prem. Inf. 1-24 Hrs.	% Mort. Prem. Inf. 24-48 Hrs.	% Mort. Prem. Inf. After 48 Hrs.	Total No. Prem. Inf. Lived	% Prem. Inf. Lived
1931.....	1,325	1.4	55	4.2	49.0	11.0	27.0	7	12.7
1932.....	1,523	2.0	108	7.1	38.0	6.5	24.0	34	31.5
1933.....	1,371	0.8	86	6.3	42.0	4.6	29.0	21	24.0
1934.....	1,359	3.2	115	8.5	27.0	0.9	32.1	46	40.0
1935.....	1,237	3.3	104	8.0	24.0	6.0	20.2	52	50.0
1936.....	2,023	2.5	156	7.7	34.0	3.2	26.9	56	35.9
1937.....	1,596	3.4	131	8.2	20.0	7.6	14.5	76	58.0
1938.....	2,631	0.46	271	10.3	9.97	2.95	4.4	224	82.65
1939.....	2,699	0.44	294	10.8	67.1	17.9	14.9	225	76.5
1940.....	2,590	0.81	297	11.4	10.4	1.6	1.6	256	86.1

In 1938 Harlem Hospital had a well established premature infant unit. The total mortality that year was 17.35 per cent. We saved 82.65 per cent. of the premature infants. We must strive for a mortality rate of less than 5 per cent. in premature infants who survive the first 48 hours.

The high mortality rate seen in the first 24 hours is explained on the basis of extreme immaturity and congenital maldevelopments. In this group are found most infants under a thousand grams. While every effort is made to save babies in the "under

1000 gram group", it is readily understood why our results thus far have been poor.

Infants dying between 24 and 48 hours are usually those of the "under 1000 gram group" who have been kept alive by adrenalin, oxygen, blood and clyses. Some of these infants have abnormalities, such as complete exstrophy of the bladder or a serious cardiac anomaly.

It is for these reasons that I do not emphasize the saving of lives in the "under 1000 gram group". Such infants later comprise our children with spasticities and varying degrees of mental retardations. Any hospital which can save more than 80 per cent. of its premature infants having lived 48 hours is doing satisfactory work.

B. *Mortality Based on Weight at Birth.* A clearer idea is obtained from mortality statistics if the premature infant weights are classified as follows:

Group 1. Under 1000 grams
Group 2. 1001-1500 grams
Group 3. 1501-2000 grams
Group 4. 2001-2500 grams

Evidence shows that the heavier the infant, the better the prognosis. The highest mortality occurs in Group 1, under 1,000 grams. Occasionally, however, especially in a multiple birth, a more viable premature infant, though weighing less, may present a better prognosis.

TABLE 10. *Premature Infant Mortality for* 1938[132]

Birth Weight Grams	Cases	Lived	% Lived	Mort. 1-24 hrs.	Mort. 24-48 hrs.	Mort. after 48 hrs.	Total Mort.	% Mort.
Under 1,000.....	1	0	0	1	0	0	1	100
1,001 to 1,500.....	11	2	18	6	1	2	9	82.0
1,501 to 2,000.....	3	3	100	0	0	0	0	0
2,001 to 2,500.....	37	35	94.9	2	0	0	2	5.1
Total	52	40	77.0	9	1	2	12	23

In discussing mortality statistics, it is important to note the percentages in each group. In Table 10, a study made in a private hospital[131] where all the premature infants were white, 71 per cent. are in Group 4, (2,001-2,500 grams). A glance at Table 5 and Table 6 will show only 58 per cent. and 55 per cent., respectively, in Group 4. Here 85 per cent. of the premature infants

are negroes. Since the heavier the infant the better the prognosis, in a study of premature infant mortality, proper consideration must be given to institutions having few infants in Group 4. However, it must be remembered that "colored infants are smaller than white infants of the same period of gestation so that a colored infant of 1,500 grams is not as premature as the white infant of 1,500 grams." (Report New York City Committee on Prematurity, Dec. 18, 1939.)

C. *Pediatrician's Responsibility.* The highest premature infant mortality rate occurs in the first 24 hours (see Table 9). Congenital maldevelopments and immaturity incompatible with life explain this high death rate. There are a few infants in this group (those dying in the first 24 hours) who can be saved by the obstetrician. The large majority of premature infants who survive the first 48 hours can be saved. That is the pediatrician's responsibility.

D. *The Obstetrician's Obligations.* The circumference and occipitofrontal diameter of the fetal head can be accurately calculated by x-ray examination.[6, 7] From this knowledge, the approximate age and weight of the fetus can be derived.

X-ray pelvimetry and pelvic architecture are used in advanced pregnancy to locate placenta, placental apoplexy, placenta praevia, tumors of the lower uterine segment causing dystocia and even abdominal pregnancy.[163] The information obtained from x-ray studies suggests the presence of prematurity and determines the induction of labor and the termination of pregnancy. This knowledge improves the chances of a viable baby being born.

A clinical plus a radiological knowledge of pelvic types gives one a better idea of an ample pelvis than clinical knowledge alone.[82] The nature of the operative delivery or the kind of forceps to use is often determined by the anatomy of the pelvis.

The obstetrician can aid materially in reducing premature infant mortality. When the occipitofrontal diameter or circumference of the fetal head shows the fetus to be three to four pounds, it may be well to terminate pregnancy in such conditions as pulmonary tuberculosis, diabetes mellitus, chronic cardiovascular disease, many of the toxemias of pregnancy and slight vaginal bleeding which threatens to be extensive (due to placenta praevia).

In order to reduce premature infant mortality, the obstetrician

must reconsider the choice of anesthesia in obstetric procedures. Ether is the anesthesia of choice when delivering a mother of a premature infant. Spinal or local anesthesia is, however, preferable to a general anesthetic where a premature infant is expected.

Morphine given the pregnant woman in labor often causes death of the fetus.[9] While 38 per cent. of infants whose mothers had received morphine on admission to the hospital died, the mortality rate for those infants whose mothers received no morphine was 23 per cent.[9] Where mothers received morphine the onset of respiration in the infant was delayed in 41 per cent. of the cases, while in those cases where no morphine was administered there was delay in the onset of respiration in only 16 per cent. of the cases. Again, it must be emphasized that morphine should not be given in Cesarean section.

Obstetricians are agreed that premature infant mortality is higher in induced labor.[9, 128] Low forceps with wide episiotomy in vertex presentation is apparently the best method of delivering the mother of a premature infant.[8] Breech extraction is decidedly harmful, while vertex delivery is somewhat safer. In short, regardless of the type of delivery, a study of fetal and neonatal deaths by one author led to the conclusion that a lowered mortality would result only from better judgment and technique in the processes of labor and delivery.

The toxemias of pregnancy (toxemic type of pernicious vomiting, nephritic toxemia, pre-eclamptic toxemia and eclampsia) are still problems of major importance in obstetrics. There were 57 cases of toxemia of pregnancy in women who gave birth to premature infants. This represented 10.2 per cent. of all the premature infants born from July 1937 to November 1939.

To the careful obstetrician treating a case of pregnancy, a slowly rising blood pressure, together with other toxemic manifestations, such as vomiting, alkalosis, positive urine findings (albumen, casts), headache, eye signs, are usually indications for bed rest, careful diet and intravenous glucose therapy. If, in spite of known therapy, the blood pressure continues to rise and the patient is worse, labor is usually induced.

It has been shown that 43.7 per cent. of women suffering from toxemias of pregnancy gave birth to stillborns when their blood pressure was 180 mm. of mercury.[5] Why not induce labor when

blood pressure readings in these women show 150 mm. of mercury? With this figure the infant is still viable.

I have mentioned but a few cases where obstetricians must reexamine their treatment of the pregnant woman in the light of newer concepts and recent findings. A beginning has been made. The pediatrician is not attempting to offer a solution or to tell the obstetrician what to do.

Already many fine obstetricians are conscious of their duties. Witness the refresher courses in obstetrics, regular lectures and discussions at leading hospitals, maternal and newborn statistical studies and hormone investigations as they relate to the pregnant woman.

We should like to mention a few specific studies by obstetricians, clinicians and pediatricians which have for their object the reduction of premature infant mortality. Among these are the use of pregnancy serum as well as corpus luteum hormone in the prevention of habitual abortion; the estimation of the level of serum choriogonadotropin as a means of reducing fetal mortality in pregnant diabetic women; the administration of vitamin K to pregnant women during labor as well as to the infant immediately after birth in order to reduce the incidence of intracranial hemorrhage and hemorrhagic disease in the newborn; the administration of dicalcium phosphate with viosterol to pregnant women in order to maintain normal mineral metabolism in both mother and infant. Finally, we must add the prophylactic administration of estrogenic hormone to pregnant women who may possibly have uterine inertia during labor, as well as the wider use of x-ray pelvimetry diagnosing the weight of the infant, prior to the induction of labor.

E. *The Need for Autopsies.* To lower the morbidity and mortality of premature infants the causes of death should be known in each case. This requires autopsies by competent men. They must be properly trained and follow a uniform, generally accepted technique.

Autopsies, properly done, often prove errors in clinical diagnosis. Many premature infants die of septicemia, intracranial hemorrhage, adrenal hemorrhage, asphyxia, congenital syphilis, erythroblastosis, pneumonia, meningitis, respiratory infections and diarrhea, yet clinical study of these infants does not reveal evidences of intracranial hemorrhage, pneumonia or other infec-

tions. The absence of fever or nuchal rigidity, negative blood cultures, negative x-ray, neurological and bacteriological findings often make a clinical diagnosis difficult. We need the autopsy for a final check and a better diagnosis in order to lead to a clearer understanding of the clinical picture accompanying these conditions which terminate fatally. At least some babies may be saved by instituting proper therapy.

It must be admitted that occasionally autopsy shows no demonstrable pathological lesions. Perhaps in this group are cases of hormone deficiency already referred to.

The following cases represent definite evidence of a need for autopsies. In each instance, in spite of careful clinical work, the autopsy findings gave the correct diagnosis.

Case 1. J—, colored female, born December 19, 1935, weight 4 pounds 4 ounces. Condition fair. Normal delivery.

January 1, 1936. Infant's general condition poor, color fair, respiration slow and deep. Examination reveals no evidence of infection. Infant probably developing a bronchopneumonia; advise isolation; doubly concentrated breast milk and oxygen. Buttocks excoriated.

January 2, 1936. Patient expired at 3:20. Diagnosis prematurity.

Post mortem Record—Anatomical Diagnosis. The body was that of a colored female baby weighing 1,200 grams. The head was opened in the usual manner and a tentorial tear 1 cm. in length was found near the posterior of the tentorium. A moderate amount of blood occupied the subarachnoid space over the left cerebral hemisphere. There were circumscribed hemorrhagic areas varying in diameter from 5 to 15 cm. in both lungs. These were firm but not friable. The liver shows a grey discoloration. No other abnormalities were found.

Gross Diagnosis. Prematurity. Laceration of the tentorium cerebelli. Subarachnoid hemorrhage. Circumscribed hemorrhage of both lungs. Parenchymatous degeneration of liver.

It is easily seen that one can hardly make a diagnosis of parenchymatous degeneration of the liver clinically. Without autopsy this lesion and the intracranial hemorrhage would have been overlooked.

Intracranial hemorrhage with birth injury was shown to be the cause of death in about one-fourth of the premature infant

deaths at the Chicago Lying-In Hospital.[142] In only one-third of the premature infant deaths was prematurity alone responsible for death.[142]

Case 2. McC—, colored male. Two day old premature infant, born "outside" of hospital, weighing 2 pounds 12 ounces.

Admitted May 18, 1936. Heart sounds poor, otherwise general examination negative. May 20, 1936: Moderate swelling left inguinal region extending into scrotum. Testicle not in scrotum. May 21, 1936: Patient pronounced dead at 2:35 P.M. Diagnosis prematurity.

Post mortem Record—Anatomical Diagnosis. Patient was a premature male infant. Birth weight 2 pounds 12 ounces. Nine days old.

Intracranial findings: Small amount of bloody fluid and blood clot above and below the tentorium cerebelli. Small tear in tentorium to right of junction with falc cerebrum. Heart and lungs: Normal. Liver and spleen: Normal. Kidneys, ureters and bladder: Normal. Adrenals: Normal. Metaphysis of right femur—regular line—orange color.

Gross Diagnosis. Prematurity, intracranial hemorrhage due to tentorial tear. Here too, autopsy reveals intracranial hemorrhage overlooked clinically.

In an infant weighing 2 pounds 12 ounces, usually brought to the hospital in extremis, with a subnormal temperature of 96.4° F., the difficulty of making a correct clinical diagnosis is evident. To sign out such an infant as premature is, to say the least, incomplete. The autopsy is necessary.

Case 3. McZ—, colored male, age 29 days.

History: Normal delivery of a premature male infant on April 19, 1937. Weight at birth 3 pounds 12 ounces. Condition fair. Respiration and circulation good.

April 21, 1937. General condition fair. Lost four ounces.

April 24, 1937. Gained one ounce. No gastrointestinal upset. Slight edema of both lower extremities.

May 6, 1937. Baby gaining weight—3 pounds 8 ounces. Feeding well. No gastrointestinal disturbance. Not dehydrated.

May 7, 1937. Feeding well. Watery stool in morning. Slight depression of anterior fontanel.

May 8, 1937. Stools watery. Abdomen somewhat distended—not rigid. Not feeding well. Sent to "isolation". Weight remained

stationary until day of death (3 pounds). Board of Health examination of stools negative. Culture of throat was negative for hemolytic streptococci.

May 19, 1937. Patient pronounced dead. Diagnosis prematurity.

Autopsy. The body was that of a poorly developed, poorly nourished, premature colored male infant. The abdomen was markedly distended. The skin showed the usual post mortem changes. The scalp was avulsed and the calvarium was opened. There was a considerable amount of congestion of the piarachnoidal vessels. No other cerebral pathology was visible. The body was opened with a median incision. Both pleural cavities contained a small amount of clear straw-colored serous fluid. The surfaces of both lungs were smooth and glistening. The right upper lung, however, showed an area of consolidation which on section presented the characteristic greyish-yellow patches suggesting bronchopneumonia. The trachea and bronchi were filled with greyish-yellow purulent material. The mucosa was moderately congested. The pericardium was filled with thick, pale green, fibrinopurulent material. The heart was normal in size. The myocardium was rather pale brown. The chambers were normal and filled with dark red currant jelly blood clot. The valves and large vessels showed no unusual changes. The stomach was normal. The intestines were filled with a moderate amount of canary yellow mucous stool. The liver was pale, reddish brown and on section showed areas of cloudy swelling. The gall bladder, spleen and pancreas showed no abnormal changes. On section, the kidneys, which were of normal size, showed pale brown cortex and medulla. The ureters, bladder and genitalia were not unusual.

Gross Diagnosis. 1. Bronchopneumonia, right upper lobe. 2. Fibrinopurulent pericarditis. 3. Right purulent otitis media. 4. Enteritis. 5. Malnutrition and dehydration.

Otitis media should not have been overlooked but a fibrinopurulent pericarditis is rather difficult to diagnose clinically and hence is easily missed. Without autopsy this latter pathological lesion, as well as the bronchopneumonia, would have remained undetected.

Case 4. M—, colored female, age 4 days, admitted November 16, 1937.

Admission Note. Premature infant weighing 2 pounds 14 ounces. Condition fair. No abnormalities. Infant gets attacks of cyanosis; temperature normal. Poorly nourished; markedly jaundiced. Head: Ears, nose and throat are negative. Chest and heart: Negative. Lungs: Moist râles bilaterally, particularly on left side. Percussion of left lung anteriorly and posteriorly, dull. Breath sounds cannot be heard distinctly over left side; somewhat diminished in intensity on right side. Abdomen: Negative; liver not palpable. Impression: Prematurity, congenital atelectasis.

Clinical Course. November 19, 1937. Condition of baby very poor, 100.2° F. The tissues are very hard and extremities are stiff. Râles heard in chest; respiration very labored. Infant not taking feedings well and jaundice is increased. Intramuscular whole blood, 10 cc. given into each buttock. Child has been vomiting old blood last two days. Prognosis very poor.

November 20, 1937. Patient pronounced dead at 6:10 A.M. Final Diagnosis: Prematurity, congenital atelectasis.

Autopsy. The body was that of a poorly nourished, poorly developed, colored female infant. The skin and conjunctivae were markedly jaundiced. The skin presented moreover the usual post mortem lividities.

Head: The scalp was incised and avulsed. The calvarium was opened in the manner usual in infants. The meninges were clear and glistening and moderately injected. There were no hemorrhages. The brain was removed and the falc cerebri and tentorium inspected and proved to be intact. The middle and inner ear and mastoid processes were clear of infection.

Body: The body was opened through a "Y" incision and the organs removed en masse.

Thorax: On opening the thorax the thymus gland was very small. The lungs were retracted, deep red, and there was no fluid in the pleural cavity. The lungs were firm and had no crepitation on palpation. Incised, they exuded deep red blood and nowhere bubbles. Both lungs were attached with a short piece of trachea and sank in water. On section the lung parenchyma was deep red and congested. There was some reddish dirty serous fluid in the trachea and large bronchi. The mucus was slightly thickened. The individual lobes also sank in water.

Heart: The heart was of normal size. There was no fluid in

the pericardial sac. The endocardium was normal and the foramen ovale was patent. The myocardium was deep brownish-red and flabby. The aorta was normal.

Abdomen: On opening the abdomen some very hemorrhagic fluid escaped from the incision. There seemed to be about 40 cc. of this fluid.

Liver: The liver was slightly smaller than normal in size and presented many subcapsular hemorrhages. On the undersurface, near the external border, was a fissure indicative of rupture of the organ. On section, the external portion of the liver was of normal consistency, except where there were subcapsular hemorrhages. There was maceration of the liver tissue in several spots.

Spleen: The spleen was normal in size, deep red coloration and on section appeared injected.

Stomach and esophagus: Appeared normal. There was no hemorrhagic matter in the stomach. The intestines were normal throughout their length.

Kidneys: The kidneys were normal in external aspect. On section the right kidney was normal, the left kidney presented a rounded mass about the size of a split pea in its central portion.

Genito-urinary tract: The ureters were normal as was the bladder.

Adrenals: There were no adrenals in their normal place and no apparent adrenals were found.

Reproductive organs: The uterus and vagina were normal as were the tubes and ovaries.

Extremities: Presented no anomaly. The epiphyseal points of ossification in the femur were absent.

Gross Pathological Diagnosis. 1. Rupture of the liver and peritoneal hemorrhage. 2. Massive atelectasis. 3. Atrophy of the thymus. 4. Absence of adrenals. 5. Tumor of the left kidney.

One has but to glance at the pathologist's report to appreciate the futility and inadequacy of a clinical study in diagnosing a tumor of a kidney and absence of the adrenal glands. Without an autopsy such pathologic lesions are practically impossible to diagnose. To state as vague and nonspecific a diagnosis as prematurity as the cause of this death is most incomplete to say the least.

Case 5. E.M—, colored male, admitted July 24, 1939.

Temperature on admission 96° F.: weight 4 pounds 9¾ ounces. Color good, breathing with a little difficulty.

Clinical Course. Baby ate well, no vomiting; did not gain well; was apparently doing well until August 1, 1939. At 11:00 P.M. condition of infant seemed weak.

August 2. Child became restless, cyanotic, no fever.

August 3. Cyanotic at intervals. Cry weak. Respirations irregular. Baby died 9:00 A.M.

Clinical Diagnosis. Prematurity.

Autopsy Diagnosis. Lung (right) multiple abscesses.

Multiple abscesses of the lung are not easily found clinically, even with the help of the x-ray. Of course, x-ray may reveal lung pathology and thus compel a diagnosis other than prematurity as the cause of death.

Case 6. N—, colored male, born August 25, 1939. General condition very poor. Birth weight 2 pounds 5½ ounces.

Progress Record. Small premature infant, cyanosis of extremities, respiration satisfactory.

August 25, 1939, 7:30 P.M. Cyanosis of legs marked; slight bleeding from mouth. Hemorrhages into skin of abdomen and thighs. History of placenta previa in mother; bag and breech extraction of 28 week-old fetus. Condition fair, faint cry present. No respiratory distress. Ten cc. of blood intramuscularly, half into each buttock.

August 26, 1939. Patient ceased to breathe.

Clinical Diagnosis. Prematurity.

Autopsy Diagnosis. Bilateral adrenal hemorrhage, prematurity.

It is rather difficult to make a diagnosis of adrenal hemorrhage. Without the autopsy this finding would have been missed. Prematurity on the death certificate would have been an incomplete diagnosis.

The value of autopsy studies is evident. Fewer deaths will be laid to prematurity. In the light of the cases just described, the clinician must admit the extreme inadequacy of a clinical examination alone in making a correct diagnosis of the majority of premature infants who die. In fact, we have autopsied many a premature infant and found no gross or microscopic lesion to account for death. On the other hand, we realize how handicapped

is one who attempts a clinical diagnosis in a case of slight tentorial tear with hemorrhage or adrenal hemorrhage or ruptured liver or absent adrenals, especially in an infant under 1,000 grams. To sign the death certificate of such babies "cause of death prematurity" is certainly misleading. Mortality statistics based on clinical diagnosis alone are not dependable.

We have found many premature infant deaths ascribed to atelectasis. There are few such infants who do not have some degree of atelectasis. It is most difficult, even at autopsy, to say how far the atelectasis was the cause of the death. My experience dictates that atelectasis as the cause of death, especially in infants weighing 1,200 grams or more, is quite infrequent. Before giving atelectasis as the cause of death, I strongly advise the clinician to seek other possible causes.

CARE AT DELIVERY

A. *Cooperation between Pediatrician and Obstetrician.* The pediatrician should be notified, in advance, if a premature infant is expected. A specially trained nurse or a pediatrician should be present at the birth of a premature infant. Our resident and house pediatrician alternate each month in assuming charge of the newborn service. They are thus on hand all day. They are prepared to remove mucus from the air passages, using sterile gauze, a soft rubber ear syringe or a soft rubber catheter. The end of the catheter may be attached to a Murphy drip glass, a piece of rubber tubing being connected with the other end. If necessary, they administer a mixture of 95 per cent. oxygen and 5 per cent. carbon dioxide, or oxygen alone.

B. *Need for Warmth.* The temperature in the delivery room should be 70° F. to 75° F. It is also advisable to have a heat lamp directed toward the perineum and the infant during the delivery.

C. *General Procedure.* 1. After the ligation of the cord, the infant should be wrapped in a warm blanket, put into a heated bed (98° F.) in the Trendelenburg position (the head and shoulders lower than the rest of the body). This position prevents the passing of mucus into the lungs where it may produce atelectasis or pneumonia.

2. Three drops of a 25 per cent. mild silver protein solution or

one drop of a one per cent. silver nitrate solution should be instilled into the eyes as a safeguard against ophthalmia neonatorum.

3. It is wise not to move the premature infant for at least an hour after birth, unless treatment is necessary. At the end of the hour the baby should be taken to the nursery reserved for premature infants.

D. *Modes of Resuscitation.* 1. We have already suggested the use of sterile gauze or catheter to remove mucus from the nose or throat in case of respiratory embarrassment in the premature infant. Mucus in the air passages may be one cause for cyanosis.

2. We have also advised a mixture of 95 per cent. oxygen and 5 per cent. carbon dioxide for cyanosis and asphyxia.

3. In some cases, where cardiovascular stimulation is needed, 1 to 3 drops of adrenalin chloride solution (1:1000), given intramuscularly, is of value.

4. Premature infants should be handled as little as possible. We deplore the use of chest-pressing; alternate hot and cold water baths; jack knifing; holding infants upside down; spanking or rubbing spines or similar procedures. They are decidedly harmful. Intracranial hemorrhage is encouraged when these infants are spanked, receive spinal massages or are held upside down, while being slapped over the soles of the feet.

5. Premature infants should be kept warm. They should be kept in the Trendelenburg position for an hour after delivery following which the Fowler position is suggested.

E. *Examining the Premature Infant.* (What to look for). 1. Congenital maldevelopments include: circulatory disturbances, including congenital heart; diaphragmatic hernia; exstrophy of bladder; absent or fibrotic bile ducts; stenotic or fibrotic portions of intestines; hydrocephalus; fragilitis ossium; clubfoot; contractures; oxycephaly; malformation of extremities or muscles; harelip; maldevelopment of scapulae; hernias; anomalies of anus; fibrotic lung; white pneumonia; massive lung collapse; sclerema neonatorum; hereditary ectodermal dysplasia; ichthyosis; mongolian idiocy; thyroid hypoplasia; spina bifida; encephalocele.

2. Birth injuries: Fracture of bones, including skull, clavicle, humerus; injuries to peripheral nerves, with facial paralysis and brachialplexus paralysis; injury to internal organs, especially rup-

tured liver, intracranial and adrenal hemorrhage, injury to spinal cord; injuries to eye, ear, nose.

3. Diseases or manifestations of disease: Sepsis; influenza; pneumonia; atelectasis; cyanosis; asphyxia; jaundice; paresis and paralysis; poor cry; failure to suck; stupor; anemia; icterus gravis neonatorum.

MAINTAINING BODY HEAT

A. *Correct Temperature and Humidity of the Nursery.* The temperature of the premature infant should be maintained at about 99.6° F. (37° C.). Higher or subnormal temperatures will mean a failure to gain in weight and a greater susceptibility to infection[18]. To insure this proper body temperature, we have found it necessary to maintain the nursery at a temperature of 80° F. Incubators are unnecessary if the room temperature can be continually maintained at 80° F. If an incubator is used, especially for the infant under 1,500 grams, the temperature in the incubator should be 80° F. to 85° F.

It is important to determine the temperature of the infant and of the incubator about every four to six hours because too high or too low a temperature will definitely harm the infant.

The humidity of the room or incubator plays a very important part in the progress of the premature infant[18]. A relative humidity of 45 to 55 per cent. is advisable. Some suggest a relative humidity between 55 to 65 per cent. for the nursery[18]. The lower the room temperature the higher the humidity requirements[18].

In the home, attempts at proper regulations of humidity in the infant's room are made by using two or three flat pans containing water. These pans may be attached to the top of the radiator, or placed on a table or shelf. Inexpensive humidity gauges are now on the market.

Premature infants are not ready to leave the incubator or a specially heated room until they are able to maintain their own temperature without external heat. When the infant reaches 5 pounds 8 ounces, if it is in good health, it may be taken from the nursery maintained at 80° F. and transferred to a nursery for premature infants kept at 72° F. This is excellent preparation for sending the hospital-confined premature infant home.

B. *Air-Conditioned Nursery.* An air-conditioned nursery pro-

vides an ideal environment. The temperature and humidity are mechanically controlled and furnish an atmosphere highly suitable for the proper growth and development of the premature infant. Sixty-five per cent. humidity is considered best in this nursery[18]. The temperature may range from 75° F. to 100° F. This wide variation in temperature is permissible and favorable and depends

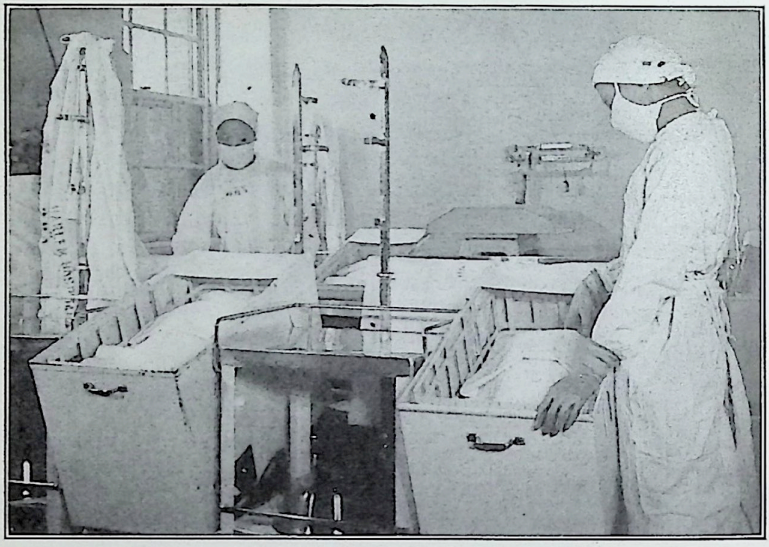

Fig. 1. A corner of the premature infant nursery kept at 80° F. Note the electrically heated bassinette; incubators kept in Fowler position. Each infant is supplied with its own table containing all necessary utensils. The infant does not leave this unit until it is "graduated" to the second unit (Fig. 2) which is kept at 72° F. for heavier infants.

upon the weight and vitality of the infant. However, wide fluctuations are not desirable once the infant has become accustomed to a certain temperature.

Automatically controlled temperatures are particularly favorable to the premature infant under 1,500 grams. With a lowering of the temperature in the ordinary room in which such an infant

is kept, it soon shows a subnormal temperature with a corresponding loss of weight. The reverse is also true. Infants kept near heated radiators often develop hyperpyrexia and fail to gain.

An air-conditioned unit reduces the incidence of infection and the mortality among premature infants. In addition, the initial weight loss and the time required for regaining it are favorably

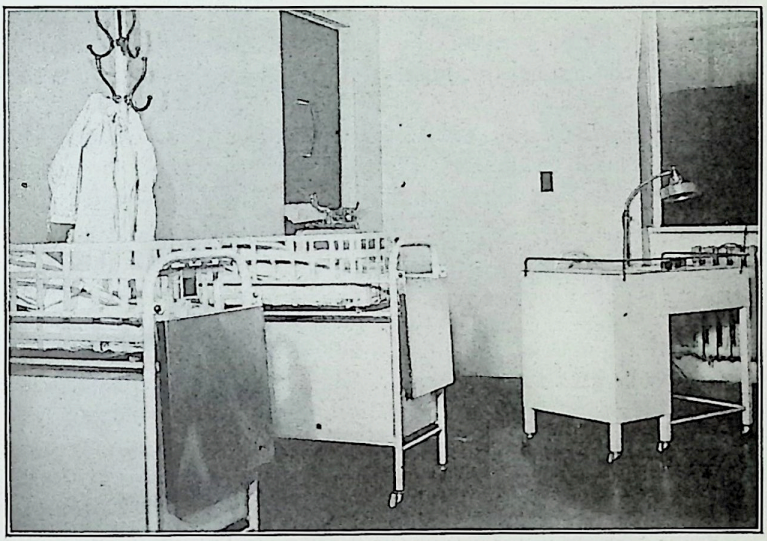

Fig. 2. Section of the premature infant unit for infants weighing 5½ pounds or more showing isolation bassinette. Some of these infants kept here were "graduated" from the premature infant unit kept at 80° F. and reserved for the smaller infants (Fig. 1). The above unit is kept at a temperature of 72° F. The infants are sent home from this unit.

affected by the air-conditioned unit[18]. The initial weight loss was 12.4 per cent. in a non-air-conditioned nursery and only 6.0 per cent. in an air-conditioned nursery with high humidity. Infants in a non-conditioned nursery required an average of 26½ days to regain their birth weight while those in the air-conditioned nursery (with high humidity) regained their birth weight in 15½ days. The mortality rate for all age-and-weight groups in the air-

conditioned unit was 7 per cent. as compared with 28.9 per cent. in the unconditioned nursery.

The amount of humidity had a direct bearing on the incidence of infection as well as on the mortality rate. Infection accounted for a death rate of 9.7 per cent. in an air-conditioned unit where the humidity was low. There were no deaths, however, in the air-conditioned unit with high humidity. In the unconditioned nursery 26½ per cent. of the infants died.

The unit may be partitioned allowing a cubicle for each infant. A cabinet, containing the necessary equipment is kept in each cubicle.

C. *Value of the Incubator.* If a temperature of 75° F. to 80° F. can be maintained in the nursery or home and practical provisions made for humidity control, an incubator is unnecessary for the larger premature infants. For the smaller infants of 1,500 grams or less, an incubator is advisable. We recommend a temperature of 80° F. for this incubator.

The simplest type of incubator supplies heat and attempts to maintain the infant's temperature at 98.6° F. to 99.6° F. A wooden box may be used as an incubator. This improvised incubator has two incandescent bulbs controlled by a switch on the outside. A canvas cover is used for the open top. For maximum heat, we use both bulbs and partly cover the top. In many cases, only one light is needed. Often, we have used one or two hot water bags instead of electric lights.

This incubator contains a sterilized blanket, covered by a sterile diaper, upon which the infant lies. The head and shoulders are slightly elevated by placing a few diapers or a folded blanket under the first blanket. We use no mattress, since blankets are more easily sterilized. Flat pans of water, placed in the room, supply the extra humidity.

Should oxygen be needed, as in the case of cyanosis, we use the funnel or forked metal nasal tip. These are in turn connected by means of a rubber tube to a tank of oxygen (see Fig. 9).

Each infant is dressed, diapered or treated on a shelf attached to this box. A thermometer is suspended inside the incubator to indicate temperature.

A type of incubator which has served us well is shown in Figs.

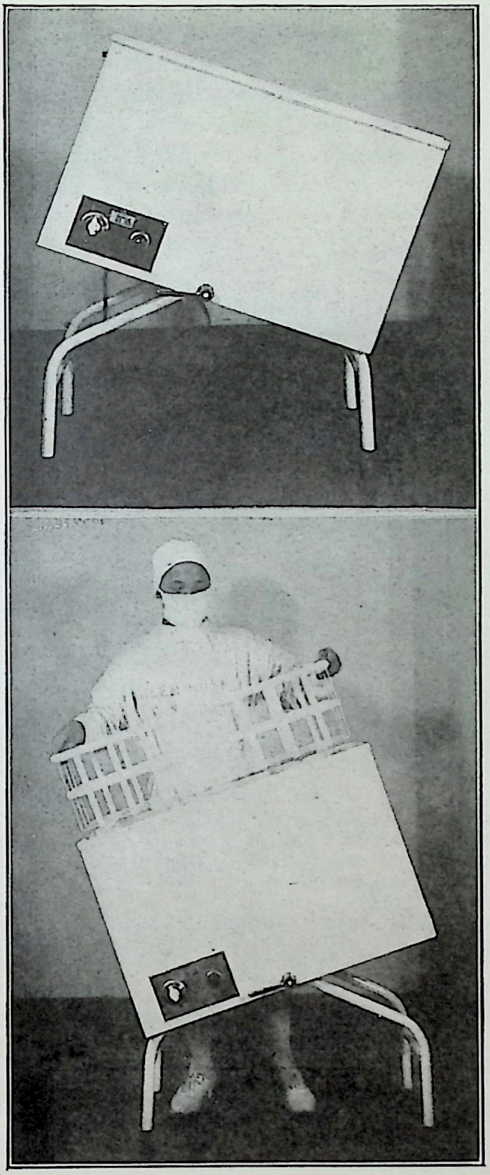

Fig. 3. (Above). Electrically heated bassinette. Note heat regulating mechanism at left lower side. This enameled steel box is tilted into the Fowler position. The bassinette is set into this box.
Fig. 4. (Below). Setting the bassinette into the electrically heated enameled steel box.

1, 3, 4. It is essentially an electrically heated bassinette. Made of enameled steel, it is 29 inches long, 20 inches high and 16¼ inches wide. It is fitted with six incandescent bulbs (space heaters are now used instead of bulbs) operated by a switch at the side. A piece of asbestos, containing 18 one-inch holes, separate those bulbs from a bassinette placed above them. This bassinette is 23¾ inches long, 14¼ inches wide and 10 inches deep. The incubator can be tilted in order to maintain a Fowler position for the infant. No provisions are made for humidity or oxygen needs.

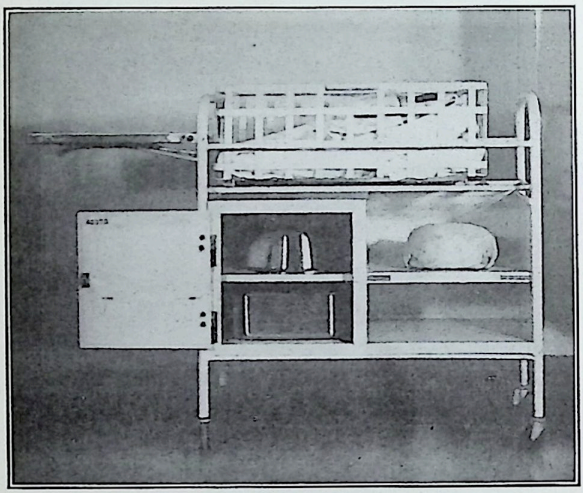

Fig. 5.. Complete unit used for the older premature infants whose heat regulating mechanisms have become fairly well established. Hot water bottles may be used, if needed. Note the autoclaved bundle on the right side (upper shelf). It contains sufficient layout for a bath and morning toilet. There is also a shelf, seen on left side, on level with bassinette, for bathing, dressing and treating the baby. The infant is not removed from this unit until it is "graduated".

The Hess water-jacketed infant bed, which has proved satisfactory to us, is heated by electricity. An electric heating plate heats the water jacket which surrounds a removable crib set into the metal bed. Oxygen can be passed into this incubator and a partial cover placed over the top of the crib. A thermometer indi-

cates the temperature of the incubator. The incubator cannot be overheated. This incubator is valuable for the tiny premature infants weighing about 1,500 grams.

Recently, another type of incubator has been described. It makes provisions for maintaining heat and humidity, provides an oxygen concentration of 25 to 46 per cent. and decreases bacterial contamination of the air[116]. The bacterial count in the incubator is less than that in the ward. A positive pressure is maintained in the incubator, thus allowing air to pass from it to the ward and not the other way. While in the ward, the incubator showed a bacterial count of 400 to 1,100 colonies. The same incubator placed in a nursery was found to have a bacterial count of nine colonies and the nursery itself 90 colonies[134].

We have treated several hundred premature infants over a period of years. We have had occasion to try at least five types of incubators. We have also seen many other incubators and an air-conditioned nursery in operation. The results in all these incubators have been good. We are inclined, however, to emphasize the need for a simple type of incubator such as the one illustrated. Elaborate incubators with mechanical and electrical contrivances are unnecessary. Besides, if the room is kept warm enough (80° F.) no incubator is needed (see Figs. 2 and 5).

D. *Provisions for Care in the Home.* Where a special incubator is impossible, the use of hot water bags in the ordinary crib is satisfactory. These hot water bags must not touch the infant; must never be full, and should be refilled with warm water at regular intervals. Body heat 98.6° F. to 99.6° F. can also be maintained by the use of a few incandescent bulbs properly placed in the bottom of the crib with a strip of asbestos separating them from the infant.

Every effort should be made to place the premature infant in a room by itself for at least the first year, at a temperature of 75° F. to 80° F. This precaution will obviate the chances for infection caused by several members of the family sleeping in the same room.

DRESSING THE PREMATURE INFANT

In an air-conditioned unit, or in an institution or at home, where proper humidity and temperature are maintained, the prob-

lem of dress is not difficult. The average premature infant requires a flannel or bird's-eye diaper; a cotton, flannel or 10 per cent. silk and wool shirt; a flannel or knit abdominal binder; a flannel wrapper with a drawstring at the bottom, and wristlength sleeves; a cotton receiving blanket, wrapped loosely around the thighs and legs; stockings; and one or two woolen blankets. After the cord falls off, the binder is no longer necessary.

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