PREMATURE AND CONGENITALLY DISEASED INFANTS

BY

JULIUS H. HESS, M.D.

PROFESSOR AND HEAD OF THE DIVISION OF PEDIATRICS, UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE; CHIEF OF PEDIATRIC STAFF, COOK COUNTY HOSPITAL; ATTENDING PEDIATRICIAN TO COOK COUNTY, MICHAEL REESE AND ENGLEWOOD HOSPITALS; CONSULTING PEDIATRICIAN, MUNICIPAL CONTAGIOUS HOSPITAL AND WINFIELD TUBERCULOSIS SANITARIUM, CHICAGO; MEMBER OF ADVISORY BOARD CHILDREN'S BUREAU, DEPARTMENT OF LABOR, WASHINGTON, D. C.

ILLUSTRATED WITH 189 ENGRAVINGS

LEA & FEBIGER
PHILADELPHIA AND NEW YORK
1922

PRINTED IN U. S. A.

In the name of

CLARA MERRIFIELD HESS

This book is affectionately dedicated to the most helpless of the human race

THE INFANT BORN PREMATURELY

particularly needful of aid in its struggle for existence

PREFACE.

In the absence of any definite collection of material on the care of prematurely born human infants, I have attempted to compile information taken from many sources. The foundation of this work with premature infants was laid in the researches of the French obstetricians and was adopted and further popularized by English and German physicians.

The growing importance of the subject is indicated by the increase in premature births during recent years revealed by vital statistics. Of 2806 deaths of infants occurring in Chicago during one year, 739 deaths in the first month of life were due to prematurity. Of 860 who died during the first twenty-four hours, 399 deaths were due to premature birth, while of 1700 who died during the first week of life, 588 deaths were due to premature birth.

A study of the etiological factors predisposing to premature birth emphasizes the necessity for proper prenatal care of the mother, thus eliminating a large group of premature births precipitated by her overwork and her anxiety, or by trauma, as well as by improper hygiene and insufficient and improperly balanced diet. The general experience of workers who have interested themselves in prenatal care of the mother in its larger aspect proves that careful following of scientific instructions inevitably decreases the number of premature births coming under observation.

Many diseases, such as syphilis and nephritis, which are direct causes of premature birth, are amenable to treatment which will prolong the intra-uterine life to the natural period, or at least to the point where prenatal development is compatible with post-natal existence. Proper preparation for the care of these infants will justify the induction of labor prior to full-term when pathologic conditions exist; moreover this procedure may be undertaken at a much earlier time than when proper facilities are not available.

In the United States the care of premature infants has not received the general attention of the medical profession which it merits. Facilities for the care of such infants are lacking, first, because special obstetrical hospitals in most instances decline outside cases, and, second, because comparatively few general hospitals are properly organized to undertake the special care required. Proper handling of these infants demands a thorough knowledge of their immediate needs. The first intimation of coming labor must be met by preparation for the infant's reception in order to avoid the dangerous period of exposure immediately after birth which is a primary cause of the high mortality. If a sudden lowering of temperature produces fatality in some cases in full-term infants, how much more likely it is to produce fatality in an immature infant, whose organs are not completely developed, who is lacking in the protective covering of body fat possessed by the mature infant, whose vitality is low and whose resistance is at a minimum. Coming from an equalized temperature of unvarying degree it is precipitated into alien surroundings, deprived of its usual nutrition and subjected to handling which, however tender, is still a shock to its delicate external and internal structures.

As a part of the great movement toward conserving and developing the individual to his highest point of health efficiency, as an important factor in national health, and as an effort directed toward the source of a considerable morbidity, the care of premature infants and the conservation of their flickering lives has a prominent place.

I desire to acknowledge my indebtedness to Dr. Martin Couney for his many helpful suggestions in the preparation of the material for this book.

J. H. H.

CHICAGO, 1922.

CONTENTS.

PART I.

ETIOLOGY, PHYSIOLOGY, PATHOLOGY.

PART II.

NURSING AND FEEDING CARE.

PART III.

GENERAL DISEASES.

PART IV.

THE OUTLOOK FOR THE PREMATURE.

LIST OF ILLUSTRATIONS.

INDEX OF TABLES.

PREMATURE AND CONGENITALLY DISEASED INFANTS.

PART I.

ETIOLOGY–PHYSIOLOGY–PATHOLOGY.

CHAPTER I.

DEFINITION.

THE term *premature*, in the precise meaning of the word, refers to those infants born before the end of the fortieth week of pregnancy, but in common usage it refers only to those infants who have undergone a gestation period of two hundred and sixty days or less, and so it may be understood that when the designation premature is used, it refers to those infants born three weeks or more before the usual termination of pregnancy.

There is another class of infants who may be considered in practically the same category as the prematures. These are the *weaklings*, infants born possibly at term, or nearly so, yet who have suffered more or less severely during their intra-uterine existence through factors which interfered with their nutrition and consequently their development. They are classed as *congenitally diseased* or *debilitated*.

In contrast to the prematures there are the full-term and mature infants. The full-term must be considered that one who is born at the completion of the normal period of two hundred eighty days of pregnancy. The mature infant is one possessed of all the faculties for extra-uterine existence and may be born before or at the expiration of normal gestation. Thus it may be seen that the functional and not the anatomical characteristics should decide maturity. While prematurity pertains to time and congenital disease or debility to function, the prematures do not need to be weaklings, whereas the full-terms may show evidence of congenital disease or debility.

The congenitally diseased are usually pale in appearance, thin, underweight, show a lack of cutaneous turgor, and have a low

2

reactive capacity, suckle and drink poorly and have a tendency to restlessness, abnormal abdominal distention and dyspeptic stools. Not infrequently this class of infants fails to gain weight in a normal manner and, therefore, often require several weeks to regain their birth weight. This indicates functional incapacity even in the absence of demonstrable organic disease. This lack of functional development varies greatly with the individuals.

With reference to this class Jaschke[1] remarks that we should admit that vital debility must be designated as a congenital functional deformity which manifests itself chiefly in a deficient resistance or very low tolerance to the conditions and variations of the extra-uterine life. The debilitated infants react on one hand with symptoms of disease toward physiological stimulus, and on the other hand their well-being is unfavorably influenced by the slightest degree of over- or understimulation.

Many premature infants, not only have been born before full-term, but also have their physical development retarded by intra-uterine disease and are below the average physical development for fetuses of a similar age.

It must be remembered that all infants born before the end of a normal term are born before the end of a full intra-uterine pregnancy, and consequently their organs are not fully developed. As a result they show certain definite body weaknesses, and a lack of resistance to the traumas of extra-uterine life. These are *immature* even though fully developed for their fetal age.

This, however, is only a *relative body weakness* in the absence of inherited constitutional debility and malformations.

It is also a fact that the younger the fetus when leaving the uterus, the greater are the difficulties to be overcome in the carrying out of the required body functions necessary to life and, therefore, the lower its vitality.

In a study of premature and congenitally debilitated infants at least two factors in the life history of the fetus must be considered:

1. The term of its intra-uterine life.
2. The state of its functional development at birth as evidenced by the presence or absence of inherited disease.

Congenital debility is dependent upon constitutional influences in the parents, and intercurrent disease during the term of pregnancy.

Notwithstanding the fact that both of the above factors must be given the most careful consideration, practically, in most instances, the influence of either factor on the extra-uterine life of the fetus in its early days cannot be definitely determined.

[1] Physiologie, Pflege und Ernährung des Neugeborenen. J. F. Bergman, 1917.

CHAPTER II.

CLASSIFICATION.

For practical clinical purposes the group of infants comprising the premature and congenitally debilitated may be classified as follows:

1. Premature infants, with no pathological changes.
2. Premature infants, with pathological changes, due to:

(a) Constitutional disease and chronic infections in the parents.

(b) Maternal factors influencing the fetal nutrition, such as overwork, undernourishment and acute illnesses during pregnancy.

(c) Local conditions in the mother.

(d) Multiple pregnancies.

(e) Constitutional defects and congenital malformations in the fetus.

(f) Infants born to parents late in life.

3. Full-term infants with pathological changes due to the same causes as those enumerated under 2.

ETIOLOGY.

The occurrence of premature birth depends upon many causes, which may be divided into those resulting in the expulsion of a healthy premature, and those which have a damaging effect upon the product of conception. In the first class may be included various injuries, falls, heavy lifting, overwork or other physical exhaustion, sudden emotional disturbances and premature rupture of the membranes, either accidental or intentional, occurring in those conditions whose existence does not affect the nutrition of the ovum, as in pelvic and spinal deformity in the mother, placenta previa, etc.

Conditions in the mother requiring operative procedure not involving the uterine cavity frequently result in premature labor either through shock and trauma, resulting from operations, as for ovarian conditions and uterine fibroids, or infection may be an added danger in cholecystitis, cholelithiasis, appendicitis, ileus and renal operations.

The cases which fall within the second category all react to a greater or lesser degree upon the fetus, some producing only momentary weakness, as the milder acute infections, others causing a weakened physical condition as a result of their long-continued action upon the nutrition and development of the fetus.

FIG. 1.—Case of congenital goiter.

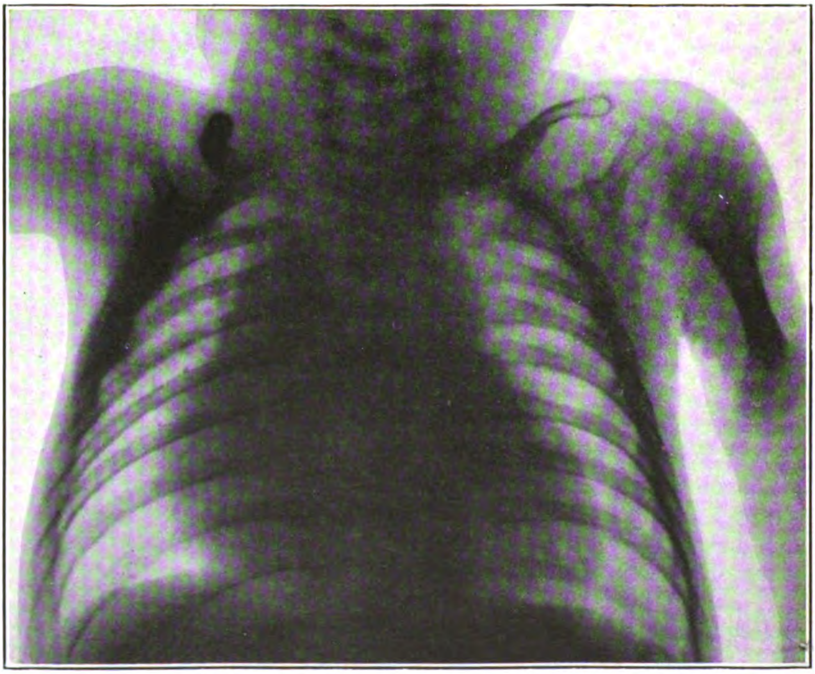


FIG. 2.—Case of congenital thymus (atrophy of gland following two exposures to roentgen ray).

FIG. 3.—Mongolian idiot.

FIG. 4.—Chondrodystrophia.

The most frequent causes are the chronic infections. *Syphilis* plays the leading rôle, and is estimated as being a factor in from 50 to 80 per cent of all cases of repeated premature expulsion of the fetus, while Lesage and Kouriansky[1] state that syphilis is a factor in the causation of congenital debility of the full-term in 25 to 35 per cent. If the luetic infection is recent, abortion is the rule; but as the infection becomes older, the succeeding pregnancies terminate

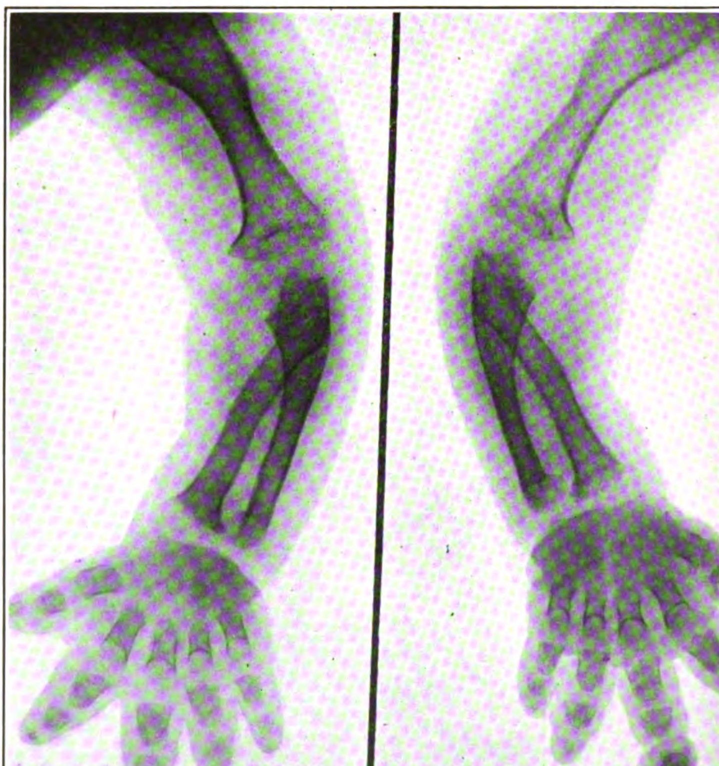


FIG. 5.—Chondrodystrophia.

later and later until a living child with or without manifestations of the disease is born, usually prematurely.

Chronic *nephritis* is one of the most frequent causes of spontaneous premature labor, and the offspring of these mothers are often puny, due, either to the systemic effect on the mother, or resulting from impaired nutrition of the fetus due to placental

[1] Congenital Debility and Atrophy, Nourrisson, Paris, July, 1919, No. 4, **7**, 193.

hemorrhages and infarcts. Nephritis in the mother is also one of the most frequent indications for the induction of premature labor.

Fig. 6.—Cretinism.

Pulmonary *tuberculosis* is less frequently the cause of premature labor, but the children, even at full-term, are often small and weak. Tuberculosis of other organs and tissues influences the fetus in proportion to the nutritional effect upon the mother or, again when involving the vertebral column or hip-joints may by their

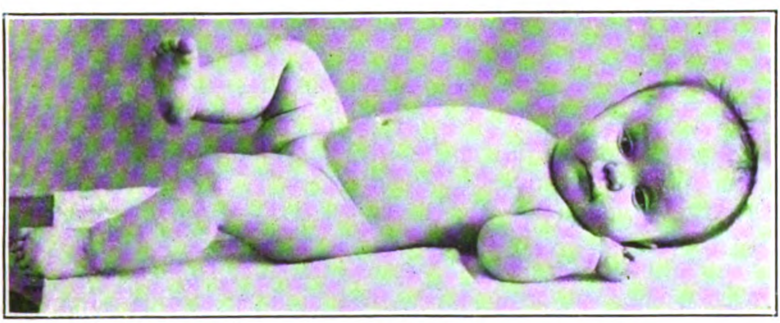


Fig. 7.—Dyspituitarism.

resulting deformities require premature induction of labor. Congenital tuberculosis is very rare, but does occur. In the majority of cases, not the disease *per se*, but the predisposition is inherited.

Premature birth occurs in 30 or 35 per cent of the cases of broken compensation in *heart disease*. The premature infants are, in these cases, often imperfectly nourished as a result of the poor aëration of the mother's blood. *Exophthalmic goiter* is occasionally the cause of premature emptying of the uterus. If chronic dyspnea exists, as a result of laryngeal or tracheal stenosis from pressure, the development of the fetus will necessarily be retarded.

FIG. 8.—Case of Siamese twins. Thoracopagus tetrabrachius tetrapus. (From the service of Dr. Ludwig Simon, Michael Reese Hospital, Chicago.)

Any of the *acute infectious diseases* may be responsible for the termination of pregnancy before the end of term. Pneumonia, influenza, typhoid fever, malaria, diphtheria, scarlet fever, measles, small-pox, Asiatic cholera and bubonic plague—all have a deleterious effect on the continuance of pregnancy. Premature labor is very common in pneumonia and influenza, being more frequent in late pregnancy.

Of *local conditions*, diseases of the decidua or endometrium, gonorrheal infection and malpositions of the uterus frequently result in premature labor, but usually before the fetus is viable. *Anomalous positions* of the fetus in the uterus may be responsible for the premature expulsion of the uterine contents.

The occurrence of *multiple pregnancy* is a fruitful source of premature labor. About 70 per cent of twin pregnancies terminate prematurely and the length of practically all triplet and quadruplet gestations is considerably shortened in most instances due to lack of room in the uterine cavity. Miller's[1] figures are slightly smaller. He states that of 3380 plural births, 2040, or 60 per cent, were premature, and had a body weight of less than 2500 gm., and a length under 45 cm. Even when mature, twins are usually small and of low body weight. This, of course, is even more true of triple pregnancies, the reserve strength possessed by the mother not being sufficient to allow three fetuses to reach their normal development. Again in the presence of several fetuses the growth may proceed unequally so that one may be born with unimpaired vitality, and the others with greatly diminished strength (Fig. 9).

Fig. 9.—Triplets.

Faulty nutrition of the fetus, such as is found in maternal overwork or from lack of sufficient food, as well as that due to wasting diseases, the blood dyscrasias (pernicious anemia and leukemia) and *intoxication* from alcohol (acute and chronic), phosphorus, arsenic, mercury or lead may—any one of them—cause either an early termination of pregnancy or so serious a lowering of nutrition of the fetus that the vitality at birth may be greatly impaired. In addition, congenital malformations in the fetus sometimes bring on premature birth. In *diabetes* prematurity is not infrequent, and the infants may show glycosuria.

Infants born to parents late in life are often born prematurely, perhaps because of the factor of undernourishment. This is also

[1] Peculiarities of the Disease of the Premature Infant, Jahrb. f. Khlk., 1886, **25**, 129.

the case in prematures born of women who have had numerous pregnancies, at short intervals.

Finally, habitual miscarriage, without evident cause, resulting in the interruption of successive pregnancies, not infrequently at about the same stage, is not rare. The author has records of several such women without a history of syphilis or other constitutional disease, and in whom uterine deformity is not demonstrable.

The *frequency* of premature labors varies greatly in different clinics. Rommel[1] quotes the following figures from various clinics, noting the number of infants under 2500 gm. in weight and below 45 cm. in length.

Miller	5.0 per cent	Orphan Asylum . . .	Moscow
Von Winckel . .	13.3 "	Maternity	Munich
Fehling . . .	25.0 "	Maternity	Halle
Budin	10.7 "	Clinique Tarnier . .	Paris
Pinard . . .	15.4 "	" Baudelocque .	Paris

It is stated that the percentage of premature births is greater during the colder months of the year.

[1] Quoted from Pfaundler and Schlossman Handb. f. Kinderh., Leipzig, 1901.

CHAPTER III.

PHYSIOLOGY.

CLINICAL FEATURES.

THE appearance and characteristics of the *healthy premature* child vary with the fetal age at the time of birth. With a lengthening of the period of gestation, the distinctive characteristics of the fetus become less and less marked until it becomes impossible to differentiate the slightly premature from the full-term infant. All the distinguishing features of the premature may also be found in the *congenitally diseased* full-term infants, and as there may be all degrees of prematurity, so we also find all stages of development between the extremes of functional and anatomical inferiority on the one hand and the normal constitution on the other. Both the premature and the debilitated infant may exhibit the following features in varying degrees.

The body is usually small and puny, though in some instances the infant may be of a considerable size, yet with a very imperfect development of its internal organs.

The weight is low, varying from amounts approximating 700 gm. ($1\frac{1}{2}$ lbs.) to 2500 gm. ($5\frac{1}{2}$ lbs.) in the viable. The latter figure may be exceeded in infants nearing maturity, and by some of the full-term weaklings, but will serve as a fair maximum.

The skin is soft and usually of a vivid red color. The epidermis is thin and the bloodvessels are easily seen.

The skin frequently hangs in folds. The adipose tissue is scant, the features are angular and the face looks old.

Lanugo is plentiful, especially upon the extensor surfaces of the extremities.

The skull is round or ovoid in contradistinction to the usually markedly dolichocephalic skull of the full-term new-born. The fontanelles are large and the sutures prominent.

The nose exhibits many small comedones. The ears are soft and small and hug the skull.

The nails have scarcely reached the ends of the fingers even in the larger infants, while in the smaller they may be very poorly developed.

The cry is feeble, monotonous and whining.

The infant lies in a deep sleep, and must be aroused for its feedings. Efforts at suction are weak or absent. All movements are slow, functions are sluggish and the child shows a remarkable degree of muscular inertia.

The temperature has a very decided tendency to remain below normal and is inclined to be irregular in character.

The urine is usually scanty.

The bowels are sluggish and constipation is the rule.

Early and intense jaundice is common.

These are the principal findings which are to be seen on superficial examination. A more critical review of these various characteristics follows. It must be remembered that any of these symptoms may vary in different individuals of the same age, depending upon the cause of prematurity, and upon the condition of health present in both the mother and the child. With increasing age, the characteristics become less marked, until the picture eventually merges into that of the full-term infant.

The determination of the exact *age of the infant prematurely born* is a matter of considerable difficulty. The information furnished by the mother as to the time of her last menstrual period, or as to the time when life was first felt, gives an entirely insufficient approximation of the probable date of confinement, and errors of a month or even more are not rare. In institutions for foundlings all data is, as a rule, absent, and other methods for determining the infant's fetal age must be relied upon. The weight of the infant is of uncertain value also, as an infant of 1500 gm. weight may be the product of a pregnancy of seven months in a healthy woman, while one of the same or less weight may be the eighth-month offspring of an albuminuric or syphilitic mother. The body measurements also vary materially with the individual. The degree of development of the osseous system is of great value in determining the anatomical development, and indirectly the condition of the bones acts as a guide to physiological development, even though they do not give absolute data as to age. Body measurements and osseous development are fully discussed later under their respective headings.

More important than a determination of the approximate term of pregnancy or a consideration of the size of the infant, at least in those infants born but a few weeks before the natural termination of the period, is a history of syphilis, tuberculosis, traumata, or other causes, operating in the mother and responsible for the early emptying of the uterus.

His[1] gives the following description of the developmental features of the fetus at varying ages:

[1] Anatomie menschlicher Embryonen, 11, Leipzig, 1882.

Fifth Lunar Month (112 to 140 days).—Head about the size of hen's egg; the skin is red and shows some fat deposit. The scalp shows indications of hair, the body is covered with lanugo, the nails can be distinguished, the eyelids remain closed. The fetus rarely lives over five to ten minutes, making feeble attempts at respiration. The heart-beats may be strong.

Sixth Lunar Month (140 to 168 days).—The body shows increased fat deposits, though still lean, the skin being wrinkled. The eyelids are separated and eyebrows and -lashes may be seen. The infant may live for several hours. The respiratory and digestive organs are underdeveloped, respirations being superficial and digestion practically impossible.

Seventh Lunar Month (168 to 196 days).—The infant has an aged appearance but the wrinkles are filling out. The eyes are open. The cry is a weak whine or grunt. Few of these infants born during the twenty-fifth and twenty-sixth weeks survive, and when they do are usually hydrocephalic, paralytic and dwarfed. Those of the twenty-seventh and twenty-eighth weeks are far more promising.

Eighth Lunar Month (196 to 224 days).—The infant is beginning to fill out, many of the wrinkles having disappeared. The bones of the head are soft and flexible. Ossification begins in the lower epiphysis of the femur. The testicles are often in the scrotum. The cry is stronger, though it may still be very weak. Under proper conditions many of these infants survive.

Ninth Lunar Month (224 to 252 days).—Panniculus adiposus develops. The wrinkles smooth out and the limbs become rounded. The lanugo begins to disappear, and the nails are at the tips of the fingers. Respiratory, circulatory and digestive organs are capable of carrying on the body functions.

Tenth Lunar Month (252 to 280 days).—The general body functions improve during this month and at the end of this period development is complete.

BODY WEIGHT AND OTHER MEASUREMENTS.

Infants born at full-term weigh on the average from 3000 to 3500 gm. The dividing line between the premature and full-term infant has been generally placed at 2500 gm. If under that figure they may be considered below par as far as concerns the strength and ability to overcome the forces which assail them on every hand. The weight of the premature varies even within greater limits than that of the full-term infant, and as one may see a child below 2500 gm., so also there are prematures with a weight above this limit.

The weight depends upon the cause of the premature birth and upon the age of the child. Those born of mothers afflicted with nephritis, tuberculosis, or other wasting diseases, and infants showing active syphilis, are usually considerably smaller than the same aged infants of healthy parents. Diseases and abnormal location of the placenta also restrict the growth of the fetus. The infant in placenta previa is often undersized, even when born at term. Multiparity may predispose to undersize.

His, in a comparison of the fetal weight and length with the age, made the following table:

	Weight.	Length.
16 to 20 weeks	250 to 280 gms.	17 to 26 cm.
20 " 24 "	645 " 1000 "	28 " 34 "
24 " 28 "	1000 " 1220 "	35 " 38 "
28 " 32 "	1220 " 1600 "	39 " 43 "
32 " 36 "	1600 " 2500 "	46 " 48 "
36 " 40 "	2500 " 3100 "	48 " 50 "

THE AVERAGE LENGTHS IN CENTIMETERS OF NORMAL FETUSES AS GIVEN BY DIFFERENT OBSERVERS.

Lunar months.	Mall.[2]	Von Winckel.[3]	De Lee.[4]	Lambertz.[5]	Ahlfeld.[6]	Schroeder.[7]
1st[1]	0.25	.....	0.75-0.9			
2d	0.55- 3.0	0.9-2.5	2.5			
3d	4.1 - 9.8	7-9	7-9	6-11		
4th	11.7 -18.0	10-17	10-17	11-17		
5th	19.8 -25.0	18-27	17-26	17-28		
6th	26.8 -31.5	28.34	28 34	26-37		
7th	33.1.-37.1	35-38	38 35	35-38	36-40	
8th	38.4 -42.5	40 43	43	38 42	40-43	41.3
9th	43.6 - 47.0	46-48	46-48	42-45	46 48	44.6
10th	48.4 -50	48-50	48 50	45-52	48-50	46.0

The weight and length as compared to the fetal age is shown in the following table from Oberwarth,[8] which gives the average length also:

Fetal age.	Weight.	Length.
26 weeks	330 to 1041 gms.	28.0 to 37.0 cm.
28 "	995 " 1408 "	36.3 " 37.5 "
30 "	797 " 1700 "	33.1 " 41.3 "
32 "	1868 " 1964 "	42.0 " 42.7 "
34 "	1286 " 2213 "	39.0 " 47.0 "
36 "	2424 " 2700 "	46.1 " 48.0 "

[1] The length for the first two months represents the measurement from the vertex to the buttocks; all the other measurements are from vertex to sole.
[2] Manual of Human Embryology, **1**, 196.
[3] Handbuch der Geburtshülfe, 1903, Bergman, Wiesbaden.
[4] The Principles and Practice of Obstetrics, Philadelphia: W. B. Saunders Co., 2d Ed., 1915.
[5] Development of the Human Skeleton during Fetal Life, Fortschr. a. d. Geb. d. Röntgenstrahlen, Suppl. I.
[6] Von Winckel's Handbuch der Geburtshülfe, I, No. 1, p. 290.
[7] Quoted from von Winckel's Handbuch der Geburtshülfe.
[8] Ergeb. d. inn. Med. u. Kinderh., 1911, **7**, 191.

These compare favorably with those given by Ahlfeld and Hecker.[1]

Fetal age.	Weight.	Length.
27 weeks	1140 gms.	36.3 cm.
29 "	1575 "	39.6 "
31 "	1975 "	42.7 "
33 "	2100 "	43.9 "
35 "	2750 "	47.3 "
37 "	2875 "	48.3 "

Potel and Hahn's[2] figures do not include the length.

Fetal age.	Weight.
27 weeks	995 to 1146 gms.
29 "	1540 " 1700 "
31 "	1881 " 1964 "
33 "	2150 " 2213 "
35 "	2400 " 2700 "

The following small group taken from my cases give the age of the fetus as computed from the date of the last menstruation. That this is an unreliable method may be recognized by noting the variation in figures in Cases 2, 3, 11, 13, 14 and 15. We therefore, place little reliance on the mother's estimate as to the date of conception.

	Fetal age, weeks.	Weight, gm.	Length, cm.	Diameters of head.				
				O. F.	Bi. P.	Bi. T.	Oc. M.	S. O. B.
1	21	700	30.0	7.5	5.5	4.5	9.0	7.5
2	22	1015	37.0	7.5	6.5	6.0	9.0	7.5
3	27	1690	40.0	9.0	8.0	6.5	11.0	7.5
4	29	1449	....	8.0	7.0	7.0	8.0	7.0
5	31	1175	37.5	9.0	7.0	6.0	11.0	8.0
6	32	1380	34.0	9.0	8.0	7.0	11.0	7.0
7	32	2040	45.0	11.5	8.5	7.5	13.0	9.5
8	33	1175	44.0	9.0	7.0	6.0	11.0	8.0
9	33	2110	45.0	10.0	8.0	6.0	12.0	8.0
10	38	3625	50.0	11.0	9.5	8.0	13.25	9.5
11	39	1610	41.5	10.0	7.75	6.25	11.75	8.5
12	39	3260	49.0	11.5	9.5	8.5	13.5	9.75
13	40	1370	38.0	9.0	7.0	6.0	10.0	8.0
14	41	1570	35.0	11.0	8.0	7.5	11.5	7.0
15	41	1810	38.5	10.0	8.0	7.5	12.5	8.5

In contrast with these measurements of the diameters of the head in prematures, the average measurements of the skull in a mature new born are noted as follows by Schauta.[3]

[1] Arch. f. Gynäk., 1872, 2. Quoted from Pfaundler and Schlossman, Leipsig, 1901.
[2] De l'accroissement en poids des enfants nés avant terme. Thèse, Paris, 1895.
[3] F. Lehr. d. ges. Gyn., 2. Aufl., Leipzig u. Wien, 1897.

1. Diameter suboccipito-bregmaticus (from the posterior edge of the great occipital foramen to the anterior angle of the great fontanelle), 9 cm.

2. Diameter fronto-occipitalis (from glabella to the occipital protuberance), 11 cm.

3. Diameter mento-occipitalis (from the point of the chin to the farthest point of the occiput), 13 cm.

4. Diameter verticalis (from the vertex to the base of the skull), 9.5 cm.

5. Diameter biparietalis (between the parietal tuberosities), 9 cm.

6. Diameter bitemporalis (between the farthest point of both coronary sutures), 8 cm.

Parents short in stature or small in build may have children who do not weigh over 2000 gm. or measure over 45 cm. in length, and yet who are neither premature nor congenitally weak.

It does not do to estimate the vitality of these infants from a consideration of their birth weight. Many of them born at or near term have a normal weight, yet they do not survive. On the other hand, infants of considerably less weight may present evidence of great vitality, a lusty cry and take nourishment with avidity. According to our experience the condition of the turgor of the prematurely born infants is of much more importance than all these. Flabby prematures with a poor turgor and a poor tonus are usually not viable. Prematures with a good turgor and a good tonus even with a low weight commonly survive.

In addition to the variations in weight and length, the premature shows variations in other measurements.

Other Measurements of the Fetus.—Von Winckel[1] regards the circumference of the head as of importance for the diagnosis of the age of the fetus and gives the following figures:

4th month	10–14 cm.	8th month . . .	25–30 cm.
5th month	13–18 cm.	9th month . . .	29–33 cm.
6th month	19–24 cm.	10th month . . .	32–37 cm.
7th month	23–28 cm.		

Reiche[2] reports the following comparative body measurements:

TABLE I.

Group. 1.	12 Children. Min.	Max.	Weight 800–1200 gm. Average.
Length of the body . .	34 cm.	41.0 cm.	37.4 cm.
Circumference of chest .	21 "	24.5 "	22.5 "
Circumference of head .	24 "	29.5 "	26.8 "

[1] Lehrb. d. Geb., Leipsig, 1889.

[2] The Growth of the Prematurely Born in the First Months of Life, Ztschr. f. Kinderh., December, 1915, **13**, 332.

TABLE I (*Continued*)

Group 2.	26 Children. Min.	Max.	Weight 1200–1500 gm. Average.
Length of the body . .	37.0 cm.	45.0 cm.	41.6 cm.
Circumference of chest .	22.5 "	27.5 "	24.8 "
Circumference of head .	26.0 "	31.0 "	28.4 "
Group 3.	**28 Children.**		**Weight 1500–2000 gm.**
Length of the body . .	41 cm.	48.5 cm.	44.2 cm.
Circumference of chest .	25 "	32.5 "	27.2 "
Circumference of head .	27 "	32.0 "	30.3 "
Group 4.	**22 Children.**		**Weight 2000–2500 gm.**
Length of the body . .	41.5 cm.	49.0 cm.	46.5 cm.
Circumference of chest .	26.0 "	30.0 "	28.4 "
Circumference of head .	29.0 "	33.5 "	32.2 "

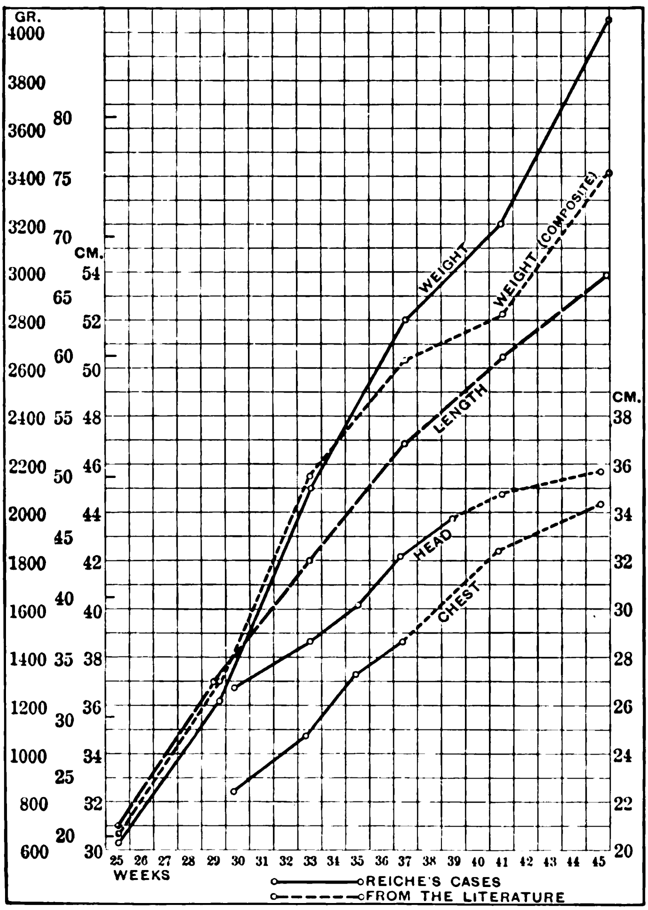


FIG. 10.—Curves showing growth in weight, length, head and chest measurements in the late fetal weeks and first weeks after maturity. (Reiche.)

These figures show a gradual and steady increase of the weight and the chest and head measurements, up to the time of maturity, when they should average 3200 gm. in weight, 50.5 cm. in length, with a chest circumference of 32.9 to 33.8 cm. and a head circumference of 34.5 cm.

We see in the eighth to the tenth month an abrupt rise of the curve of chest circumference, the curve flattening somewhat soon after birth. This increase in the circumference of the chest in the last fetal months is considerably higher than that of a mature child during the first months after birth. In the latter the circumference of the chest increases from 32.5 to 37.2 at the end of the third month to 41 at the end of the sixth month, therefore in the first six months of life approximately about as much as in the last three fetal months.

In the curve of the growth of the skull the flattening appears even somewhat earlier. The ratio, however, between the growth of the skull in the last three fetal months and that in the first six months of life is the same as in the circumference of the chest. Also the circumference of the head grows absolutely and relatively considerably more in the last fetal months than in the first six months of life.

A proof for the correctness of these figures Reiche[1] finds in the fact that the corresponding figures are considerably lower in children who die shortly after birth. They are premature weaklings whose intra-uterine development in spite of sufficient body weight did not attain such a degree that it might be completed in the extra-uterine life.

The corresponding figures are, as follows:

	7 Children.		Weight 800–1200 gm.
Group 1.	Min.	Max.	Average.
Length of the body	34.0 cm.	38.5 cm.	37.0 cm.
Circumference of chest	18.0 "	23.5 "	20.6 "
Circumference of head	21.0 "	27.5 "	25.0 "
Group 2.	9 Children.		Weight 1200–1500 gm.
Length of the body	39.0 cm.	42.0 cm.	40.1 cm.
Circumference of chest	21.0 "	27.0 "	23.8 "
Circumference of head	26.0 "	31.0 "	28.8 "
Group 3.	5 Children.		Weight 1500–2000 gm.
Length of the body	41.5 cm.	47.0 cm.	43.6 cm.
Circumference of chest	25.0 "	27.5 "	25.9 "
Circumference of head	28.0 "	31.0 "	29.6 "

From these figures Reiche[2] concludes that in premature weaklings the length of the body does not vary greatly from that of

[1] The Growth of the Prematurely Born in the First Months of life, Zeitschr. f. Kinderh., December, 1915, 13, 332.

[2] Ztschr. f. Kinderh., 1915, **13**, 349.

healthy children, but on the other hand the measurements of the circumference of the chest and of the circumference of the head are considerably smaller.

Ylppö[1] recently studied the relation of the chest circumference to that of the head in prematures and full-term infants. He found that at birth the circumference of the head is greater than that of the chest, and the greater the prematurity the more marked is the relative disproportion between the head and chest circumferences. These facts are borne out by his table:

Weight of infants, Grams.	Number.	Circumference of head.	Circumference of chest.	Breast cumference, per cent of head circumference.
Under 1000	16	25.0	20.8	83.2
1001–1500	78	31.8	24.5	77.0
1501–2000	75	30.0	26.3	87.7
2001–2500	74	32.3	29.5	91.3
New born 3000–3500	100	33.5	31.0	92.5

In comparison with the preceding tables on prematures we note the conclusions drawn by von Reuss[2] from his own work and the tabulations compiled by Weissenberg[3] on the mature new-born infant.

Body measurement.	Boys. Min.	Boys. Max.	Boys. Average.	Girls. Min.	Girls. Max.	Girls. Average.
Body length	47.5	54.0	50.8	43.5	53.0	50.0
Span of arms	45.0	52.0	48.6	42.0	52.0	48.0
Vertex-shoulder	11.5	13.5	12.4	10.5	13.5	12.1
Sitting-height	31.2	36.5	33.8	30.0	36.4	33.3
Breadth of shoulders	9.0	12.2	10.7	9.0	12.0	10.4
Breadth of hips	7.0	8.7	7.8	6.8	8.3	7.7
Circumference of head	30.5	35.5	32.7	29.0	35.0	32.6
Girth of chest	25.5	32.0	28.2	25.0	32.0	28.5
Length of trunk	19.5	24.0	21.4	19.0	24.0	21.2
Length of arm	19.5	23.5	21.4	18.5	22.5	21.0
Length of leg	18.0	22.2	20.5	17.0	21.8	20.3
Length of hand	5.8	7.0	6.4	5.8	7.5	6.4
Length of foot	7.3	8.3	7.8	6.5	8.3	7.8

The peculiarities of the proportions of the body characteristic of the full-term new born consist therefore of the following: Not only the sitting height, but also the height of the trunk proper is greater than the leg. The length of the trunk proper is greater

[1] Pathologisch-anatomische Studien bei Frühgeburten, Ztschr. f. Kinderh., March 25, 1919, Orig. Bd. 20.

[2] Die Krankheiten des Neugeborenen, Julius Springer, 1914.

[3] Die Körperproportionen des Neugeborenen, Jahr. f. Kinderh., 1906, **64,** 839.

than that of the arm. The arm is longer than the leg. The circumference of the head is usually greater than that of the chest. Occasionally the circumference of the head and chest are equal; in strongly built infants the circumference of the chest often exceeds that of the head. The body length approximates 47 to 54 cm. and errors in statements of length result because of the lack of consideration for the deformity of the skull and caput succedaneum (von Reuss[1]).

Jaschke,[2] in a recent study of the premature and debilitated child, came to the conclusion that there was less variability in

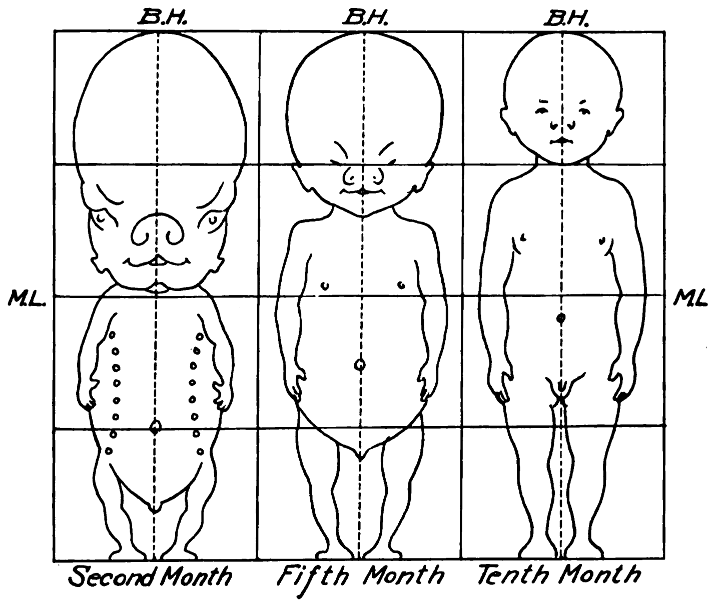


Fig. 11.—Changes in body proportions in fetal life. *B.H.*, Body height; *M.L.*, Midline.

certain relations between measurements of the body than was commonly thought. "In immature infants the fronto-occipital circumference of the head always is greater than the circumference of the shoulders (Frank and others), while in mature infants the opposite is true; also the proportion between the height of the head and the height of the body (Stratz) is disturbed since the height of the head is greater than one-fourth of the length of the body; this is due especially to relatively shorter legs" (Fig. 11).

[1] Die Krankheiten des Neugeborenen, Julius Springer, 1914.
[2] Physiologie, Pflege und Ernährung des Neugeborenen, Wiesbaden, 1917.

INTERNAL ORGANS.

Gundobin[1], studying the average weight of the inner organs of the mature new born in grams, noted the following:

Brain	389–354.5
Heart	17.24–16.5
Lungs	57 (Lt. 25; Rt. 32)
Liver	120–130
Pancreas	2.63
Spleen	7.2
Kidneys	11–12
Suprarenals	2.5
Testicles	0.2
Epididymes	0.12
Ovaries	0.2
Thyroid	1.6 (Max. 2.8; Min. 1.3)
Thymus	11.7

In contrast with these figures, we may quote from the anatomical studies of Ylppö on premature infants.

BRAIN WEIGHT OF INFANTS (YLPPÖ).[2]

Age.	Number of cases.	Boys. Average weight in grams. Of body.	Of entire brain.	Ratio of brain to body weight.
Fetus of eight months	3	2440	248	1 to 10
Newly born	3	2785	389	1 to 7.2
1 month	3	3860	517	1 to 7.5
2 months	5	4400	538	1 to 8.2
3 "	5	4480	555	1 to 8.1
4 "	5	4890	568	1 to 8.6
5 "	5	5614	632	1 to 8.9
6 "	5	6035	668	1 to 9.0
7 "	3	6560	702	1 to 9.3
8 "	3	6460	768	1 to 8.4

Ylppö found several instances in which the large brain weight seemed to be out of proportion to the figures of other observers. His studies led him to believe that the brain of the premature (even the smallest) grows at the same rate as if the fetus were in utero and that it develops in extra-uterine life after certain given laws of Nature; thus, the small body weight having relatively little to do with the brain. In these cases of marked disproportion he found that when one compares the absolute age of the prema-

1 Quoted from von Reuss: Krankheiten der Neugeborenen, Julius Springer, Berlin, 1914.
2 Pathologisch-anatomische Studien bei Frühgeburten, Ztschr. f. Kinderh., March 25, 1919, Orig. Bd. 20, 212.

ture, from the time of conception, with that of a normal infant, it is seen that the brain weight of the two compare favorably. His conclusions were that the size of the brain has nothing to do with a hydrocephalic process, since it is not explained by an abnormal water content, and that the "megacephaly" of prematures is a physiological process.

Tonsils.—In prematures there appears at the site of the palatine tonsils only one or two small cavities. Only after four to five months does a glandular structure appear.

Thyroid Gland.—This is very small, but it has a very rich blood supply. In one case of a seven-months premature Ylppö observed an enlargement of the thyroid (1.5 gm.): weight of infant, 1270 gm.; length, 44 cm. Microscopically there were large quantities of colloid in the center of the follicles, but no hemorrhages or evidence of degenerative changes.

Thymus Gland.—In prematures of 1000 to 2000 gm. it is between 1 and 3 gm., while in full-terms it may be as much as 20 gm. Gundobin[1] estimated it in prematures of similar weight as on the average of 2.5 gm.

Heart.—The heart on the average is from 0.5 to 0.75 per cent of the body weight of prematures. In those from 900 to 1200 gm. Ylppö found that the weight ranged from 4.5 to 7 gm. In full-term infants and those with a longer intra-uterine growth (of the prematures), the relation between heart and body weight was found to remain about the same by Lomer, thus:

4000 gm. infant	– 27.6 gm. heart	= 0.7 per cent body weight.
2–3000 gm. "	– 20.7 gm. "	
1–2000 gm. "	– 11.4 gm. "	

The ductus Botalli closes more slowly and later in prematures. On the average blood ceases to pass through after the end of the first or second week of life.

Liver.—The liver is the largest of the internal organs of the premature body. The smaller the premature, the greater is the relative size of the liver.

WEIGHT OF THE LIVER IN PREMATURES (YLPPÖ).

Weight of infant, Grams.	Number of cases.	Average weight of liver. Grams.	Liver weight, percentage of body weight.
Under 1000	11	43.73	4.8
1001–1500	12	53.17	4.3
1501–2000	4	56.75	3.3
2001–2500	3	102.33	4.5

[1] Die Besonderheiten des Kindesalters, Berlin, 1912.

With the increase of body weight the liver weight slowly increases. The figures for the group of 1501 to 2000 gm. are too small, and are based only on four observations. The weight of the liver in prematures has to do with the richness of its blood supply.

Spleen.—The spleen, as the liver, is very rich in blood.

WEIGHT OF THE SPLEEN (YLPPÖ).

Weight of infant. Grams.	Number of cases.	Average weight of spleen. Grams.	Spleen weight percentage of body weight.
Under 1000	14	1.5	0.17
1001–1500	12	2.8	0.21
1501–2000	4	4.4	0.22
2001–2500	8	7.2	0.28

As with the liver, the spleen increases in size with increase in the body weight.

Kidneys.—The ratio between the weight of both kidneys and the body weight is greater in prematures than in full-terms and older infants:

WEIGHT OF KIDNEYS (YLPPÖ).

Weight of child. Grams.	Number of cases.	Average weight of kidneys. Grams.	Kidney weight percentage of body weight.
Under 1000	15	5.2	0.59
1000–1500	17	8.9	0.76

Gundobin showed that in full-terms the percentage was 0.38 per cent.

Vierordt[1] showed that in men between nineteen and twenty-five years of age the percentage was 0.48 per cent.

The embryonic features of the kidneys are very marked. The fetal markings disappear fairly rapidly. In one case of a sixth to seventh embryonic month premature of 1000 gm. birth weight, the fetal markings were gone after five to seven weeks of life (Ylppö).

BODY TEMPERATURE.

During the intra-uterine life the child receives gratis the material necessary for its maintenance, for the development and regeneration of its cells. The maternal blood stream brings to the level of the placenta the oxygen and other substances needful for its

[1] Gerhardts Handbuch d. Kinderh., 1881, **1**, 1, part 2, p. 386.

nutrition, and the passing of these foods into the antenatal circulation requires no effort on the part of the fetus other than the cardiac contractions. From birth on, however, the child is an independent being and it must fight that it may live.

The upkeep of the somatic tissues is dependent upon the functions of the respiratory system and the digestive tract, and these activities require of the new-born infant an expenditure of energy of which it has had no previous experience. Before birth the energy resulting from intracellular combustion was transformed into that amount of heat necessary to the performance of the new cellulo-chemical reactions occurring in the fetus. After birth a much greater amount of energy is necessary because of the more extensive reactions taking place within the tissues and because of the appearance of motion. Increased metabolism is, therefore, necessary to the accomplishment of the digestive and respiratory functions and to enable the infant to fight against external physical agents, principally cold.

Cause and Nature of Hypothermia.—Heat regulation is one of the least developed functions of the premature infants, their body temperature showing marked fluctuation with a tendency to hypothermia. This is due to several factors:

1. *Faulty Heat Regulation Due to Lack of Development on the Part of the Nervous System.*—It is possible to imagine that in a premature infant where the development of the brain is still going on, and the separation into the white and gray matter has not been completed, that the nervous system is not sufficiently matured to function normally.

2. *Loss of Heat Through Radiation.*—The extent of the heat loss from the body of an animal by conduction, radiation, evaporation from the skin and the surface of the lungs is determined by the extent of the surface and by the thickness of the ill-conducting subcutaneous fatty layer; the heat loss, therefore, is in greater part proportional to the extent of the surface of the body. In a premature infant the body surface is relatively greater than in a full-weight new born, since the size of the body is absolutely smaller. Wrinkled skin and absence of the fat deposits in the skin are responsible for the greater loss of heat. It is these physical conditions which make it difficult for the premature to retain its own heat and predispose to the readiness with which the subnormal temperature can occur.

3. *Insufficient Oxygen Combustion.*—Due to a poorly developed respiratory center causing asphyxia.

Babak[1] found that the lower the temperature in the respiratory

[1] Ueber die Wärmeregulation der Neugeborenen, Pflügers Arch., 1902, **89**, 154.

chamber, the greater the consumption of oxygen, this corresponding to the irradiation of heat. The average values in one hour per gram of body weight amounted to:

Temperature in chamber. Deg. C.	Consumption of O_2. cc.
24.0	378
23.2	562
20.0	581
19.9	632
17.1	636
12.9	739
12.1	874

From the results of this experiment it is clear that the infant's organism attempted to equalize the physical minus with the chemical plus. But in spite of the more intensive exchange of gases, the body temperature was sinking with a low external temperature and also when the infant was insufficiently covered. The increase in oxidation processes, therefore, was not sufficient to compensate for the increased heat radiation.

4. *The Circulation.*—The circulation as affected by its nervous mechanism and weak cardiac action is another important factor.

5. *Insufficient Heat Production Due to Lack of Food or Improper Metabolism.*—This cause of hypothermia is of minor importance in the premature infant which is fed a sufficient quantity of breast milk and shows ability to assimilate the same. As the sucking centers are too poorly developed to enable the infant to obtain sufficient nourishment, most of these infants cannot be trusted to their own resources in obtaining their food.

A careful consideration of all of the factors tending to hypothermia make it evident that we cannot depend on an equalization of the heat loss from the body surface by the internal production of heat, and therefore in order to maintain a uniform temperature it becomes necessary to assist the infant by giving it an artificial environment of good air sufficiently heated to maintain a normal body temperature.

THE GROWTH OF THE PREMATURE.

Initial Weight Losses.—Loss of body weight during the first days of life occurs so constantly in full-term infants that moderate losses must be considered physiological. This is also true of premature infants although in most instances it is relatively greater. Premature infants lose relatively more and regain their birth weight more slowly, often requiring a month (De Lee[1]) and also, as a

[1] See page 30, Ref. 4.

general rule, the nearer the prematures are to full term, the lower is the relative loss of weight as expressed in percentages.

The average loss in weight in the premature and in other infants of relatively low birth weight during the first days of life is shown in the following table adapted from Reiche:

Weight.	Length.	Average decrease.
800–1200 gm.	32.0–40 cm.	71 gm.
1200–1500 "	37.0–44 "	97 "
1500–2000 "	40.0–48 "	137 "
2000–3500 "	41.5–50 "	177 "

Gundobin's figures are considerably higher, as he came to the conclusion that the initial loss of weight in infants with a birth weight under 2000 gm. gm. amounted on the average to 148 gm.

The artificially-fed infants lose more weight than the breast fed, but no differences were noticeable between those infants nursing at the mother's breast and those fed by a wet-nurse (Reiche).

In children of multiparous women both the absolute and also the relative percentage value of the weight loss is smaller than in those of primiparous, which is undoubtedly due to better nursing conditions, milk appearing sooner in multiparæ and being usually more abundant.

The loss of weight is also relatively larger the less the birth weight of the infant, as the following table taken from Pies[1] will show:

Initial weight.	Primiparæ. Average decrease.	Multiparæ. Average decrease.
2500 gm.	240 gm. = 11.2 per cent	195 gm. = 8.2 per cent
2510–3000 "	235 " = 8.3 "	180 " = 6.2 "
3010–3500 "	295 " = 9.0 "	265 " = 8.1 "
3510–4000 "	360 " = 9.7 "	325 " = 8.7 "
4010–4500 "	245 " = 8.4 "	366 " = 8.3 "
Average	275 gm. = 9.3 per cent	266 gm. = 7.9 per cent

Initial loss in weight rests upon the fact that the new-born infant gives off more than it takes in. The meconium is accountable for a considerable part of the loss. This averages in weight according to Camerer[2] from 70 to 90 gm.; according to Hirsch[3] from 150 to 200 gm. In addition to that, the urine voided before the child receives much fluid must be considered, though this is probably small. The water lost through the lungs and skin, the loss of the stump of the umbilical cord, and, in some cases, the vomiting of swallowed liquor amnii during the first twenty-four

[1] Ueber die Dauer, die Grösse und den Verlauf der physiologischen Abnahme der Neugeborenen, Monatschr. f. Kinderh., 1911, **9**, 51.

[2] Beitrag zur Physiologie des Säuglingsalters, Ztschr. f. Biol, 1900, **39**, 37.

[3] Die physiologische Gewichtsabnahme der Neugeborenen, Berl. klin. Wchnschr., 1910, **2**.

hours, are all factors in reducing the weight of the new born. Furthermore, it has been shown that there is a loss of the body tissues, of the fat, glycogen and albumin, as evidenced by the loose and wrinkled condition of the infant's skin, and lost turgor of the tissues in general. Landois[1] found that the loss of weight in infants in whom the cord was tied late was 5.9 to 7.4 per cent less than those in whom the cord was tied and cut early.

Gundobin[2] found that the lowest weight was usually reached sometimes between the fourth and sixth days in the full-term infant and that the birth weight was regained on the eleventh to the sixteenth day. Very frequently, however, and especially in weaklings and prematures, the birth weight was not regained as early as the sixteenth day, twenty or thirty days being required to make up the initial loss. The artificially-fed regained the loss later than the breast-fed infants.

Pfaundler,[3] in his observations on 1000 new-born infants came to the conclusion that the physiological weight loss occurred in 42 per cent by the fourth day. The loss in the infants of from 1500 to 4000 gm. birth weight averaged 7.8 per cent of the latter, and was about the same for the heavy as for the light, although it was relatively slightly greater in the former.

Birth weight.	Loss in weight.				
Over 4000 gm.	325 gm.	= 7.6 per cent	of the	birth weight
3500–4000 "	300 "	= 8.0 "	"	"
3000–3500 "	250 "	= 7.7 "	"	"
2500–3000 "	210 "	= 7.6 "	"	"
2000–2500 "	190 "	= 8.4 "	"	"
1500–2000 "	130 "	= 7.4 "	"	"
	Average	7.8 per cent		

Ramsey and Alley[4] noted in 300 cases that the average loss of weight continued for three days and was regained by the tenth day by only one-fourth of the infants.

Shick,[5] believing that the initial loss of weight was avoidable, gave each infant 10 per cent of its body weight of breast milk the first twenty-four hours, increasing the amount until 15 per cent was given at the end of the third twenty-four hours. He employed the milk of mothers having infants less than a week old and was able to prevent the initial loss in all of his twelve cases.

The increase in weight of the prematures is noted in the table on p. 44 in a group of the author's cases.

[1] Zur Physiologie der Neugeborenen, Monatsschr. f. Geb. u. Gyn., 1905, **32**, 194.
[2] Besonderheiten des Kindesalters, Berlin, 1912.
[3] Körpermass-Studien an Kindern, Ztschr. f. Kinderh., March 28, 1916, 151–152.
[4] Observations on the Nutrition and Growth of New-born Infants; an Analysis of 300 Clinical Charts, Am. Jour. Dis. Child., June, 1918, **15**, 408.
[5] Zur Frage der physiologischen Körpergewichtsabnahme der Neugeborenen, Ztschr. f. Kinderh., 1916, **13**, 257.

Case.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Birth-weight, gm.	740	690	1360	1335	1435	1420	1080	1440	1935	1930	1750	1890	1950	2010	1765	1875	1960
Gain by weeks, gm.:																	
First	165[1]	155[1]	40	15	185[1]	135	65[1]	200	300[1]	260	130[1]	130	175	240	50	85	25[1]
Second	20[1]	35[1]	75	100	15	65	15[1]	....	50	250	45	175	235	150	120[1]	190	65
Third	....	5[1]	45	85	75	130	20	105	125	260	100	35	300	320	155	110	150
Fourth	25	35	45	90	65	290	80	105	50	265	175	35	300	375	210	190	90
Fifth	15	5	125	....	100	25	95	150	85	....	175	90	300	375	....	150	180
Sixth	5	5	175	....	135	225	95	125	275	....	125	....	....	360	190		
Seventh	10	20	75	....	....	210	65	65	140								
Eighth	15	10	75	....	....	300	100	240	100								
Ninth	5	15	125	....	....	....	100										
Tenth	20	10	50	....	....	....	75	140	140								
Eleventh	....	....	125	....	....	....	75	112									
Twelfth	....	....	85	....	....	....	175	50									
Thirteenth	....	....	....	....	....	....	100	40									
Fourteenth	....	....	....	....	....	....	115	30									
Fifteenth	....	....	....	....	....	....	120	50									
Loss in weight:[1]																	
Daily gain, gm.	....	....	12	14	17	27	24	24	15	37	21.5	18	40	43	23	23	25
Range of calories	....	....	137 to 220	95 to 161	116 to 148	115 to 144	132 to 170	130 to 137	90 to 171	105 to 131	102 to 119	127 to 170	111 to 120	94.5 to 118.5	114 to 125	104.5 to 120	105 to 132

[1] These infants were all fed human milk exclusively and all except Infants 1 and 2 left the hospital in good health at the end of the period tabulated.

The growth of the premature infant has been well shown by the tables of Camerer,[1] who figures out the daily average increase in ten infants who had a birth weight ranging from 1330 to 1970 gm.

Week of life	0	2	4	8	12	16	20	24	28	32	36
Weight in grams	1630	1830	2090	2636	3272	3906	4430	4068	5367	5717	6217
Average daily gains in grams	....	9	19	23	22	20	14	12	10	10	

Camerer compared the increase in weight in breast-fed and bottle-fed premature infants with an initial weight of from 1590 to 1740 gm.

	Doubled weight.	Trebled weight.	Quadrupled weight.
Breast fed	10th week	22d week	33d week
Bottle fed	11th "	24th "	40th "

Camerer's further figures also show that the artificially-fed full-term infant is much slower in its weight increase than the breast-fed child.

	Average birth weight.	Number of infants.	Doubled weight, weeks.	Trebled weight, weeks.	Quadrupled weight. weeks.
Breast fed	1680	8	12	24	52
Artificially fed	2420	18	18	44–48	

The average daily increase in weight of the premature of different periods as well as for the premature child is shown by Friedenthal:[1]

Fetal months.	Average daily increase in weight.
6th to 7th	19.5 gm.
7th to 8th	29.3 "
8th to 9th	23.3 "
9th to 10th	13.3 "
1st month of mature child	25.0 "

The growth in length proceeds slowly from month to month, diminishing in rate (Friedenthal).

Age.	Growth in length per month.
6th to 7th fetal month	6.0 cm.
7th " "	5.0 "
8th " "	4.5 "
9th " "	4.0 "

If these figures of Friedenthal's are plotted into a curve it is seen that the curve of the body weight and that of the body length run parallel up to the seventh or eighth month, at which time the length curve rises less abruptly than the weight curve.

Pfaundler[2] found that the rate of growth in an infant born three months prematurely became the same as that of a maturely born child when the premature had reached the age of three months.

[1] Med. Wchnschr., 1909, No. 34. [2] See p. 43, Ref. 3.

These figures apply, of course, to the healthy prematures only and not to those debilitated from disease or by unfavorable environment or food.

Reiche's[1] investigations have shown that the growth of the prematures follows the same rules of growth that hold good for the corresponding months after impregnation. In healthy prematures there is no difference between the intra-uterine and extra-uterine growth in the same months, so that the birth in itself causes no disturbance of growth provided that the infant has reached a certain stage of development, compatible with the exercise of certain indispensable functions, *e. g.*, respiration, circulation and digestion. This stage of development is seldom reached before the twenty-eighth week of life, when the infants are about 34 cm. long and weight approximately 1 kg. It has, therefore, been proposed to designate the age of the infant from the time of conception rather than from the time of birth. Serious chronic diseases of the mother (especially lues and tuberculosis) exert a growth-inhibiting influence upon the infant. Their progress is not governed by the same laws that hold good for healthy premature infants.

Reiche has also studied the relation between the growth in weight and the growth in length and has introduced the term length-weight coefficient, by which is understood the weight of a unit of length. The following table shows the birth-weight coefficient for different groups of prematurely born infants:

Birth-weight.	Length of body.	Length-weight coefficient.
800-1200 gm.	32.0-40 cm.	28.0 gm.
1200-1500 "	37.0 44 "	33.8 "
1500-2000 "	40.0-48 "	43.2 "
2000-2500 "	41.5-50 "	48.7 "

Langstein[2] formulated the following law from the observations of Reiche and others: Both the growth in mass and the growth in length of these organisms in whom the transition from intra-uterine to extra-uterine life had to occur prematurely, proceeds according to the same laws that correspond to the period of time after impregnation.

The majority of multiple pregnancies terminate prematurely and therefore the percentage of twins among the prematurely born is considerably higher among mature children. By the development of more than one child in the mother's womb the growth may be impaired, and this consists, as a rule, in impairment of growth in mass, only in exceptional cases in impairment of growth in length.

[1] Ztschr. f. Khk., Dec., 1915.
[2] Ernährung und Wachstum Frühgeborener, Berl. klin. Wchnschr., 1915, 24.

But even in these prematurely born, twins have a tendency in their first months of life to make up this loss. The curves of growth of twins run, as long as no intercurrent diseases interfere, parallel to each other and also to the curve of those children in whom a larger difference in growth was present at birth. The proportions of growth between the circumference of the thorax and the circumference of the head are scarcely influenced by multiple pregnancy. In individual twins even these curves run parallel to each other.

Weight in Relation to the Body Surface.—Ssytcheff[1] gives the following table comparing the surface area and the weight in the premature and in older children.

Age.	Weight. gm.	Surface area, sq. cm.	Surface area per kg. of weight. sq. cm.
Premature four days old	1505	1266.4	841.4
New born	2097	1476.0	704.0
3 months old	3520	2279.0	647.0
6 "	5138	2961.0	576.2
1 year old	9095	4800.0	527.0

Thus it is seen that the larger the volume (weight) of the infant the smaller the surface area relative to that weight.

In estimating or comparing heat loss or other metabolic processes relating to or dependent upon surface area, it is evident that one should have an exact method of determining that area. Meeh,[2] in 1879, was the first to construct a formula for this purpose, the basis for which was the observation of Molischott that the volume of bodies of similar composition and form varies in the ratio of the cube root of their weight and their surface areas in the ratio of the square root of their volume.

Recent investigations have given us two reliable formulæ for the rapid *estimation of the body surface* of the infant, those of Dubois and DuBois[3] and of Howland and Dana.[4]

The formula of Dubois and DuBois, which is entirely independent of the body weight, predicates the division of the body into several regions, the various measures of length of these regions being multiplied by the sums of the various measurements of the width, and the figure thus obtained multiplied by the constant for the given region. These constants have been worked out by the investigators and represent the reciprocal of the average factor for that particular combination of length and breadth measurements which showed the smallest variations.

[1] Quoted from Gundobin, Die Besonderheiten des Kindesalters, Allg. mediz. Verlagsanstalt, Berlin, 1912.
[2] Oberflächen Messungen des menschlichen Körpers, Ztschr. f. Biol., 1879, vol. **15**,
[3] Arch. Int. Med., 1916, 863.
[4] A Formula for the Determination of the Surface Area of Infants, Am. Jour. Dis. Child., 1913, **6**, 33.

In the formula proposed by Howland and Dana[1] the data supplied by Meeh and Lissauer[2] were used. Meeh had included 3

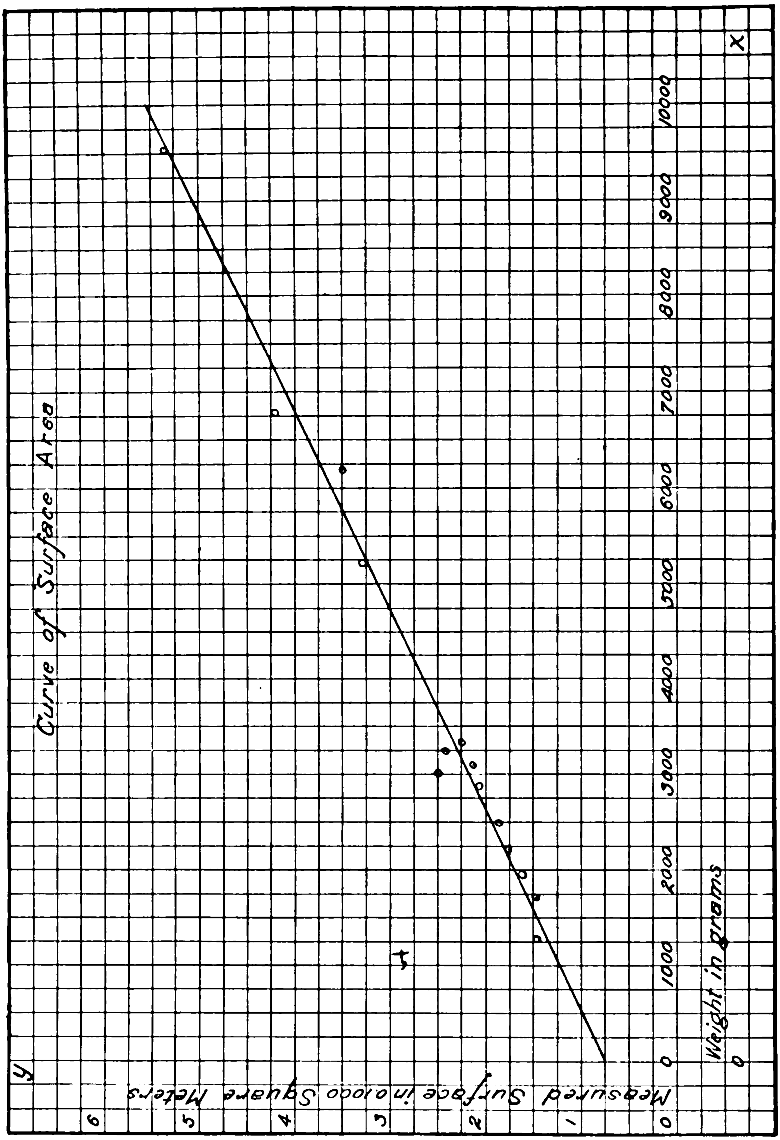


FIG. 12.—Chart showing weight and surface area of infants. (Howland and Dana.)

[1] See p. 47, Ref. 4.
[2] See p. 47, Ref. 2.

infants among his observations and Lissauer had measured the area of 11, making 14 in all. Howland and Dana first plotted on a chart the weight and surface area of these 14 cases and then drew a curve as nearly as possible to all these points so that the distance from any point would be as small as possible.

This curve (Fig. 12), by its distance from the axes *ox* and *oy*, represents an average of the observed data, so that when drawn

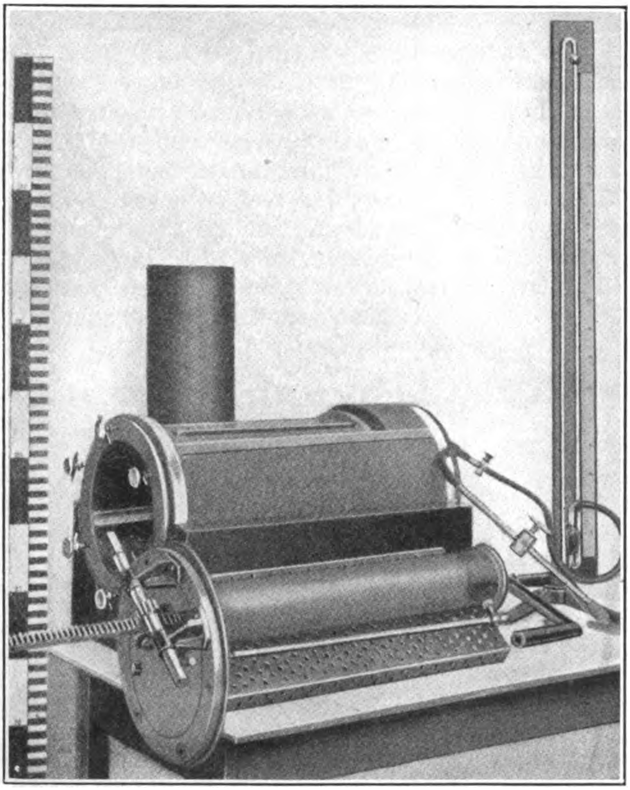


FIG. 13.—Dermatograph. Apparatus for measuring body surface. (Pfaundler.)

to the proper scale, the point on the curve representing any known weight of the child may be marked on the chart and the surface area read off directly. Thus, if one has an infant weighing 7000 gm. and it is desired to know its surface area, one finds where the 7000 gm. line intersects the curve. Carrying this point horizontally to the left, it is seen to intersect the *oy* axis at a point corresponding to 4100 sq. cm.

This formula, *u* equals *mx* plus *b*, is the algebraic representation of this form of curve, and in it x and y represent the abscissas and ordinates of the curve, b represents the distance along the y axis, and m represents the tangent of the angle that the curve marked with the x axis.

In this formula:

y = surface area of child in square centimeters.
x = weight of child in grams.
m = 0.483.
b = 750.

The factor b was read directly from the chart and m was obtained by dividing 5560 minus 730 by 10,000. Having these last three quantities, it becomes possible to obtain the y or surface area by simple computation—the weight times 0.483 plus 730.

Pfaundler,[1] in 1916, reviewed the previous methods of measuring body surface and elaborated a new method based on the principle that the body surfaces are usually in the form of a cylinder or obtuse cone. The body was divided into sixteen regions by use of an instrument—dermatograph, and the areas added to give the total surface. This instrument is illustrated in Fig. 13.

CHARACTERISTICS OF VARIOUS ORGANS.

Respiratory Tract.—One of the most marked features of the premature and of the congenitally weak are the poor respiratory efforts, indeed, Billiard[2] has defined congenital weakness as "the incomplete establishment of respiration." The premature in response to the need of air, inspires at birth, but its muscular power is weak and its efforts are insufficient to raise the thoracic wall and thus dilate the pulmonic cavity. As a result, though the large bronchi are filled with air, many of the small bronchioles are not dilated and a large portion of the lung continues to remain in a fetal stage, and may require several weeks for its complete expansion. The reason for this poor functioning of the organs of respiration lies in the lack of development of the respiratory centers in the medulla.

Most observers state that the *chest wall* of the premature infant is more or less immobile, moving but slightly with each respiration, but it has been our experience that quite constant evidence of prematurity is shown in the flexibility of the thorax and its tendency to retraction with each inspiration, the seeming immobility being the result of the poor effort on the part of the muscles of respiration,

[1] Körpermass-Studien an Kindern, Ztschr. f. Kinderh., March 28, 1916, Bd. **14**, 1-148.

[2] Traité des maladies des enfants nouveau nés, 1833, 73.

due to their weakness. The chest walls can expand but the muscular power is insufficient to make them do so. This muscular inertia, which is so well evidenced in these infants, is therefore partly the result of poorly developed muscles and partly the result of deficient innervation due to a similar lack of development of the cerebral centers.

Accompanying the deficient oxygenation of the blood are attacks of *cyanosis*, during which respiration ceases entirely. This apneic interval lasts for one or two minutes and then breathing is resumed. These attacks are not at all infrequent during the first fortnight and often appear without warning. In those cases in which recovery occurs the attacks become less frequent and less severe, but when unrelieved they are of grave significance and not uncommonly result fatally.

Clinically the weakened respirations are manifested by the monotonous, feeble, whining cry and grunting expirations with comparative immobility of the thorax, and the superficial and often irregular character of the respirations, which become abdominal in type. While a child born at the sixth month may breathe for hours or days, previous to that time respiration is not fully established. Even though respiratory exchange does not occur, the heart may be found beating several hours after birth.

The *frequency of respiration* in the sleeping premature immediately after birth is frequently as high as 40 to 50 per minute. When awake the rate is about 50 or more unless the infant is crying, when it is much less than in ordinary breathing. The type of respiration in the premature is essentially diaphragmatic, superficial and irregular, showing interruptions particularly during crying when these pauses may be quite long. The soft and yielding character of the thoracic wall in the premature permits of slight degrees of retraction of the lower intercostal spaces during the deeper inspirations.

The *physical findings* over the lungs of premature infants are uncertain. On inspection and palpation the thorax shows deficient mobility, on percussion the sounds over the bases are lower than over the balance of the chest, and on auscultation the vesicular murmur is hardly perceptible. At autopsy these signs are confirmed and the lower parts of the lungs particularly are seen to be atelectatic, at times the major portion of the organ being involved, making gaseous interchange very difficult.

The complete establishment of respiration may be prevented not only by the weakness of the respiratory movements but by the aspiration of liquor amnii or mucus during the last moments of delivery, which mechanically prevents the entrance of air into the pulmonary alveoli. (See Atelectasis.)

Parrot,[1] Billiard[2] and others have noted a condition which is spoken of as life without respiration, of which the characteristic manifestations are the absence of thoracic movements, the presence of a pulse and of movements of the extremities, and the absence of asphyxia immediately after birth. The persistence of the ductus arteriosus renders this condition supportable, as it allows the blood to pass directly into the aortic current without passing through the lungs. Such infants remain in their intra-uterine state of apnea until the respiratory centers become sufficiently irritated by the increasing venous blood to evoke respiratory action. This life without respiration should not be confounded with the apparent death of children born at or before term. Apparent death has two forms: The syncopal form, which is characterized by pallor of the skin and absence of pulse, and the asphyxiated form, distinguished by cyanosis of the skin and the presence of a pulse beat. (See Apparent Death.)

The nasal passages of the new-born prematures are particularly narrow, favoring the easy occurrence of stenosis in inflammatory conditions involving the nasal mucosa.

Interference with respiration also results from the aspiration of food or vomited matter into the larynx or trachea, the lack of development of the pharyngeal and laryngeal reflexes being responsible for the not infrequent occurrence of this accident. Attempts at drinking sometimes result in mechanical hindrance of obstruction to inspiration during the act of swallowing. Aspiration of food is often followed by a pulmonary infection and thus atelactasis of the lung may be said to predispose to a pneumonia which not infrequently leads to death. (See Infections of the Lungs.)

Jaschke[3] considers the deficient function of the respiratory apparatus as being due to the fact that the irritability of the respiratory center is so low that a large accumulation of carbonic acid in the blood is necessary to make it act. With the sinking of the carbonic-acid tension with stronger respirations, the depth of respiration decreases again, because of lowered stimulation of the respiratory center and finally a point is reached in which the blood is arterialized, when the respiratory center no longer responds. A pause in respiration sets in and lasts until excess of carbonic acid stimulates new respiratory movements.

A further point is brought out by Jaschke. There appears to be a disturbance of the gaseous interchange, which is probably explained by the peculiarity of the blood serum of debilitated premature infants. This was first noted by Pfaundler.[4] The blood serum shows a diminution of the OH ions, and a correspond-

[1] L'athrepsie, Paris, 1877.
[2] See p. 50, Ref. 2.
[3] See p. 18, Ref. 1.
[4] Quoted from Jaschke.

ingly greater concentration of the H ions, which condition makes the draining of carbonic acid from the tissues more difficult. Jaschke believes that this agrees with Finkelstein's[1] theory that the attacks of cyanosis are to be regarded as an expression of a chronic carbonic-acid intoxication.

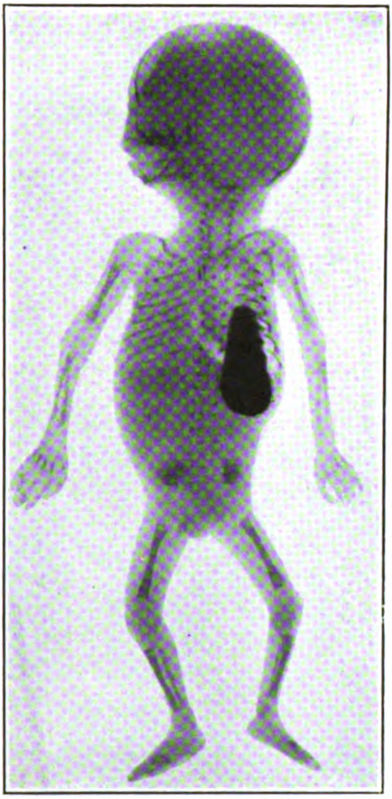


FIG. 14.—Roentgenogram showing position of stomach in a sixteen weeks' fetus.

The Digestive Tract.—1. *Anatomy.*—The muscles of the buccal region, of the tongue and of the soft palate are weak.

The stomach of the premature infant before its first feeding, as seen in autopsy, is in an almost vertical position and tubular in its form. In the premature infant which has been fed the fundus is fairly well developed and causes the stomach to assume a more oblique position. This is corroborated by a roentgen-ray examination (Figs. 14 and 15).

[1] Quoted from Jaschke.

A. F. Hess[1] was able to demonstrate that the gastric canal of the infant is more nearly vertical than horizontal, and that therefore from a functional standpoint the infant's food traverses the gastric canal in a vertical rather than a horizontal path, even though the stomach lies more or less horizontally. This fact is even more true of the physiological path of the food in the premature (Fig. 16).

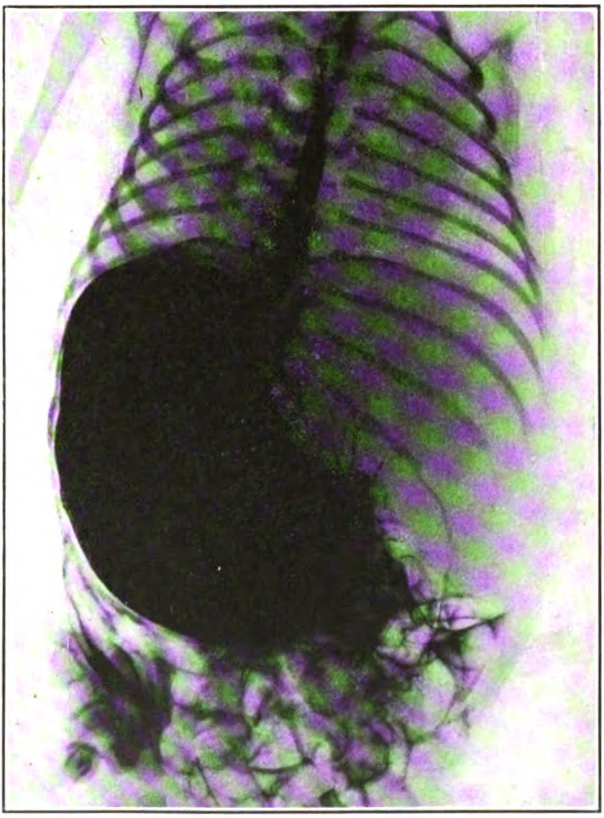


Fig. 15.—Roentgenogram showing position of stomach in a still-born, full-term infant.

The cardiac end of the stomach is found well to the left and usually about the level of the tenth dorsal vertebra. The cardiac sphincter is usually poorly developed (Fig. 17). This in part accounts for the ease with which the premature infant regurgitates its food. The pylorus lies somewhat higher than that of the full-

[1] Am. Jour. Dis. Child., 1912, **3**, 133.

term new-born, in whom it is found about midway between the ensiform cartilage and the umbilicus. Before feeding it is almost always found to the left of the median line. The pyloric musculature is usually quite well developed, even in the new-born premature (Figs. 18, 19 and 20).

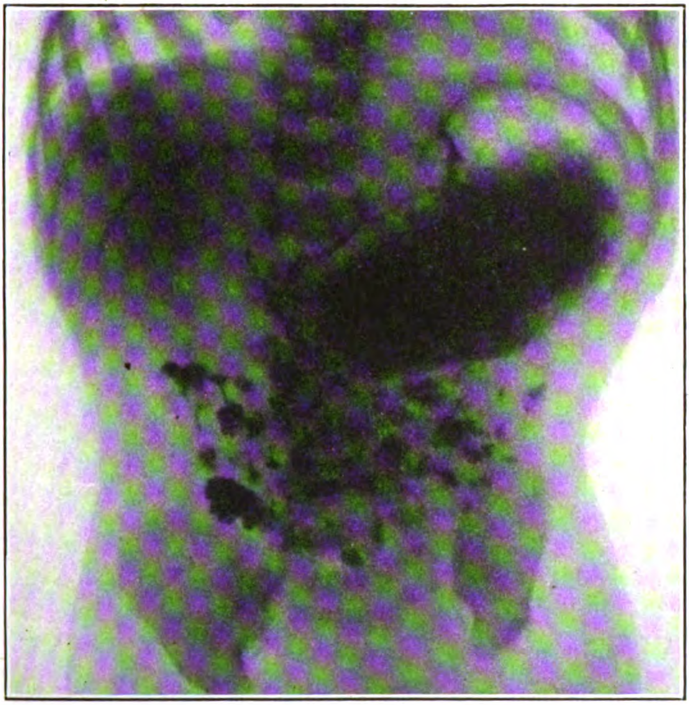


Fig. 16.—Roentgenogram of stomach immediately after feeding showing oblique position and early passage of food through the pylorus.

The musculature of the stomach at autopsy in the new-born premature is in a state of contraction, giving the stomach a tubular appearance. In the living, however, this tubular appearance quickly disappears with the administration of food, the fundus enlarging much more rapidly than the balance of the stomach in order to meet the physiological demands.

Gastric Capacity.—Although many authors have measured the full-term infant's stomach as to its capacity, both at autopsy and in the living, their figures vary considerably.

Mosenthal,[1] after a careful study of full-term infants measured

[1] Arch. Pediat., 1909, **26**, 761.

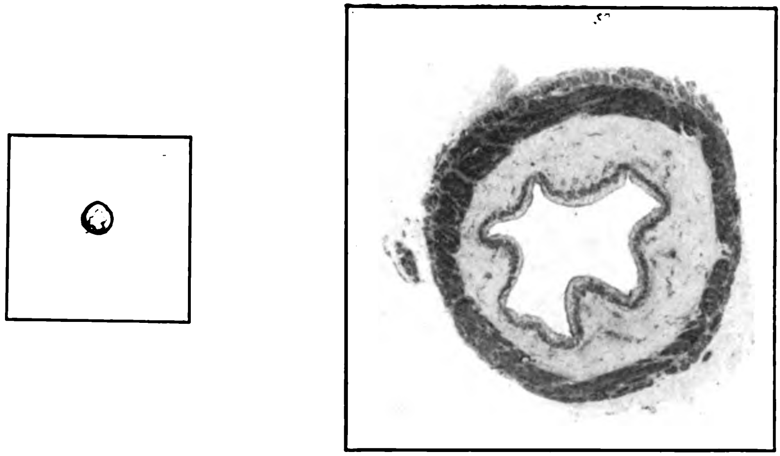


Fig. 17.—Section through the esophagus near its junction with the stomach of a fetus, aged thirty-two weeks. Normal size and enlarged 10 diameters. Section taken from stomach shown in Fig. 24.

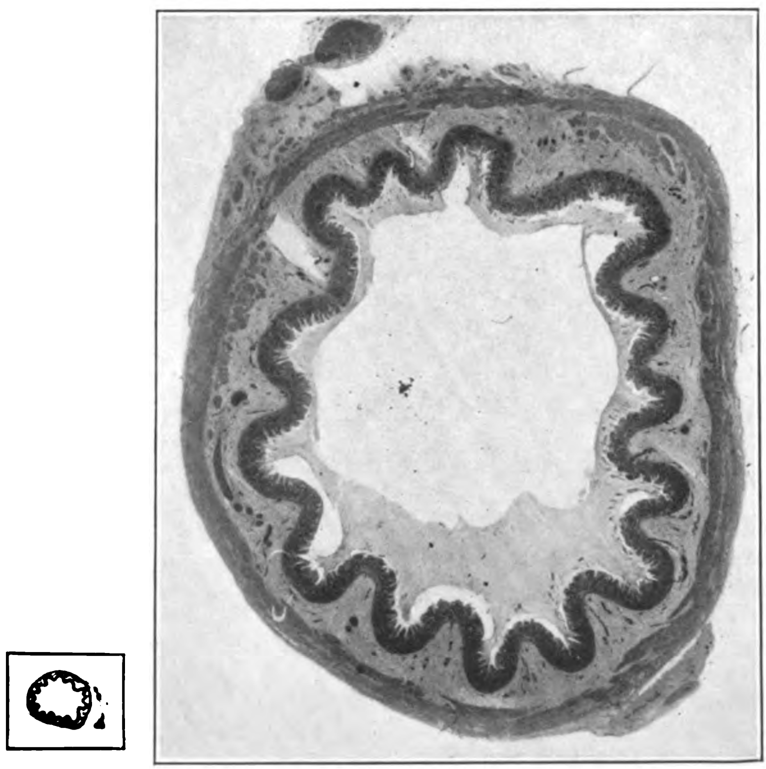


Fig. 18.—Transverse section through the middle of the fundus of the stomach of a fetus of twenty-two weeks. The glands have shallow crypts, in this case filled with coagulated mucin. The glandular portion of the section is not so thick as in the adult. There are a few parietal cells at the base of the fundus glands. Normal size and enlarged 10 diameters.

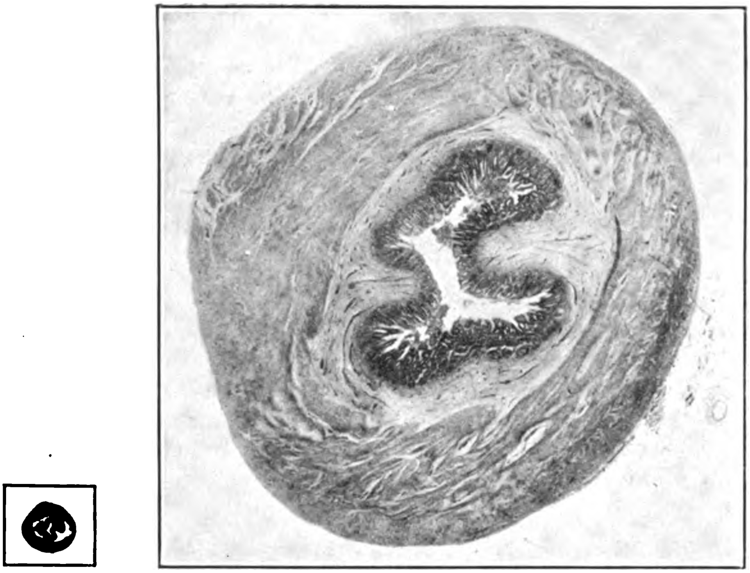


FIG. 19.—Transverse section through the pyloric end of the stomach of a fetus of twenty-four weeks. The long pyloric glands have deep crypts between them, representing a close approach to the adult type. The absence of Brunner's glands removes it from the immediate vicinity of the pyloric sphincter. Normal size and enlarged 10 diameters. Taken from stomach in Fig. 23.

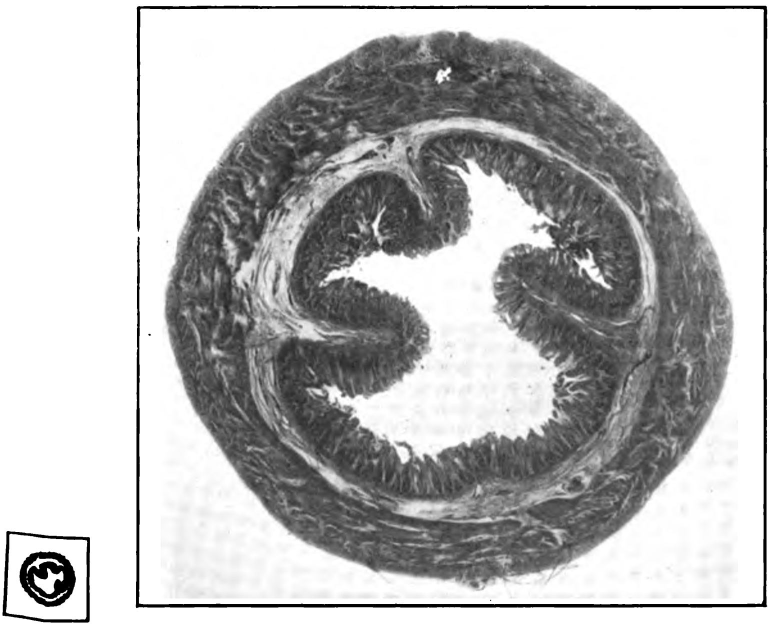


FIG. 20.—Transverse section through the pyloric end of the stomach of a fetus of twenty-eight weeks. Normal size and enlarged 10 diameters, taken from stomach in Fig. 23.

during life and post mortem, states that the physiological capacity of the stomach exceeds the anatomical gastric capacity during life because of the rapid passage through the pylorus of the individual feedings during the act of nursing. This fact is corroborated by the roentgen ray (Fig. 16) in several of our cases. Therefore, the gastric capacity, as measured post mortem by filling the stomach with water under pressure of 15 cm. of water with the pyloric end of the stomach ligated, must also fall short of giving the exact functional capacity.

Pfaundler's[1] figures for the stomach capacity during the first three months of life for the full-term infant are 90,100 and 110 cc.

Holt[2] gives the following averages for stomach capacity in a series of studies made on infants dying during the first four weeks of life and examined post mortem.

Age.	No. of cases.	Capacity.
Birth	5	36 cc.
Two weeks	7	45 "
Four weeks	4	60 "

Notwithstanding the fact that distention of the stomach according to the method of Pfaundler at autopsy is far from an ideal method of estimating the physiological capacity of the stomach, the author has undertaken to measure the stomach capacity for the various fetal ages after the sixth month by this method, and to illustrate the same graphically by photographs which represent the actual size of these stomachs at various fetal ages. This has been done more especially to illustrate the dangers of individual overfeedings which are so disastrous to the life of the premature.

Figs. 21 to 26 are photographs taken with specimens immersed in oil and represent the exact size of the stomach under 15 cm. of water pressure at different ages.

The stomach of the premature infant on a diet of breast milk is usually found empty at the end of one and one-half to two hours. That of the artificially fed requires a considerably longer period of time, depending upon the nature of the food administered, even in the case of feeding with predigested milk.

2. *Physiology.*—The digestive functions of the healthy premature infant are proportionate to the age at the time of birth. At the sixth or seventh month most of the functions and secretions are rudimentary and insufficient, while in the older infants the lessening of digestive ability is not so great.

The sucking ability in the prematures and weaklings is feeble as a result of the lack of muscular strength necessary to operate

[1] Magenkapazität in Kindersalter, Wien. klin. Wchnschr, 1897, 44.
[2] Diseases of Infancy and Childhood, New York and London, 1911, p. 309.

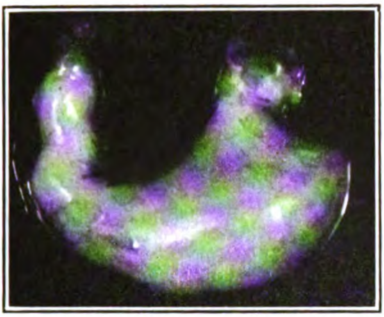


FIG. 21.—Stomach estimated fetal age twenty-four weeks capacity, 5 cc.

FIG. 22.—Stomach estimated fetal age twenty-six weeks capacity, 8 cc.

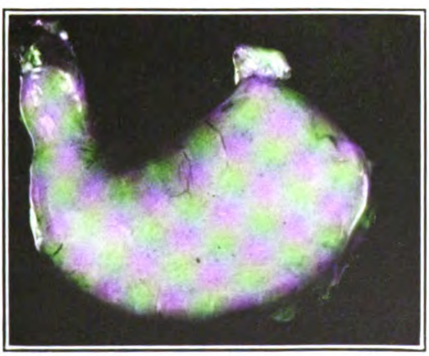


FIG. 23.—Stomach estimated fetal age twenty-eight weeks, capacity 10 cc.

the suction, the muscles of the buccal region, of the tongue and of the soft palate being weak. Accompanying this muscular asthenia is an inactivity of the salivary glands, as a result of which the mouth is dry. The lack of sucking movements tends also to retard the development of these glands.

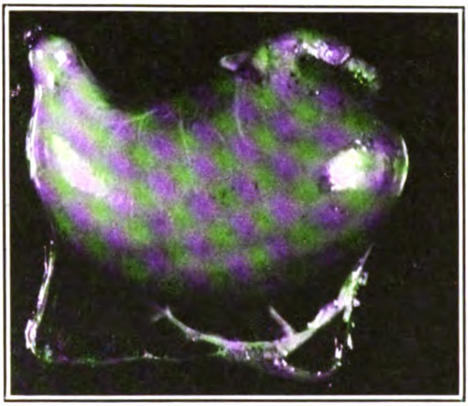


FIG. 24.—Stomach estimated fetal age thirty-two weeks, capacity 18 cc.

The strength to swallow is also diminished in the premature. In the weakest a few drops of milk placed in the mouth remain

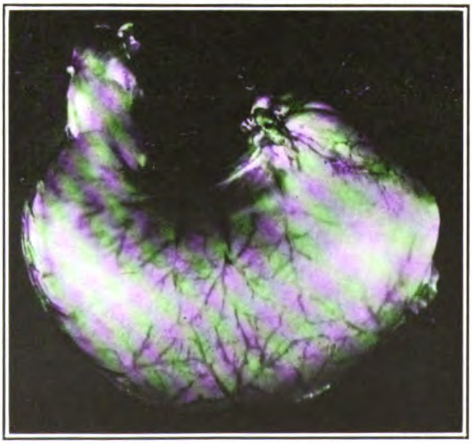


FIG. 25.—Stomach estimated fetal age thirty-six weeks, capacity 25 cc.

there; in the stronger, though at first they nurse, they soon tire and their efforts to swallow cease.

"Hunger contractions" were studied by Taylor[1] in 5 premature and 40 full-term new-born infants. A comparison of the contractions in the new born with several older children showed that the hunger contractions in the former were greater than in the latter. Reflex inhibition from the presence of food in the stomach was present in infants of all ages. The time of appearance of hunger after feeding in healthy infants gaining in weight and receiving a sufficient amount of food was: For *premature infants* under one month, one hour and forty minutes; in full-term infants under two weeks, two hours and fifty minutes; in infants from two weeks to four months, three hours and forty minutes.

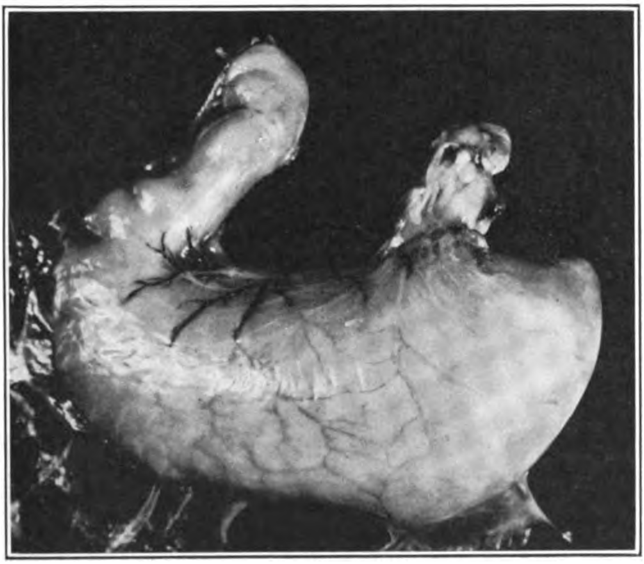


Fig. 26.—Stomach estimated fetal age forty weeks, capacity 45 cc.

The *ferments* of the gastro-intestinal canal are most conveniently discussed from the standpoint of action. The first group are those that aid in the splitting up of protein substances.

Pepsin is present in the gastric mucosa as early as the fourth fetal month, though not in such quantities as in the older children. It increases in amounts up to about the third month of life and then remains at about that level. Hydrochloric acid and *rennin* are also present in fetal life. Hess[2] was able to demonstrate free hydrochloric acid in 54 out of 55 cases immediately after birth.

[1] Hunger in the Infant, Am. Jour. Dis. Child., October, 1917, **14**, 233.
[2] Gastric Secretion of Infants at Birth, Am. Jour. Dis. Child., 1913, **6**, 264.

Lipase was found to be present in small quantities by Ibrahim[1] in a fetus of 800 gm. and plainly present in those from 1100 gm. upward. Sedgwick[2] had previously demonstrated it in 1906.

Trypsin is present in the pancreatic extract of the new born. Ibrahim found trypsinogen as early as the sixth fetal month and enterokinase was also found by him in an extract of intestinal mucosa from premature infants. The lower third of the small intestine is most active in the production of enterokinase.

Secretin, the ferment which activates the pancreas, was found in the small intestine of the full-term new born by Ibrahim and Gross,[3] but its activity was slight. In the premature it is probably even more deficient.

Erepsin splits albumoses and peptones and originates from the mucosa of the small intestine. It has been demonstrated in the premature by Langstein,[4] Jæggis,[5] Cohnheim[6] and others.

The next group consists of the carbohydrate ferments, of which the milk-sugar ferment *lactase* is found in the intestinal contents, the stools and the intestinal mucosa. It is frequently absent from the intestinal tract of the premature, as it makes its appearance rather late in fetal life. Nothmann[7] was able to demonstrate it in the stools of the mature new born in only a few cases. The presence of relatively large amounts of milk sugar in the infant's food probably increases the amount and activity of the lactase. The deficiency of lactase at birth is indicated further by the finding of lactose in the urine of new-born infants (Nothmann). This would seem to point to a lack of milk-sugar fermentation (von Reuss).

The cane-sugar splitting ferments, *invertin* and *saccharase,* are present at an early date in embryonal life, although there is no use for them in those fed on human milk or where lactose is used artificially, for a long period of time. They are found in the intestinal walls and in the meconium.

Maltase is present, according to Ibrahim,[8] in all parts of the small intestines and in the intestinal contents of prematures. *Diastase,* the amylolytic ferment, is present in the salivary glands and in the pancreas of the new born. *Ptyalin* is found in the parotid and in the submaxillary secretions, although it is not required until the beginning of the starch feeding. Ibrahim believes

[1] Verhandl. d. deutsch., Gesellsch. f. Kinderh., Koln, 1908, p. 36.
[2] Arch. Pediat., 1906, **23,** 414.
[3] Jahrb. f. Kinderh., 1908, **68,** 232.
[4] Jahrb. f. Kinderh., 1908, **68,** 9.
[5] Zentralb. f. Gyn., 1907, 1060.
[6] Ztschr. f. Physiol. Chem., 1903, **37,** 467.
[7] Monatsschr. f. Kinderh., 1909, **8,** 377.
[8] Ztschr. f. Physiol. Chem., 1910, **64,** 95.

that the pancreatic function of the new born and especially of the premature new born is somewhat below that of the older infant, and, therefore, the instructions of the older clinicians not to feed these infants mixtures containing much starch were correct from a physiological point of view (von Reuss).

The third and last group of ferments are those which act upon the fats. *Steapsin* was found in the pancreatic secretion by Zweifel,[1] and Ibrahim[2] showed that it was also present in the premature. The meconium contains this ferment. *Lipase* is very active in the gastric mucosa of prematures.

In general the premature may be said to possess nearly all the ferments necessary for the breaking-down of its food. Some of them, such as diastase and ptyalin, which are not present during fetal life or only in the most insignificant quantities, are called forth even in the premature, by the administration of food, and though they may be deficient both in amount and in activity at this time, the continued stimulation offered by food soon results in a material increase in both qualities, at least in the case of prematures who possess a sufficient degree of vitality. All necessary ferments being present, it is of little advantage to feed the premature infant predigested human milk.

Ferment therapy also is of little value in premature infants as is also true in older children. If the required ferments are present they will increase with the giving of food. It is not the absence of ferments that is responsible for the peculiarities of action of the digestion of the prematures, but rather the way the food is broken down and absorbed; and a clear realization of these differences is necessary to an understanding of the peculiarities of the digestion of the new born, both premature and at term.

The normal *gastric mucosa* provides only for the absorption of salts and carbohydrates.

Ganghofer and Langer[3] found that up to the fourth day of life the *intestinal tract* is permeable to foreign proteins and the importance of this is great. The permeation of these through the intestinal wall results in the formation of antibodies in the tissues, and the danger of sensitization of the organism to that particular protein. Herein lies one of our most important indications for feeding with *human milk*.

The intestinal canal is more frail than in the full-term infants and the intestinal musculature is weak and easily distended and often times unable to expel the contained meconium.

The *meconium* begins to be formed at the fourth fetal month.

[1] Untersuchungen über das Verdauungsapparat der Neugeborenen, Berlin, 1874.
[2] See p. 62, Ref. 1.
[3] München. med. Wchnschr., 1904, 1497.

It is made up of the secretions of the gastro-intestinal tract, vernix caseosa, threads of mucus, desquamated epithelium, biliary acids and salts, cholesterol, fat droplets, fatty-acid crystals and liquor amnii which has been swallowed. That which is passed on the first day is dark green, thick, sticky, homogeneous and odorless. Its excretion lasts from twenty-four to ninety-six hours. During the first few hours it is free from bacteria and even later the number of organisms present is small. The characteristic yellow color of the breast milk stool is scarcely established before the fifth and sixth day and then only when the milk taken is rich. The sour odor of the breast-milk stool may also be recognized at this time.

Hymanson and Kahn,[1] investigating the properties of meconium found that there were traces of ammonia and amylase, but no uric acid, trypsin, erepsin, lactase or lipase. Their analysis of the inorganic constituents is given in the table which follows:

	1.	2.	3.	4.	5.
Parts per thousand:					
Water	732.3	801.7	784.5	697.7	718.6
Dry matter	267.7	198.3	215.5	302.3	281.4
Organic matter	245.2	180.1	197.7	280.5	257.9
Ash	22.5	18.2	17.8	21.8	23.5
Ash percentage of:					
Total meconium	2.25	1.82	1.78	2.18	2.35
Dry matter	8.3	9.1	8.2	7.2	8.3
Analysis of meconium ash (per cent):					
Fe_2O_3	3.17	1.17	2.24	0.92	1.44
CaO	18.24	......	17.55	21.18	16.34
MgO	4.21	8.05	6.17	6.18	4.75
P_2O_5	12.62	8.62	......	......	11.70
SO_3	23.14	25.63	18.47	28.30	24.32
Cl	5.86	7.12	6.89	5.34	
K and Na	24.19	......	33.72		

3. *Bacteriology of the Gastro-intestinal Tract.*—The gastro-intestinal tract of a healthy premature is sterile at birth and remains so for a short time afterward, the meconium remaining sterile for about twelve hours. This is followed by invasion of bacteria, most probably with the first feeding, and during the next two days the gastro-intestinal flora is very variable, depending chiefly on the surroundings of the infants. After the third day, however, a typical intestinal flora develops, the type depending chiefly upon the diet of the infant.

In an infant fed with human milk saccharolytic bacteria predominate, the chief one being Bacillus bifidus, which is especially

[1] Study of the Intestinal Contents of Newly Born Infants, Am. Jour. Dis. Child., February, 1919, **17**, 112.

numerous in the large intestine up to the sigmoid flexure. This portion of intestine also contains the largest number of bacteria. Bacillus coli is also present, especially in the region of the ileocecal valve and cecum, but still Bacillus bifidus predominates. The flora of an infant on human milk are much more homogeneous than that of an infant artificially fed.

In artificially-fed infants there is a relative increase of Bacillus coli and of proteolytic bacteria and a diminution of Bacillus bifidus. However, the flora of artificially-fed infants are much more variable and depend chiefly on the chemical composition of the food.

Human milk low in protein and high in sugar leads to the flora of fermentation, while cows' milk which is high in protein and low in sugar leads to flora of putrefaction.

Carbohydrates favor the development of fermentative organisms, lactose favoring especially Bacillus bifidus and maltose and dextrin compounds favoring Bacillus acidophilus.

Proteins favor the development of organisms of putrefaction, especially when given in excess.

Fat seems to have no distinctive action on the intestinal flora.

Metabolism of Premature Infants.—The following facts are quoted from Jaschke,[1] who states that there is not sufficient material on hand at the present time for comparing the metabolism of prematures both healthy and debilitated with the metabolism of mature normal infants.

"The expenditure of energy as related to the unit of body surface is in the premature much greater than in the mature new born (Camerer), when the age is calculated from birth; on the other hand, however, they are almost the same, if age is calculated from the time of conception (Pfaundler) which very well agrees with the curve of the potential of life. The nitrogen under balance in the premature lasts longer than in the mature new born, which is probably dependent in the first place upon the small food intake. There are not sufficient experiments on the gaseous exchange and on the insensible perspiration to enable one to draw conclusions that would be of general value. There is nothing known of mineral metabolism."

Nervous System.—The lack of development of the cerebrospinal nervous system is greater than that of the sympathetic system. It is most markedly evidenced by the muscular inertia shown by the infant. Many of them lie in a state of stupor or somnolence from which they must be aroused to be fed. Others can be aroused by external stimulation which calls forth only a weak cry and slight movements of the body. These movements are slower than those

[1] See p. 18, Ref. 1.

of the full-term infant and the child tends to relapse into a deep sleep as soon as the stimulus is removed. Also depending to some extent upon the incomplete development of the nervous centers are the weak respiratory functions and the feeble efforts at sucking. At this time the development of the brain is still going on and the separation of the white and gray matter is not yet completed.

The nasal and pharyngeal reflexes are particularly weak in children born before term. Abdominal reflexes are almost never present in the premature; in fact they are rarely seen in the newborn infant.

Among many neurologists the opinion is prevalent that prematurity predisposes to idiocy, imbecility and epilepsy. However, it appears in these instances it is not so much the premature birth that is responsible, but rather there seems to be a common cause leading to retarded development and premature expulsion of the fetus.

Cardiovascular System.—As compared with other organs the *heart* is relatively well developed. That the heart should be strong is not surprising, as from the first months of pregnancy the precocious development of this organ is found to be in complete accord with the importance of its function. The high position of the diaphragm and the equality of the diameters of the thorax causes the long axis of the heart to lie in a more nearly transverse position. Because of this position the apex beat is found in the fourth interspace, 0.5 to 1 cm. outside the mammillary line.

The variability of the *pulse-rate,* which is quite marked in the premature new born, ranges from 90 to 200 per minute, with an average of about 120. This variability is the result of the lack of development of the cardiac inhibitory centers.

At birth the thoracic respirations determine a considerable flow of blood through the pulmonary artery to the lungs. The function of the ductus arteriosus ceases at this time and the blood current is diverted from the foramen ovale through the tricuspid orifice into the right ventricle. Within twenty-four to forty-eight hours after birth the ductus arteriosus is almost completely closed normally, while the foramen ovale is soon completely occluded by the rapid growth of its valvule. If, however, the ductus arteriosus is not closed, as is frequently the case in the premature infants, due to non-expansion of the lungs with a resulting increased resistance in the lesser circulation, cyanosis may result.

The heart is usually only secondarily involved in asphyxial attacks, the tones becoming weak and slow during the spells of cyanosis. The heart action often persists for hours after the respiration ceases. Myocardial asthenia in the premature may also result in cyanosis and is frequently accompanied by edema. (See Cyanosis.) General circulatory difficulties may also be the cause of subnormal temperature in these infants.

Blood-pressure in the mature at birth and for the first few days of life is low and in the prematures and weaklings it is still lower. In the new born the pressure ranges around 80, while the figures for the premature and the weaklings will vary from 60 to 70 mm. of mercury (Trumpp).[1]

The *vascular walls* in the premature are weaker than in the infant at term and because of this these children are subject to hemorrhage following relatively slight traumata. This is particularly true of the intracranial vessels and thus we see that hemorrhages in this region are relatively more frequent in the premature.

The intracranial hemorrhages are usually followed by early death and in many instances undoubtedly these are interpreted as respiratory deaths because of the influence of pressure on the respiratory center.

This tendency to hemorrhage in the premature in some cases is due to deficient coagulability of the blood.

In a study of the new-born Rodda[2] found by his method that the average coagulation time was seven minutes, with a normal range between five and nine minutes.

There is a prolongation of coagulation and bleeding times from the first day to the maximum on the fifth day of life, with a return to the average first-day determination time before the tenth day. It is significant that this coincides with the age incidence of hemorrhagic disease and cerebral hemorrhage.

In icterus neonatorum normal coagulation and bleeding times were found.

Several cases of melena neonatorum gave markedly prolonged coagulation times—up to ninety minutes—and bleeding times of hours, days or until the condition was controlled.

Suspected and mild cases of congenital syphilis gave normal findings. Severe and progressive cases gave prolonged times.

Pfaundler[3] found a low alkali reserve in debilitated prematures and believed this to be an important factor in the low immunity to infection. The blood also shows an increased viscosity due in all probability to the increase of water loss over intake during the first days. Rusz[4] has also suggested, as a second factor, the delayed tying of the cord, with a resulting flow of blood from the placenta, causing a relative overloading of the fetal circulation.

The *cell content* of the blood of the premature does not differ greatly from that of the new-born infant, though it does possess certain special characteristics (Kunckel).[5]

[1] Jahrb. f. Kinderh., 1906, **63**, 43.
[2] Am. Jour. Dis. Child., April, 1920, **19**, 269.
[3] Verhandl. d. Ges. f. Kinderh., Breslau, 1904, **21**, 24.
[4] Monatsschr. f. Kinderh., 1911, **10**, 360.
[5] The Changes in the Blood of the Prematurely Born and Weaklings, Ztschr. f. Kinderh., July 26, 1915, **13**, 101.

The *erythrocytes* are slightly less in number and diminish readily under the influence of infections, jaundice and edema. Macrocytes and microcytes are very numerous and poikilocytosis is also often observed. *Nucleated erythrocytes* are characteristic of the blood of the premature infant and the farther the child is from term the more numerous are these nucleated cells. In the mature infant nucleated cells are only found during the first few days of life, while in the premature they are found as late as ten days after birth. A large number of these cells is incompatible with life. They reappear quickly with the onset of any infection and are slower to disappear when the temperature is subnormal. With redevelopment of a subnormal body temperature during the first weeks of life, the nucleated reds tend to reappear.

The *leucocytes* are less numerous or only slightly increased. Instead of 12,000 to 13,000 leucocytes, as found in the normal full-term new-born infant, there are on the average 8000 in one cubic millimeter in the premature.

The *differential count* shows a high percentage of mononuclears and abnormal elements, such as mast cells (basophiles) and myelocytes. These cells possess little activity, which is an important factor in the low resistance of these infants to disease. What bearing the lowered alkalinity has on the feeble reaction of the white cells is still an open question. The reaction to infection is, as a rule, very feebly polynuclear and may even be replaced by one of transitional forms and abnormal elements, myelocytes and mast cells, as if the hematopoietic organs were only capable, in their deficiently developed states, of putting into the circulation immature elements. The polynuclear eosinophiles are fewer in number in the premature and disappear when an infection occurs. In congenital syphilis they are usually increased.

While in the normal full-term infants the *hemoglobin content* gradually sinks and at the end of the fourth week amounts to about 85 per cent (by Sahli's hemoglobinometer), in the prematurely born infants its value at this time is 50 to 60 per cent; therefore, in prematurely born infants there is a distinct and very early hemoglobin impoverishment of the blood, which reaches its maximum in about the third to the fourth month. While the hemoglobin content shows a marked deviation from the normal, the number of erythrocytes is only little below the normal and therefore the hemoglobin content of the individual blood corpuscle is considerably less than normal. This accounts for the constant and early development of anemia in prematures during the first three months of life. The cause of this hemoglobin deficiency seems to be an insufficient iron content of the premature's blood, which is easily understood when we recall that Hugouneng[1] has proven that the quantity of iron stored up by the fetus in the last third of pregnancy is twice as large as that during the first two-thirds.

Lichtenstein's[2] studies on a large number of premature infants showed a considerable degree of anemia in a large percentage of his cases.

In a study of the blood findings in 90 prematurely born infants (those of known syphilitic and tuberculous parentage were excluded) in greater part born one or two months before term, Lichtenstein recorded the following findings:

In 10 cases he found:

Age.	Hemoglobin.	Red blood cells.	White blood cells.
First day of life . . .	96.7	6,135,000	7.512
Third day of life . . .	90.7	5,799,000	5.755
Tenth to twelfth day of life	85.8	5,376,000	8.572

The hemoglobin and red cell counts were relatively those of the full term, showing an absence of congenital anemia. The white blood cells were below the averages given for full-term infants and presented an absolute leukopenia.

There was also a more marked anisocytosis, polychromatosis and erythroblastosis than is seen in the blood picture of the full-term new-born.

Subsequent examinations of the blood in 19 premature infants breast fed over two weeks by healthy mothers gave the following averages:

Age.	Hemoglobin.	Red blood cells.	White blood cells.
3 to 4 weeks	76.0	4,023,000	7,560
2 months	50.5	3,616,000	8,720
3 "	40.2	2,945,000	7,042
4 "	40.5	3,065,000	7,969
5 "	44.0	3,733,000	7,969
6 "	40.0	3,740,000	7,969

These results, when compared with examinations of wet-nurses' infants, showed a decided oligochromemia (controls never under 55); and oligocythemia (controls rarely under 4,000,000). The red cells increased toward the end of the first half year of life. The white cell counts for full-term infants usually averaged between 10,000 and 12,000, those of the prematures after the fourth week between 7000 and 9000.

There is also a constant anisocytosis and anisochromemia which changes run parallel with the oligochromemia. Erythroblasts were found in some cases as late as the fourth month.

[1] Compt. Rend. de l'Acad. des Sc., April, 1899, 128.

[2] Svenska Lakaresa As Kapets Handlinger, Stockholm, December 31, 1917, No. 4, 43.

The percentages of the various white cells do not vary greatly from the picture of the full-term infant. Metamyelocytes were occasionally seen as late as the second month. The figures are tabulated in the following tables:

WHITE BLOOD CELL PERCENTAGES (LICHTENSTEIN).

Day of examination.	Neutrophile leucocytes, per cent.	Eosinophile leucocytes, per cent.	Small lymphocytes per cent.	Other lymphocytes per cent.	Large mononuclears, per cent.	Metamyelocytes, per cent.
1st day . .	45.8	1.8	11.6	18.5	8.2	13.4
3d day . .	31.0	3.1	23.5	27.5	8.7	5.9
10 to 12 days	27.9	3.2	20.3	33.2	11.2	3.8

WHITE BLOOD CELL PERCENTAGES IN THE LATER MONTHS (LICHTENSTEIN).

Age of infants.	Leucocytes.			Lymphocytes.			Large mononuclears.			Eosinophile.
	Max. per cent.	Min. per cent.	No. cases.	Max. per cent.	Min. per cent.	No. cases.	Max. per cent.	Min. per cent.	No. cases.	Per cent.
0.5–1 month	33.5	5.8	5	76.5	51.5	5	14.5	1.0	5	2–3
2 "	20.3	7.3	8	82.8	56.5	8	17.3	6.3	8	2–3
3 "	41.5	9.5	6	79.8	44.0	6	12.0	3.5	6	2–3
4–6 "	35.0	29.8	4	60.8	48.0	4	14.3	4.8	4	2–3

Lande[1] examined a group of 70 prematures born from the sixth to the eighth month of pregnancy, with weight from 830 to 2500 gm. The majority were fed on human milk, made an average monthly gain of 450 gm., and were relatively free from infection and congenital lues. The results of examination of the hemoglobin content and the percentage of the red and white blood cell elements in the newly born prematures during the first weeks of life are shown in the following table:

Age of infants.	No. cases.	Hemoglobin.			Erythrocytes.			Erythroblasts.		White blood cells.		
		Max. per cent.	Min. per cent.	Commonest value, per cent.	Max. cc.	Min. cc.	Commonest value.	Max.	Min.	Max.	Min.	Commonest value.
1 day	12	140	100	110–120	5.8	3.8	4.3–5.0	7000	400	20,000	3800	10,000 to 15,000
2–4 "	15	135	100	125	6.7	4.1	4.6–5.4	6700	0	16,000	3600	8,000 to 12,000
6–8 "	6	105	100	....	5.6	4.0	....	160	0	11,400	6600	....

[1] Ztschr. f. Kinderh., 1919, 22, 299.

Age of infants.	No. of cases.	Leukocytes.			Lymphocytes.			Large mononuclears.			Eosinophiles.	Mast cells.	Myeloblasts and myelocytes.
		Max., per cent.	Min., per cent.	Commonest value per cent.	Max., per cent.	Min., per cent.	Commonest value per cent.	Max., per cent.	Min., per cent.	Commonest value per cent.			
1 day	12	54	12	40–50	85	39	45–55	12.5	1	7–10	0.5–1.5	0–1.5	3–12.5
2–4 "	15	64	11	40–55	87	30	40–55	12.0	2	.5–10	0–5.0	0–2.5	2– 6.0
6–8 "	6	68	29	.....	65	26	.....	8.0	6	....	0–2.0	0–2.0	0– 2.0

From these tabulations Lande drew these conclusions: Opposed to the findings in full-term infants, there is in prematures a greater richness of nucleated red blood corpuscles, a more frequent appearance of myeloblasts and myelocytes during the first days of life, a lesser development of absolute and relative leukocytosis, and a greater number of immature leukocyte forms.

The blood picture from the third week of life to the age of six months is expressed by the following figures (Lande):

Age of infants.	No. of cases.	Hemoglobin.			Erythrocytes.		
		Max., per cent.	Min., per cent.	Commonest value, per cent.	Max., Millions.	Min., Millions.	Commonest value, Millions.
1.0 month	13	105	70	80–85	5.5	3.3	3.6–4.6
1.5 "	9	95	50	60–70	3.9	2.7	3.2
2.0 "	17	80	50	60–70	4.4	2.4	3.0–3.6
2.5 "	7	80	50	60–65	4.0	2.3	3.0–3.6
3–3.5 "	24	80	50	60–70	4.9	2.4	2.9–3.9
4–4.5 "	18	75	50	60–70	5.2	2.7	3.3–4.0
5–5.5 "	15	85	55	65–75	4.7	3.4	3.9–4.6

Lande noted a fall in the hemoglobin content from 80 to 85 per cent to 60 to 65 per cent in the third month, which slowly rises to 65 to 75 per cent in the sixth month. At the same time the number of erythrocytes sinks from about 4,000,000 to 3,300,000, in order to again approach the normal value by the end of the first year.

Nathan and Langstein[1] have found the blood in the new-born very low in antitoxic, bactericidal and hemolytic properties.

Blood-sugar determinations in three healthy prematures fed on mother's milk were studied by Heller.[2] In no case was sugar

[1] Ztschr. f. Kinderh., 1919, **22**, 299.

[2] Der Blutzuckergehalt bei Neugeborenen und Frühgeborenen Kindern, Ztschr. f. Kinderh., 1913, **9**, 44.

found in the urine, this being explained by the fact that in no instance was there an evident hyperglycemia. The percentage of blood sugar noted between ten and a half and twelve hours after birth was 0.095, 0.089, 0.082; these figures are for infants weighing respectively 1500 gm., 1380 gm. and 930 gm. The diets were increased so that on the seventh day the two heavier infants were both getting 160 gm. while the smaller was given 80 gm. of milk. The percentage of blood sugar was then noted; 0.104 for both heavy infants (twins) and 0.065 for the other. All observations were taken from one-half to two hours after the feeding. There was a steady fall in blood sugar in the twins until the sixth day.

These blood-sugar findings are similar to those of Götzky,[1] who found an average of 0.085 mg. in the full-term new born, somewhat lower findings in prematures, and 0.095 mg. in later infancy, as compared with 0.102 mg. in later years. Because of the relationship of blood sugar to diet, comparative studies must be undertaken with a knowledge of the quantity and quality of food and the time to the meal.

Lymphatic System.—This is well developed and does not differ materially from that of the new born, unless possibly its circulation is slowed as a result of the slowing of the general circulation.

Thymus and Thyroid Glands.—These organs present the highest degree of development of any glandular structures. In fetal life these organs contribute to the formation of blood and during the first few weeks of life have a phagocytic action.

Genito-urinary System.—In the female the labia minora usually overlap the labia majora, while in the male the testicles are often high in the inguinal canal, though it can happen that they are found in the scrotum as early as the seventh month.

An examination of the urine of the premature throws but little light upon the *metabolism* of these infants. The proportion of ammonia N to the total N is below normal, while $\frac{C}{N}$ is increased. This speaks for an increase in the processes of decomposition. Nobecourt and Lemaire[2] found that the freezing-point of the urine of prematures was lowered.

The amount and character of the *urine* during the first few days of the life of the premature depend upon the intake of fluid, upon the absolute body weight and upon the absolute and relative amounts of water within the body tissues.

If the quantity of fluids taken is small the amount of urine secreted is proportionately small. When the quantity is larger, as is usually the case if the infant is given feedings to substitute

[1] Ztschr, f. Kinderh., 1913, **9**, 44.
[2] Quoted by Pfaundler.

the mother's milk and frequent feedings of water, the relative as well as the absolute amount of urine secreted is larger. Cramer[1] found that with an abundant supply of milk during the first few days of life the urinary output amounted to 54 to 60 cc for every 100 cc of milk consumed.

The frequency of urination during the early days of the premature is less than at an older age. While the infant may urinate during its passage through the birth canal or immediately after, yet, as a rule, during the first few days the urinations are very infrequent, at most three or four and more often only one or two times during the twenty-four-hour period. It is not at all uncommon that no urine is passed during the first day. This failure to urinate during the first day of life is not of much moment, but in those instances of absence of urination for as long as four days, as have been reported, some anomaly was undoubtedly present. With the increase in the fluids taken, which occurs usually on the third or fourth day, the frequency of urination also increases.

During the period of greatest concentration the *reaction* of the urine is strongly acid. As it becomes more dilute, the acidity becomes less marked.

Albuminuria is a symptom shown by almost all infants just after birth, full-term as well as premature. The length of time during which this persists is short, seldom more than the first few days, and the quantities of albumin present are small: 0.25 gm. of albumin per 100 cc of urine is a maximum which is frequently reached in the full-term infant. Von Reuss[2] found the urine of only 4 per cent of new-born infants to be free from albumin during the first four days of life. After that time the amount of albumin present rapidly falls, unless the concentration of the urine remains high, and it retains the turbidity characteristic of infant urine, when the albumin persists for a longer period.

Albumin in the urine of the new born would seem to be somewhat of a physiological condition, certainly having no relation of a causal nature to the infections or other toxic factors of the later periods of life. Albuminuria at this time seems to have a certain analogy with the orthostatic albuminuria of older individuals, both probably to be accounted for by circulatory disturbances of the kidney. Von Reuss believes the condition is most easily explainable on the basis of circulatory stagnation which occurs in a more or less pronounced degree after every delivery. Uric-acid infarcts may also have some bearing as a cause of albuminuria. The deficient blood supply of the kidney and the lack of water passing through the organ as a result of the small quantity taken during

[1] Arch. f. Kinderh., 1901, **22**, 1.
[2] Verhandl. d. Ges. f. Kinderh., Münster, 1912, **29**, 145.

the first few days of life probably increases the amount of albumin passed.

Nothmann[1] found *milk sugar* in the urine of premature infants who were breast fed, and he reports that he found no such cases among the full-term infants. *Sugar* was found by Hoeniger[2] in the urine of several infants delivered by forceps. It was excreted for three or four days and then gradually disappeared. It was believed to be the result of the force used during the operative delivery.

Acetone bodies are found in small quantities in the urine of poorly nourished and underfed weaklings.

During icterus neonatorum *bilirubin* occurs in the urine in the form of a precipitate. It is also found in solution in septic conditions and in hemorrhage of the new born.

Creatin and *creatinin* have not been studied in the premature.

Occasionally *hyaline casts* in small numbers, often covered with urates, are seen. These are probably due to the same causes as the albumin and have no pathological significance.

Special Senses.—Over the eyes of the youngest prematures occasionally there can be seen more or less well-marked vestiges of the pupillary membrane, the cornea is inclined to be somewhat thicker, the anterior chamber less deep and the iris less pigmented. Strong light impressions are followed by reflex closure of the lids, but sudden movements are not followed by such closure, as the reflex is psychic, depending upon fear.

The *eye movements* of the premature infant are incoördinated, motion being most often in a horizontal direction, occasionally outward, but more often and in a comparatively strong manner, inward. It is not uncommon to see this tendency to convergence persist until the second month. The *light reflex* is present before birth and the pupil, when exposed to a strong light, contracts only to dilate again in two or three seconds. This secondary dilatation is particularly well marked in the premature as a result of the poor development of the nerve fibers, which are easily fatigued (Furmann).[3] The convergence reflex is absent in prematures as well as in the more mature infant because fixation does not occur.

Skin and Adnexa.—The skin is thin, soft and usually of a more or less vivid red appearance, occasionally of a peculiar cyanotic hue, and the transparent dermis allows the circulatory network to be clearly distinguished. The skin is partly or completely covered with lanugo hairs which are seen most commonly between the

[1] Monatschr. f. Kinderh., 1909, **8**, 377.
[2] Deutsche med. Wchnschr., 1911, 500.
[3] Die Reflexe der Säuglinge. Diss., Petersburg, 1903, Loc. cit., Gundobin, Berlin, 1912.

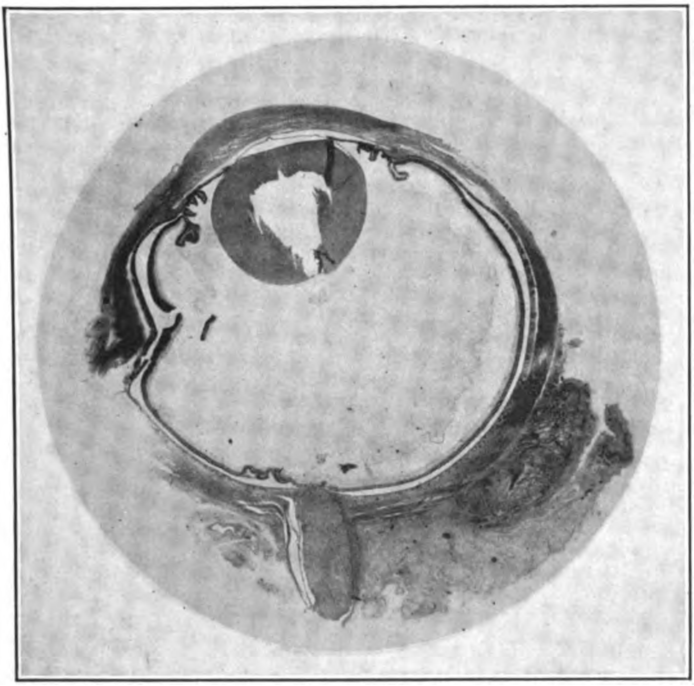


FIG. 27.—Embryological eye section. (Normal size and enlarged 5 diameters.)

The *conjunctiva* has reached its full development and shows subconjunctival lymph-follicles beginning to develop into separate entities.

The *cornea* is still in the developmental stage and shows some interesting conditions. The corneal epithelium is uniform and is a two-cell layer well developed and without mitotic figures. Bowman's membrane is just beginning to be differentiated from the anterior corneal stroma, but does not form an entity as yet. The development of the membrane is not uniform throughout but appears in scattered areas and without continuity. (This would tend to place the specimen in the first half of the fifth month). The anterior corneal stroma is well developed and is dense. The posterior corneal stroma is well developed. Both stroma show fixed corneal cells. Decemet's membrane is fully developed and is intact from angle to angle.

The *anterior chamber* has begun to form by the retraction of the anterior lens capsule and pupillary membrane from the posterior surface of the cornea and the iris is pushing into the anterior chamber in front of the lens. The chamber angle is already differentiated and wide spaces exist in the pectinate ligament, much wider than in adult life.

The *iris* is recognizable as a separate entity. The anterior surface is smooth and uniformly covered with smooth endothelium. No crypts have developed as yet. (This speaks for an age of less than six months). The iris stroma is still very thin and loose, but is well vascularized. The retinal pigment epithelium of the iris is differentiated and well-developed, although the posterior layer is thinner and less heavily pigmented than the anterior. The sphincter iridis can be recognized as a separate entity and already fills the pupillary margin of the iris fairly well. Individual dilator fibers are present but the muscle as a whole is still undeveloped.

The *ciliary body* is still small and is posterior to the position occupied in adult life. The retinal pigment layer and the ciliary processes are well-developed and are fairly well anterior. But the main body is well back, is thin, and is still undifferentiated into its component parts. Bruecke's muscle can be recognized, although it has not formed into the complete muscle as yet; but Mueller's muscle is still missing.

The *lens* is nearly spherical and in the periphery can be found a few proliferating lens fibers. The anterior capsule is a two-cell layer and in intimate association in the pupillary membrane which has not entirely disappeared. No trace of vascularization remains. The posterior capsule is missing. No zone of Zinn fibers can be found.

The *vitreous* is missing.

The *retina* is distinctly behind the rest of the eye in its development. A definite separation of the layers is present, but a differentiation of rods and cones has not yet taken place. Even differentiation of the cones (the first to appear as an entity) cannot be recognized, although the external limiting membrane is developed and in place. Nerve fibers are in the process of development and their presence has swollen the optic nerve head to its usual size. There is much glia in this area. Just anterior to the optic nerve head is a bit of hyaloid artery still visible, although in the process of absorption.

The *optic nerve* is fairly well developed although there is more glia than usual in an eye of this size.

The *chorioid* is well developed and is fairly well vascularized.

The *sclera* is well developed but is rather loose in structure. (Description of specimen by Dr. Harry S. Gradle, Chicago.)

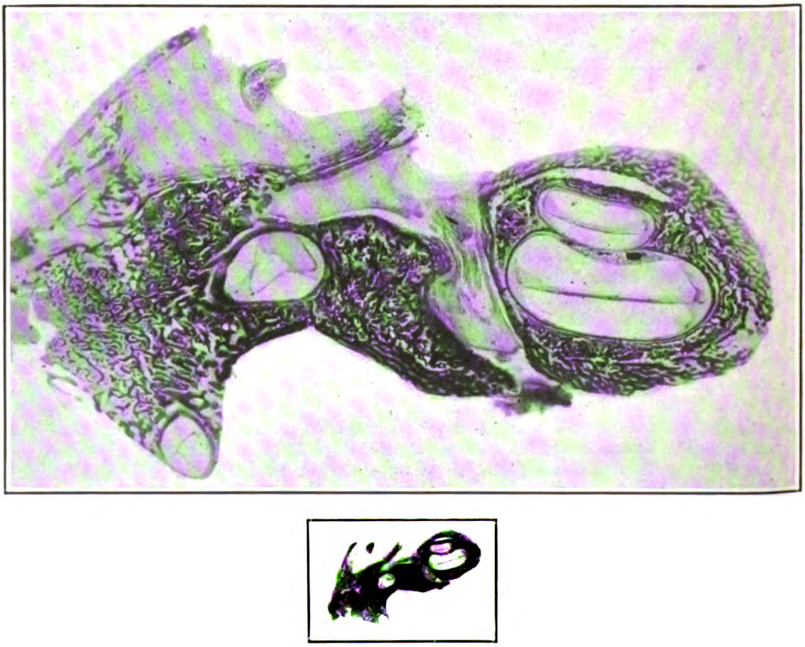


Fig. 28.—Embryological section of petrous portion of temporal bone. (Normal size and enlarged 5 diameters). Vertical section through the petrous portion of the temporal bone of a fetus of five and a half months, exposing the cochlea, the cochlear nerve, two semicircular canals and adjacent caseous tissue.

shoulder blades, but also frequently upon the face and upon the extensor surfaces of the extremities. There is also noted extensive milium and flaccidity of the auricle and alæ nasi, whose cartilage is not properly developed.

Icterus is usually more pronounced than at term and erythema is slower to disappear. If hypothermia develops the redness of the skin usually fades.

The absence of subcutaneous fat betrays itself by an angular appearance of the face, the chin is pointed, the head is small and narrow and the wrinkles of the skin impart an oldish appearance to the face which is especially marked after a few days when the loss of weight has been material and the skin often hangs in folds over the muscles and bones. Not infrequently the skin appears glossy as if on tension and this is seen especially in small prematures in the presence of sclerema and scleredema. Patches of skin may be absent, especially over the heels.

The hairs on the scalp are short and feebly colored, the nails are often poorly developed and do not reach the end of the fingers or toes and the nose is covered with small white comedones. The navel is closer to the symphysis than at term.

Mammary Glands.—The mammary glands are, as a rule, poorly developed, usually not palpable and particularly in the younger prematures do not often attempt to secrete milk. If fluid is present, as it may be in the older prematures, it usually makes its appearance about the eighth day, is most abundant up to the fifteenth day and may last until the third month. It is equally common in either sex. In most cases the secretion amounts to only a few drops, but occasionally larger quantities are seen.

Skeletal Development.—The lack of exact anatomical data as to the skeletal development of the premature infant has caused the author to resort to the use of roentgenographic studies. The stage of ossification of the skeleton of the fetus as observed in roentgenograms is of considerable practical importance in determining the age of the fetus. In addition observation by the roentgenographic method is more reliable than determination of age based on length and other measurements, since osseous development is more regular and offers many more factors for consideration. Pathology may often be readily recognized. Our studies thus far have shown that in the early months more accurate determination is possible than in the later months, because many more new centers appear in the first months, and the time of appearance is more constant.

The study of the roentgenograms for diagnostic purposes discloses that the cephalad segments, including the upper axial skeleton and upper extremities, are far more constant as to time of development of their osseous centers than the caudad segments and those of the lower extremities. *This should be borne in mind in making comparative studies.*

The figures as to length and other measurements of the fetus have been discussed earlier (p. 29). Basing our facts on the roentgenographic studies of a series of 55 normal cases, whose ages were determined from the history of menstruation and pregnancy and from their measurements, the normal process of development of the human skeleton was found to be as follows:

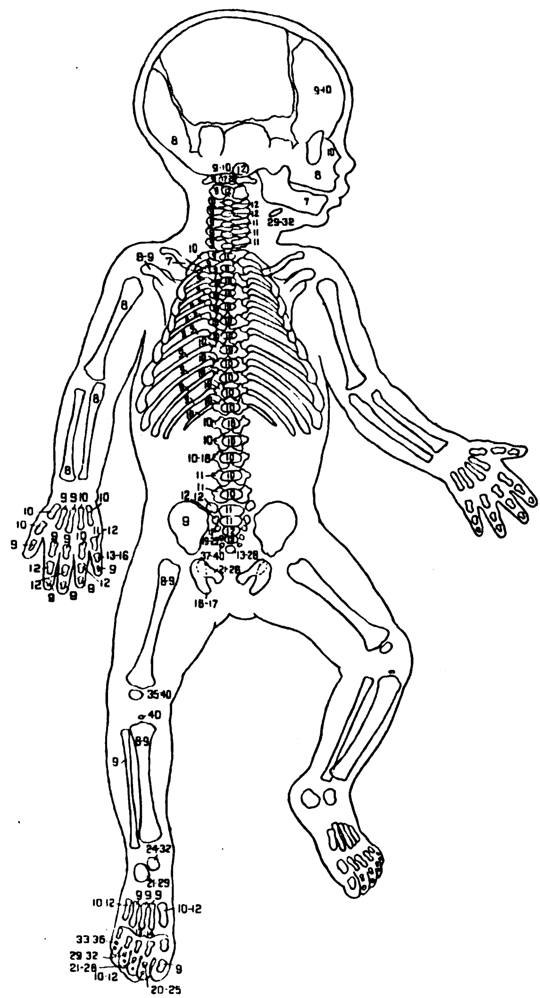


FIG. 29.—Development of centers in weeks. Diagram showing osseous development of infant at full term, and development of ossification centers in weeks. Centers shown which are frequently absent at birth: (1) head of tibia; (2) coccyx. Centers omitted in outline: (1) sternum; (2) hyoid.

Other Measurements of the Fetus.—Von Winckel regards the circumference of the head as of importance for the diagnosis of the age of the fetus and gives the following figures:

4th month	10–14 cm.	8th month	25–30 cm.
5th "	13–18 "	9th "	29–33 "
6th "	19–24 "	10th "	32–37 "
7th "	23–28 "		

The weight is entirely unreliable for the estimation of the age of the fetus, because it is subject to too many variations and is much influenced by the mother's general condition, and more especially by her diet.

Thus, it is seen that even the length, which up to this time has been regarded as the most reliable criterion for the determination of the age of the fetus, has many shortcomings and may result in an error of several weeks.

The ossification of the human skeleton begins in the upper part of the body and spreads very rapidly in both directions.

FIG. 30

FIG. 31

FIGS. 30 and 31.—Roentgenogram Fig. 30 and diagram Fig. 31 of fetus at seven weeks, actual size.

Seventh Week.—The first centers of ossification develop in the clavicles in the sixth to seventh week of intra-uterine life (Kreibel-Mall, Rauber-Kopsch[1]), but they do not become visible in the roentgenograms until in the seventh week. The ossification center appears in the middle of each clavicle and spreads rapidly in both directions.

Soon after the ossification has started in the clavicle one center appears in each half of the mandible.

Outside of these centers of ossification usually no other centers, except occasionally that of the maxilla, are visible in roentgenograms of the seven weeks' old fetus.

[1] Lehrbuch der Anatomie des Menschen, Thieme, Leipzig, 1914, **2**, ed. 10.

Eighth Week.—Osseous development makes rapid progress in the eighth week, and a large number of centers of ossification become visible at this time.

Fig. 32

Fig. 33

Figs. 32 and 33.—Roentgenogram Fig. 32 and diagram Fig. 33 of fetus at eight weeks, actual size.

The following bones show centers of ossification demonstrable in roentgenograms.

Skeleton of the head: The squamous portion of the occipital bone and superior maxilla. In the latter the ossification begins soon after that of the mandible, the center appearing above the region where the alveolus of the incisor tooth is later located.

TABLE 3.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

HEAD

Mandible	7th week
Occipital bone (squamous portion)	8th week
(lateral and basilar portion)	9th to 10th week
Superior maxilla	8th week
Temporal bone (petrous, mastoid and zygoma)	9th week
Sphenoid (inner lamella of pterygoid process)	9th week
(great wings)	10th week
(lesser wings)	13th week
(anterior body)	13th to 14th week
Nasal bone	10th week
Frontal bone	9th to 10th week
Bony labyrinth	17th to 20th week
Milk teeth (rudiments)	17th to 28th week
Hyoid bone (greater cornua)	29th to 32d week

Usually no centers of ossification are present in the axial skeleton in this week.

Shoulder girdle: In the scapula a center of ossification usually appears in the eighth week, sometimes in the ninth week. The center corresponds to the position of the middle of the spine of the scapula.

Upper extremity: The humerus is the first bone of the free extremities to show a center of ossification, which appears in the diaphysis early in the eighth week. Radius and ulna follow in the order given, the centers appearing very soon after those of the humerus.

The ribs begin their ossification in the eighth week, an ossification center appearing in the region of the angle and extending slowly toward the veretebral column, but rapidly in the opposite direction. The fifth, sixth and seventh ribs, which ossify first, are visible in this period. From this region the process of ossification progresses with equal rapidity both cephalad and caudad. The last ribs to ossify are usually the first pair. Shortly before the first pair, the twelfth pair usually ossifies, but this is very irregular and we found it absent in several of our cases in old fetuses although other bones of the body and all the other ribs were very well developed.

TABLE 4.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

BODY	
Clavicle (diaphysis)	7th week
Scapula	8th to 9th week
RIBS.	
Ribs, 5th, 6th, 7th	8th to 9th week
2d, 3d, 4th, 8th, 9th, 10th, 11th	9th week
1st	10th week
12th (very irregular)	10th week
STERNUM.	
Sternum	21st to 24th week
UPPER EXTREMITY.	
Humerus (diaphysis)	8th week
Radius (diaphysis)	8th week
Ulna (diaphysis)	8th week
Phalanges, terminal	9th week
basal, 3d and 2d	9th week
basal, 4th and 1st	10th week
basal, 5th	11th to 12th week
middle 3d, 4th, 2d	12th week
middle 5th	13th to 16th week
Metacarpals, 2d and 3d	9th week
4th, 5th, 1st	10th to 12th week

Lower extremity: Centers of ossification may be occasionally seen in the diaphyses of the femur, but usually they become visible in the ninth week. The femur is the first to show a center, the tibia starting in its ossification a little later, and the fibula following very soon after the tibia.

Ninth Week.—Portions of the hand and of the foot enter the stage of ossification, these being the most important new developments in this week, The following additional centers of ossification are visible in the head: Inner lamella of the pterygoid process of sphenoid and mastoid portions of the temporal bone. The zygomatic process of the temporal bone begins to cast a shadow, its shape being somewhat pointed anteriorly and somewhat convex externally, thus resembling a needle. Bony trabeculæ are often seen in the posterior root of the mastoid process. The superior maxilla forms at this time a simple triangular plate, the base of which is parallel to the margin of the maxilla, the apex pointing toward the root of the nose. The malar bone may become visible toward the end of this week or during the next week.

TABLE 5.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

VERTEBRÆ

Arches, all cervical and upper 1 or 2 dorsal	9th week
all dorsal and 1 or 2 lumbar	10th week
lower lumbar	11th week
upper sacral	12th week
4th sacral	19th to 25th week
Bodies from 2d dorsal to last lumbar	10th week
from lower cervical to upper sacral	11th week
from upper cervical to lower sacral	12th week
5th sacral	13th to 28th week
1st coccygeal	37th to 40th week
structural arrangement	13th to 16th week
odontoid process of axis	17th to 20th week
Costal processes, 6th and 7th cervical	21st to 33d week
5th cervical	33d to 36th week
4th, 3d, 2d cervical	37th to 40th week
Transverse processes, cervical and dorsal	21st to 24th week
lumbar	25th to 28th week

Axial skeleton: Arches of all the cervical and upper one or two dorsal vertebræ show centers of ossification, usually no centers for bodies being visible. One center develops in each arch, the process beginning in the first cervical vertebra and proceeding caudally.

Shoulder girdle: The acromion process of the scapula begins to ossify in this week. The first formations of these centers are difficult to study in roentgenograms on account of their small size, but the later stages can be easily demonstrated. Development of the centers of ossification in terminal phalanges is followed by the appearance of centers in the metacarpals which become visible in the ninth to tenth week. The following is the order of ossification in the metacarpals: second, third, fourth, fifth, first, of which the second and the third are usually visible in the ninth week.

Ribs: All the ribs, except the first and the twelfth cast shadows.

Pelvic girdle: The ilium usually appears in this week, rarely at the end of the eighth week. Ossification begins in the region of the greater sacrosciatic foramen and near the acetabulum.

Lower extremity: Centers of ossification in femur, tibia and fibula are seen. Centers begin to develop in the phalanges, the first one being a center for the diaphysis of the terminal phalanx of the big toe. Diaphyses of the metatarsals follow in the same sequence and almost at the same time as corresponding portions of the hand, but with far less regularity.

TABLE 6.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

PELVIC GIRDLES.	
Ilium	9th week
Ischium (descending ramus)	16th to 17th week
Os pubis (horizontal ramus)	21st to 28th week
LOWER EXTREMITY.	
Femur (diaphysis)	8th to 9th week
(distal epiphysis)	35th to 40th week
Tibia (diaphysis)	8th to 9th week
(proximal epiphysis)	40th week
Fibula	9th week
Os calcis	21st to 29th week
Astragalus	24th to 32d week
Cuboid	40th week
Metatarsal, 2d and 3d	9th week
4th, 5th and 1st	10th to 12th week
Phalanges, terminal 1st	9th week
terminal 2d, 3d, 4th	10th to 12th week
terminal 5th	13th to 14th week
basal 1st, 2d, 3d, 4th, 5th	13th to 14th week
middle 2d	20th to 25th week
middle 3d	21st to 26th week
middle 4th	29th to 32d week
middle 5th	33d to 36th week

Tenth Week.—Comparatively few new centers of ossification are added in this week.

Skeleton of the head: Nasal bone and frontal bone show centers of ossification. The great wing of the sphenoid becomes visible.

Axial skeleton: Bodies of the vertebræ begin to cast shadows. The process starts in the bodies of the lower dorsal vertebræ, progressing from this region with unequal rapidity in both directions. Usually the lower ten dorsal and all the lumbar vertebræ show centers of ossification in their bodies in this week. The process of ossification of the arches, progressing downward, has become more or less advanced in all the thoracic vertebræ, invading occasionally the upper lumbar region.

Shoulder girdle: Ossification of the scapula spreads to the supraspinous fossa.

Upper extremity: Diaphyses of basal phalanges of fingers develop centers of ossification, the following being the sequence: third, second, fourth, first and fifth. Of these, usually the third, only, shows a center in this week.

Ribs: At this time ossification, as a rule, is seen in all the ribs, the twelfth behaving very irregularly. It was found absent in some comparatively old fetuses far beyond the tenth week.

Lower extremity: Beginning with this week centers of ossification are present also in the terminal phalanges of the second and of the third toes.

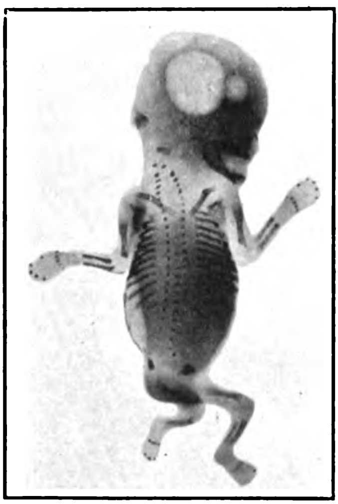


FIG. 34

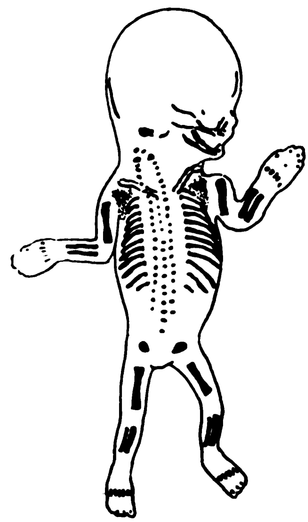


FIG. 35

FIGS. 34 and 35.—Roentgenogram Fig. 34 and diagram Fig. 35 of fetus at ten weeks, actual size.

Eleventh to Twelfth Week.—In this period almost as many centers of ossification are present in the fetal skeleton as at the time of birth, so that but few are added during the period of development following the third month, and further changes in the fetal skeleton consist mostly of growth of the centers of ossification, of their fusion and of the formation of the internal structure of the bones. A fine, somewhat irregular, medullary cavity forms in the long bones, usually being seen first in the tibia.

Skeleton of the head: The tympanic ring usually becomes visible in this week, rarely at the end of the eleventh week. In pictures taken from the side, its shadow lies between the angle of the mandible and the basilar portion of the occipital bone. The median lamella of the pterygoid process reaches considerable size and is visible as a hook-shaped, curved plate with concavity posteriorly, lying behind the lower portion of the perpendicular part of the palate bone. The malar bone joins the end of the zygomatic process of the superior maxilla and that of the temporal bone.

Four centers are now present in the occipital bone. The anterior sphenoidal body begins to ossify.

Axial skeleton: Ossification of the arches invades the lower lumbar region. The ossification of the bodies now appears in the lower cervical region and in the upper part of the sacrum, the intermediary bodies having been visible previously. There are, however, considerable variations in the time of appearance of centers of ossification in the sacral vertebræ.

Shoulder girdle: No new centers develop, the old ones increasing in size.

Upper extremity: The diaphyses of all the basal phalanges cast shadows. Middle phalanges of the third, fourth and occasionally of the second finger develop centers of ossification in their diaphyses. The middle phalanx of the fifth finger ossifies much later.

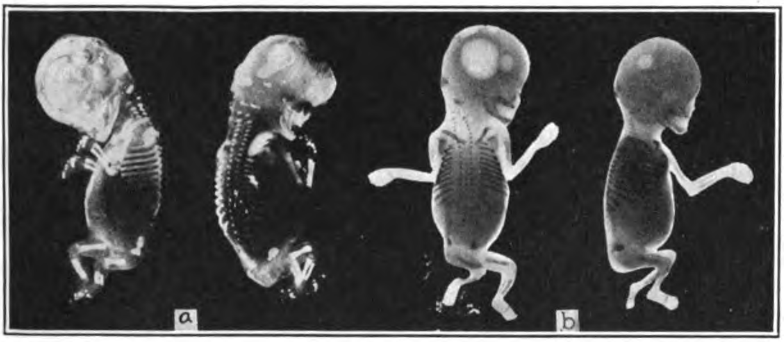


Fig. 36.—Photograph (*a*) and roentgenogram (*b*) of transparent specimens of fetus at ten weeks. One-half actual size.

Up to the end of the third month the bony diaphyses of the humerus, radius and ulna remain longer and thicker than the corresponding bones of the lower extremity.

Pelvic girdle: Either in this period or shortly after, a third center of ossification develops in the ilium, being situated ventrally from the fused first and second centers. There is a marked irregularity in the time of appearance of the third center of the ilium, since occasionally it may appear almost three weeks after this time.

Lower extremity: The terminal phalanges of the fourth and fifth toes usually develop centers of ossification; in the fifth, however, the center may occasionally appear as late as in the thirteenth week. The bony diaphysis of the femur, which up to this time has been shorter and thinner than the bony diaphysis of the humerus, has almost reached the length of the latter, remaining, however, still somewhat thinner.

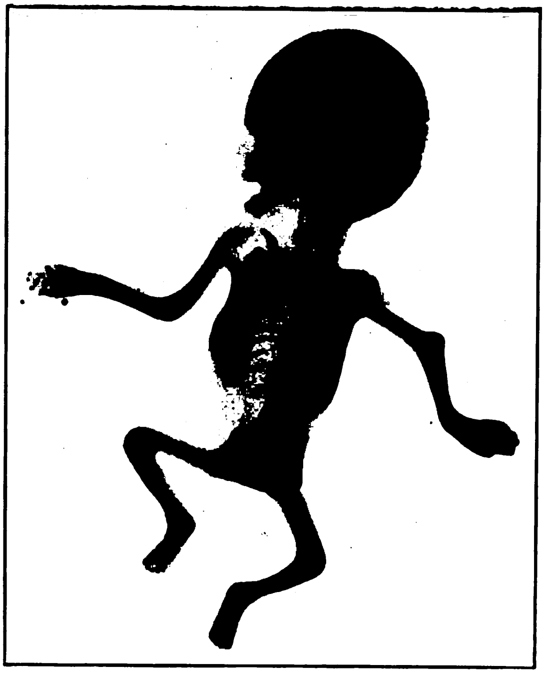


Fig. 37

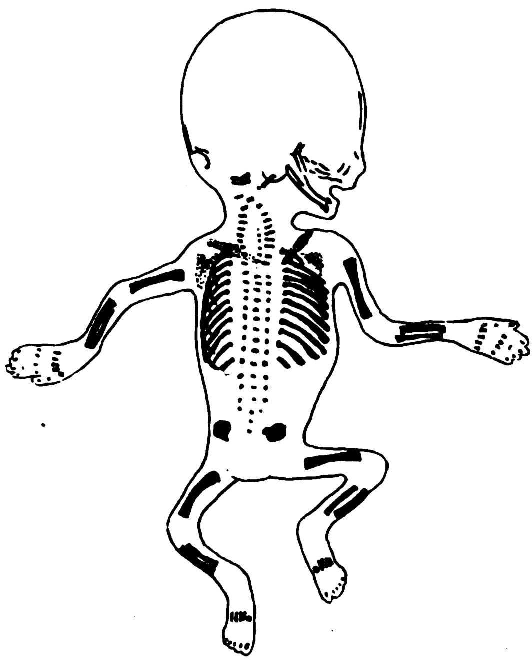


Fig. 38

Figs. 37 and 38.—Roentgenogram Fig. 37 and diagram Fig. 38 of fetus at eleven to twelve weeks, actual size.

Thirteenth to Sixteenth Week.—Characteristic in the osseous development of this period is the appearance of structural arrangement in the bodies of some vertebræ and the presence of centers of ossification in the diaphyses of all of the long bones of the hand and of the foot, except the middle phalanges of toes.

Skeleton of the head: The lesser wing of the sphenoid is visible at the beginning of this period. The posterior body of the sphenoid appears about the fourteenth week.

Fig. 39

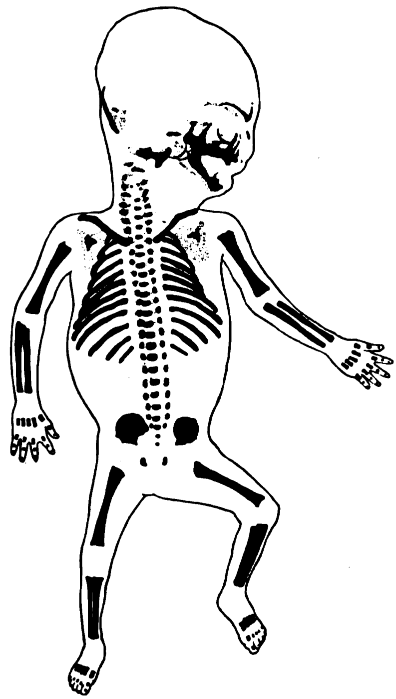


Fig. 40

Figs. 39 and 40.—Roentgenogram Fig. 39 and diagram Fig. 40 of fetus at thirteen to sixteen weeks, one-half actual size.

Axial skeleton: At the end of this period all the vertebræ, with the exception of first and second lower sacral and the coccygeal, have at least one center of ossification. Arches are ossified also in the upper sacral region and the bodies from the upper cervical down to the lower sacral region. Structural arrangement becomes visible in the bodies of some vertebræ. Upper and lower plate, casting denser shadow, become differentiated. A zone of lighter shadow is seen between these two plates and in the central portion of the body a flat, darker shadow appears. The greatest diameter of this darker shadow corresponds to the longitudinal axis of the fetus in lumbar and lower dorsal vertebræ; in other dorsal vertebræ it lies horizontally. These shadows appear in the bodies of the vertebræ in the region in which the primary centers made their first appearance.

Upper extremity: In the fifteenth to sixteenth week a center of ossification appears in the diaphysis of the middle phalanx of the fifth finger, so that at this time the diaphyses of all the long bones of the hand possess centers of ossification.

Pelvic girdle: At the end of this period or somewhat later a center becomes visible in the descending ramus of the ischium. Instead of one center, two separate centers may develop in this portion of the innominate bone and they may remain separate for a long time afterward.

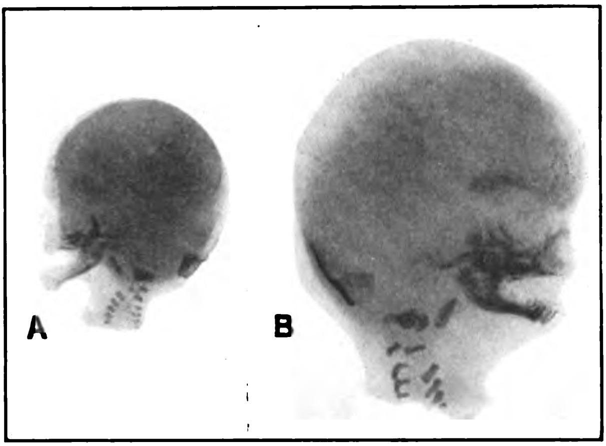


Fig. 41.—Roentgenograms of skull showing ossification centers at (*a*) eleven to twelve weeks and (*b*) thirteen to sixteen weeks, actual size.

Lower extremity: In the thirteenth week a center of ossification develops in the diaphysis of the terminal phalanx of the fifth toe, if it did not appear earlier. In the fourteenth week ossification in the basal phalanges begins, first in the big toe, and proceeds toward the fibular side in other toes, and at the end of this period it usually reaches the last toe.

Seventeenth to Twentieth Week.—In this period the bony labyrinth first appears and bone tissue begins to be formed in the rudiments of the milk teeth.

Skeleton of the head: Several new centers of ossification appear in the petrous portion of the temporal bone, but they do not show well in roentgenograms. The bony labyrinth begins its development. In the rudiments of milk teeth, bone tissue begins to be formed and casts a shadow. The process starts in the lower incisors.

Axial skeleton: A center of ossification appears in the odontoid process of the axis. The darker shadows in the bodies of the vertebræ become more distinct and external formation and internal structure of osseous bodies of vertebræ become visible in roentgenograms. Ossification of the arches may reach the fourth sacral

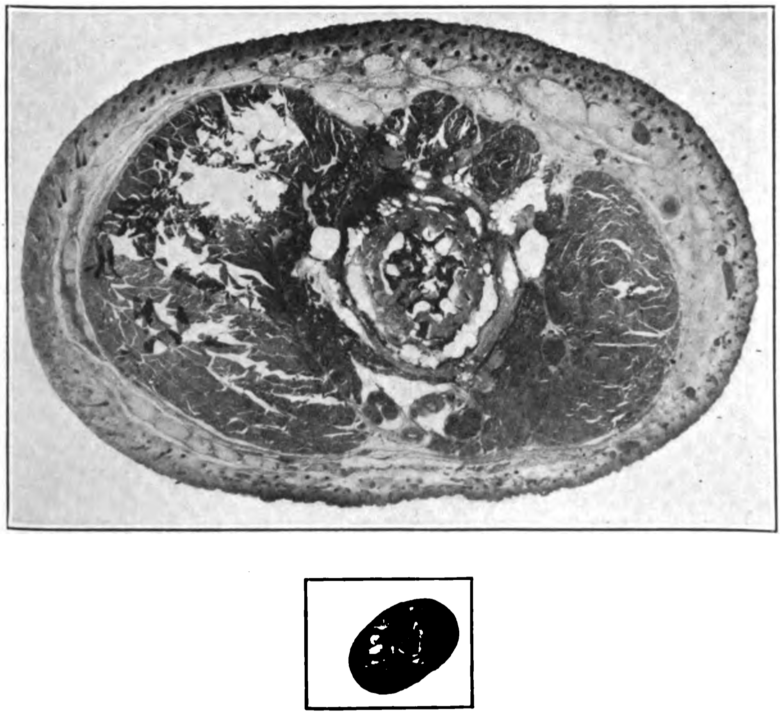
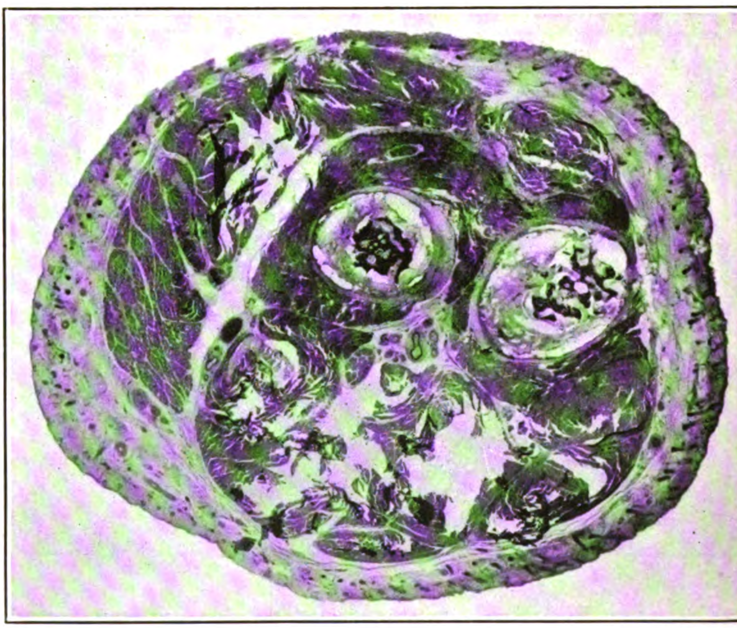


Fig. 42.—Photomicrograph of cross section of arm of twenty-two weeks fetus. Enlarged 6 diameters. Small figure actual size.

vertebra at the end of this period, although this frequently occurs later.

Pelvic girdle: The twentieth week is the earliest time of appearance of a center in the horizontal ramus of the pubic bone; this, however, varies between the twentieth and the twenty-eighth week. The center is located near the margin of the obturator foramen, two centers developing occasionally.

Lower extremity: In the twentieth week a center of ossification may develop in the middle phalanx of the second toe, but this usually occurs in the twenty-first to the twenty-fourth week and frequently even later than this. On the whole, there are marked differences and also individual variations in the time of appearance of centers of ossification, and also in the sequence of ossification in the phalanges of toes, especially in the basal phalanges and even more so in the middle phalanges. In the hand, however,



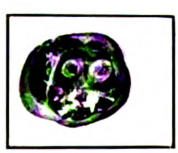


Fig. 43.—Photograph of cross-section of forearm of twenty-two weeks fetus. Enlarged 6 diameters. Small figure actual size.

the sequence of ossification in the phalanges is far more constant and the time of appearance of the centers is much less changeable than that of the centers in the phalanges of toes.

Twenty-first to Twenty-fourth Week.—In this period ossification usually starts in the tarsus, os calcis being the first to show a center of ossification. The sternum begins to develop by several centers of ossification, but there are considerable variations in the arrangement and size of these centers and also in the time of their appearance.

Skeleton of the head: The superior maxilla shows a large amount of spongiosa. Toward the twenty-fourth week the alveolar portion of the superior maxilla begins to overhang the level of the palatal plate, but develops as a real process only during the cutting of the teeth.

Axial skeleton: The costal process of the sixth cervical vertebra starts in its ossification. Shadows of transverse processes are seen in vertebræ down to the twelfth dorsal.

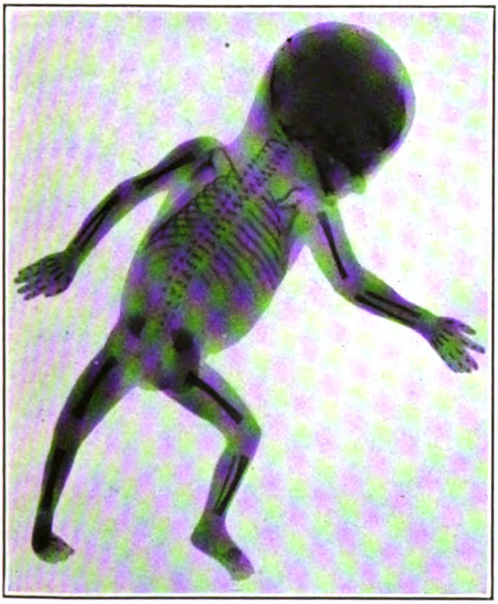


Fig. 44

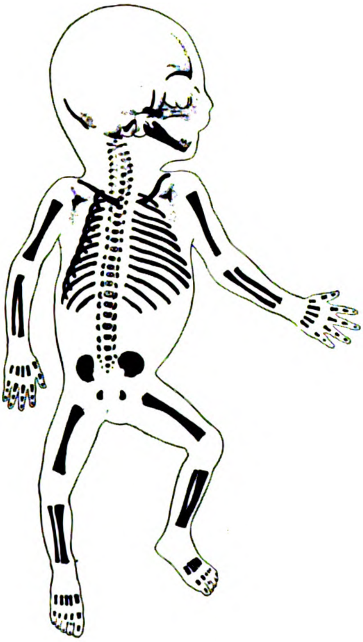


Fig. 45

Figs. 44 and 45.—Roentgenogram Fig. 44 and diagram Fig. 45 of fetus at seventeen to twenty weeks, one-third actual size.

Upper extremity: In this period the ossified portion of the diaphysis of the humerus reaches the articular ends and begins to overlap these so that at the distal end of the humerus both fossæ (olecranon and cubital) and ulna and olecranon become visible, and later, on the proximal end of the humerus an indication of medial and posterior portion of the neck appears.

The sternum begins its ossification. Usually one center forms in the manubrium first and this is followed soon afterward by several centers in the body of the sternum. The centers form a longitudinal row first, and soon they assume a round or elliptical form. Not seldom the first centers of ossification appear in the upper part of the body between the second and the third costal cartilages. The position of the ossification centers of the sternum corresponds usually to the level of the intercostal spaces.

Fig. 46

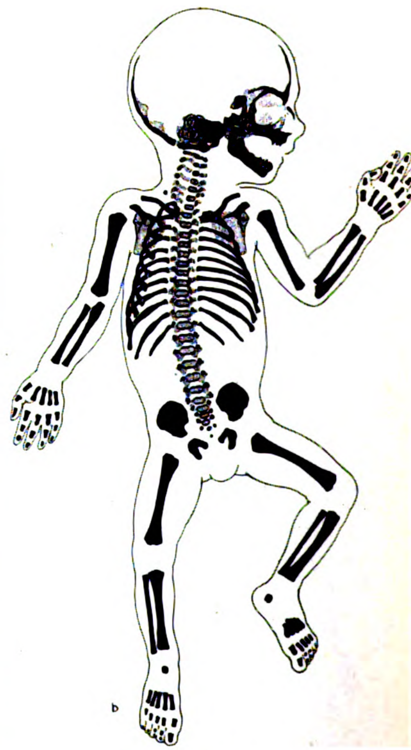


Fig. 47

Figs. 46 and 47.—Roentgenogram Fig. 46 and diagram Fig. 47 of fetus at twenty-five to twenty-eight weeks, one-fourth actual size.

Lower extremity: A center of ossification develops in os calcis, its appearance being occasionally delayed by from four to eight weeks. Sometimes it is followed by the appearance of a center in the astragalus. The middle phalanx of the second toe, and occasionally that of the third toe, acquire a center of ossification in their diaphyses.

Twenty-fifth to Twenty-eighth Week.—The rudiments of all the milk teeth have entered the stage of ossification in this month.

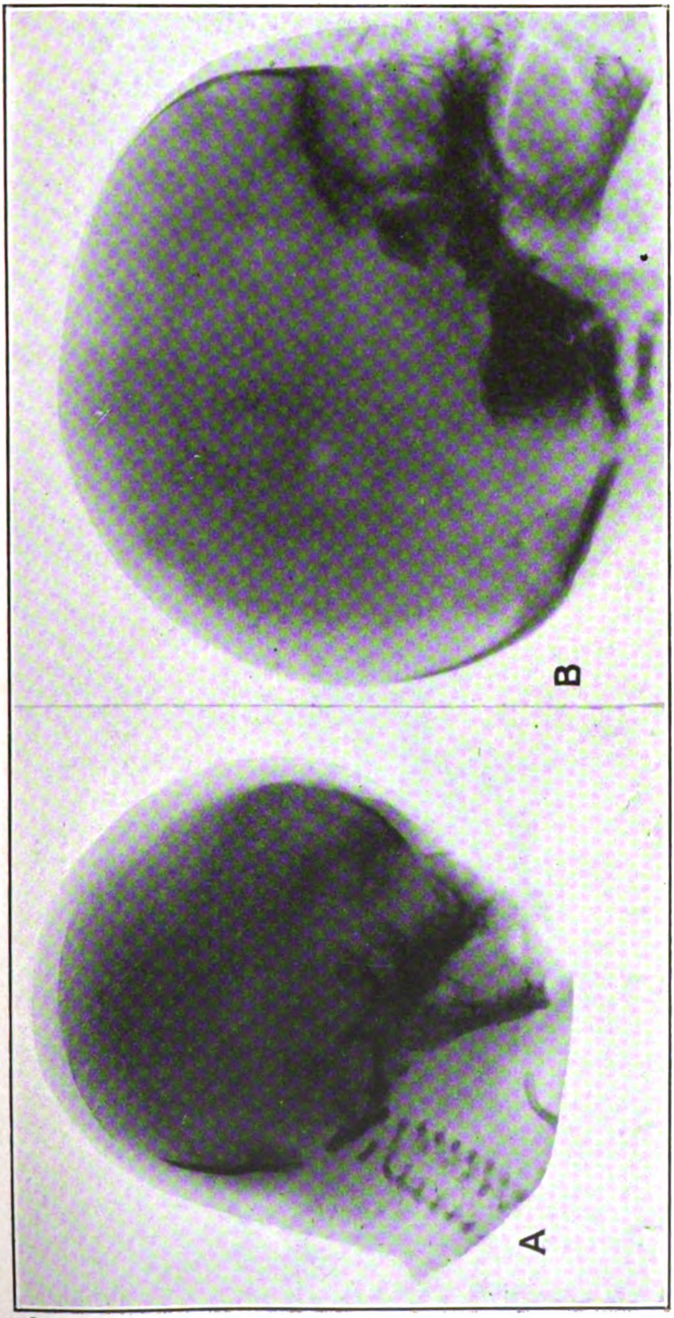


FIG. 48.—Roentgenograms of skull of fetus showing ossification centers at (*a*) seventeen to twenty weeks and (*b*) twenty-five to twenty-eight weeks, actual size.

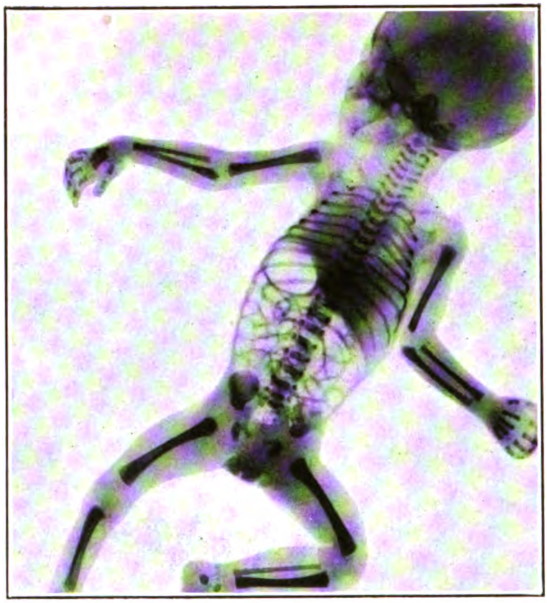


FIG. 49

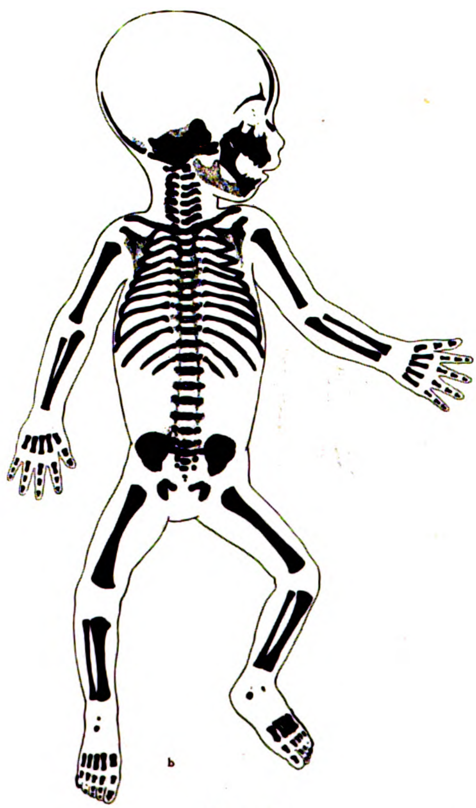


FIG. 50

FIGS. 49 and 50.—Roentgenogram Fig. 49 and diagram Fig. 50 of fetus at twenty-nine to thirty-two weeks, one-fourth actual size.

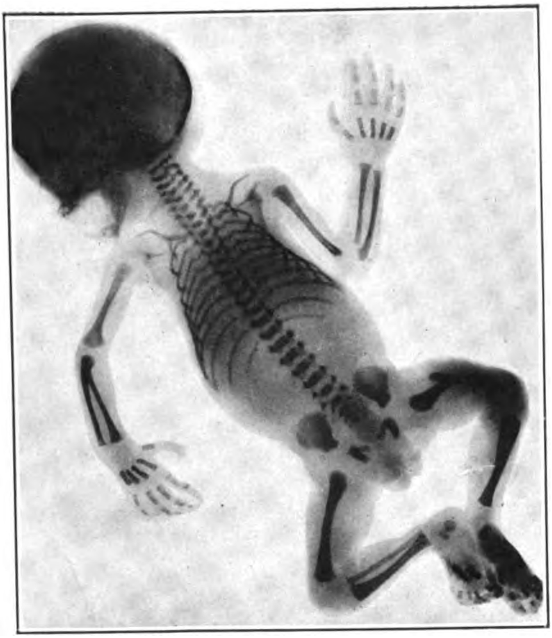


Fig. 51

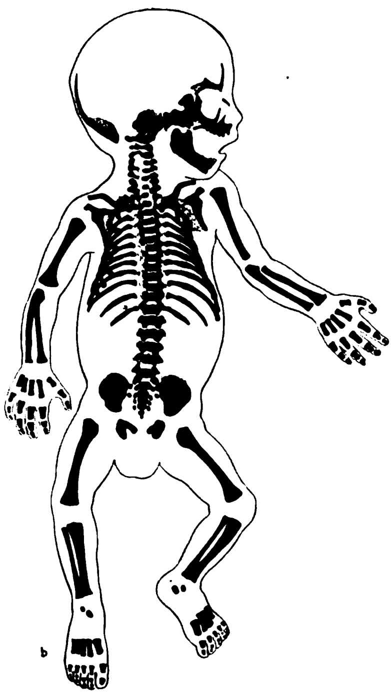


Fig. 52

Figs. 51 and 52.—Roentgenogram Fig. 51 and diagram Fig. 52 of fetus at thirty-three to thirty-six weeks. Roentgenogram one-fourth actual size. Diagram somewhat less.

The development of the transverse processes of the vertebræ progresses down to the last lumbar vertebra. At the end of this period a center of ossification may develop in the lateral masses of the first and of the second sacral vertebræ. The body of the fifth and the arches of the fourth sacral vertebræ become ossified at this time, rarely earlier.

A center of ossification develops in the astragalus.

In the horizontal ramus of the pubic bone the center may develop as late as in this period.

Twenty-ninth to Thirty-second Week.—The greater cornua of the hyoid bone usually become visible, appearing as cone-shaped processes directed obliquely upward at the level of the second cervical vertebra.

The lateral masses of the first and second sacral vertebræ ossify usually at this time.

In the sternum three or more large centers of ossification are visible.

The middle phalanx of the fourth toe frequently begins its ossification during the period.

Thirty-third to Thirty-sixth Week.—This period is the earliest time at which the first epiphyseal center may appear, that of the distal epiphysis of the femur. Usually, however, this center appears later, at about the time of birth.

The costal process of the sixth and of the fifth cervical vertebræ begin their ossification.

Thirty-seventh to Fortieth Week.—The middle turbinates ossify at the end of the fetal period and shortly before birth the rudiments of the first permanent molar teeth begin to ossify.

The costal process begins to ossify in the fourth, the third and the second cervical vertebræ; the first coccygeal vertebra usually ossifies during the last weeks before birth and vertical arrangement of trabeculæ becomes visible in the bodies of the vertebræ.

A center of ossification appears in the proximal epiphysis of the tibia just before birth in a majority of cases, and ossification in the cuboid frequently starts before birth, usually by several centers, although in some cases it may not be visible even in the new born.

The New Born.—A center of ossification in the distal epiphysis of the femur is so frequent in the new born that Lambertz calls it a sign of maturity. This is frequently the only epiphyseal center present in the new born. Poirier[1] gives a summary of the literature on the time of the appearance of the epiphysis at the distal end of the femur. Schwegel found it to appear between birth and the third year. Casper in the ninth fetal month. Hart-

[1] Traité d'anatomie, **1**, 227.

mann found it lacking in 12 per cent of cases at birth and in 7 per cent of cases present as early as the eighth fetal month.

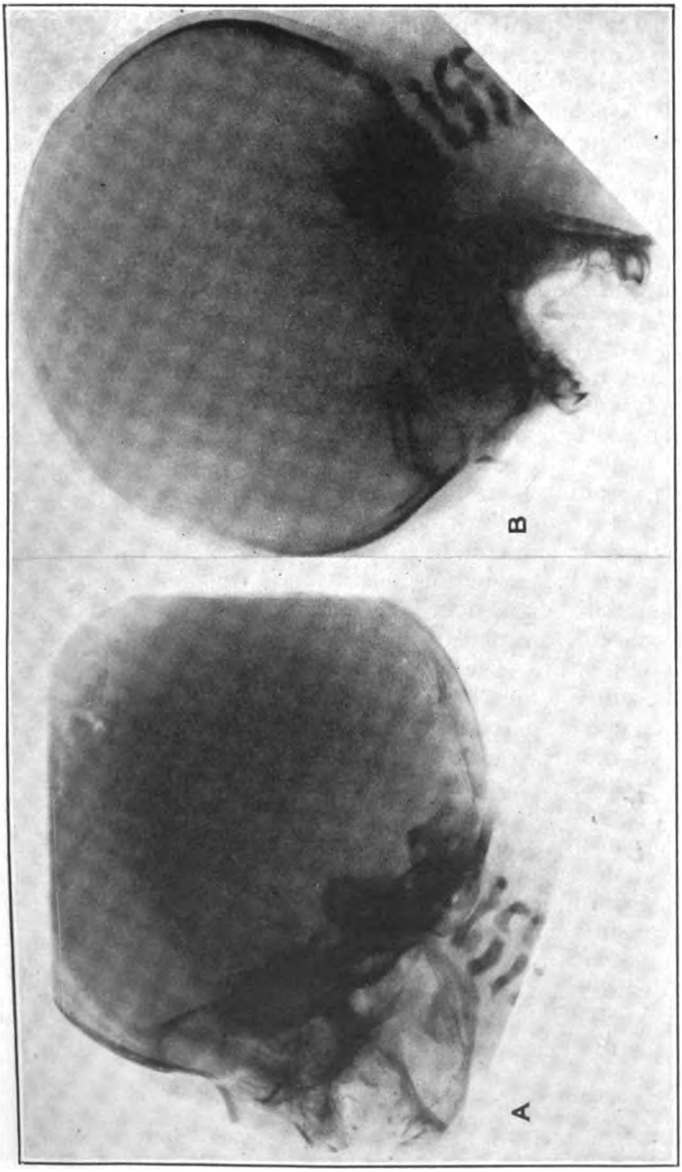


FIG. 53.—Roentgenograms of skull of fetus showing ossification centers at (*a*) twenty-nine to thirty-two weeks and (*b*) thirty-three to thirty-six weeks, three-fourths size.

The four parts of the occipital bone (basilar, two lateral and the squamous) are separated from each other by thin layers of cartilage. The mastoid portion of the temporal bone is not ossified in its entire extent, a serrated line marking the boundary between bony and cartilaginous portions of the mastoid part. The lateral halves of the frontal bone are separated. The body of the hyoid bone is usually ossified. Both halves of the mandible, as a rule, are united by connective tissue.

The vertebræ are ossified in all their essential parts, including transverse and articular processes of the arches, but the centers of ossification are separated from each other by cartilage. The first coccygeal vertebra is usually ossified by this time.

In some cases the proximal epiphysis of the humerus is ossified. In the hand all bones are ossified except the carpus, in which centers of ossification in os magnum and unciform may be seen only very rarely.

At birth the ossified portion of the os pubis surrounds usually a portion of the anterior boundary of the obturator foramen, but the region of the symphysis and upper margin of the horizontal ramus of os pubis remain cartilaginous. The following portions of the innominate bone are not ossified in the new born: The crest of the ilium with superior spines, acetabulum, spine of ischium and ascending ramus of ischium.

The middle phalanx of the fourth toe is frequently, that of the fifth toe always, cartilaginous in the new born; in the fourth toe, however, the middle phalanx may start in its ossification in the eighth fetal month. The following portions of the leg are usually not ossified in the new born: Proximal epiphysis of tibia and of the fibula, epiphyses of metatarsal bones and of phalanges, the cuboid and the three cuneiform bones.

Other Methods of Studying Osseous Development Compared.—We have compared the process of ossification, as observed in the roentgenograms of the fetuses studied with the roentgenographic studies of Alexander,[1] Bade,[2] Hasselwander[3] and Lambertz, and found that the time of appearance of centers of ossification pretty well agrees, in general, there being minor differences only.

Compared with the studies of Mall, who used transparent specimens of embryos and fetuses for observing the appearance of centers of ossification, we find that by the use of these specimens he was able to demonstrate the minute centers of ossification gen-

[1] The Development of the Osseous Vertebral Column, Fortschr. a. d. Geb. d. Röntgenstrahlen, Suppl. 13.

[2] Short Description of Ten Roentgenologically Examined Fetuses, Centralbl. f. Gynäk., 1899, p. 1031.

[3] Studies of Ossification of the Skeleton of the Human Foot, Ztschr. f. Morphol. u. Anthropol., 1903, 5, 438.

erally about one week earlier than they are demonstrable by roentgenograms. This observation also agrees with text-books of anatomy (Rauber-Kopsch, Gray[1]) which have been consulted for this purpose, and it is found that they place the time of appearance of various centers about one week ahead of the time at which the centers cast shadows large enough to be visible in roentgenograms.

By courtesy of Dr. Roy Lee Moodie, of the Department of Anatomy of the University of Illinois, we obtained transparent specimens of a pair of twins from his embryologic collection and made roentgenograms of them. By studying these roentgenograms and specimens (Fig. 36) we found the following differences:

	Roentgenograms	Transparent specimens
Basal phalanges of fingers	3d	2d, 3d, 4th
Terminal phalanges of toes	1st, 2d, 3d	1st, 2d, 3d, 4th
Bodies of vertebræ	9 lower dorsal	9 to 10 lower dorsal, respectively
	All lumbar	All lumbar
	1st sacral	1st, 2d sacral
Bodies of vertebræ	Upper 3 lumbar	Upper 3 to all lumbar, respectively

Thus the transparent specimens show in the tenth week centers that become visible in the roentgenogram only in the eleventh to twelfth week.

Variations in Osseous Development.—There are, as might be expected, some variations in the normal process of ossification, and it is also influenced by pathological conditions of the mother and of the fetus (for example, syphilis, rickets, osteogenesis imperfecta, etc.). In general, these pathological processes may well be diagnosed in the roentgenograms so that an error may easily be prevented. In some portions of the skeleton the ossification is less regular than in others, and as a general rule the more caudad the portions of the skeleton are, the more they are subject to variations in the process of ossification; and the centers which develop at a later period of fetal life are also more variable. Thus, there are considerable variations in the time of appearance of centers of ossification in the sacral vertebræ. The foot, as a general rule, is unreliable as an indicator of the age of the fetus. The ossification of the sternum is also irregular in the time of appearance, size and arrangement of the centers of ossification. The twelfth rib is also very irregular, and we found it absent in roentgenograms of the fetus from the thirteenth to sixteenth week, and also in some other older ones, although, as a rule, the twelfth rib appears in the tenth or in the eleventh week. Some of the

[1] Anatomy, Descriptive and Applied, Ed. 18, Lea & Febiger, Philadelphia and New York, 1910.

centers, although demonstrable by careful examination, are so small as to be easily overlooked, and this may lead to an error. For this reason it is necessary to know what centers we may expect at that particular age of the fetus, and we should look for them in good light with a magnifying glass.

Bade has examined roentgenograms of twin fetuses, one of which was 5.8 cm. long, weighing 8 gm., and the other 6.3 cm. long, weighing 11 gm. The only difference in the stage of ossification was that the larger fetus showed two more centers in the arches of the vertebræ and two additional centers in terminal phalanges of the fingers.

In the twins from Dr. Moodie's collection, which we have studied, the only differences in the stage of ossification are in the axial skeleton, one fetus showing centers for seventeen bodies and twenty-four arches on each side and the other only fifteen bodies and twenty-two arches on each side.

The process of ossification is more constant for a particular age than the length of the fetus. Mall,[1] in his article on ossification in embryos up to one hundred days old, concludes that "the remarkable regularity of the appearance of the bones makes of them the best index of the size and of the age of embryo we now possess."

Limitations of Accuracy.—In the first half of pregnancy the estimation of the age of the fetus may be made with greater accuracy because many more new centers appear in the first months, and also because the time of appearance of the earlier centers is more constant. In later months most centers in the lower part of the skeleton are available for study, although these are less constant in the time of their appearance. We have intentionally made our groupings broad enough to cover minor errors in diagnosis, but more careful subsequent studies may refine the diagnosis to such a degree that determination of age will be possible within the period of one week in the first half of the pregnancy, and within two weeks in the second half of the pregnancy.

Different Values of the Different Portions of the Body.—In the very early period (second month) the stage of ossification of clavicle and mandible is of chief importance, and on the basis of presence or absence of these centers determination of the age is made. Both roentgenograms and transparent specimens show that the time of appearance of these centers is almost constant, which makes them of cardinal value in diagnosis.

Next in importance are the centers of the upper extremity, and especially of the hand (metacarpals and phalanges) which are very regular, not only in the time of their appearance, but also in their

[1] On Ossification Centers in Human Embryos Less Than One Hundred Days Old, Am. Jour. Anat., 1906, 5, 433.

sequence. The ossification of the diaphysis of the long bones of the arms extends from the eighth to the sixteenth week, and during this period the determination of the age may frequently be made from a good roentgenogram of the hand alone.

The progress of ossification of the head is also of considerable diagnostic importance, but the centers in many bones of the head are very difficult of demonstration. Those, however, that can be well demonstrated are of much value in the determination of the age. This is especially true of the occipital bone, superior maxilla, tympanic ring, nasal bone and hyoid bone.

The axial skeleton (the vertebral column) is less reliable than the foregoing named portions of the skeleton, and especially its lower portion is of little value in diagnosis of age. It is not the absolute number of arches or of the bodies ossified which decides the diagnosis as to the age of the fetus, but more the region involved and the extent of the development in the particular region of the vertebral column (cervical, dorsal, lumbar, sacral). On the other hand, however, the facts that the process of ossification of the vertebral column extends from the ninth week throughout the life of the fetus, and all its centers, as a rule, are well demonstrable, make it of special value for at least approximate determination, although it must not be forgotten that occasionally the process of ossification may be delayed in the vertebral column, while it is normal and regular in other portions of the body.

The sternum is unreliable as an index of age and its centers are frequently difficult to demonstrate. The ribs are fairly constant, except the twelfth pair, which, as previously mentioned, may not show at all in roentgenograms of comparatively old fetuses.

While the ossifications of the long bones of the legs are pretty regular, since they appear at an early period, ossification in the foot is very irregular and the stage of ossification of the foot is of little value in the determination of the age of the fetus. The osseous development of the foot extends from the ninth week to the end of the fetal period (not being, however, completed even at this time) and during this time there are very marked variations, especially in the centers which appear late in the fetal period.

From the above it may be seen that, as a general rule, the earlier a center appears the more regular it is, and since the process of ossification starts in the cephalic region and spreads caudally, it is also true that the more caudad a skeletal segment is situated the more it is subject to variations and irregularities.

Advantages of the Roentgenographic Method.—The peculiar advantage of the roentgenographic method for determining the age of the fetus lies in the fact that while in the determination of age according to the length we base our final conclusions usually on one, rarely on two or three measurements expressing different lengths of the fetus, in the roentgenographic method many centers of ossification are the factors taken into consideration before arriving at a final conclusion; and they act as check on each other and quite frequently the roentgenograms alone give us information as to whether the fetus is normal or not, a point which seldom may be determined from measurements alone.

Technic.—In studying the roentgenograms it is well to use a reading glass of about four inches in diameter, since some centers of ossification may be so small as to be very easily overlooked when sought for with the naked eye.

If only one exposure of the fetus is made then the best position to show as many ossification centers as possible is as follows: The back lying flat on the plate, head turned completely to one side so that the side of the head lies on the plate and lateral exposure is obtained. (It should be remembered in the study of the skull that both halves of the skull are usually visible.) Arms and fingers should be extended and fingers spread as far as possible from one another. One hand should be pronated and the other supinated, the lateral exposure, which is often of so much value in roentgenograms taken for the purpose of surgical diagnosis, not being of much value, since in this position shadows of phalanges of fingers and of metacarpals are superimposed and cannot be well differentiated. The legs should also be extended and feet put into such a position that all metatarsals and phalanges are shown.

CHAPTER IV.

PATHOLOGICAL FINDINGS IN PREMATURES.

VERY little careful work has been done with reference to the pathological changes in the premature infant. The discussion which follows is a summary, largely taken from the recent excellent work of Arvo Ylppö.[1]

Premature infants must be classified into two groups: Those that are born "weaklings" owing to congenital deformities or malformations, congenital diseases, especially lues, and the congenital weaklings born of nephritic, eclamptic or tuberculous mothers, or those suffering from chronic toxemia. In the second group are those fully developed and normal for their fetal age.

One is often amazed at the life energy of these prematures, in view of the high grade pathological changes in the various organs, especially the hemorrhages into the brain and spinal cord.

Premature birth should be considered a traumatic process, in which the characteristic pathological processes are most frequently noted in three groups of organs, for which there appears to be a predilection:

1. The skull with the brain and its membranes, inclusive of the spinal cord.
2. The lungs.
3. The gastro-intestinal canal.

Intracranial hemorrhages are especially important. Ylppö believes that they are responsible for 30 per cent of the deaths of prematures in the early days of life. In the skull there are subarachnoidal or intrapial hemorrhages, while in the spinal column they are extradural.

The so-termed subdural hemorrhages and those from tears of the tentorium, which are present in full-term infants, are only exceptionally seen in prematures.

Ventricular hemorrhages are frequently found in prematures, but hemorrhage into the brain substance proper is quite rare. High-grade edema of the pia is, as a rule, also present along with these intracranial hemorrhages. Bacteria easily settle in the injured brain membranes and meningitic processes are not uncommon.

[1] Arvo Ylppö, Ztschr. f. Kinderh., xx, 212, 1919.

In the later life of the premature the appearance of spastic states (Little) and of disturbances in intelligence are often seen and are explained as a rule, undoubtedly, as a consequence of old hemorrhages into the brain and spinal cord.

The condition spoken of in the literature as hydrocephalus of the premature, has, as a rule, nothing to do with hydrocephalus. The brain represents one-fourth of the body weight and appears normal macroscopically and developmentally. Ylppö suggests the term "megacephalus." This megacephalus is due to the fact that the brain develops at practically the normal rate, while the growth of the body is retarded.

Hemorrhages into the lungs appear not only under the pleura, but are scattered through the entire parenchyma. The alveolar septums are thickened because of the extravasations of blood. The normal circulation in the lung is hindered and in the extra-uterine life there appears a stasis, which hinders the taking up of air and predisposes secondarily to atelectasis. Following stasis and bacterial changes, there may appear in the lungs of prematures a high grade, almost total, inhibition of the circulation of the blood.

Inflammatory changes in the lungs or bronchi appear infrequently and atypically in the first days of life. Bronchopneumonia, after the second week of life, begins to play an important part in causing death.

Epicardial hemorrhages are of common occurrence. They appear just as do the subpleural hemorrhages and those in all the other organs.

Subcapsular liver hemorrhages are on a par with other sub-serous hemorrhages. They are important only insofar as they may be extensive, and with rupture of the capsule may result in hemorrhage into the peritoneal cavity with death.

Hemorrhages into the kidney are frequent. They have two predilections: In the interstices of the apices of the pyramids, or in the neighborhood of the venæ et arteriæ arciformes. Hemorrhages into the Malpighian bodies and cortex are rare. Infarcts appear in the same sites as the hemorrhages.

The hemorrhages of the digestive tract are next in importance to those of the brain. In small prematures, dying shortly after birth, one often finds hemorrhages scattered through the entire tract. The areas of predilection are: The lower portion of the esophagus, the cardia and fundus of the stomach, the mucous membrane folds in the corpus ventriculi, the duodenal margin of the pylorus, and the entire duodenal mucosa. In the deeper portions of the intestines, hemorrhages occur infrequently about the ileo-cecal valve and in the mucosa of the large bowel. These hemorrhages appear chiefly under the epithelial cells in the tunica propria. Blood often appears in the bowel lumen after rupture of the mucosa. These hemorrhages in extra-uterine life are important only insofar as they predispose to infection, which readily occurs. As a result, within one and a half days prematures may show a marked mucous membrane necrosis and peritonitis.

Inflammatory processes within the digestive tract, especially in the esophagus and duodenal mucosa are also common. From the inflamed intestinal mucosa, bacteria easily invade the blood, with a following general sepsis.

The mucosa of the stomach is frequently involved in the septic processes of the prematurely born, especially in their early days of life. Involvement of the stomach is often followed by peritonitis and by Bacillus coli septicemia.

The histopathological inflammatory processes, due to bacteria, appear atypically in prematures. This is associated with a very ineffective exudation of fibrin and scanty mobilization of leucocytes. Because of these factors general sepsis in all infections appears easily.

The hemorrhages are due to diapedesis, rhexis, or both, and vary with the intensity and duration of stasis and the grade of the infections—toxic damage to the capillary walls.

The preceding summary of the pathological changes in the premature has been concerned chiefly with the question of hemorrhage. (Specific pathology will be discussed later under the "Diseases of the Prematurely Born.")

PART II.

NURSING AND FEEDING CARE.

CHAPTER V.

MATERNAL NURSING.

NURSING AXIOMS.

THE following may be laid down as nursing axioms:

A diet similar to what the mother was accustomed to, with moderate limitations, may be taken.

There should be one bowel evacuation daily.

From three to four hours daily should be spent in the open air in exercise which does not fatigue

At least eight hours out of every twenty-four should be given to sleep.

There should be absolute regularity in nursing and expression.

There should be no worry and no excitement.

HYGIENE OF THE MOTHER.

The Diet of the Mother.—A plain, more or less restricted diet is desirable. This must be enforced in the management of the wet-nurse, but to a less degree with the mother.

Nursing is a perfectly normal function, and a woman should be permitted to carry it out along the natural lines. Inasmuch as there are two lives to be provided for instead of one, more food, particularly of a liquid character, may be taken than the mother may be accustomed to. It is our custom to advise that milk be given freely. A glass of milk may be taken in the middle of the afternoon, and 8 ounces of milk with 8 ounces of oatmeal or corn-meal gruel at bedtime, if it does not disagree with the mother. Our only evidence that a food is disagreeing is the condition of the digestion. When any article of food disagrees with the mother, or if she is convinced that it disagrees, whether or not such be really the case, the food should be discontinued. In a general way, milk (1 quart daily), eggs, meat, fish, poultry, cereals, fresh vegetables and fruits constitute a basis for selection.

For more detailed suggestions see page 122.

The Bowel Function.—A very important and often neglected matter in relation to nursing is the condition of the bowels. There must be one free evacuation daily. For the treatment of constipation in nursing women we have used different methods in many cases. The dietetic treatment by increasing the whole cereals, rough breads and cooked vegetables with plenty of recreation and exercise promise most. Manipulation of the diet should not be such as to interfere with the milk production Three other methods are open to use; massage, local measures and drugs. Massage is available in comparatively few cases. Local measures consist in the use of enemas and suppositories. Every nursing woman under our care is instructed to use an enema at bedtime if evacuation of the bowels has not taken place during the previous twenty-four hours. For a laxative in such cases, and in many others, a capsule of the following composition has served well:

℞—Extracti nucis vomicæ	0.015 gm. (¼ gr.)
Extracti cascaræ sagradæ	0.325 gm (v gr.)

Sig.—To be taken at bedtime.

The amount of the cascara sagrada may be varied as the case may require. In not a few instances we have found it necessary to give two capsules a day in order to produce the desired result. Neither the nux vomica nor the cascara appears to have any appreciable effect on the child.

Air and Exercise.—Outdoor life and exercise are not only as desirable here as they are under all other conditions, but to the nursing woman, with her added responsibility, they are doubly valuable. In order to get the best results exercise or work should be so adjusted as not to reach the point of fatigue. The mother whose nights are disturbed should be given the benefit of a midday rest of an hour or two. It should be our duty, however, to explain to the mother and to other members of the family that an important element in satisfactory nursing is a tranquil mind.

Care of the Breasts.—A well-established routine should be instituted for the care of the breasts. To facilitate this a readily accessible tray with the necessary utensils should be provided. This should contain a glass-stoppered bottle with a saturated solution of boric acid, a jar of cotton pledgets on toothpicks, to be used as applicators for the boric acid, a graduated glass or beaker. The nipples should be thoroughly washed before and after nursing with a saturated solution of boric acid poured fresh from the bottle for each cleansing, and the surplus thrown away. The boric acid should be applied with the cotton pledgets. The fingers should not come in contact with the nipples if the child is to nurse directly at the breast. If the nipples are tender they should be anointed with a sterile mixture of 5 per cent tincture of benzoin in liquid vaseline.

All utensils, including the breast-pump, if one is in use, should be sterilized by boiling. In case of the breast-pump the rubber bulb may be removed for this purpose. Where the milk is to be expressed the hands must be thoroughly disinfected by washing with soap and water and rinsing before manipulation of the breasts. Under all conditions soap and water should be freely accessible, and their use required before handling the breast of the mother.

CONDITIONS INFLUENCING THE BREAST MILK.

Secretion.—Spontaneous failure of lactation is extremely rare and probably always occurs in consequence of an incomplete emptying and an insufficient stimulation of the breasts. This is especially true in the feeding of premature infants, and nursing must be supplemented by other methods of emptying the breasts, such as expression, pumping, or the nursing of a second infant.

The ability to restore the milk supply in breasts which have not been nursed for days and even weeks, when proper stimulation is applied is the best proof of this assertion.

When the milk supply is temporarily insufficient the necessary complemental feedings should be obtained from some other source and only as a last resort should mixed feeding be instituted.

Fissures.—Fissures offer serious difficulties to nursing because of the severe pain and danger of mastitis.

Relief of the pain is frequently accomplished by elevation of the breasts by a binder. Among the best local applications are silver nitrate solution 5 per cent, followed by an ointment. (Balsam of Peru 1, castor oil 30; or silver nitrate 1, balsam of Peru 2 and petrolatum 30.)

The nipples must be thoroughly cleansed before each nursing.

Simple Engorgement.—The first essential to relief is the restriction of fluids by mouth and the administration of laxatives. In our experience compound jalap powder in teaspoonful doses once or twice daily is best. Saline laxatives are effective but more likely to pass into the milk. Citrate of magnesia is least likely to do this. The breasts are tightly bandaged and an ice-bag is applied to each, external to the bandage. If this does not relieve the breasts massage and expression should be practised and the bandage and ice-bags reapplied.

If the cold applications produce discomfort as they occasionally do, hot boric dressings, protected by oil silk may be used, a compression bandage being applied external to the dressings. These should be repeated at hourly intervals.

The infant should be put to breast regularly at four-hour intervals if able to take them. The wet-nurse's baby may be used for this purpose if at hand.

Mastitis.—Ice-bags are best applied early. Later, warm moist applications are more useful. When incision is necessary it should be radial and must not enter the mammilla. This should be performed as soon as pus is localized and is to be followed by expression through the incision. In order to prevent further congestion of the breasts gentle expression should be practised at regular intervals. This not only relieves the congestion, but, in a very large percentage of cases, it tends to localize infection and a normal secretion is retained after the healing of the incision.

Menstruation.—The advent of this physiological function is frequently attended by a lessened milk secretion which leads the infant to become fretful due to underfeeding. Occasionally menstruation is attended by attacks of colic or indigestion in the infant, but, under no circumstances, should the advent of menstruation be considered as an indication for weaning, as all of the symptoms disappear within two or three days.

Factors influencing the *mental condition of the mother*, such as anger, fright, worry, shock, distress, sorrow, or the witnessing of an accident may affect the milk secretion sufficiently to cause no little discomfort to the child, and oftentimes the lessening of the flow for a day or two. At times, especially when the mother is under the influence of shock or grief, it may be necessary to substitute artificial feeding for a few nursings during these periods, until the mother has again resumed her mental equilibrium, her breast being emptied by mechanical means in the meantime.

Asthenia and Anemia without a definite underlying organic pathology must not be considered sufficient causes for weaning. Most of such women receive benefit to their own health, increasing in weight and strength and often overcoming their anemia. This is probably due to the more complete involution during the puerperium and stimulation of the glands of internal secretion and blood-making organs.

Drugs.—Alkaloids of opium, hyoscyamus, belladonna and similar drugs, when given in large quantities not infrequently pass into the milk, and should therefore never be administered in large quantities to the nursing mother. Belladonna may cause a decrease in milk secretion, and should be administered with caution during the period of lactation. Mercury, iodides and the newer salts of arsenic are also secreted in the milk, and may be used to advantage when a luetic mother is nursing a luetic infant.

THE NURSING PROPER.

Regularity in Nursing.—The breast which is emptied at definite intervals invariably functionates better than does one which is not, not only as regards the quantity, but also the quality, of the milk, thus regular habits in breast-feeding are as essential to milk production as to its digestion and assimilation. *The baby should be wakened to be fed.*

The average mother will supply the needs of the individual meal with *one breast,* and the breasts should be alternated in successive feedings. Thorough emptying of the breast should be encouraged under all circumstances, as this is our best method for increasing the milk supply, and the baby is the best means at hand by which this is accomplished. This should be encouraged in every instance. It is most readily thwarted by allowing a lazy baby to partially empty both breasts. This will soon lead to a diminished milk secretion. Expression or the nursing of a second baby will usually prevent the loss of milk supply. Massage will often be of great assistance in retaining the milk flow. It should be carefully and gently applied at regular intervals.

Sometimes, however, it is advisable to give *both breasts* at each feeding, *i. e.,* under the following conditions: (1) During the first few days to stimulate secretion, and a little later to relieve the congested breasts; (2) to weak babies when there is an abundance of milk, and they are not strong enough to get the last milk that comes harder; (3) to overfed babies, where it is desirable to give them only the first and weakest milk, and to lessen the yield of milk from the breast; (4) as the milk supplied by one breast fails to meet the needs of the infant, both breasts should be given at each nursing—the first breast should be thoroughly emptied before allowing the baby to take the second breast, and the next nursing started on the second breast given in the last feeding.

When to Begin First Feedings.—Little is to be gained by placing a premature infant to the mother's breast during the first twenty-four hours and as they do not stand starvation the limited supply of milk needed should be obtained from some other mother. Water should be administered four or five times during the first twenty-four hours. When the premature is unable to take the breast, massage and expression should be begun on the second day and continued at first four, and later six, times daily. When a wet-nurse's baby is available it should be left to suckle the mother's breast at stated intervals.

Number of Feedings in Twenty-four Hours.—During the second, third and fourth days the infant may be placed at the breast at four- to six-hour intervals, and if strong enough to nurse these may be increased so that it will be nursed every three or four hours. If it does not obtain sufficient food by this means it may be given hand feedings of expressed milk between nursings.

Length of Nursing.—As a rule, a robust baby takes three-fourths of the milk obtained from a good breast in the first five minutes of a twenty-minute nursing. Fifteen to twenty minutes should be the limit for the nursing period. If a baby is doing well on shorter periods and seems satisfied, let it be its own judge of the nursing time. While premature infants may nurse well during their first three or four days of life, frequently when they become intensely jaundiced they develop a marked apathy and under such circumstances they must be awakened during the nursing period to keep them at work. At such times they must at least be partially hand fed. It may also be necessary to feed them more frequently.

Administration of Water.—At least one-twentieth to one-twelfth of the body weight of the infant, in the form of inert fluids, should be fed daily during the first two days. A 1 per cent milk-sugar solution (boiled) will answer. For further fluid intake needed see Tables I, II and III, pages 181 and 182. Otherwise there will be unnecessary loss of weight and perhaps a high degree of fever due to inanition. A high temperature during the first days of life is more commonly due to "inanition" than infection in present-day obstetrics. The best differential test is administration of water or sugar water at regular intervals. In case of water inanition sufficient fluid intake results in a rapid drop in the temperature.

Nursing in Difficult Cases.—When the weight curve remains stationary or the gain is less than should be expected the possibility of underfeeding as the cause must not be lost sight of. The estimation of the twenty-four-hour secretion of milk is of first importance because of the relationship between demand and supply. The quantity taken by the infant at each nursing should be measured by weighing before and after feeding at the breast, and also by measuring the amount of milk fed by hand. Conclusions should be made only after such estimation for a period of at least twenty-four to forty-eight hours. Expression of both breasts after each nursing may be of advantage to the mother even though the baby is only nursing on one breast. Expression when thoroughly and properly applied will, in itself maintain a full and free milk supply without placing an infant at the breast. In some instances this may be continued for many months. It may be stated that the small flat breast offers greater difficulties to proper manipulation than the full conical breast. For details as to the method of expression see page 126.

The classes of cases which are most likely to necessitate hand feedings are those suffering from cleft palate and harelip and those in which there is deformity of the mother's nipples. We have recently had an opportunity to observe some of the cases being treated by the Minneapolis Breast Feeding Bureau. Among these a very severe case of harelip and cleft palate, nine months of age, and a case of congenital absence of nipples in the mother, whose infant was five months of age. Both of these infants had been fed exclusively on expressed milk and had attained the average weight and development of breast-fed infants of their respective ages.

CHAPTER VI.

WET-NURSING.

THE WET-NURSE. HER SELECTION AND HER BABY.

The Problem.—When there is a positive inability on the part of the mother to nurse her offspring, either through inadequate functioning on the part of the breast or systemic disease, we are confronted with the problem of securing human milk from another source, as notwithstanding the numerous isolated reports on successful raising of premature infants on artificial foods, the statistics of infants fed by artificial foods, when compared with those of infants fed on human milk are so strikingly in favor of the latter that the obtaining of human milk must be considered imperative.

How Obtained.—In our experience, even in a large city, great difficulty has been met in obtaining a regular supply of wet-nurses. On several occasions various charitable and hospital societies have attempted to establish a wet-nurses' registry as a clearing house for the several maternity and general hospitals of Chicago. These attempts have not been successful for two reasons: (1) Because of the irregularity in the demand, and (2) because of the lack of coöperation on the part of the various institutions caring for this class of cases.

The Nationality of the Wet-nurse.—This is of considerable significance where the supply allows of a selection. The phlegmatic temperaments as seen in women of Northern and Central Europe of Teutonic and Slavic descent, offer the ideal material, while other nationalities, such as Italians, and the Southern negroes when removed from their home environment to a Northern climate with the consequent change in diet, secrete a milk poor in quality. However, even the latter in an emergency should not be neglected.

Requirements of a Good Wet-nurse.—1. She should be in good health, and, especially, free from all contagious and infectious diseases, and also from local diseases of any kind, such as those involving the nose, throat, skin, etc.

2. Her mammary glands should be of such quality that she can secrete sufficient milk of good quality, and the nipples sufficiently developed to allow of nursing, or proper expression of the milk (Figs. 54 and 55).

3. Whenever possible her age should be not less than eighteen and not more than thirty-five years.

4. The age of her baby, as compared with that of the baby she is to nurse, is a matter of indifference in most instances. However,

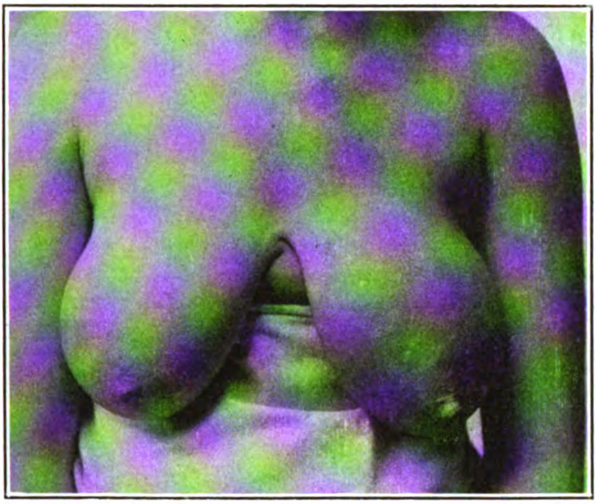


Fig. 54.—A good secreting spherical breast with well developed nipples. The breast is composed largely of glandular tissue. The engorged veins are plainly visible. This young primipara acted as a wet-nurse for over eighteen months. See p. 124.

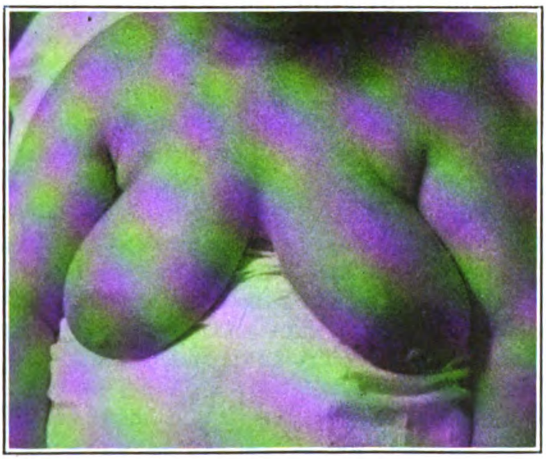


Fig. 55.—Large, pendulant breasts composed mainly of fat and connective tissue, the type to be avoided in the selection of a wet-nurse.

the first weeks, or if possible the first two months, of lactation should be avoided, because of the presence of colostrum and the rapidly changing quality of the breast milk, which not infrequently causes serious gastric and intestinal disturbances in very susceptible infants, as evidenced by vomiting, colic and diarrhea. Multiparity may be considered an asset, if the nurse has demonstrated her ability to care for and feed previous cases. A multipara is also less likely to be affected by her new surroundings, especially if this be a private home. When the wet-nurse has more or less direct charge of the infant, one who has been nursing her own or other infants will be likely to meet the technical difficulties in the care of her charge.

Examination of the Wet-nurse.—The examination of the wet-nurse should always be made in a systematic manner to insure against overlooking important things.

1. A careful history should be taken as to the number of her children, miscarriages and the presence of constitutional diseases in her family.

2. She should be thoroughly examined, all parts of the body being exposed, and the examination should include the skin and hairy parts of the body for the presence of skin lesions and parasites, as well as for old luetic scars. The organs of the chest and abdomen should be subjected to careful examination.

3. The breasts should be examined.

4. The genitalia, including the cervix and the urethra, and in all cases a cervical (and where suspicious, a urethral) smear should be taken and examined for gonococci. As a single smear is often misleading, in cases of the slightest suspicion where a girl baby is to be nursed the examination of the cervical and urethral smears should be repeated.

5. An examination and search should be made for chronic infections, especially for syphilis. A Wassermann test should be made in every case, and reported upon before she is allowed to supply milk, as it is well known that a syphilitic mother in a very great number of cases shows no clinical evidence of syphilis. The mouth and pharynx, neck, anus and genitalia, entire skin and lymphatic glands should also be examined for evidence of syphilitic lesions.

Tuberculosis.—The lungs, glands and osseous system should be examined, and a careful history as to susceptibility to colds and to recurring bronchitis elicited.

6. *Acute Infections.*—She should be questioned as to exposure to contagious disease, and she should be examined for evidence of acute infections of the nose, throat and ears.

7. Her teeth should be examined and defects and pyorrhea corrected, if necessary, at the expense of the family.

8. The urine should be examined (*a*) for evidence of nephritis, (*b*) for evidence of diabetes. It should, however, be remembered

that a positive reaction for sugar should not be overestimated, unless the sugar is proven to be dextrose, as very commonly in our experience during the early weeks of lactation a lactosuria is present. The kind of sugar can easily be determined by the phenylhydrazine test, followed by a microscopical examination of the crystals.

9. Nervous and psychic disturbances, such as epilepsy, insanity, hysteria, should, if found, by all means exclude the subject.

10. Her child should be examined for evidence of syphilis. Possibly one of the best arguments for the non-employment of a wet-nurse during the first two months of her lactation is the possibility of a latent syphilis. Where there is the slightest doubt, a Wassermann reaction should be made on the infant. The general condition of the child gives us the best evidence both as to the quantity and to the quality of the maternal milk. Unless the source of the nurse be known, it is well to be certain that she is nursing her own baby. In case of its death or its absence, every effort should be made to obtain its condition at birth and its later development.

So far as possible she should not be subjected to annoying questioning on the part of the family, which is entirely unnecessary, if she has been properly examined by the physician. It has been our experience that such unnecessary questioning has led to nervousness, and not infrequently has caused her to decline the position, at a time when she was most needed.

Her Place in the Household.—She should be treated neither as a guest nor as a menial, but so far as possible should be graded according to her previous station in life. There is grave danger of mental depression on the part of a woman, well-born and sensitive, who, through misfortune or necessity, is forced to seek this means of employment, and also of an exaggerated estimate of self-importance on the part of a woman but little accustomed to the luxuries of life upon her entrance into the home of employment, particularly if attentions are paid to her. As has been previously stated, all instructions and demands should be made by the person best qualified in the individual case. A divided responsibility will always lead to future complications.

Her quarters should be well located; their ventilation should be supervised, and she should be held responsible for their general cleanliness. The wet-nurse's baby should always be kept in the room with her, so that she may feel the full responsibility for its health and care.

The Quantity of Milk to be Expected from a Good Wet-nurse.—The quantity and quality of milk supplied must vary greatly with the glandular development of the individual wet-nurse, the state of her health, and the factors quoted elsewhere which would affect it temporarily. The amount and variety of stimulation applied to the breasts, of which the direct nursing by a full-term infant is the most valuable (at least for the purpose of stripping the breasts), must be given due consideration. In view of the many emergencies and influencing factors, no absolute standard for quantity and quality can be set for general rule. A wet-nurse who does not secrete sufficient milk during the first few days in her new employment should not be discharged until every effort has been made to improve her milk production. Frequently the change in environment is sufficient to reduce it temporarily.

Cost of Milk.—The wet-nurses in the Sarah Morris Hospital receive their board and room and $10 per week. Figuring the former at $8 per week, this would total a cost to the institution of $18 per week for each nurse. With an average of 30 to 40 ounces of milk per nurse daily, or 210 to 300 ounces per week, the average cost will be about 6 to 9 cents per ounce, or approximately $2 to $3 per quart.

When milk is dispensed to patients outside of the hospital, a charge of 15 cents an ounce is made for it, which is a reasonable price when all of the contending factors are taken into consideration.

Number of Nurses Needed.—Each good wet-nurse can care for the needs of about two infants, depending upon their weight and development, beside allowing the strippings for her own child.

Length of Lactation.—No time limit is placed upon the employment of a wet-nurse as long as the quality and quantity of her milk is sustained, and she continues in good health. One of our nurses had a lactation period of eighteen months. Such long periods of lactation, however, are not to be advised.

The Wet-nurse's Baby.—The presence of the wet-nurse's baby predisposes to her peace of mind, and wherever possible, she should take it with her. Her baby's state of health is by all means the best indication as to her ability as a nurse, and, with this, of the presence of constitutional disease in herself. It may be of immense value, if the baby is strong and healthy, to keep up the flow of of milk, in case the baby to be nursed is a weakling. It may also be used to estimate the functional capacity of a wet-nurse by nursing at regular intervals, and weighing before and after the nursing for twenty-four-hour periods. If in perfect health it may be put to the breast, after the weakling has taken such milk as it has strength to draw. If this is not practicable then the weakling should be nursed alternately with the well baby on each breast. It is also of immense value in emptying the breast after the wet-nurse has removed as much milk as it is possible by expression or by the breast-pump, if this is the means of drawing the milk for the weakling. It is a well-known fact in all institutions where wet-nurses are used, that the greater the degree to which the breasts are stimulated by suckling infants, the greater will be the reward in production. If the milk is insufficient for both babies, partial or entire meals of artificial food may be substituted for the wet-nurse's infant.

At the first sign of an acute illness on the part of the wet-nurse's baby, it should be separated entirely from the other baby, and removed from the breast; its illness should be given the same serious consideration as that of the other infant, so that the mother's anxiety may be relieved. It should receive as much of its mother's milk as can be spared. This can be expressed from the breasts and fed from a bottle.

Feeding of the Wet-nurse's Baby.—When a single infant is to be nursed the second baby is often a necessity in the promotion of the development and stimulation of her breasts. No breast can be developed to its fullest capacity with the breast-pump or hand expressions. It is a well-known fact that the breasts will respond in proportion to the demand placed upon them, and in most instances during the first few weeks of the premature's life, when its demands are met by from 4 to 16 ounces of milk, the wet-nurse can supply sufficient milk for both babies. When her supply becomes insufficient to meet the demands, her baby can be put upon partial bottle feedings of the strength as indicated by its age and development. The progress of the wet-nurse's baby has great influence on her peace of mind, which may spell success or failure in her ability to carry out her work. When the premature infant gives evidence of sufficient strength to be placed upon the breast, we have found the application of the wet-nurse's baby to the other breast a very valuable expedient in aiding the flow of milk into the breast which is to be nursed by the weakling. In many instances we have seen the milk flow from the second breast by this method so freely that but very little effort was required on the part of the weakling to obtain its food.

THE HYGIENE OF THE WET-NURSE.

In general, everything that has been said in the chapter on hygiene of the nursing mother applies also to the wet-nurse—of course, with the proper modifications, made necessary by peculiarities of her position.

Clothes.—Her clothes should be simple, and in every part washable. As the care of her undergarments is of even greater importance than her outer clothing, it is well that her laundry should be done with the family work, so that the family laundress who is trusted by the family may be charged with its inspection.

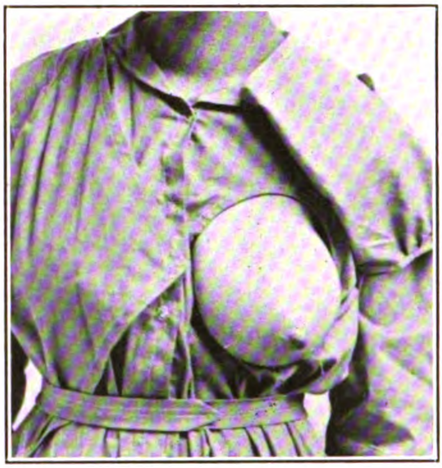


Fig. 56.—Wet-nurse uniform. Her dress should be of a simple type, and made of a material of different color from that worn by the nursing staff. One lapel of dress raised and thrown over shoulder, one lapel of undervest raised and breast exposed for nursing.

To simplify nursing or the drawing of milk, the author has devised two garments for wet-nurses. The material used for the

Fig. 57.—Wet-nurse uniform. Undervest, with one lapel raised, exposing breast.

outer garment is of yellow gingham, such as is used in the making of hospital uniforms—the yellow color being selected to distinguish the wet-nurse from the blue, as used by the nursing corps. The corset-waist is to be made of heavy muslin. The corset, if worn at all, should be of a very low type so as to avoid all pressure on the breasts. It is best of a cheap quality so that it can be replaced frequently for sanitary reasons. Each wet-nurse should be supplied with four uniforms and six nursing corset-waists (Figs. 56 and 57).

The Diet of the Wet-nurse.—There is danger of the creation of indolent habits through neglect of regular exercise and the lack of regular household duties, but even greater danger lies in the direction of overfeeding with unusual foods. The average wet-nurse is either obtained from an institution or a home in which the luxuries of life are limited, and she has been accustomed to a simple nutritious diet. Every attempt should be made to supply the nursing woman with a well-rounded diet of simple foods, with milk and cereals as the basis, and these supplemented with meats, soups, the common vegetables, limited amounts of fruits and plain desserts. Insofar as possible the aromatic vegetables, unripe and highly acid fruits, fried meats and rich pastries are to be avoided. We believe that, on the whole, too great stress has been laid upon the danger of the diet in the mother of a full-term infant, and in most cases the average mother can partake of a very full diet. However, in the case of the woman nursing premature infants, it should become a custom to allow only such foods during the first few days after her installation as can be given with perfect impunity. When a full, free flow of milk is established other vegetables and fruits can be added, one at a time, and after each addition to the diet a try-out should be given the milk. We have on numerous occasions seen marked intestinal distention and diarrheal attacks following even seemingly slight indiscretions of the diet on the part of the wet-nurse.

The diet should be so constituted as to meet the following requirements:

1. Furnish enough food of the proper kind to satisfy hunger and meet the physiological requirements of her body and produce a milk of good quality. This includes keeping the food elements in their proper proportions.
2. Prevent the presence of any obnoxious substances in the milk.
3. Prevent gastric and intestinal indigestion, constipation, or anemia in the wet-nurse.
4. Maintain the weight of the wet-nurse with little or no variation.

It is our hospital practice to furnish each wet-nurse with two quarts of good wholesome milk daily, and at least one pint of cereal gruel, preferably farina or cornmeal. A mixture of milk and cereal gruels makes a very good combination for drinking midway between meals. The remainder of the milk may be taken with the meals, either pure or in the form of cocoa, tea or weak coffee, in whichever form it is best taken by the individual woman. The latter is of considerable importance, as in the forced diets which are required, where an abundance of milk is demanded, distasteful foods soon become obnoxious.

DIET FOR WET-NURSES, PARTICULARLY FOR PREMATURE BABIES.

Meats.—Beef, lamb, chicken, fish, bacon.

Eggs.—Soft cooked only.

Vegetables.—Potatoes, carrots, spinach, lettuce (no vinegar), beets, string beans, canned corn, squash, asparagus, celery.

Fruits.—Prunes, apples, oranges, peaches, pears, apricots, raspberries, blackberries, cherries, strawberries (stewed only).

Cereals.—Rolled oats, rice, farina, cream of wheat, hominy grits, Wheatena, Pettyjohn's and all cooked wheat, oats, rice and corn cereals.

Fats.—Cream, butter, olive oil.

Desserts.—Soft puddings, gelatines.

Breads.—Wheat, rye, bran, corn, crackers, zwieback, coffee cakes and plain cakes.

Liquids.—Milk, buttermilk, kazol, cocoa, weak tea and coffee, malted milks.

Soups.—Broths and soups made with beef, chicken or lamb. Vegetable soups made with milk or with meat stock and vegetables.

Avoid.—Aromatic vegetables (onions, cabbage, turnips, cauliflower); acid vegetables (tomatoes, pie-plant, cucumbers); acid fruits; highly spiced or seasoned foods; salads with acid dressings; raw fruits, except oranges; fried foods.

MENU FOR ONE DAY.

Breakfast:

Fruit (orange, prunes or apple-sauce).
Cereal with cream and sugar.
Bacon (2 slices), or some other easily digested meat if desired.
Bread, toast or rolls.
Butter.
Cocoa or milk or weak coffee.

Dinner:

Broth or soup.
One meat from list given (roast beef or broiled chop).
Potatoes (old) in any form except fried.
Vegetables (squash, beets).

Light dessert (custard, gelatin).
Bread (white, rye or bran).
Butter.
Cocoa or milk or weak tea or coffee.

Supper:

One meat from list given (chicken).
Potatoes (creamed).
One vegetable (asparagus).
Cereal with cream and sugar (rice).
Stewed fruit (peaches).
Bread and butter.
Cake occasionally.
Milk or cocoa.

No candies should be allowed except as a dessert with one of the main meals.

If the nurse's appetite demands more food because of the large amount of milk secreted, or if insufficient fluids are taken with the meals to cover the fluid requirements, as previously stated, one or two midday, and one night luncheon may be given. These should consist of milk, milk and tea, malted milk or cereal decoctions, with crackers, coffee cake, etc.

Beers, malt-extracts and other rich drinks are not forced upon the nurse unless she is accustomed to them, and feels their need. It must always be remembered that an excess of fluids would naturally tend to dilute the milk unless the secreting gland be of exceptional development. Excessive feeding by giving of too frequent meals in the presence of anorexia will retard rather than increase the milk flow.

Exercise of the Wet-nurse and Her Work.—She should be impressed before her engagement with the fact that she will be required to do a moderate amount of work and exercise regularly out of doors. The former will be of service in promoting her general health, and both the work and the exercise will serve as a nerve tonic and prevent her becoming indolent. This does not mean that she should become a drudge, but that she should at least be required to care for her own room and her own infant's clothes, and should be made to feel that in return for her laundry work she would be requested to do some light general work about the house. Her exercise in the open air should so far as possible be at regular times. The question as to the care of the napkins of both babies is open to considerable discussion; and it may be stated that whenever it becomes necessary for the nurse to express her milk by hand, she should not be subjected to the handling of soiled napkins, whenever this can be avoided.

OTHER CONDITIONS INFLUENCING THE QUALITY OF THE BREAST MILK.

The Nervous and Mental State of the Nurse.—The nervous and mental state of the nurse is of the utmost importance, and wherever possible an emotional, nervous, erratic woman should be excluded, because of the tendency of these influences to suppress the flow of milk. Therefore, whenever possible, a woman of more or less phlegmatic temperament is to be selected. This is especially true in the case of a woman who is to be in close contact with and is to nurse an infant with neurotic tendencies. There is also the possibility of the same influence being manifest in time of slight indisposition on the part of her own infant, and such an individual is also more likely to resent the necessity of partial or entire artificial feeding of her own child to the advantage of the premature infant, when it has reached such an age when it may make greater demands on her supply.

Menstruation.—Menstruation rarely produces any serious disturbances. It is always a safe procedure to dilute the milk during the first and second day of menstruation when the nurse suffers considerable pain at these times.

Period of Lactation.—Period of lactation may or may not be a considerable factor, depending upon the individual woman. We had in our employ a nurse who had been with the institution for sixteen and a half months, and whose infant was eighteen months old, and who supplied us with the largest quantity and the best quality of milk of the four nurses in the institution.[1]

When possible a nurse should be selected after the first few weeks of lactation, at which time the colostrum has disappeared from the milk, and the quantity and quality of her milk has become established. After the first few weeks of lactation but little or no attention is to be paid to the age of the wet-nurse's baby as compared with that of the infant to be fed, and we have never noted any ill effects following the rule.

[1] The milk of this nurse was examined in the laboratories of the University of Chicago after seventeen months of lactation with the following result:

Albumin	1.30	per cent
Casein	0.69	"
Fat	3.54	"
Lactose	7.025	"
Salts	0.1885	"

It must be remembered that this is an exceptional case, and but few women under the stress of ordinary life can properly nurse their infants after the ninth to twelfth month.

THE NURSING.

The Infant's Bedroom.—Under ideal circumstances, this should be separated from that of the wet-nurse. This is especially true where a trained attendant has care of the infant. It should under all circumstances also be separated from the wet-nurse when she is of a low degree of intelligence and of a type not to be trusted with the care of the infant.

Method of Drawing Milk.—Numerous methods of obtaining milk from the breasts have been described, but only those most practicable of application will be detailed. These should be divided: (1) Into those in which the baby is placed directly at the breast, and (2) those methods by which the milk is drawn from the breasts and fed to the infant. Two methods are especially applicable where the baby is fed directly on the breast, and needs assistance because of its weakness.

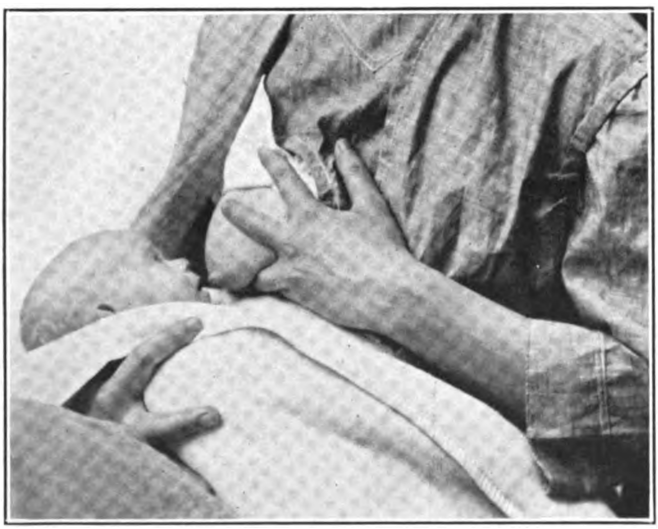


Fig. 58.—Proper method of holding baby during nursing. The nurse is seated on a low nursing chair with her right foot elevated on a low stool.

1. The premature infant is placed at the breast, and is supported there by the nurse's right arm while nursing at the right breast, and the left hand is used to grasp the breast just above the nipple between two fingers and the milk is expressed directly into the baby's mouth. In this way the baby is taught to take the breast, and at the same time receive its food with little effort.

This method can be continued until the baby has gained sufficient strength to nurse without assistance.

2. Much the same result can be accomplished by placing the wet-nurse's baby on the opposite breast during the nursing period, whereupon the simultaneous nursing on both breasts will cause a free flow of milk into both sides.

The methods by which the milk is drawn from the breasts and fed to the infant by hand or by other means are:

1. By the breast-pump. The modification of Holz vacuum apparatus, as devised by the author (Fig. 60), by which means the milk is drawn directly into two graduated 2-ounce flasks, which

Fig. 59.—Premature infant nursing one breast and wet-nurse's baby nursing the other. If there is a choice of breasts the premature should have the better one reserved for its use. This leaves the strong infant to develop the poor breast. If the premature is unable to empty its breast, nursing should be followed by expression or application of the wet-nurse's baby, if both are well.

can be filled to the quantity desired, and stoppered for future use, so that the milk is free from handling, and thereby avoid contamination. This type as well as other hand pumps are less practical than drawing milk by expression. Dr. I. A. Abt, of Chicago, has recently designed an electric breast-pump which promises to be of great value.[1]

2. By direct expression which is by all odds the method of choice and which is performed as follows:

Scrub the hands and nails with soap, warm water, and a nail-brush for at least one full minute. Wash the nipple with fresh

[1] Tr. Am. Ped. Soc., 1921.

absorbent cotton and boiled water or a freshly made boric solution. Dry the hands thoroughly on a clean towel and keep them dry. Have a sterilized graduate glass tumbler or large-mouthed bottle to receive the milk.

(*a*). Grasp the breast gently but firmly between the thumb placed in front and the remainder of the fingers on the under surface of the breast. The thumb in front and the first finger beneath should rest just outside of the pigmented area of the breast.

(*b*). With the thumb a downward pressing motion is made on the front against the fingers on the back of the breast, and the thumb in front and fingers behind are carried downward to the base of the nipple.

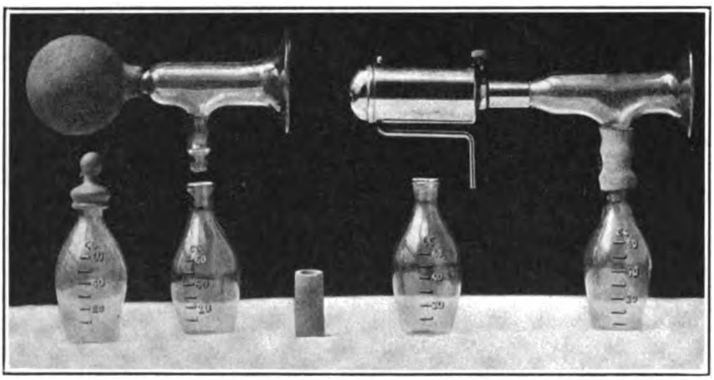


FIG. 60.—The pump is made in two types, the first fitted with a large rubber bulb of a size considerably larger than is ordinarily sold with breast pump, and the second attachment to which the Holz vacuum pump can be fitted. In place of the ordinary collecting bulb at the lower surface, an arm is so constructed as to allow the milk to flow into specially designed graduated 2-ounce milk flasks.

(*c*). This second act should end with a slight forward pull with gentle pressure at the back of the nipple, which causes the milk to flow out.

The combination of these three movements may be described as "back-down-out."

It is not necessary to touch the nipple.

This act can be repeated thirty to sixty times a minute after some practice.

Both breasts may be emptied if necessary, or they may be used alternately.

The act should be carried through with such gentleness as to cause little or no inconvenience to the nurse even in the first days

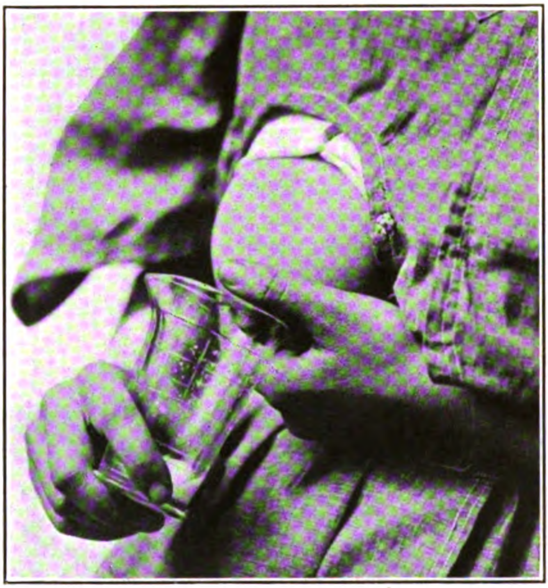


FIG. 61.—Direct expression, first motion.

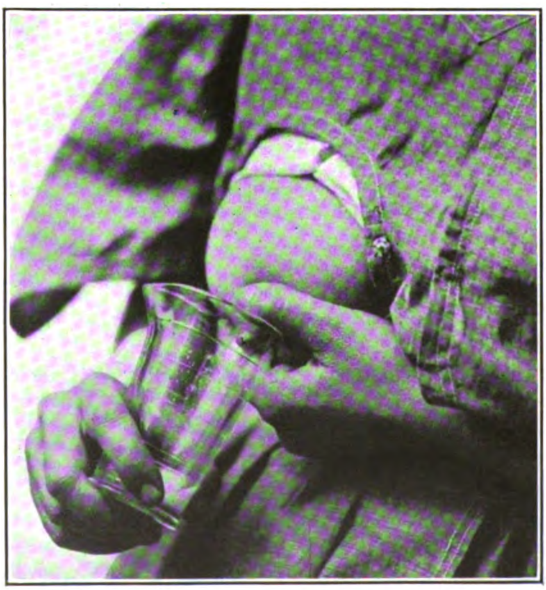


FIG. 62.—Direct expression, second motion.

of lactation. Some nurses prefer to use one hand for both breasts, others become ambidextrous and prefer to change hands.

By this means, following a little practice, the nurse can express from 6 to 8 ounces of milk from two good breasts in fifteen to twenty minutes. While drawing, each 2 ounces of milk is poured directly into sterile, stoppered bottles to prevent the fingers of the nurse coming in contact with the milk by overfilling the glass.

The milk should be covered at once by a sterile cloth held in place by a rubber band and kept on ice until used.

Daily Number of Expressions.—Expression is performed six times daily at regular intervals of four hours during the day and night.

HOSPITAL RULES FOR HANDLING WET-NURSES.

Samples of breast milk should be examined from each wet-nurse at regular intervals. Her breasts and method of expression should be inspected. It is not uncommon for wet-nurses to dilute their milk by adding cows' milk to increase the quantity when they experience a shortage.

Sick babies are not permitted to nurse from the wet-nurses' breasts; the expressed milk should be fed to the sick baby, whenever possible while it is yet warm.

If there be any question as to the reliability of the wet-nurse the milk must be drawn in the presence of a second person.

Wet-nurses for prematures must not be allowed to go to a general table for their meals, but must have their meals brought to them where they may partake of their food under the eye of a nurse who understands what their diet is to be. Wet-nurses have precarious appetites, as a rule, and they are more likely than not to have a craving for something that will either diminish the amount of their milk or impart some condition that will make it disagree with the sick babies.

Wet-nurses should be kept rigidly within regular hours in the institution. They should not be permitted to go out after night because they will do indiscreet things, eat foods calculated to interfere with their efficiency as wet-nurses, drink alcoholic stimulants, and so upset themselves generally and the milk supply will be diminished. On the other hand, the wet-nurses should be made comfortable, and should be given a sufficient amount of work in the institution to keep them busy. They are disposed to resent restraint and unless their time is fully occupied, they will be sure to fret and thus diminish their milk supply.

The wet-nurse should be obliged to observe the laws of health and cleanliness; they should be obliged to bathe regularly and it should be the duty of the head nurse of the department to see that their bowels are kept in proper condition and that their genitals are clean and healthy.

Wet-nurses should never be employed until the Wassermann test has been made, and until a competent physician has given them a thorough examination to determine the presence or absence of specific disease. They should never be permitted to go on duty with running ears, sore eyes, sore throat, bad teeth or any discharge from a mucous membrane, or any skin eruption.

The wet-nurse should be given a certain number of babies to feed, and as long as her milk agrees with them, and she is in perfect health, should be kept to the same babies without any admixture of the milk of any other nurse This acts as a check on any indiscretion as it would be reflected in the baby.

CHAPTER VII.

CARE AND NURSING OF PREMATURE INFANTS.

All infants born three weeks or more before full term should be considered premature and treated as such. Every infant born after the sixth month should be given an opportunity for life by the administration of necessary care and diet. Healthy premature infants when properly cared for will frequently reach the full development of the full-term infant by the end of the first year, and the majority of those surviving usually develop a normal body and mind, notwithstanding the fact that they are more commonly subject to megacephalus, rickets, spasmophilia, anemia, gastro-intestinal, respiratory and circulatory affections, all of which can be overcome without leaving any sequelæ, unless based upon some congenital anomaly.

To be successful with these infants a certain routine must be followed:

1. Preparation for their home or hospital care must be made, whenever possible before labor begins.
2. Their immediate care after birth is of greatest importance.
3. Their general care must be adapted to their individual needs.
   (*a*) Refrigeration must be prevented.
   (*b*) Skilled nursing is essential.
4. Human milk must be provided for those born before the thirty-sixth week.
5. The daily routine must be adapted to the infant's age and development.
6. Contact between the infant and individuals not concerned in its immediate care must be avoided. Attendants ill with colds and other forms of infection should observe most rigid rules of asepsis to avoid cross infections.

PREPARATIONS FOR THE INFANT'S BIRTH.

In case of expected premature labor immediate preparations must be made for the reception of the infant into a proper environment. The preparation must not be delayed until labor has begun, otherwise many viable premature infants will be lost. If the proper facilities cannot be furnished in the home, the mother should be persuaded to enter a hospital before confinement. She should be impressed with the fact that every day of added intra-uterine life will improve the infant's chances not only for life, but also for normal development.

Preparation for the proper conduct of labor should be complete whether in the home or hospital. The mother should be prepared with great care and every effort made to conduct an aseptic labor. The room should be selected and prepared to meet the needs for labor and the requirements of the infant. It should be well ventilated and properly heated to at least 70° F. Blankets and pads into which the baby is to be received should be warmed. The basket-bed or incubator-bed should be prepared for its reception by proper sterilizing and heating, so that all exposure to cold will be avoided. Everything must be in readiness for the care of the cord, eyes, mouth, skin and treatment of asphyxia. These should include a catheter and hot bath, and facilities for transportation of the infant to a hospital, if necessary.

IMMEDIATE CARE OF THE PREMATURE INFANT.

Asepsis.—The greater susceptibility of the prematures demands even more painstaking observation of the rules that hold good for new-born infants in general. These infants succumb more readily to infection and are much less resistant than are the full-term infants. Again, the frequently complicated feeding technic gives more opportunity for disturbances of the digestive tract so that in every form of indirect feeding careful attention to details must be insisted upon. Also the danger of infection of the respiratory passages by careless exposure and aspiration of food are not to be underestimated.

Reception of the Infant.—A warm sterile pad, towel or preferably a blanket should be in readiness to receive the infant. As soon as the head is born the face and eyelids should be gently sponged with sterile warm water, and the mucus should be removed from the air passages by carefully wiping the nose and mouth with a soft pledget of gauze. The body and cord should be protected from all contact with feces and other infected matter. After the body is born the infant should be placed so that the head is dependent, allowing the mucus and secretions which may have accumulated in the respiratory passages to escape.

Preservation of Body Temperatures.—The preservation of temperature demands a very careful supervision immediately following birth, proper attention must be paid to the thermolability and tendency to subnormal temperatures. The chief object in the preservation of the temperature is the prevention of excessive heat loss, which in itself may be a danger to the infant. This will also diminish the energy loss. The infant must be wrapped in material with poor heat conduction, and then placed in a warmed bed. Both are essential to a successful maintenance of body temperature.

The preservation of heat must be begun immediately after birth of the infant, preferably on the confinement bed itself, as the extent of the initial temperature loss is of no mean consequence to a premature infant. After severing the cord the infant should be placed in a heated basket or incubator-bed, which should be a part of the equipment of the delivery-room.

In the home, hot-water bottles, a properly protected electric pad (p. 224), or an improvised incubator (p. 223) will answer the purpose. It should be remembered that these infants are easily burned and such burns are usually fatal.

In small prematures the cotton-pack, completely enveloping the infant, except for the face and genito-anal region, answers very well. To the genital region and anus a napkin of cotton or gauze combination may be applied. A jacket may be placed on the outside of the cotton to hold it in place.

Treatment of the Cord.—The time of tying and section of the cord will depend entirely on the general condition of the infant and to some extent on the obstetrician's ability to prevent undue exposure of the infant to cold. In the absence of marked asphyxia it is well to allow the pulsation of the cord to become weakened or to disappear before ligation. This usually requires from one to five minutes during which time the infant will receive from 30 to 60 cc of blood from the placenta. This blood should be conserved, when possible.

The cord should not be tied too close to the skin. Great care must be exercised in tying the cord to prevent cutting it in two with the ligature which is easily accomplished in the premature, therefore it is always well to leave sufficient space for a second ligature behind the first in case of an accident.

Asphyxia.—The possibility of asphyxiation of the premature infant must be borne in mind throughout the entire labor. The heart tones should be carefully watched and in cases of prolapse of the cord, if it cannot be successfully replaced, it may be necessary to induce a rapid delivery of the infant. Any accumulated secretions or aspirated material must be removed by inversion of the child and if necessary by aspiration by means of a catheter. In more extreme degrees of asphyxia early separation of the cord may be necessary so that artificial respiration and a hot bath may be instituted (p. 244).

The irritation of the catheter in the pharynx will frequently reflexly stimulate respiration. It should, however, be remembered that the use of the catheter is not without danger to the operator because of the frequency of syphilis as a cause of premature birth. If these procedures fail to bring about the desired result the infant should be suspended by the feet, the forehead resting lightly on the bed or table so as to deflect the chin and straighten out the trachea and then the chest is compressed between the thumb of the right hand resting on the back and the four fingers of the same hand resting on the anterior wall of the chest.

This act should be repeated from sixteen to twenty times a minute by compressing and suddenly relaxing the chest wall. This should be continued for at least one minute in severe cases to insure success. At the same time a nurse or assistant should wipe the excess of mucus from the nose and throat. The child is then placed in a warm bath (about 105° F.) for five minutes, and then placed in a heated bed. In extreme cases the procedure must be repeated. Administration of oxygen, about 120 bubbles per minute, may be of value, if administered through a catheter inserted in the mouth or a properly constructed mask. Careless handling and traumatizing the infant or too rapid performance of artificial respiration is productive of more harm than good and must therefore be avoided. There must be definite indications for all manipulations undertaken. If the infant appears to be recovering spontaneously it should be left alone.

It must be borne in mind in the conduct of all premature labors that the anesthetics, if used in labor, tend to weaken the uterine contractions, thus prolonging labor and favoring asphyxia and a sufficient quantity of the drug may pass into the infant to seriously affect it, which is especially true of scopolamine-morphine anesthesia.

All premature infants whether asphyxiated at birth or not should be carefully watched for cyanotic attacks during the first days of life, as such attacks may develop suddenly and without warning. They may be due to a disturbance in the pulmonary circulation, to a congenital atelectasis, or to injury of, or hemorrhage into the respiratory center in the medulla. At other times they are precipitated by intra-abdominal distention which may interfere with cardiac or respiratory action. For further discussion of this condition see Cyanosis (p. 241).

Care of the Mouth and Nose.—Every effort must be made to avoid trauma of the mucous membranes of the nose and mouth, because of the danger of secondary infections. Cleansing of the nose should be done by the use of soft cotton pledgets or applicators. In wiping out the mouth only soft material is permissible. Much can be accomplished by facing the child with the mouth downward or laterally with the trunk elevated, so that the mucus can gravitate toward the mouth.

Care of the Eyes.—One per cent silver nitrate solution or 25 per cent argyrol should be used to prevent ophthalmia neonatorum. The nitrate of silver solution should be neutralized with a normal saline solution instilled in the eyes. Not infrequently the application of silver nitrate will result in some inflammatory reaction of the conjunctiva in the first six to twelve hours after its application. This is especially frequent in premature infants and is usually relieved by the application of cold boric-acid solution to the lids. It is not to be confused with the more serious specific ophthalmia which develops on the second or third day. In case of doubt a microscopic examination of the purulent discharge must be made. In all cases an old silver nitrate solution which has undergone decomposition should be avoided, as such solutions are far more prone to irritate the sensitive conjunctiva.

Care of the Skin and Genitalia.—It is of the greatest importance that premature infants shall be handled as little as possible. And when there is doubt as to the advisability of giving the initial warm bath, it is best omitted, because of the danger of causing a collapse. When the bath can be given without chilling it is indicated in most infants weighing 1500 gm. or more. In smaller infants and those showing evidence of atelectasia or asphyxia, it may be needed to stimulate the respiratory functions. Oiling the body is unnecessary and is to be avoided. The genitalia should be carefully cleansed with a boric-acid solution or sterile water without trauma. The same is true of the buttocks, after which a small pad of cotton or combination is applied to the genitalia and buttocks.

Dressing the Cord.—Either a dry or alcohol dressing should be applied. The cord usually dries by mummification and drops off in most instances by the end of the first week, averaging somewhat later than in full-term infants. Every precaution should be taken to prevent trauma of the stump and secondary infection. This applies more especially to the bathing of the infants in emergencies for cyanotic spells and hypothermia.

Examination for Congenital Anomalies and Disease.—Before the infant is left by the physician it should be examined for congenital anomalies and evidence of syphilis and other diseases.

GENERAL HYGIENE AND ENVIRONMENT.

Requirements of a Hospital Nursery Unit.—This depends greatly upon the method used for maintaining external heat.

1. Superheated rooms without heated beds.
2. Individual heated beds.

When the *superheated rooms* are in use separate rooms for the

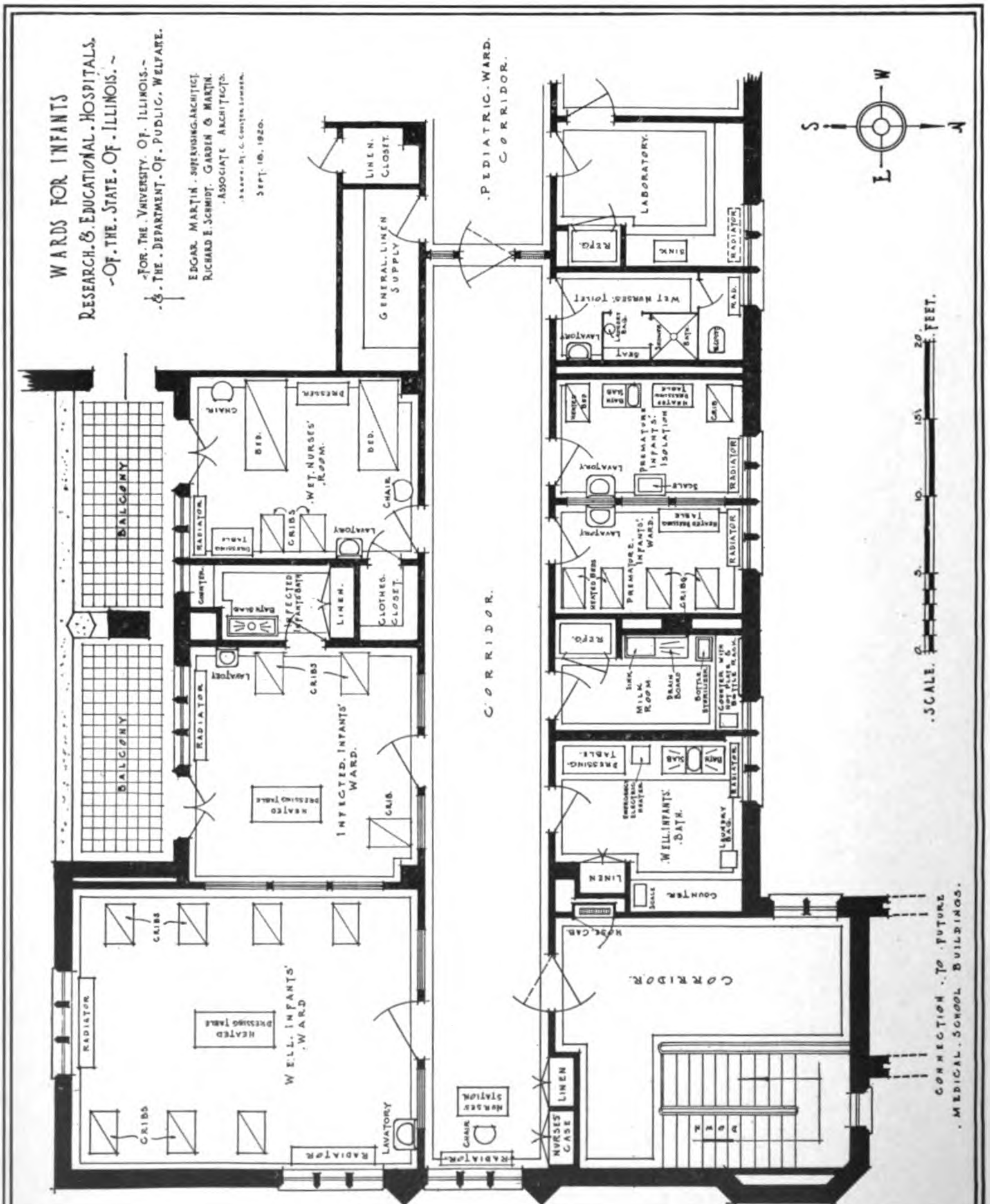


Fig. 63.—Floor plan of infant ward for a general hospital with limited floor space. Wards for infected infants are equipped for complete isolation.

older and better-developed infants must be supplied to gradually accustom them to ordinary room temperature. However, this extra room is not necessary when external heat is applied in individual beds in which the temperature can be regulated to meet the needs of each infant. In using the latter the room can be held at a temperature approximating 70° F. In point of economy of space and special care for the infant the latter method has every advantage.

When individual heated beds are used the following units are required in a properly regulated department.

Room Containing Heated Bed.—A room with a south exposure is preferable. In such a room the matter of ventilation will depend to a large extent upon the type of heated bed which is used. When the old type of closed incubator is used, it must necessarily receive fresh air through a pipe passing through the wall of the building or an opening in a window, thereby supplying the bed with air from the outside (Fig. 136). When an electrically heated bed or home improvised bed is used the infant is dependent upon the general ventilation of the room for its supply of fresh air.

Such a room is best constructed with double windows and transom which can be regulated at will according to the season and existing weather conditions. Such a system of ventilation should be sufficiently flexible to permit regulation to meet exigencies which may arise due to instability of the general heating plant. It has been our experience that when a well-constructed superheated bed is used, variations of from 6° to 8° F. in the room temperature during the twenty-four hours cause little inconvenience to the infant.

It should be remembered that the beds should not be placed in a direct line of draft between the windows and the doors. The room should be built or selected with this in mind. Such a room should also contain a hygrometer and special thermometers which register not only the present temperature but also the extremes for twenty-four hours (Taylor Instrument Company). Such a thermometer is one of the best methods of testing an efficient nursery. Further discussion of incubator rooms, incubators, superheated beds and similar apparatus are covered under the special chapter on Incubators.

This room is to be used only for well new-born prematures in their individual beds and older infants who have been gradually accustomed to ordinary room temperature.

The Nursery.—The nursery should be a room independent of the station in which the superheated beds are kept. It should be provided with double windows, a good system of heating, and must be kept immaculately clean. Good ventilation and general cleanliness are essential. Unless a special bathroom can be pro-

Fig. 64.—Hospital bathroom, located between two small wards for infants, showing two metal water jackets resting on a porcelain sink. These can be filled with water and have a registering thermometer for indicating the temperature before giving the bath. They are covered with a clean towel for each baby. Baby is showered from an automatic mixing tank which registers temperature of the water in the tank. The room further contains a scale and a low dressing table with the various dressings, powders and ointments to be used. Also low nursery chairs, collapsible bags for soiled linen and waste basins.

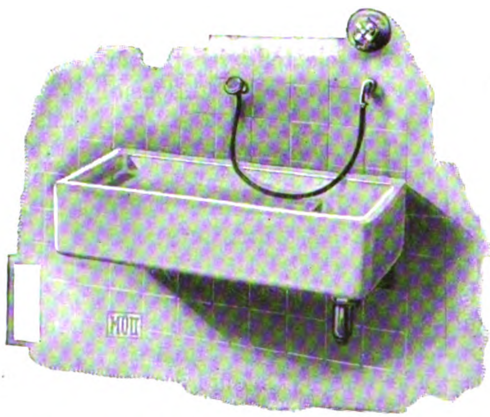


Fig. 65.—Divan bath with thermostatic mixing control.

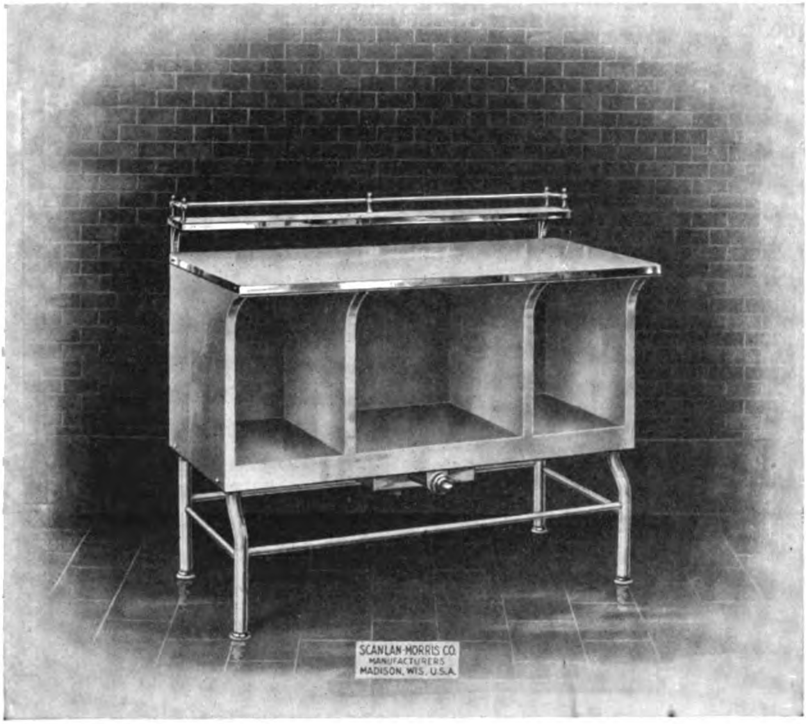


Fig. 66.—Electrically warmed dressing table. (DeLee.)

Fig. 67.—Large unheated dressing table, provided for dressing of two babies. Scale in center and closed cabinet for clothes. (Couney.)

vided, the nursery should be furnished with the following equipment.

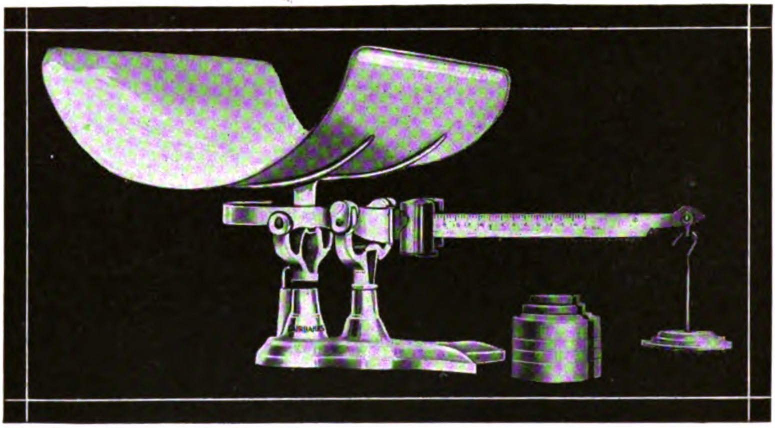


FIG. 68.—Scale for weighing infants.

FIG. 69.—Thermometer registering present and extreme room temperature during the twenty-four hours. It is to be adjusted by a small magnet once daily.

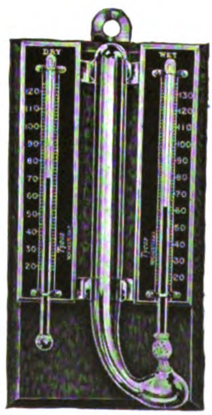


FIG. 70.—Hygrometer. Wet and dry bulb.

1. A bathing slab or board. We find a metal jacket which can be filled with warm water very serviceable (Fig. 64). The Divan bath with thermostatic mixing valve is well designed for this purpose.

2. A heated dressing table provided with cabinets for storing and warming clothes.
3. Supply closets for linens.

Reading of dry bulb ther.	1°	2°	3°	4°	5°	6°	7°	8°	9°	10°	11°	12°	13°	14°	15°	16°	17°	17.5	18°	18.5	19°	19.5	20°	20.5	21°
	RELATIVE HUMIDITY.																								
65°	95	90	85	80	75	70	66	62	57	53	48	44	40	36	32	28	25	23	21	19	17	15	13	12	10
66°	95	90	85	80	76	71	66	62	58	53	49	45	41	37	33	29	26	24	22	20	18	17	15	13	11
67°	95	90	85	80	76	71	67	62	58	54	50	46	42	38	34	30	27	25	23	21	20	18	16	15	13
68°	95	90	85	81	76	72	67	63	59	55	51	47	43	39	35	31	28	26	24	23	21	19	17	16	14
69°	95	90	86	81	77	72	68	64	59	55	51	47	44	40	36	32	29	27	25	24	22	20	19	17	15
70°	95	90	86	81	77	72	68	64	60	56	52	48	44	40	37	33	30	28	26	25	23	21	20	18	17
71°	95	90	86	82	77	73	69	64	60	56	53	49	45	41	38	34	31	29	27	26	24	22	21	19	18
72°	95	91	86	82	78	73	69	65	61	57	53	49	46	42	39	35	32	30	28	27	25	23	22	20	19
73°	95	91	86	82	78	73	69	65	61	58	54	50	46	43	40	36	33	31	29	28	26	24	23	21	20
74°	95	91	86	82	78	74	70	66	62	58	54	51	47	44	40	37	34	32	30	29	27	25	24	22	21
75°	96	91	87	82	78	74	70	66	63	59	55	51	48	44	41	38	34	33	31	30	28	26	25	23	22
76°	96	91	87	83	78	74	70	67	63	59	55	52	48	45	42	38	35	34	32	30	29	27	26	24	23
77°	96	91	87	83	79	75	71	67	63	60	56	52	49	46	42	39	36	34	33	31	30	28	27	25	24

FIG. 71.—Humidity table for use with wet and dry bulb hygrometer.

FIG. 72.—A milk station consisting of three rooms. Room 1.—For all used bottles, bottle washers and steam bottle sterilizers. Room 2.—A clean room for preparation of formulæ. This room also contains milk separator, fat testing apparatus and butter churn. Room 3.—Pasteurizing and sterilizing apparatus.

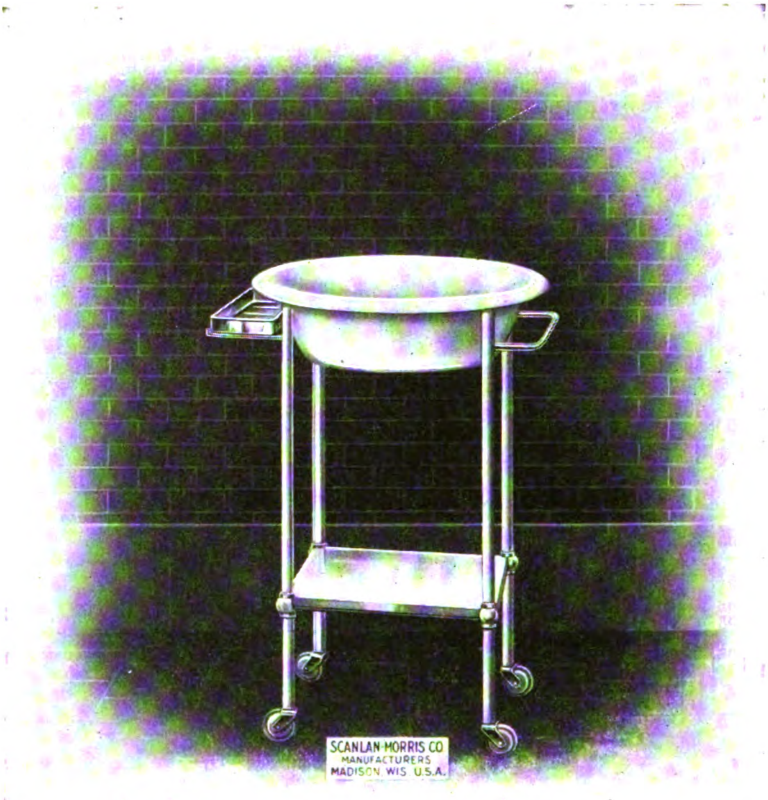


FIG. 73.—Portable bath basin for individual use of infected infants. Basin can be removed for sterilization.

FIG. 74.—Individual bed with utensil compartment for infected cases.

4. A well-constructed balance scale graduated to 4 gm.
5. A hygrometer (Figs. 69 and 70).
6. Thermometers registering the present and extreme temperatures for twenty-four hours (Fig. 70).
7. A time clock should also be provided and all feedings registered by this method, so that the supervisor may have a constant check on the activities of her assistants.

The general hygiene and care of the infant in the nursery is second only in importance to an ample supply of human milk and a maintenance of the body temperature of the infant.

Milk Stations.—A milk station for preserving and dispensing breast milk and artificial diets should be a part of the equipment of every general and special hospital (Fig. 72).

Wet-nurses' Quarters.—Wet-nurses' quarters should provide living and sleeping-rooms for the wet-nurses and their babies. The ideal requirements for such a unit are described under the chapter on Wet-nurses, p. 117.

A shower bath and toilet facilities should be provided for the special use of wet-nurses but not in living quarters.

SPECIAL QUARTERS FOR SICK INFANTS.

It is of the greatest importance that infected premature infants be grouped according to their ailments and that complete facilities for caring for these infants be established, in order to avoid cross infections. Two such units should be provided whenever it is expected that a considerable number of premature infants are to be cared for, and should include facilities for bathing, feeding, and the general care of patients. Gastro-intestinal and respiratory infections must be kept separated and treated as septic cases. Syphilitic infants and cases of gonorrheal ophthalmia must also be provided with separate quarters. Thrush and furunculosis which frequently develop into severe types should also be isolated.

Aseptic nursing is imperative to the welfare of the department. Soiled linens, clothes, bottles, thermometers and all other utensils must be handled as infected material.

A *complete department* should therefore provide for:

A. *Well Infants.*—A room containing heated beds for the early care and cribs for graduates. The further needs are: A heated dressing table, a supply closet, thermometer (high and low), hygrometer, time clock, electric heater for emergency, screens and a lavatory.

A nursery with bathing facilities, supplied with: A bath slab, a lavatory, a heated dressing table, shelves for toilet articles, a gas or electric plate, an electric heater for emergency, a scale, thermometer, supply closet.

A special bath-room when possible should be provided so that bathing in the nursery may be avoided.

Quarters for wet-nurses with independent bath and toilet facilities, equipped with: Beds, cribs, chiffoniers, dressing table, nursery

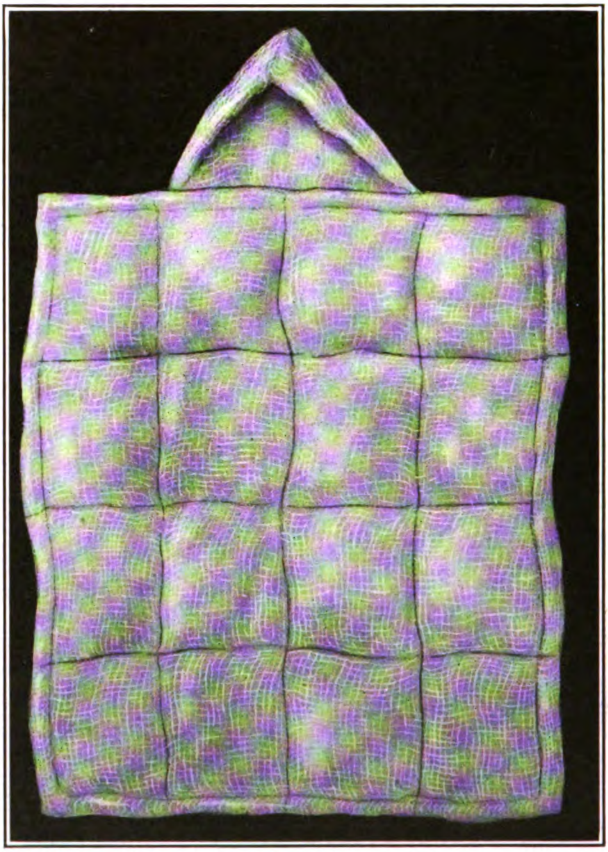


Fig. 75.—Emergency robe with hood made of gauze and cotton combination.

chairs and lavatory. The bath room should have a shower bath, dressing room, toilet and lavatory.

A milk station containing a sink, refrigerator, work table, tubs for washing utensils, steam sterilizer, bottle and food racks.

Nursing staff including a directing nurse and assistants.

Wet nurses.

B. *Infected Infants.*—Room equipped with heated beds and cribs and provided with bathing facilities. This room should further contain a lavatory, heated dressing table, scale, thermometer, hygrometer, emergency electric heater, supply closet and screens. The bath tub in this room may be of the small ambulatory type or of the Divan slab type. Both may be easily sterilized.

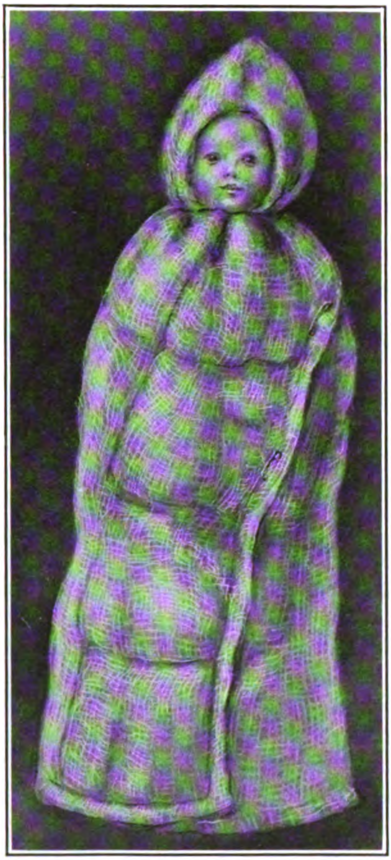


FIG. 76.—Emergency robe applied to infant.

The *nursery* should be considered as the center of the unit and when a separate bath room is provided, the former may be used for housing the graduates. The temperature of this room should range between 78 and 80° F. during the hour of bathing, at other times 70 to 75° F. The entire station must be thoroughly cleaned at least every second day and disinfected by scrubbing immediately after the diagnosis and removal of infectious cases.

The Nursery Staff.—The selection of a personnel for the nursing staff of a unit established for the care of premature infants requires great care. Nurses assuming these responsibilities must be intensely interested in their work. They must be willing to make many necessary sacrifices while the infant is passing through the critical stages. They must, at all times, be prepared to meet the emergencies of asphyxia and to counteract the spells of cyanosis. These two factors in themselves require almost constant diligence, otherwise the work of previous days will go unrewarded. They must use good judgment to prevent over- and underfeeding, as to a very great extent the size of the individual meal will be dependent upon the physical condition of the infant at the time of feeding. In no other class of patients is it so necessary to change or modify on short notice previous orders for diet. The nurse must know the indications for and the methods of administering catheter feedings, colonic flushing, tubbing and the application of artificial respiration.

In our hospital wards we have found the constant changing of nurses, as is so frequently the case in meeting the curriculum for nurses' training in general hospitals, to be of the greatest disadvantage. Far better results are obtained when the nurse in charge has under her care assistants who need not necessarily be nurses in training, but preferably young women who are especially preparing themselves for the care of young infants, and who can be relied upon to stay in the station for long periods of time. Such women become expert in the handling of these infants, can frequently feed them with a minimum of excitement of their reflexes, and soon learn to bathe and give them their exercise and massage, which is so essential to every infant in order to prevent "hospitalization."

The *ideal nursing staff* for such a station is, therefore, one consisting of a well-trained supervising nurse and a corps of assistants desiring this training, and who are willing to remain in this service for a long period of time.

DAILY ROUTINE.

Removal of Infants from Their Beds.—The position of the infant in bed should be changed at regular intervals. The removal of infants from their beds should be practised with forethought. The small infants should, so far as possible, be manipulated only upon a definite indication: (1) For cleanliness, including bathing; (2) exercise, including gentle massage after the first week or two. In most instances the food, when administered other than by catheter, can be given without removing the baby from the bed. Catheter feeding in infants not subject to cyanotic spells can often be performed to advantage without removal from the bed. When cyanosis is present or easily precipitated the infant should be removed from the bed during feeding.

In preparing the infant for *permanent removal* from the heated bed the room temperature should be gradually lessened until 70° F. is approached.

Next the infant is placed in an infant's crib, the sides of which have been padded to prevent extreme currents of air from coming in contact with the infant and thereby increasing radiation. These cribs may remain in the same room as the individual heated beds, or may be kept in the nursery if it be the more desirable room of the two, when there is a separate bath room. The infant should not be kept permanently in a room in which a considerable number of infants are being bathed throughout the day. There is no need for shortening the stay of the infant in the heated bed if the temperature of the surrounding air is gradually being lowered as the infant develops. Depending upon the age and development, the average length of time in a heated bed varies from one to six weeks. It is good practice to place the older infants in the crib during the day and to replace them in the heated bed during the night when the heating of the house or ward is uncertain.

The Bath.—In the very weak infants it is frequently advisable to omit the first and the daily bath for two or three days. It may, however, be necessary to use the warm bath to stimulate the infant during its cyanotic attacks.

It should be a fixed rule in the care of premature infants to handle them as little as possible, because of the danger of provoking cyanotic attacks and the regurgitation of food. It should be our object to keep the skin clean and active. The practice of oiling the infant as a routine measure is to be avoided. If the bath cannot be undertaken without danger of chilling the infant, it should be either dispensed with or postponed for a more opportune time; or a partial bath may be given without removing it from the heated bed by washing the face, buttocks and genitalia.

Indications for and Methods of Administering Baths.—The earliest baths should consist of a sponging with water at 105° F., one part of the body only being exposed at a time to prevent chilling and the process carried forward as rapidly as possible in a room of not less than 75° F., otherwise it is best omitted in the very small infants.

As infants grow older they may be dipped in or sprayed with water heated to 100° F., and this may be gradually lowered to 95° F.

Under no circumstances should the infant be bathed without first taking the temperature of the water and the room.

Infants with subnormal temperature may frequently be stimulated and the temperature raised by placing them in a warm bath which is held between 103° and 106° F.

In cases of hyperpyrexia a bath from 4° to 5° lower than the infant's temperature with cold to the head is of therapeutic value.

In the presence of cyanotic attacks the plain warm bath or weak mustard bath with slight friction repeated as indicated are probably the best therapeutic measures. During such attacks the infant should be handled gently as not infrequently careless and rough handling will result in death during these cyanotic attacks.

All bathing before separation of the cord should be carried out with the idea of promoting surgical cleanliness.

Gentle friction and light massage are of great value following the bath. Neither of these methods of stimulating the circulation must be overdone. Bathing should always be done before feeding.

Care of the Eyes.—If properly cared for at the time of delivery and if there is no reaction to the solutions used at that time, they require no further attention except ordinary cleanliness. The nurse should be warned against getting bath water, or more dangerous, mustard water in the eyes. In cases of ophthalmia the treatment is practically that as used for full-term infants with greater care for the prevention of trauma and destruction of the eye.

The Nose and Mouth.—Unless there is a direct indication due to plugging of the nose or an infection of the nose and mouth, there should be no manipulation of these mucous membranes, because of the danger of abrading them and opening fresh surfaces for infection. In the presence of upper respiratory tract infections or stomatitis, the greatest care should be taken in applying local treatment as advised in the special chapter dealing with these diseases.

The use of the nasal catheter is always a dangerous procedure and even the passing of the catheter through the mouth may result in trauma if not carefully performed.

The Breasts.—In simple mastitis the breasts should be anointed with camphorated oil and a light pad of cotton held in place by a snug breast binder. The dressing may be changed every second, third or fourth day as indicated. In case of abscess formation, which is of very infrequent occurrence in prematures, incision and drainage should be performed.

The Genitalia.—The genitalia more especially the vulva in girls should be handled with extreme care in order to avoid trauma and infection. Small cotton combination pads should be applied to the buttocks and genital organs in order to receive the feces and urine. They should be frequently changed in order to avoid irritation from the excreta. By the use of these small pads which are described under the chapter on clothing, the frequent change of diapers can be avoided.

When there is evidence of infrequent or painful urination, which is more especially true in a male infant, it should be immediately inspected for evidence of occlusion due to the drying of secretion or exudate in the presence of an ulcer at the meatus. The buttocks are easily irritated by the decomposing urine and acid stools, and these parts readily become infected. In most instances the napkin can be changed without removal from the bed. In the treatment of all lesions about the genitalia an attempt should be made to keep the parts dry and clean. If water proves irritating a starch water may be substituted or the parts may be cleansed with benzoated lard. The parts are then dusted with stearate of zinc or rice starch. When these simple methods fail, a 1 per cent mixture of balsam of Peru in castor oil or lanolin may be used. Our best results have been obtained in older infants when the buttocks are exposed to warm dry air through the medium of an incandescent electric light or sunlight if the latter is possible without the danger of chilling the infant. In small prematures the parts may be left uncovered in the heated bed. Small rolls of cotton may be used to separate the folds of the skin.

The present-day use of washing powders, which are retained in improperly rinsed diapers and which lead to a rapid decomposition of the urine, may be a source of intertrigo.

Delayed urination is not infrequent and should lead to an inspection of the genital organs. A delay of twenty-four hours in the passage of the first urine is quite common in premature infants. If the infant is otherwise apparently normal, it should not be a cause for too great concern, and it is to be remembered that a small quantity of colorless urine may dry out and go unobserved. The best treatment is the administration of fluids approximating one-twelfth to one-twentieth of the body weight of the infant during the first day or two, and later approximating one-sixth of the body weight. This is inclusive of all fluids administered. A warm moist pad over the lower abdomen and pelvis or a warm bath will frequently cause spontaneous urination.

Uric-acid crystals and urates are very commonly found in the urine of the premature causing a pinkish stain on the napkin and are most commonly due to marked concentration of the urine. At autopsy, however, more frequently than in the full term, do we find these salts deposited in the kidneys. Considerable pain may be caused by the passage of these deposits through the ureter. In every case fluids should be pushed.

The Bowels.—The anus should be carefully inspected shortly after birth to ascertain the presence or absence of anomalies. Delay in passing the first stool may be due to one of many causes, such as delayed peristalsis, weak abdominal wall, contracted sphincter and accumulation of feces, most commonly in the sigmoid or cecum.

We believe it is a good custom to attempt to promote a bowel movement before the beginning of milk feedings. Frequently the administration of inert fluids per mouth will promote peristalsis. We do not hesitate to give a 1- or 2-ounce normal saline colonic flushing. The amount used depends upon the development of the infant. A small glycerin or soap suppository answers. If there remains doubt as to the patency of the intestinal tract, a small dose of castor oil, 0.5 to 1 cc (8 to 15 drops), may be administered per mouth. Once the patency of the intestinal tract has been established, intestinal evacuations are usually spontaneous, more especially so with infants fed on breast milk or with high carbohydrate mixtures. For further treatment see Constipation.

Care of the Skin.—The skin of the premature is very delicate and covered with lanugo and prominent sebaceous glands. There is a great tendency for the skin to dry and crack and to desquamate in large flakes. This is especially true in infants suffering from marked jaundice. There is also great tendency for papular, vesicular and pustular eruptions of various types to develop. Erythematous eruptions are of frequent occurrence. All of these conditions will call for a modification of the daily routine, insofar as the baths and local skin care are concerned. The greatest danger is due to secondary skin infections which is especially true of the syphilitic infant. The various forms of dry treatment of these lesions offer the best results with the least danger of spreading. The application of silver nitrate to each pustule and vesicle after cleansing with alcohol have given us the best results, except in the case of syphilitic infants where local mercurial treatment is indicated.

The daily care of the skin should therefore consist of the avoidance of trauma and exposure to secondary infections in the bathing and handling of the infant, the removal of all excretions, the separation of irritated folds by a layer of cotton, and the dry treatment of all non-suppurating skin lesions, and antiseptic treatment, cauterization or specific treatment of open lesions.

Delayed Separation of the Cord.—Delayed separation of the cord may be hastened by the application of 5 per cent silver nitrate solution or 50 per cent alcohol dressings. In the use of the latter a few drops of alcohol may be applied to the dressing at regular intervals. When the hard, dry cord remains intact far beyond the usual time for separation it may be necessary to cut through the remaining strands, using great care to avoid the live tissues. Granulations are best treated by the application of silver nitrate solution or hard stick.

Body Temperature.—The body temperature must be taken through the rectum. It should be recorded morning and evening. An individual thermometer should be furnished for each infant. Fluctuations in body temperature are more marked than in the full-term infant with a tendency toward *hypothermia*. A minimum of 97° F. should be considered the lowest compatible with progress. Attempts should be made to limit the daily fluctuations to 1.5° F.

Subnormal temperature may result from undue exposure at birth, subsequent carelessness, lack of development of the nervous system, absence of a good layer of subcutaneous fat, respiratory insufficiency circulatory weakness and insufficient heat production due to lack of food or defective metabolism.

These etiological factors are to be counteracted by definite therapeutic measures.

Prevent undue exposure and trauma from the moment of birth.

The infant should be placed in a heated bed of proper construction and kept there under constant supervision. The temperature of the heated bed should be varied with the needs of the individual infant. Small prematures and congenital weaklings with marked hypothermia should temporarily have a surrounding temperature varying from 85° to 95° F. Older and stronger infants are better placed in a bed at 75° to 80° F. As the infant develops its vital functions and the subcutaneous fat increases, the temperature of the bed should be gradually lowered to that of the nursery, which should be kept at about 70° to 75° F. It should be the rule to regulate the temperature of the heated bed by the rectal temperature curve, and while it may be impossible to bring the body temperature to normal, the degree of hypothermia is our best guide in the application of external heat.

It may be necessary to place the infant in a hot bath to raise the temperature and stimulate respiratory and cardiac function following syncope.

Removal from the bed should follow definite indications, ordinary feeding, changing napkins and the ordinary routine measures can be carried out in the bed.

The body must be insulated by proper clothing to be described.

The body fluids, after the first few days, must be maintained by an intake of from one-sixth to one-eighth of the body weight in fluids in twenty-four hours, and this must include a caloric intake of more than a sustaining diet, 70 calories per kilo after the first ten days of life (p. 180).

Respiratory and circulatory functions must be protected and at times stimulated.

Hyperpyrexia frequently results from an overheating of the bed, and when a high temperature is noted the temperature of the bed should be considered as a possible cause.

Infections of all kind tend to the development of fever, but on the whole the reaction is less than in the full term, however, the exception may be true. We have found massive pneumonias at autopsy which were unassociated with temperature above the average normal.

The Pulse.—The pulse may be imperceptible in the extremities and require auscultation of the heart for timing. The cardiac action will usually range from 100 to 180 per minute in the small and weak infants, although occasionally a very slow pulse is noted, which latter usually precludes a bad prognosis. The best indicator of proper cardiac function is the infant's general circulatory condition; it gives far more information than the number of heart beats.

Respiration.—The respirations normally vary from 20 to 60 per minute in different infants and are to a large extent dependent on the heart action in infants not suffering from atelectasis or central disturbances. During cyanotic attacks they become almost imperceptible and may be temporarily suppressed. Again the general condition of the infant is the best guide.

Weighing should be done at a specified time each day as part of the general routine, with a good scale. The infant should, unless contraindicated, be undressed for this purpose, and this is best done before the bath. The relation between the time of the last feeding and passing of feces should be noted.

In older well infants daily weighing may not be indicated but in prematures it should be done as a routine, more especially in difficult feeding cases. Those fed at the breast must be weighed before and after nursing, and the food taken is to be recorded.

Loss of Body Weight during the First Days of Life.—This occurs almost constantly in premature infants, the percentage loss being greater in the premature than in the full-term infant, and, on the whole, they are much slower in regaining their birth weight. In the group of cases studied by the author the average loss in the cases weighing between 1000 and 2000 gm. was 10.9 per cent. More recently we have been able to reduce the initial loss to approximately 5 per cent in a number of cases by carefully increasing the fluid intake after the first twelve hours.

Most of our cases have regained their birth weight by the eighteenth to the twenty-first day, with a daily gain averaging from 12 to 40 gm. after reaching their lowest weight, which is usually about the fifth day. Infants under 1500 gm. may be considered as progressing satisfactorily on an average of from 10 to 20 gm., and doubling their birth weight in seventy-five to one hundred days; and those from 1500 to 2000 gm. when they are making a daily gain of from 15 to 25 gm. after they have reached or passed their birth weight with a doubling of that weight in from fifty to one hundred days.

The Infant's Clothes.—The wardrobe should be planned and completed in advance of labor. In emergencies this may not be possible. It is imperative to remember that preservation of the body heat must be begun immediately after birth; on the confinement bed itself. Insulation of the body is the prime thought to be borne in mind when planning the wardrobe. The clothes must fit the body snugly, providing only for a thin layer of air between the body and the dress. The material must be selected with some knowledge of the method by which external heat is to be supplied. The head, except the face, and the extremities must be equally protected with the body.

At *birth* the infant is received into a warm blanket and immediately placed in a heated basket, heated bed or incubator.

In supplying external heat it should be remembered that these infants are easily burned, and such burns are usually fatal.

In small prematures for temporary emergency use a sterile cotton-pack which completely envelopes the infant, except for the face and genito-anal region, may be applied. It should, however, be remembered that cotton is far inferior to wool in prevention of heat radiation. An improvised jacket, preferably of flannel, may be placed on the outside of the cotton to hold it in place.

To the genital region and anus an easily changed small pad of cotton or gauze combination may be applied. Whenever the infant becomes soiled, it is only necessary to change the pad. This should not be neglected.

If special outer garments are not available, the infant should at once be wrapped in a small heavy woolen blanket, or cotton combination, which can be fastened about the body loosely by bandages or safety-pins in papoose fashion. The greatest disadvantage of such a dress is the limitation of body movements, which is of considerable importance even in these infants. All pressure and constriction must be avoided (Figs. 75, 76 and 77).

In a well-equipped station several sets of special cloths should be provided. These should be kept sterilized in packets. The outfits will differ somewhat, depending upon whether the open or closed incubator beds are used.

With the *open type of heated beds*, all garments next to the body, except the napkins, should be made of light-weight flannel.

A set of clothing should consist of woolen bands of small size; small woolen undershirts; overshirts; pinning skirts; woolen stockings; diapers; pads; bibs; and a woolen bag, with an attached head-piece, with a slit over the upper part in front to allow passing over the head. The bag should be open at the bottom to allow of its being raised for changing of napkins, dressing the cord and general care of the infant (Fig. 77). The overshirt should be somewhat longer and larger than the undershirt and may to very good

Fig. 77.—Woolen bag with hood. For further protection it may be drawn together beneath the infant's chin.

advantage be made from French pique which is less impervious to air than flannel.

In the absence of a sleeping bag the infant may be wrapped in a light flannel blanket, so applied that the upper part will form a hood.

With the *closed type of bed*, the sleeping bag and blanket are unnecessary.

A *complete outfit* for use with an open bed should contain:
Four bands 12 inches long and 4 inches wide (flannel or knit wool).
Four undershirts with blind sleeves and draw string at neck (flannel).
Four overshirts (flannel or French pique fleeced).

Fig. 78.—Wool flannel undershirt with closed sleeves.

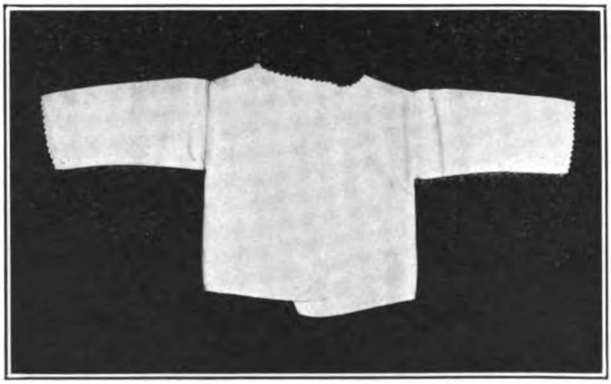


Fig. 79.—Heavy overshirt made from French pique.

Four pinning skirts (French pique 24 by 28 inches).
Two bags with hoods 30 inches long and 20 inches wide (woolen).
Or two blankets 1 yard square (flannel, knit wool or cashmere).
Four pairs of stockings (woolen).
Two dozen diapers size 18 by 20 inches (fine bird's eye).
Small genital pads (absorbent cotton and gauze).
Bibs (same material as jackets).

How to Dress the Baby.—The clothes must be put on quickly without undue exposure. First, the abdominal band is applied, if needed

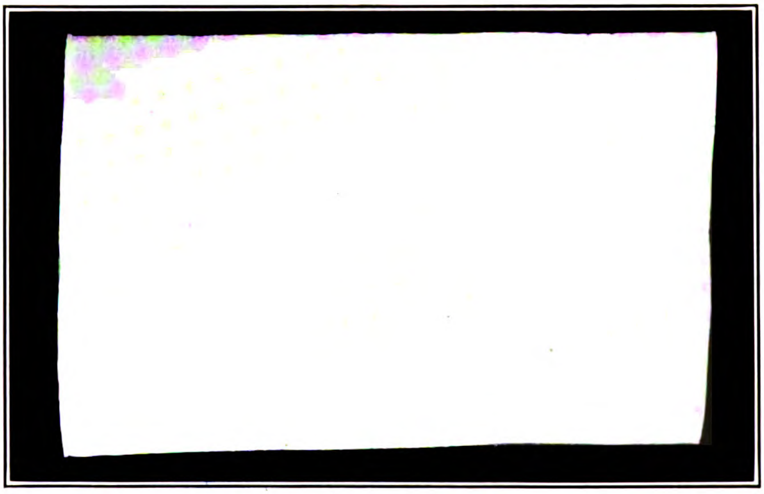


FIG. 80.—Pinning skirt or blanket for the lower half of the body. (Made from French pique.)

FIG. 81.—Bib.

to retain cord dressing, otherwise it may be omitted, then the undershirt, followed by the overshirt, both of which are pinned at the side, next the small genital pads and diapers, to be followed by the pinning blanket, the latter being turned up over the feet and pinned at the back.

The infant may then be placed directly in its bed and its head and body covered by a blanket, or it may be put in one of the woolen bags before being put in its bed. The selection of the last article of dress will depend largely on the condition of the infant.

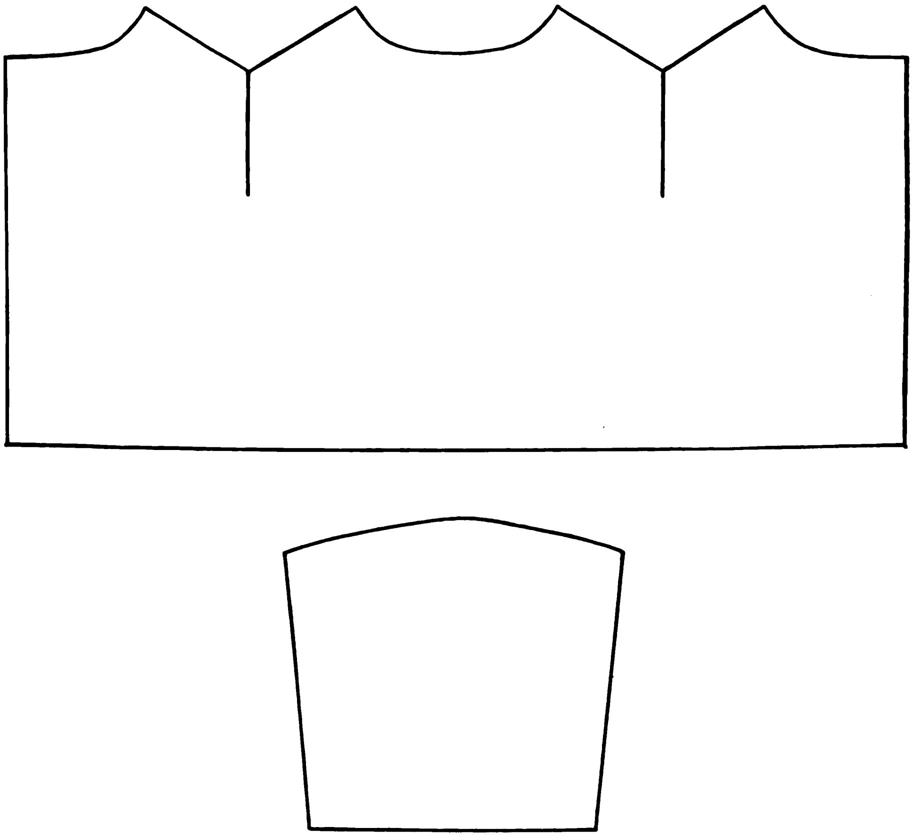


Fig. 82.—Pattern for designing under- and overshirts. Diagram of body and sleeve patterns.

The essentials of the dress are:

1. Good insulation.
2. Cleanliness.
3. Protection from changes in temperature.
4. Ease of application and removal with a minimum manipulation of the infant.

In the emergency and in very small and weakly infants these indications may be met temporarily by a complete envelopment in cotton, but as soon as safe and convenient the infant should be dressed in the simple and easily applied garments described. These

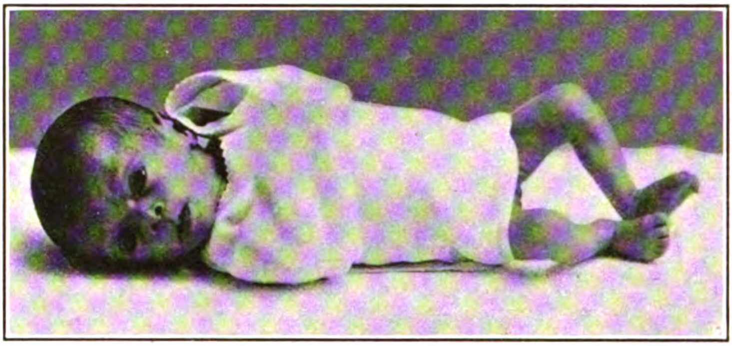


FIG. 83.—Dressing the baby. Under- and overshirts applied.

garments are so applied that they may be described as upper and lower garments. For the changing of soiled napkins the upper half of the clothes need not be removed. Complete undressing is required only for the purpose of bathing.

FIG. 84.—Dressing the baby. Under- and overshirts and pinning shirt applied.

Many of these little infants vomit repeatedly and if it were not for the heavy texture of bib and jacket, it would necessitate very frequent complete undressing of the infant, instead of removal of the soiled linen only, which is but part of his dress. These clothes are easily ironed. Absorbent cotton can be used as a bib.

We also provide for fresh bedding preferably by the use of untarred jute in our bed and pillows, if the latter are used, which can be thrown away at will because of its cheapness.

The infants should be watched very closely and the wet and soiled linen changed immediately to prevent intertrigo, as the urine dries very quickly in the heated bed and when concentrated erodes the skin, which is severe and disastrous to these children. After each change the infant should be carefully cleansed, either with water, benzoated lard or mineral oil, before being replaced in the heated bed. The clothes should fit snugly and are to be preheated before applying and must be absolutely dry. This especially applies to diapers. In laundering the baby's clothes no bluing, lye or strong alkaline soaps should be used, the best for this purpose being a neutral or nearly neutral soap of the type of which Ivory soap is an example. The clothes should then be rinsed in pure water before drying.

The child should not be wiped with the soiled diaper, but with absorbent cotton which can then be destroyed. The same should apply for bathing purposes, where cotton is far more cleanly than a sponge.

Arranging the articles on and in a heated dressing table expedites dressing the infants with the above style of dress. The child can be dressed in one or two minutes without undue manipulation.

Watch for Sickness.—The possibility of grave pathological changes with minor clinical manifestations must be constantly borne in mind in the care of prematures. In order to diagnose and properly counteract the dangers which may follow the overlooked simple ailments, at least one daily general inspection and examination, quickly but carefully performed, is required. The exception to this rule is the immediate danger due to handling extremely delicate infants.

In no other group of infants is a careful study of the individual functioning of the heat centers and the respiratory, circulatory, nervous, genito-urinary and gastro-intestinal organs so imperative.

The Hospital Records.—The records should include the following forms:

1. A history and physical examination blank (Fig. 85).
2. A graphic record chart (Fig. 86).
3. A special feeding card for recording the amount of individual feedings and stamped by the time clock. Time of urination and stools and a description of the latter can be recorded on this same card. Inspection of the infant at feeding times will prevent neglect in changing the infant and assist in the prevention of local and ascending bladder infections. The data from the feeding card should be transposed to the graphic record sheet daily (Fig. 87).
4. Temperature chart for room and bed. On this sheet is recorded the temperature of the bed in which the baby is kept. It should be charted at six-hour intervals, best at 6 A.M., 12 M. and 6 and 12 P.M. These are the most likely times for maximum changes

in the ward temperature which might call for an increase or decrease in the external heat to be applied to meet the desired bed temperature. At the same time the ward temperature should be recorded and the humidity in the room and bed should be noted and recorded (Fig. 88).

5. Physician's order blank (Fig. 89).
6. Milk station order blank (Fig. 90).
7. Wet-nurse's record blank (Fig. 91).

The Clinical Record.—A careful history is most important, as much evidence which will have a direct bearing on the prognosis will frequently be elicited as well as suggestions for feeding and therapy. The maternal history as to illness, previous pregnancies and their outcome must be elicited. The paternal history is also of prime importance. The presence or absence of acute illness in the home, more especially whooping-cough, scarlet fever, diphtheria and septic infections should be investigated before the infant is discharged.

Every hospital record should show the data of at least two social-service investigations. This, while usually neglected, frequently reveals conditions in the home which make the early discharge of these retarded infants impossible, if their lives are to be conserved.

The first investigation should be made in the shortest time possible after the infant enters the hospital, the last just previous to the infant's discharge.

Conserving the Mother's Breasts.—If the mother does not accompany the infant she should be encouraged to conserve her breast milk. This may be accomplished by one of several methods: (1) By expression at regular intervals, and if this is the method used she should be encouraged to send her milk to the hospital once or twice daily, if for no other reason than to keep a record of her faithfulness. (2) By nursing a neighbor baby, one loaned to her from the hospital or some other source. Later by having her come to the institution to nurse her own or a full-term hospital baby. (3) By placing a puppy to her breasts. While this latter at first thought may seem repulsive, it has in our own experience proved to be a most desirable expedient.

REQUIREMENTS FOR THE CARE OF PREMATURE INFANTS IN THE HOME.

The establishment and maintainance of properly equipped hospital stations are essential to the lowering of mortality, more especially in the large cities and particularly among the poorer classes. A careful consideration of the requirements for and results to be expected from their care in the home is equally essential.

PRESENT AND PAST ILLNESSES:

Asphyxia	Vomiting	Pemphigus	Cerebral Hemorrhages
Cyanosis	Meteorism	Furunculosis	Encephalitis
Hemorrhages	Diarrhea	Adenitis	Otitis
Stridor	Constipation	Cord (Condition of)	Pyelitis
Rhinitis	Atelectasis	Sepsis	Rachitis
Icterus	Bronchitis	Megacephalus	Spasmophilia
Convulsions (Early, Late)	Pneumonia	Hydrocephalus	Anemia
Difficult Nursing	Edema	Meningitis	Scurvy

Lues (Evidence of)____________________

____

Conitgenal Deformities____________________

Operations____________________

Important details of diseases__________ During first week__________

____

Later____________________

____

____

____

FEEDING HISTORY:

Before entering hospital—Breast, bottle, mixed____________________

____

First ten days (Breast, bottle, mixed). Number__________, Interval__________hours

Amount__________ccm. Administered (Breast, dropper, bottle, catheter)__________

Subsequent feedings. Kind__________ Method__________ Number__________

Interval__________hours, Amount__________ccm.__________

Other details____________________

____

Present Feeding (Able to nurse—Yes, No)____________________

MOTHER'S general health: ______ Is she available? (Yes, No) ______

Quality of breasts (good, fair, poor) ______ Nipple (good, bad, inverted) ______

Is she pumping, expressing, other methods ______

Why was nursing discontinued? ______

Does the baby take all of its feedings? ______

Does the baby vomit? ______ How much? ______

Does the baby have colic? ______ When? ______

How many times a day do the bowels move (average)? Well, sick ______

Color ______ Consistency ______ Mucus ______ Curds ______ Blood ______

PHYSICAL EXAMINATION

Temperature ______ Pulse ______ Respiration. (Underline each word describing condition)

INSPECTION: Bright Apathetic Cyanotic Anemic Icteric

GENERAL CONDITION: Fat Thin Good Fair Poor

SKIN:

Normal	Prickly Heat	Pemphigus	Furunculosis	Nails
Tissue turgor	Seborrhea	Impetigo	Edema	Lues

MUSCLES: Biceps and thighs firm flabby

HEAD: Normal Deformities ______ Fontanelles ______

Craniotabes ______ Megacephalus ______ Hydrocephalus ______

EYES: Pupils equal, unequal ______ React to light ______ Nystagmus ______

Blepharitis ______ Conjunctivitis ______

NARES: Clear Crusted Discharge Character ______

MOUTH: Normal ______ Stomatitis (type) ______

Deformities, hare-lip, cleft palate, etc. ______

RECOMMENDATIONS:

FEEDING____________________

EXTERNAL HEAT____________________

MEDICAL____________________

SURGICAL____________________

SUMMARY OF HISTORY AND EXAMINATION: Born in hospital, home__________

Father's History____________________

Mother's History. Para__________

Diseases____________________

Miscarriages____________________

Pathology of Pregnancy____________________

Pathology of labor. Length__________hours. Character__________

Infant. Single, Twins, Triplets. No. living__________Which__________

Length of gestation__________days. Age when received__________days

Temperature when received__________°F. Condition when received__________

Congenital disease (Lues) (Evidence)____________________

Other____________________

Congenital deformities ______

Birth injuries ______

Post natal diseases. First week ______

Later ______

Previous care. (Artificial heat, etc.) ______

Previous feeding. Kind ______ Amount ______ How Given ______

Birth weight ______ Initial loss of weight ______ Present weight ______

Age when B. W. regained ______ Age when B. W. doubled ______ Trebled ______

Anemia ______ Rachitis ______ Spasmophilia ______

Other pathological findings ______

Condition on discharge. Age, etc. ______

Apparent cause of Prematurity ______

Cross Index ______

Future Development (Mental, Physical) ______

SUBSEQUENT TREATMENT

Date	Age	Weight	Temp.	

it a
infl
use

Case No.______________

Name____

Date of Admission__________________________________

Present Weight____________________________________

TIME	FOOD	AMOUNT	STOOLS
TOTAL			

Fig. 87.—Individual diet record sheet.

TEMPERATURE CHART FOR HEATED BED

NAME____________________ DR.____________________

DAY OF ENTRANCE____________________ AGE__________ TEMPERATURE__________

BIRTH WEIGHT________________ PRESENT WEIGHT________________ CASE NO.________________

DATE	6 A.M.				12 A. M.				6 P.M.				12 P.M.			
	ROOM		BED		ROOM		BED		ROOM		BED		ROOM		BED	
	Humid.	Temp.	Contact	Temp.	Humid.	Temp.	Contact	Temp.	Humid.	Temp.	Con-tact	Temp.	Humid.	Temp.	Contact	Temp.

FIG. 88

PREMATURE INFANT STATION

FEEDING ORDERS

DATE	NAME	AGE	WEIGHT	BREAST MILK	NUMBER	AMOUNT	WATER	OTHER FOOD

FIG. 89

PREMATURE INFANT STATION

FOOD REQUIREMENT FROM MILK STATIONS

DATE	NAME	AGE	WEIGHT	BREAST MILK	NUMBER	AMOUNT	WATER	OTHER FOOD

Fig. 90

BREAST MILK SUPPLY

NAME ____________________

DATE	HOUR	AMOUNT	DISPOSITION

FIG. 91.—Wet-nurse milk supply record sheet.

In many instances the premature is born unexpectedly with little time for preparation for its reception. The expectancy of a premature labor is almost always associated with more or less excitement in which thought for the baby's needs are likely to be overlooked, the mother usually being given first consideration.

It will, therefore, be our object to outline a proper routine for the establishment of an emergency home unit.

In the home care of these infants the same rules for hygienic maintainance of body temperatures, breast feeding, and daily routine must be maintained as suggested for their hospital care.

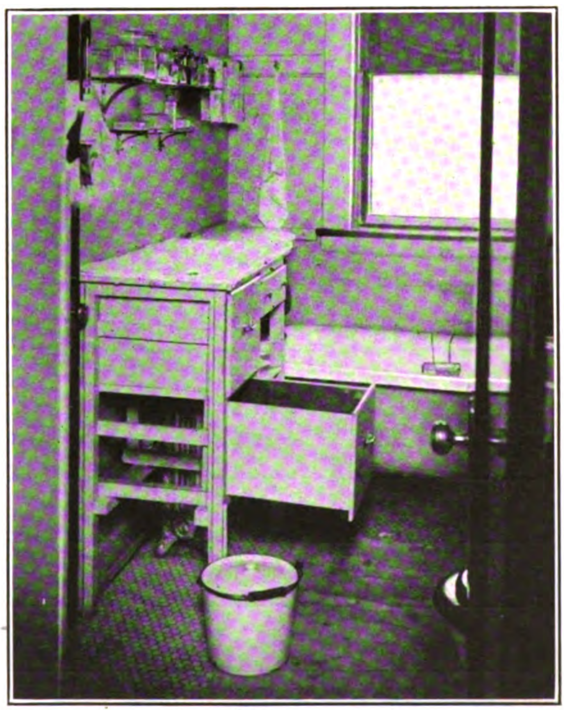


FIG. 92.—Special bath room equipment for private home, showing dressing table (padded) with drawers, built over radiator. Shelves for dressing, etc., above the table. Bathing board over one end of bath tub.

The Nursery Unit.—Whenever possible two rooms should be set aside for the infant's use; one equipped as a nursery with furnishings similar to those described for the hospital nursery. The second room is to be used for sleeping quarters and must be equipped with a heated bed. These rooms must be well ventilated and at the same time well heated. In both these rooms all draperies and unnecessary furniture must be removed.

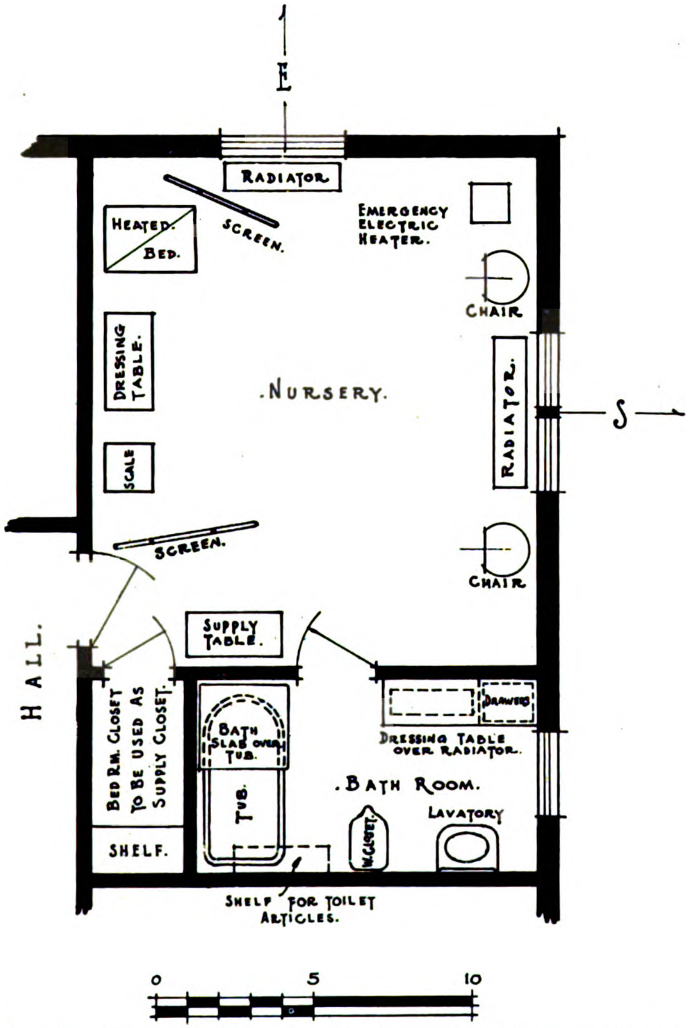


Fig. 93.—Plan for arrangement of stations in a private home, consisting of one large, well ventilated and heated room and a bath room.

A bath room properly equipped (Fig. 92) makes a splendid second room in which the general care of the infant can be administered.

This room should be given over to the exclusive use of the infant. When such a bath room is available only one other room is needed.

While the baby is being dressed or bathed the nursery or bathroom temperature should be in the neighborhood of 80° F. A gas or electric stove will be of assistance in accomplishing this. When a superheated bed is in use the sleeping room may be kept between 70 to 75° F. These rooms should be devoid of all excessive furniture and draperies. *All visitors other than the attendants and physician must be excluded.*

The equipment of the room or rooms should include a heated bed, a dressing table, preferably heated, or placed over a warm radiator, a small electric or gas stove for emergency use, a scale, bathing and feeding utensils, a thermometer, a hygrometer and surgical supplies (Fig. 93).

The Superheated Bed.—In the home hot-water bottles, a properly protected electric pad or an improvised incubator will answer the purpose (p. 223).

A thermometer should be placed alongside the baby as too great emphasis cannot be laid on the dangers and fatalities due to overheating and burning of prematures. There is a great tendency to hyperthermia which must be recognized and properly interpreted. There is usually a rapid return to the normal body temperature without bad effects upon removal of the cause unless too long continued. The general care of the heated bed has been described on p. 218.

The Nurse.—She must be experienced in the feeding and handling of such infants and must be tireless in her efforts to prevent complications. She must be diplomatic in order to permit the overcoming of the mother's anxiety, with its consequent effect upon her milk secretion. She must be able to control the habits of the wet-nurse, if one is employed. She must insist upon taking orders from the physician and no one else. She must be able to keep a careful record, practise aseptic nursing, avoid accidents and be cleanly in her personal habits.

A second person should be present, who can assist and relieve the nurse. She must be willing to work under the nurse's supervision. Such a person is indispensable in the presence of emergency. Only those directly interested in the care of the baby should come in contact with it.

The Infant's Food.—Breast milk should be considered indispensable and during the first days of life it may be necessary to obtain a temporary supply from a neighboring mother, a wet-nurse or a hospital. A small amount, 90 to 240 cc daily will usually meet the emergency. Only when these sources of supply fail absolutely should artificial feeding be instituted.

Preparation for Labor.—The protection of the infant must begin with the first stage of labor. The room, receiving clothes and its bed must be properly warmed. Refrigeration is the direct cause of more deaths among prematures than any other extraneous factor. All routine measures described for the hospital care in Chapter VII should be, so far as possible, observed in the home.

Clothes.—The clothes best suited have been described. Simplicity in dress with a minimum manipulation or changing being the object to be attained, because of the dangers of exposure, trauma and infection. The infant should be received into warm blankets. One of the most common errors is to allow the infant to remain in such a loosely applied robe, which does not provide for proper insulation of the skin because of the large air space between the blanket and the infant. This allows rapid radiation of the body heat. Therefore, at the earliest possible moment the infant should be protected from head to foot by closely applied warm clothes. The body, if woolen clothing is not at hand, should be wrapped in cotton. The cotton should be applied in two parts, the upper half encircling the head except the face, together with the trunk and upper extremities, the lower half should encircle the lower extremities, a small pad being applied to the genitalia and buttocks. This allows for cleansing the genital region with a minimum of manipulation. The upper part of the body may then be covered by a small-sized infant's shirt on the outside of the cotton jacket and the infant is then wrapped in a woolen blanket in its bed. The clothes best adapted for later use can be made according to the description on page 155, and should be supplied as soon as possible. A woolen blanket should cover about three-quarters of the basket, the head being left open.

The Bath.—The advisability of giving a warm bath has been discussed but it is our desire to emphasize the conclusion that the initial bath is to be omitted whenever there is danger of unduly exposing the infant. In a proper environment the warm cleansing bath should be given in the absence of cardiac and respiratory complications.

Further Early Care.—The baby must, under all circumstances, be under constant observation during its first hours because of the dangers of cardiac and respiratory complications, over- and underheating, overcovering, and overlying, the latter due to careless placing of the infant in the bed. Whenever feasible the infant should be placed in a properly prepared room away from the mother. Its personal attendant, other than for special care, need not necessarily be a trained one. When a dependable person is not at hand it should be kept in the room with the mother.

The general care should be that as described for hospital care.

The Results Obtained.—With human milk, a skilled nurse, an adequate bed, a good nursery and proper feeding and nursing technic the same good results are to be expected as in hospital care.

Transportation to a Hospital.—Removal to a hospital station should not be delayed when nursing and feeding needs cannot be fulfilled in the home. It should be moved in a specially prepared bed so that it will not be exposed *en route*. When the infant is to be sent to the hospital which is provided with a transportation incubator, the institution should be called upon to transfer the infant.

CHAPTER VIII.

METHODS OF FEEDING.

It is necessary to consider these infants as belonging to two large groups:

1. Those able to nurse at the breast.
2. Those too weak to nurse at the breast.

INFANTS NURSING AT THE BREAST.

This presupposes that the infant has the proper physical development to withdraw milk from the human breast in the presence of an abundant supply and well-developed nipples. Such an infant may be placed at the breast two or three times during the last half of the first day after the circulatory and respiratory functions are well established. Following the first day it should be placed at the mother's breast regularly for two- or three-minute periods at three- or four-hour intervals, even though the breast contains little milk. Following these attempts at nursing, food should be supplied from another mother or a wet-nurse, whenever such a supply is obtainable rather than to institute artificial feeding. In the hospital it is our custom to give these additional feedings by hand; in private practice the infant may be placed to the wet-nurse's breast, one of the breasts being set aside for this purpose, and if there is a difference in the breasts the better one is selected for the premature. Whenever possible this is the best method of getting the food to the baby, as it prevents contamination of the milk, stimulates the breasts and develops the baby's independence as well as his sucking muscles. However, it is to be remembered that congenital syphilis is to be excluded, both in the infant and wet-nurse, in all cases where the infant is put directly to a breast other than the mother's. Not infrequently great assistance may be given the infant in securing its milk by one of two methods: Either by expressing the milk directly into the baby's mouth or by placing the wet-nurse's baby on the opposite breast (Fig. 94) which reflexly stimulates the flow of milk into the opposite breast, thereby assisting the weak infant in obtaining its food. *Overfeeding* becomes a danger in this direct application of the infant to the breast, and weighing before and after nursing should be practised. *Underfeeding* is an even greater danger, and here again the infant must be weighed before and after feeding to ascertain the amount of food taken. If insufficient, further food can be supplied by hand feeding.

Fig. 94.—Feeding premature infant by direct expression from right breast. Wet nurse's baby on left breast assists in stimulating the flow of milk into the right breast.

INFANTS TOO WEAK TO NURSE AT THE BREAST.

In this group of infants careless exposure must be avoided. In the absence of cyanosing they may be fed without removal from the bed. If cyanosis threatens they should be fed on the dressing table. The inability to nurse may be due to improper development of the nursing center, or lack of coördination on the part of the pharyngeal muscles and tongue. The latter is usually made evident by a return flow of milk from the mouth. Again, the infant may be too weak to nurse, or it may not have learned to suck, or vomiting or perhaps cyanosis may prevent its feeding properly. In this group of infants we may, of necessity, resort to one of several procedures:

The use of a fruit *spoon* (Fig. 95) or, better a large size medicine dropper (Fig. 96).

In those infants who can assist themselves:

A small *nursing bottle* (Fig. 97). The 1-ounce bottle is provided with small nipples the size of those commonly sold on doll nursing bottles, which can usually be obtained of proper quality. Such a nipple can be made by perforating the rubber bulb of a better quality medicine dropper. Our 2-ounce bottle has a larger neck which takes the ordinary size nipple. One with a small mouth piece must be used. It should be made of a soft elastic rubber. The semitransparent nipples usually answer best.

Fig. 95.—Fruit spoon which can be used for mouth or nasal feeding. The latter is not recommended.

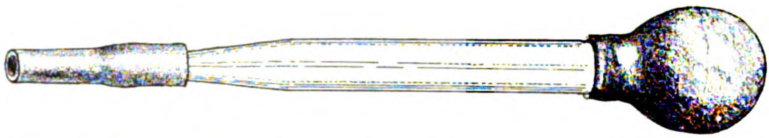


Fig. 96.—Large medicine dropper with a short piece of soft rubber tubing over lower end to prevent injury to the baby's mouth. Most infants soon learn to suckle on the soft rubber tube when inserted into the mouth. When sufficiently developed a small bottle and nipple can be substituted.

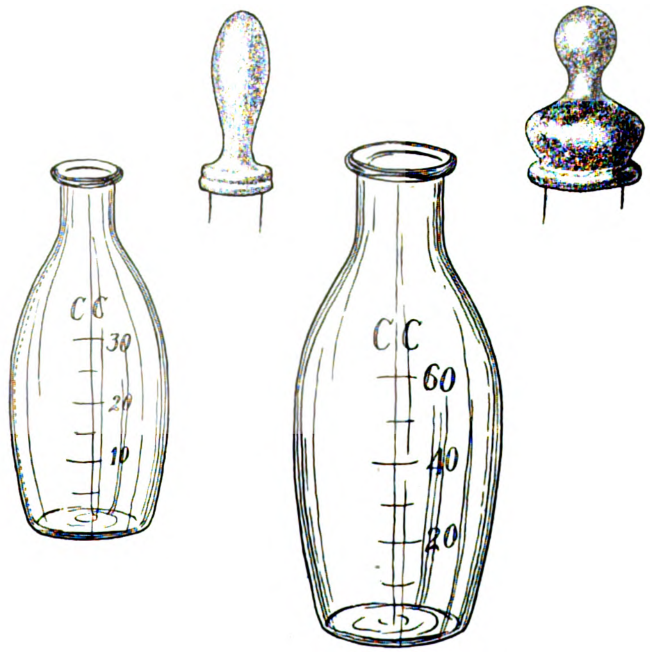


Fig. 97.—One ounce graduated nursing bottle with small nipple approximately the size of the end of an ordinary medicine dropper. Two ounce graduated bottle with a special nipple with a small mouth piece. This nipple will fit on the larger neck bottle sold on the market and can also be inverted for cleansing. Another good nipple is that shown in type two Breck feeder. (Fig. 98.)

The Breck feeder in the original, or a modification which can be made by flanging the ends of a urethral syringe, using a heavy rubber finger-cot on one end and a small nipple or perforated soft medicine-dropper tip on the other, will usually suffice (Fig. 98). The second type illustrated has the disadvantage of having the milk enter the bulb on filling from the large end. The bulbs are difficult to clean.

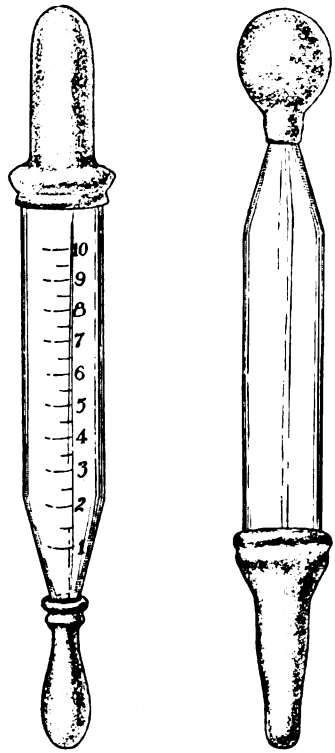


Fig. 98.—Modified Breck Feeders. Type I: with a small nipple at the lower end and an ordinary finger cot at the upper end. Type II: has a large nipple at the lower end and a medicine dropper bulb at the upper end. The latter is not a safe model because the milk must be poured into the large end and therefore enters the bulb which is difficult to clean. Type III can be made by flanging a straight piece of large tubing and using the large nipple at one end and the finger cot at the other. The glass part can be blown by any specialty glass company or the barrel of an ordinary glass syringe can be drawn and flanged to take the rubber parts.

Direct expression of milk into the infant's mouth has proved one of the most valuable expedients in our hands as a method of teaching the infant the act of nursing.

Catheter feeding is the simplest and best method of procedure in the smaller infants, if carefully practised by an experienced nurse.

Catheter feeding should be instituted as soon as fatigue or cyanosis is noted following other methods of feeding. A catheter (No. 12 French, No. 8 American, No. 5 English) about 14 inches in length may be attached to a small funnel, graduated glass tube, or, in case of emergency, the glass barrel of a small syringe may be used. All food should be carefully measured and administered slowly with a *minimum elevation* required to obtain a free flow of the milk. The

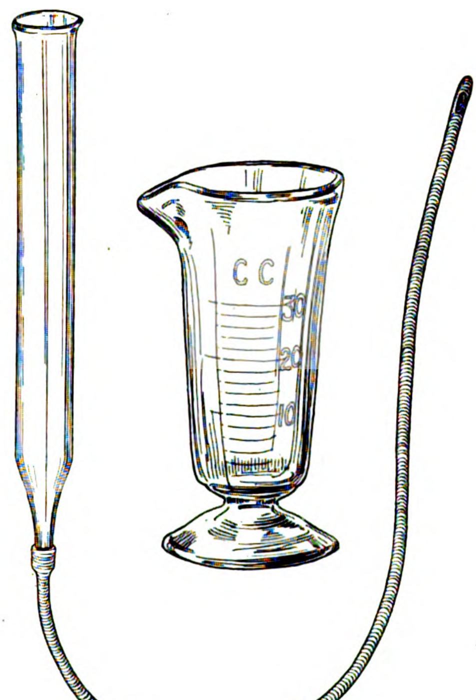


Fig. 99.—Utensils for catheter feeding. Glass barrel of syringe, No. 12 French catheter and one ounce graduate glass. The catheter should be marked at 2 cm. intervals between the distances 12 to 20 cm. above the tip.[1]

infant should be upon its back on a flat surface with the head either in the median line or turned to the right. The passage of the catheter is usually effected without difficulty by passing it in the midline to the pharynx, gradually pushing it into the esophagus. The poorly developed reflexes rarely cause retching. The dis-

[1] As there are no short catheters marked in the metric system on the market it is advisable to mark several for ward use between 12 and 20 cm.

tance to which the catheter is to be passed is of great importance when we consider that this procedure must be repeated at least six to eight times daily over a considerable period of time. It has been our rule to measure the distance from the bridge of the nose to the tip of the ensiform cartilage, which is usually in the neighbor-

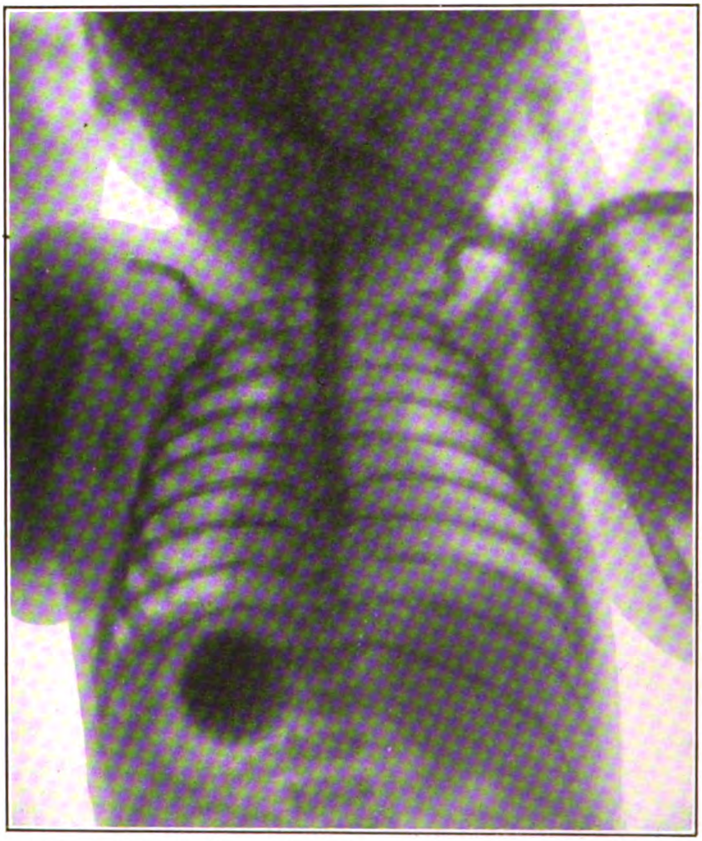


Fig. 100.—Catheter feeding. The catheter has been passed for a distance equal to that from the bridge of the nose to the tip of the ensiform cartilage measured with the chin at right angles to the body. The lower end is seen about 1 cm. above the cardia in A, Fig. 101.

hood of 12 to 15 cm. (Full-term new-born infants average about 16 cm.) The catheter is marked at this point with indelible ink and is passed to this point or about 1 cm. further than this distance which allows it to reach the lower end of the esophagus just above the cardia, from which point the food will flow through the patent cardia. We thereby avoid irritating the gastric mucosa and stimulation of the reflexes at the cardia. One soon learns the

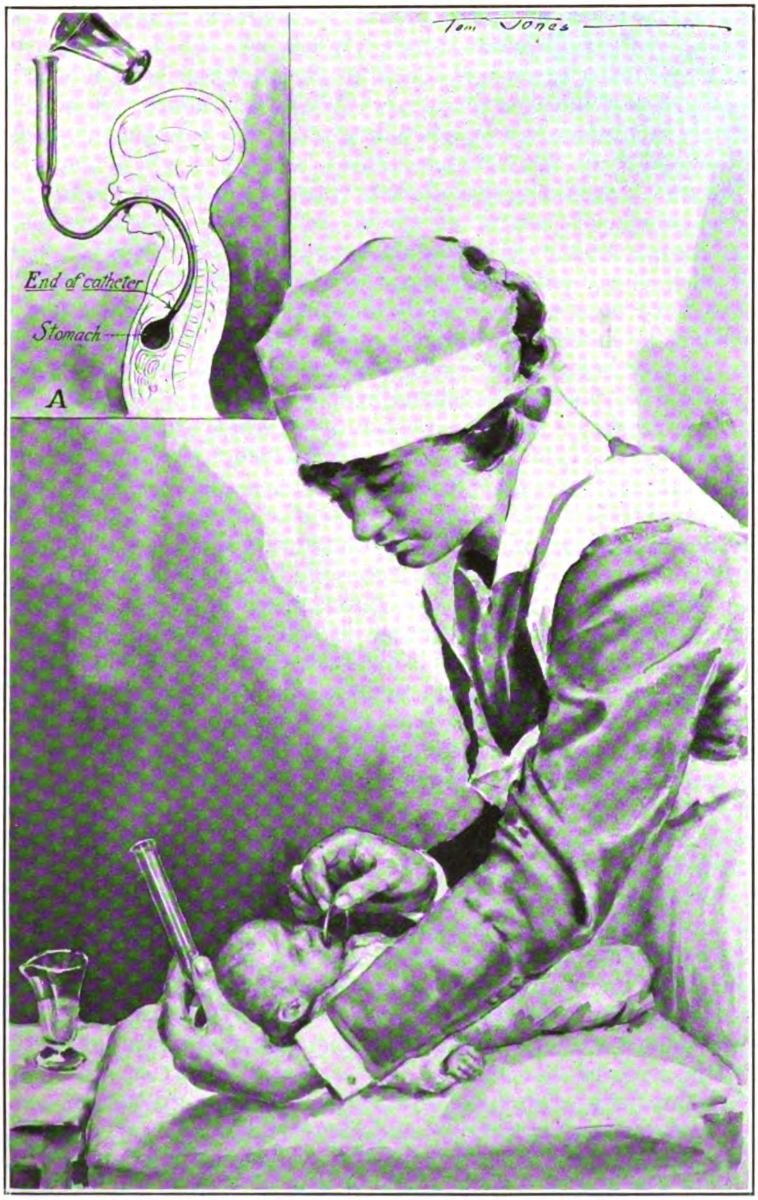


Fig. 101.—Feeding baby with catheter. Catheter feeding as carried out by one person. The head is held at right angles by the left hand, the catheter is passed with the right hand. Next the funnel is passed to the left hand and elevated to allow the air to escape from the stomach. The catheter is now compressed and slightly elevated and part or all of the feeding is poured in from the graduate and allowed to flow slowly into the stomach. The small sketch illustrates the point to which the lower end of the catheter should be passed.

distance the catheter can be passed in each case in order to avoid retching. The milk is now allowed to flow into the stomach slowly, the funnel being raised only slightly above the level of the body, usually 6 or 8 inches will suffice. After the feeding the catheter is firmly compressed to avoid spilling milk into the pharynx during its removal (Figs. 99, 100 and 101).

The infant should be turned on its right side following the feeding. In the presence of gastric distention, raising the infant before and after feeding to the vertical position, avoiding flexion of the body, will allow of the eructation of air and frequently prevent cyanosis. When the stomach is noticeably distended with gas before feeding the catheter should be passed 1 or 2 cm. further than the mark on the catheter before starting feeding, in order to allow the gas to escape. It is then retracted as directed and feeding started. The catheter should be passed with the funnel empty, so as to allow of this procedure. The catheter should then be compressed and the milk poured into the glass funnel. This allows the air in the funnel to escape thereby preventing overdistention of the stomach by the mixture of food and air. The feeding period should be as short as possible without undue haste. Too rapid feeding is more dangerous than too slow. Usually one to three minutes are needed. Two nurses can be used to advantage in catheter feeding, but, as so frequently happens, only one is available during the night feedings. Every nurse should be trained to undertake catheter feedings without assistance. We believe that turning the infant on the right side following feedings reduces the emptying time. Its position should be changed at least once between feedings to avoid localized pulmonary congestion.

The Number of Feedings.—This will, of necessity, depend in many instances upon the question of catheter *versus* other methods of feeding. Larger infants fed by catheter can often be given sufficient food at four-hour intervals to meet their needs. In small infants fed by dropper, bottle or other methods we have experienced great difficulty in administering a sufficient quantity of food by the long-interval feeding. As the attendants in charge are frequently not to be trusted with the catheter feeding, the short-interval feeding must be resorted to.

For this purpose we have grouped our infants into two classes—those weighing under 1500 gm. and those weighing above this figure. These figures are arbitrary and will not require rigid adherence. The classification is based on the tendency of the smaller infants to become exhausted when the feedings are too long continued. The smaller hand-fed infants are fed at two-hour intervals during the day and three hours at night. The larger on the three-hour basis. When catheter feeding is the method of choice, even in the smaller infants six to eight is usually the maximum number needed in twenty-four hours. It must be remembered that all feedings are dependent on the general development of the infant in relation to its digestion and metabolism and its ability to retain the food administered, as well as on the attendant complications to feeding, such as asphyxia, cyanosis and gastric distention.

When to Start Regular Feeding.—This is a question of great importance to these infants, because of the tendency to develop acute inanition. Therefore a regular feeding regimen must be started early. Human milk is essential to a low mortality. As little can be expected from the mother for several days, it becomes necessary to obtain the limited supply necessary from another mother, preferably a wet-nurse. If for any reason it is unlikely that the mother may be depended upon, either because of illness or local breast conditions, immediate search should be begun for a supply of breast milk.

Feeding During the First Day.—During the *first day* it is our custom to withhold milk for twelve hours until the respiratory and circulatory functions are well established. During the second twelve hours one to three feedings of breast milk may be started if the infant's condition warrants.

Feeding from the Second to the Tenth Day.—The second to the tenth days may be grouped together as the second feeding period for practical purposes.

From the second day they should be fed regularly, day and night, the number and time of feedings depending to a great extent on whether the food be given with or without the use of a catheter; second, upon the gastric capacity; third, upon the infant's general condition.

Further fluids, preferably inert, such as water or 1 per cent lactose solution, are administered to compensate for the loss of body fluids through the kidneys, bowels, lungs and skin. The infant requires about one-sixth of its body weight of water, inclusive of that contained in the milk, in twenty-four hours while in the heated bed. Such quantities, however, should not be attempted on the first days; usually it will be possible to approximate one-eighth of the body weight by the fourth day. The early feedings must necessarily be small and the increases gradual.

Each Infant Fed Individually.—They must be considered individually, as it is impossible to formulate definite rules for feeding, at least during the first ten days.

1. We must have a definite idea of the minimum food requirements for life.

2. The amount of food necessary to maintain at least a stationary weight.

3. The amount of food needed to meet the requirements for growth and development.

Approximately one-seventh of the body weight of fluids and human milk of a food value of 70 calories per kilo every twenty-four hours are required to maintain life. Little can be expected in the way of weight increase until 90 calories are reached, and depending on their weight, body surface and physiological development, their later needs will approximate 100 to 140 calories per kilo body weight (Table IV). In exceptional cases it may be necessary to feed breast milk in amounts equaling 160 to 200 calories per kilo. Such infants are usually markedly underweight for their fetal age.

Infants, to fulfil all their needs, will therefore require from 140 to 200 cc of breast milk per kilo, or about one-seventh to one-fifth of their body weight daily. They can, however, maintain life on 100 cc and hold their weight in most cases on 130 cc per kilo. Exceptionally, we have fed as high as 300 cc per kilo in underweight infants. The latter must be carefully observed for signs of overfeeding, such as vomiting, gastric dilatation and cyanosis.

Beginning (in most cases by the second day) with 20 to 40 cc human milk per kilo of body weight, the quantity may be increased by 8 to 15 cc daily per kilo until, usually by the tenth day, feedings averaging from 80 to 140 cc per kilo can be fed (Tables I, II and III).

AVERAGE HUMAN MILK DIETS REQUIRED BY PREMATURES DURING THEIR FIRST TWENTY-ONE DAYS.

After the *tenth day* in larger infants the milk can be increased more rapidly, usually by 15 and occasionally 20 cc per day, until from 140 to 200 cc (100 to 140 calories) per kilo are fed, the methods of giving food, as well as its frequency being dependent on the general development of the infant.

The size of individual feedings will vary with the method of feeding. When *catheter* fed, six to eight feedings a day are given, with an average of from 4 to 6 cc per feeding during the second day. The feedings are now increased daily by an average of 2 cc per feeding. When feeding from the *bottle* or by *dropper*, smaller feedings are usually given more frequently—usually from eight to ten daily, although twelve may be needed when larger feedings are not retained. Begin with 2 to 4 cc and increase by 1 or 2 cc per feeding on each succeeding day, until 140 to 200 cc per 1 kilo per day is reached.

The food and water to be administered should be noted in writing for the nurse's instruction each day, after a thorough inspection of the infant and its clinical chart.

The diet of a premature infant making a satisfactory gain in weight should not be changed arbitrarily without a well-defined indication.

TABLE I.—INFANTS APPROXIMATING 1000 GM (2 POUNDS) IN WEIGHT.

Day.	Milk for twenty-four hours, cc.	Bottle fed.		Catheter fed.		Additional water.	Total fluids.	Calories, per kilo.	Fluid intake *vs.* body weight.
		Number.	Amount.	Number.	Amount.				
1	4–12	3 to 2	2–4	3–2	4	45	50–60	3–8	1/20
2	20–40	10 to 8	2.0– 5.0	6	5	60	80–100	14–28	1/12
3	30–50	"	3.0– 6.0	"	5–8	70	100–120	21–35	1/9
4	35–60	"	3.5– 7.5	"	6–10	80	115–140	24–42	1/8
5	45–70	"	4.5– 9.0	"	7–12	"	125–150	31–49	1/7
6	50–80	"	5.0–10.0	"	8–13	"	130–160	35–56	"
7	60–90	"	6.0–11.0	"	10–15	"	140–170	42–63	"
8	65–100	"	6.5–13.0	"	11–16	"	145–180	45–70	1/6
9	75–110	"	7.5–14.0	"	12–18	"	155–190	52–77	"
10	80–120	"	8.0–15.0	"	13–20	70	150–190	56–84	"
11	90–130	"	9.0–16.0	"	15–22	"	160–200	63–91	"
12	95–140	"	9.5–17.0	"	16–23	"	165–210	66–98	"
13	105–150	"	10.5–18.0	"	18–25	"	175–220	73–105	1/5
14	110–160	"	11.0–20.0	"	19–26	60	170–220	77–112	"
15	120–170	8	13–21	"	20–28	"	180–230	84–119	"
16	125–180	"	15–23	"	21–30	"	185–240	87–126	"
17	135–190	"	17–24	"	22–31	50	185–240	94–133	"
18	140–200	"	18–26	"	24–33	"	190–250	98–140	"
19	150–210	"	19–27	"	25–35	40	190–250	105–147	"
20	160–220	"	20–28	"	26–36	"	200–260	112–154	"
21	165–230	"	21–29	"	27–38	30	195–260	115–161	"

The caloric requirements are figured on the basis of a retained birth weight. Water additions recommended are calculated on the average between high and low milk requirements. The necessity for further water after the twenty-first day must of necessity vary with the individual case. Water may often be discontinued when one-fifth of the body weight in breast milk is being fed in twenty-four hours.

TABLE II.—INFANTS APPROXIMATING 1500 GM. (3 POUNDS) IN WEIGHT.

Day.	Milk for twenty-four hours. cc.	Bottle fed.		Catheter fed.		Additional water.	Total fluids.	Calories, per kilo.	Fluid intake *vs.* body weight.
		Number.	Amount.	Number.	Amount.				
1	6–15	3 to 2	3–5	3–2	5	60	65–75	3–7	1/20
2	30–60	10 to 8	3–7	6	5–10	90	120–150	14–28	1/12
3	40–75	"	4–9	"	6–12	100	140–175	18–34	1/10
4	50–90	"	5–11	"	8–15	"	150–190	24–42	1/9
5	60–105	"	6–13	"	10–17	120	180–225	28–49	1/8
6	70–120	"	7–15	"	12–20	"	190–240	32–56	1/7
7	80–135	"	8–17	"	13–22	"	200–255	36–63	"
8	90–150	"	9–19	"	15–25	"	210–270	42–70	"
9	100–165	"	10–21	"	17–22	"	220–285	46–77	1/6
10	110–180	"	11–23	"	18–30	"	230–300	52–84	"
11	120–195	"	12–24	"	20–32	"	240–315	56–91	"
12	130–210	"	13–26	"	22–35	"	250–330	60–98	"
13	145–225	"	14–28	"	23–37	"	260–345	66–105	1/5
14	150–240	"	16–30	"	25–40	"	270–360	70–112	"
15	160–255	8	18–32	"	27–42	100	260–355	74–119	"
16	170–270	"	20–34	"	28–45	80	250–350	80–126	"
17	180–285	"	22–36	"	30–47	"	260–365	84–133	"
18	190–300	"	24–38	"	32–50	60	250–360	88–140	"
19	200–315	"	26–40	"	33–52	"	260–375	94–147	"
20	210–330	"	28–42	"	35–55	40	250–370	98–154	"
21	220–345	"	30–44	"	37–57	"	260–385	102–161	"

Water administration recommended is for infants taking the average between low and high milk requirements. If diet is well taken and one-fifth the body weight in milk is being fed, the water may now be discontinued.

TABLE III.—INFANTS APPROXIMATING 2000 GM (4 POUNDS) IN WEIGHT.

Day.	Milk for twenty-four hours, cc.	Bottle fed.		Catheter fed.		Additional water.	Total fluids.	Calories, per kilo.	Fluid intake vs. body weight.
		Number.	Amount.	Number.	Amount.				
1	20–30	4	5–8	3	7–10	80	100–110	7–10	1/20
2	40–80	8	5–10	6	7–13	100	140–180	14–28	1/12
3	55–100	"	7–12	"	9–16	120	175–220	20–35	1/10
4	70–120	"	8–15	"	11–20	"	190–240	24–42	1/9
5	85–140	"	10–18	"	14–23	160	245–300	30–49	1/8
6	100–160	"	12–20	"	16–27	"	260–320	35–56	1/7
7	115–180	"	14–22	"	19–30	"	275–340	40–63	"
8	130–200	"	16–25	"	21–33	"	290–360	45–70	"
9	145–220	"	18–27	"	24–37	"	305–380	50–77	1/6
10	160–240	"	20–30	"	26–40	"	320–400	56–84	"
11	175–260	"	22–32	"	29–43	"	335–420	61–91	"
12	190–280	"	24–35	"	32–47	"	350–440	66–98	"
13	205–300	"	26–37	"	34–50	"	365–460	72–105	1/5
14	220–320	"	28–40	"	37–53	140	365–460	77–112	"
15	235–340	"	30–42	"	39–57	120	355–460	82–119	"
16	250–360	"	32–45	"	42–60	100	350–460	87–126	"
17	265–380	"	34–47	"	44–63	80	345–460	92–133	"
18	280–400	"	36–50	"	47–67	60	340–460	98–140	"
19	295–420	"	38–52	"	49–70	40	335–460	103–147	"
20	310–440	"	40–55	"	52–73	20	330–460	108–154	"
21	325–460	"	41–57	"	54–77	0	345–460	113–161	"

The necessity for further water diet after the twenty-first day, or increases over amounts recommended in the schedule must of necessity vary with the individual case.

TABLE IV.—CALORIC REQUIREMENTS PER KILOGRAM BODY WEIGHT.

VALUES RECOMMENDED BY DIFFERENT AUTHORS.

Salge[1]	130 to 150
Samelson[2]	115 to 150
Oppenheimer[3]	120 to 130
Czerny-Keller[4]	100 to 120
Langstein-Meyer[5]	120 to 130
Budin[6] (average)	140
Birk[7]	100 to 160
Reiche[8] (in those under 2000 grams)	120 to 130
(in those over 2000 grams)	95 to 110
Oberwarth[9]	120 to 160
Morse and Talbot[10] (average)	120
E. Moll[11]	110 to 120
Cook, P.[12]	120 to 200

[1] Einführing in die moderne Kinderheilkunde, Berlin, 1909.
[2] Ztschr. f. Kinderh., 1911, **11**, 18.
[3] Med. Klin., 1908, **6**, 92.
[4] Des Kindes Ernährung usw. Leipsig u. Wien, 1912.
[5] Säuglingsnahrung u. Säuglingsstoffwechsel, Wiesbaden, 1914.
[6] The Nursling, London, 1907.
[7] Säuglingskrankheiten, 1913.
[8] Ztschr. f. Kinderh., 1914, **12**, 369.
[9] Ergeb. d. Inn. Med. u. Kinderh., 1911, **7**, 191.
[10] Diseases of Nutrition and Infant Feeding, Macmillan & Co., New York, 1920.
[11] Ztschr. f. Kinderh., 1919, **21**, 329.
[12] Arch. Pediat., 1921, **37**, 201.

These feedings should, as rapidly as possible, be supplemented by water or sugar water by mouth, or saline by rectum to meet the required 140 to 200 cc per 1 kilo of fluids required daily.

Initial Weight Loss.—The lower the birth weight, the greater is the percentage weight loss to be expected. Artificially-fed infants lose more weight than breast-fed infants in whom the diet is started early. An average loss of not more than 8 to 12 per cent of the birth weight may be considered as satisfactory. By regular administration of inert fluids during the first days the total loss can frequently be reduced to 5 per cent.

Daily Gains.—These are not necessarily in proportion to the changing quantity of milk administered, as many factors, such as condition of the bowels, quantity of urine passed, temperature of the infant's surroundings and numerous other factors will necessarily influence the weight.

An average daily gain greater than 20 gm. is unusual when the infant's food is limited to one-sixth of its body weight.

Although occasionally an infant holds its birth weight, most infants do not regain their birth weight before the end of the second or third week.

In the very small prematures an average daily gain of 10 to 15 gm. with a doubling in birth weight in from seventy-five to one hundred days may be considered satisfactory. In the larger infants a gain of 15 to 20 gm. may be expected with a doubling in birth weight in from fifty to one hundred days. The birth weight is frequently trebled within one hundred and eighty days.

Special Feeding Rules.—1. Food requirements which have been recommended must, of necessity, be considered as relative, variations being to a great extent influenced by the physiological and anatomical developments and to a not inconsiderable extent by the temperature and humidity of the air surrounding the infant and the type of clothes in which it is dressed.

2. Each day the total amount of food as indicated for the individual infant is to be estimated, in order that the required food and water will be administered. The number and amount of feedings will, of necessity vary, but each must also be estimated for each day.

3. When a number of infants are to be fed by one wet-nurse, careful calculation of the day's needs of each infant must be made by the floor nurse for the information of the nurse in charge of the milk supply.

4. Expression of breast milk should be performed at regular intervals, preferably six times a day at four-hour periods day and night. The sixth expression during the night may, however, be omitted if the supply is in excess. It is only by regular and com-

plete emptying of the breasts by expression that a milk supply can be maintained for an indefinite period, unless there is a second baby which can be placed at the breast.

5. Human, as well as cows' milk, must be obtained under aseptic conditions and kept clean and cool until ready for the infant. To preserve milk properly, the ice box must register less than 50° F. The food should be slowly warmed before feeding.

6. The boiled water to be fed must be carefully calculated, and it must represent the difference between the total fluids indicated which will usually average from one-eighth to one-fifth of the body weight of the infant for twenty-four hours and the amount of fluid given as milk. *The water for each day should be measured and set aside in an individual stoppered bottle each morning.*

It should be administered between the milk meals or occasionally there may be an indication for diluting the milk with part of it. In order to administer the full day's water supply in some of the small infants and those who vomit, it may be necessary to give water in small quantities one, two and even three times between milk feedings. If unable to swallow properly water must be given by catheter. In larger infants only a few water feedings a day may be needed, and usually by the third or fourth week, one-seventh or one-fifth of the body weight in milk can be fed daily. At this time the water may be discontinued unless it is necessary to supply external heat of considerable degree, or the infant has a fever, both of which necessitate increased amount of fluids.

The maximum feeding figures as given in the tables for 1000-, 1500- and 2000-gm. infants from the seventeenth to the twenty-first days and which range from 140 to 161 calories may seem excessive, but it should be remembered that in figuring these feedings they are based on birth weight without allowance for weight increase which is usually seen during the third week of life of these infants. Allowance for these weight increases are covered by the maximum total diet recommended. Not infrequently the infant requires more rapid increases in its diet than those quoted as the maximum feedings. The physician must be the best judge of the needs of the individual case.

Feedings After the Twenty-first Day.—Usually by the twenty-first day, the food requirements of the infant are quite well established and a careful observation of the infant's weight, stools, disposition and equally important, its body temperature will decide the future food requirements.

The water requirement will, to a great extent, be dependent upon the supply of artificial heat and the presence of fever. Ordinarily by the beginning of the fourth week, one-seventh to one-fifth (140 to 200 cc or 100 to 140 calories per kilo) of the infant's body weight in the form of breast milk is needed to maintain proper growth. Occasionally it is necessary to exceed these amounts in the poorly nourished premature. If the physiological functions are seemingly normal the *scale* is the deciding factor in indicating food increases or decreases.

As the infant takes on weight and becomes fat with a rounding of the features and the body as is the case in successfully breast-milk-fed prematures, the total milk administration can be held at one-sixth and not infrequently one-seventh of the body weight, and normal weight increases may still be maintained.

Mixed Feeding.—When human milk, even though in small quantities, is available, it should form the basis of the diet, and cows' milk mixtures should be supplemental (Cases 1 to 14). Experience has taught us to expect a rapidly increasing mortality when less than 50 per cent of the food is other than human milk.

FIG. 102.—Baby Juanita. Age, one day; weight, 1070 gm.

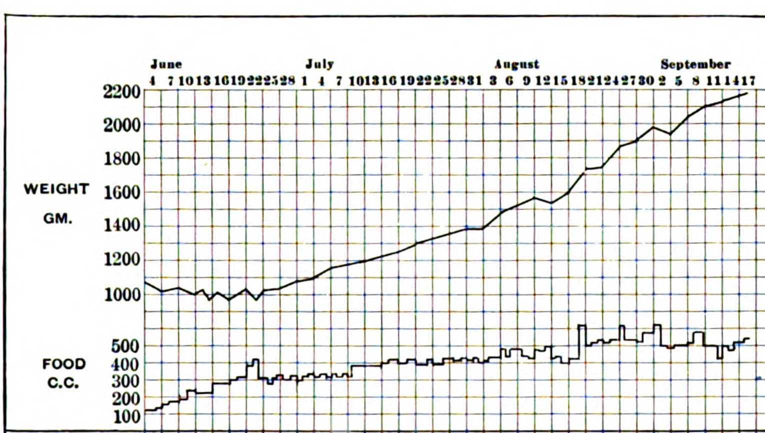


FIG. 103.—Baby Juanita. Weight and food curves and calories per kilogram weight. The patient entered June 4, aged one day; weight, 1070 gm.; condition fair. Discharged September 18; aged one hundred and five days; weight, 2180 gm. An ideal curve with an energy quotient ranging between 132 and 170, and an average daily gain of 12.5 gm. over a period of ninety days.

FIG. 104.—Baby Silvis B. Age when taken, seven months; weight, 13 pounds.

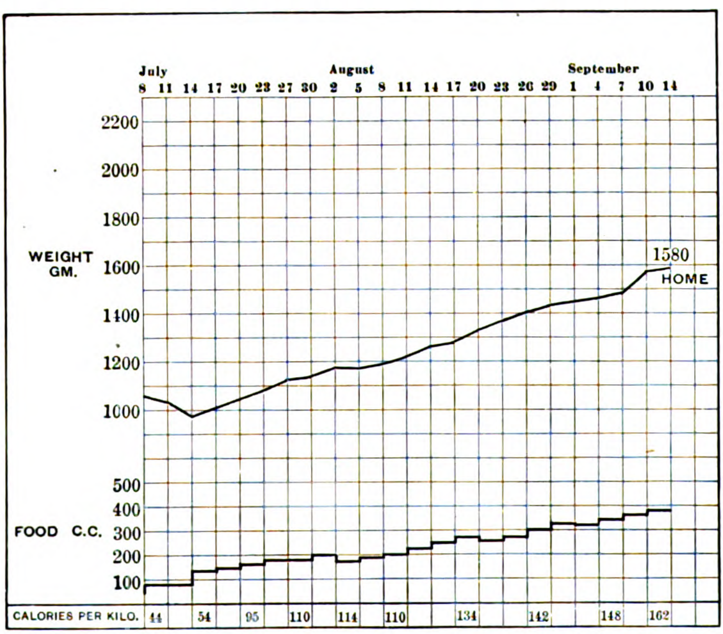


FIG. 105.—Baby Silvis (Italian). Weight and food curve and calories per kilogram. Born July 5, admitted July 8; weight, 1050 gm.; lowest weight, 980 gm., on his tenth day of life. Initial loss, 70 gm. Discharged September 11; aged sixty-five days; weight, 1580 gm. Regained entrance weight fourteenth day in hospital when seventeen days old.

AVERAGE DAILY GAINS IN GRAMS.

1	week	average	loss	per day	= 8.57	gm.;	average	48.9	cal. per kilo.
2	"	"	gain	"	= 10.0	"	"	91.0	" "
3	"	"	"	"	= 4.28	"	"	109.0	" "
4	"	"	"	"	= 8.5	"	"	121.8	" "
5	"	"	"	"	= 10.0	"	"	119.0	" "
6	"	"	"	"	= 10.0	"	"	137.6	" "
7	"	"	"	"	= 14.0	"	"	136.0	" "
8	"	"	"	"	= 11.8	"	"	142.6	" "
9	"	"	"	"	= 11.8	"	"	142.6	" "

FIG. 106.—Baby Allen B. Age, eight days; weight, 1135 gm.

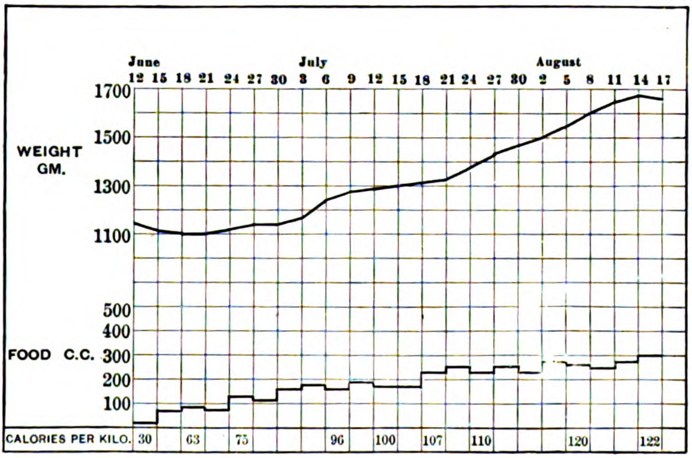


FIG. 107.—Baby Allan B. Weight and food curves and calories per kilogram. Born June 5, admitted June 12; weight 1135 gm. Discharged August 16, weight, 1655 gm.; age, sixty-four days. Regained entrance weight fifteenth day in hospital Initial loss = 35 gm.

AVERAGE DAILY GAIN IN GRAMS.

1	week average	loss	per day	=	5.0	gm.;	average	30.2	cal. per kilo.
2	" "	gain	"	=	2.14	"	"	62.87	" "
3	" "	"	"	=	6.4	"	"	75.3	" "
4	" "	"	"	=	14.28	"	"	94.7	" "
5	" "	"	"	=	5.7	"	"	96.4	" "
6	" "	"	"	=	7.1	"	"	105.6	" "
7	" "	"	"	=	15.7	"	"	107.1	" "
8	" "	"	"	=	14.1	"	"	95.6	" "
9	" "	"	"	=	12.1	"	"	112.6	" "

FIG. 108.—Baby Peggy. Age, three days; weight, 1185 gm.

FIG. 109.—Baby Peggy. Age, forty-three days; weight, 2155 gm.

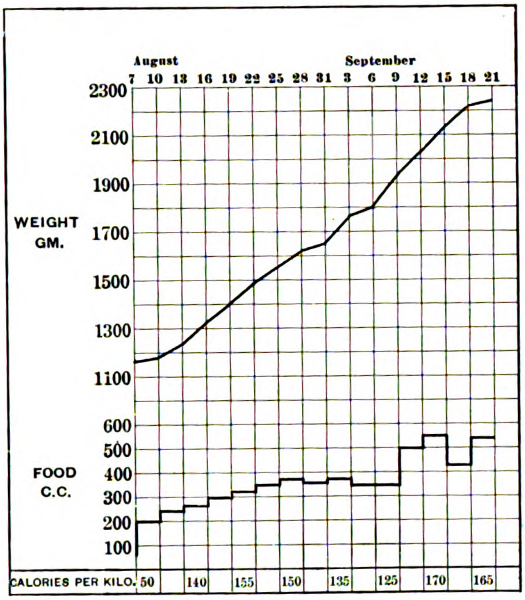


FIG. 110.—Baby Peggy. Weight and food curve and calories per kilogram. Admitted August 7; weight, 1185 gm. Discharged September 19; weight, 2155 gm.; age, forty-three days. No initial loss.

AVERAGE DAILY GAIN IN GRAMS.

1 week average gain per day	=	7.14 gm.;	average	105.7	cal. per kilo.
2 " " " "	=	22.8 "	"	142.5	" "
3 " " " "	=	15.7 "	"	155.9	" "
4 " " " "	=	20.0 "	"	114.15	" "
5 " " " "	=	30.7 "	"	145.3	" "
6 " " " "	=	33.1 "	"	146.0	" "
7 " (3 days) " "	=	28.33 "	"	149.0	" "

FIG. 111.—Baby Grace A. Age, three days; weight, 1180 gm.

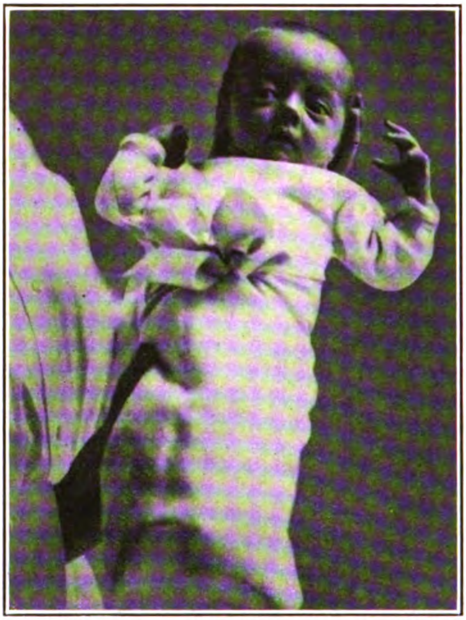


FIG. 112.—Baby Grace A. Age, eighty-nine days; weight, 1875 gm.

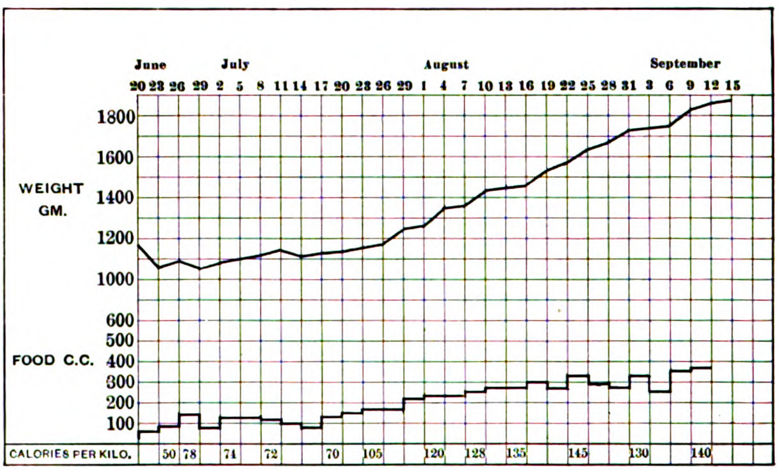


FIG. 113.—Baby Grace A. Weight and food curve and calories per kilogram. Born June 17, admitted June 20; weight, 1180 gm. Discharged September 14; weight, 1875 gm.; age, seventy-seven days. Regained entrance weight thirty-sixth day in hospital. Initial loss, 110 gm.

Average Daily Gain in Grams.

1	week	average	loss	per day	= 13.57	gm.;	average	36.36	cal.	per kilo.
2	"	"	gain	"	= 2.14	"	"	74.5	"	"
3	"	"	"	"	= 1.4	"	"	72.5	"	"
4	"	"	"	"	= 1.4	"	"	70.2	"	"
5	"	"	"	"	= 5.7	"	"	82.2	"	"
6	"	"	"	"	= 15.0	"	"	105.6	"	"
7	"	"	"	"	= 15.0	"	"	120.4	"	"
8	"	"	"	"	= 12.14	"	"	128.9	"	"
9	"	"	"	"	= 16.4	"	"	131.1	"	"
10	"	"	"	"	= 15.0	"	"	129.2	"	"
11	"	(4 days)	"	"	= 20.0	"	"	113.3	"	"

Fig. 114.—Peter P. Taken when sent home. Weight at birth, 1220 gm.; weight when sent home, 2810 gm.

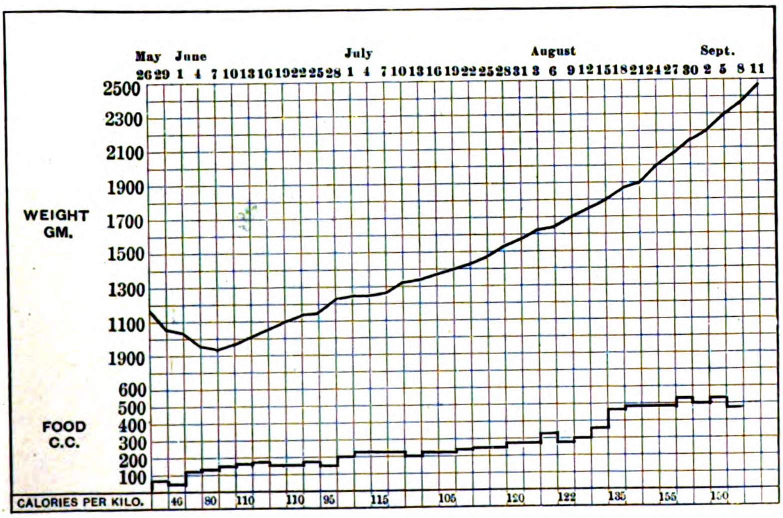


Fig. 115.—Baby Peter P. Weight and food curves and calories per kilogram. Born May 26, admitted May 26; weight 1175 gm. Discharged September 11; weight 2480 gm.; age, one hundred and eight days. Regained entrance weight on thirty-second day in hospital, when thirty-two days old. Initial loss = 220 gm. Double birth weight in one hundred days.

Average Daily Gain in Grams.

1	week	average	loss	per day	= 15.0	gm.;	average	22.6	cal. per kilo.
2	"	"	"	"	= 12.14	"	"	82.23	" "
3	"	"	gain	"	= 13.57	"	"	107.0	" "
4	"	"	"	"	= 12.14	"	"	111.0	" "
5	"	"	"	"	= 10.0	"	"	102.0	" "
6	"	"	"	"	= 8.7	"	"	115.0	" "
7	"	"	"	"	= 10.0	"	"	113.8	" "
8	"	"	"	"	= 8.5	"	"	108.0	" "
9	"	"	"	"	= 15.7	"	"	117.0	" "
10	"	"	"	"	= 15.0	"	"	126.0	" "
11	"	"	"	"	= 14.2	"	"	132.0	" "
12	"	"	"	"	= 22.8	"	"	143.0	" "
13	"	(in 12 days)		"	= 22.0	"	"	150.0	" "
14	"	(in 12 days)		"	= 28.75	"	"	145.4	" "

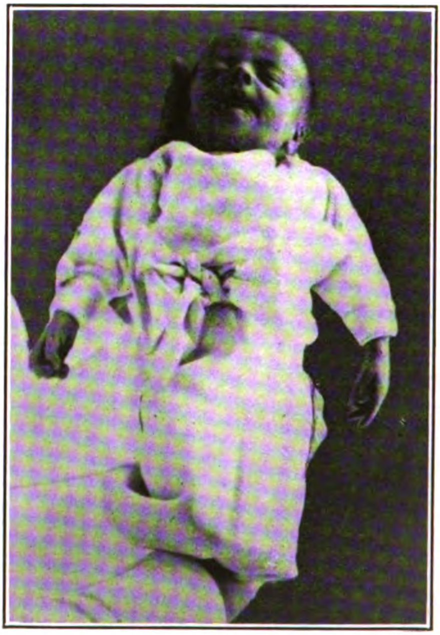


Fig. 116.—Ethna H.

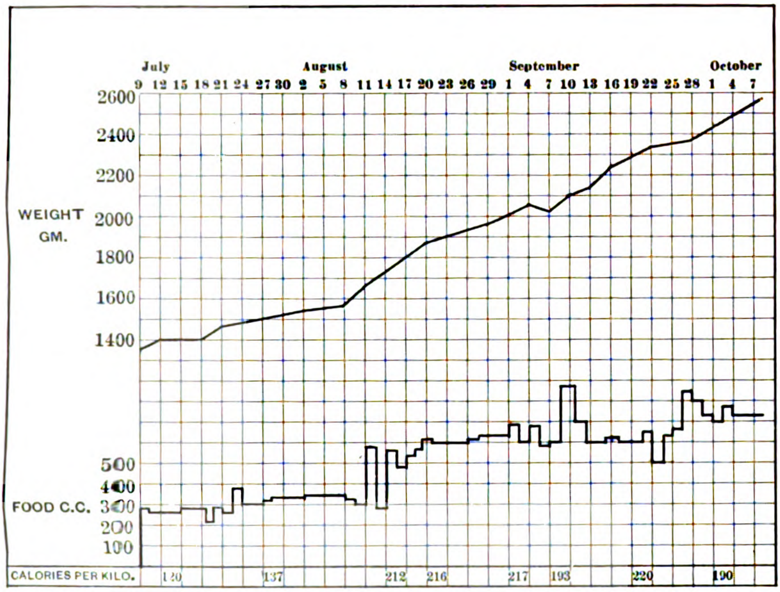


FIG. 117.—Baby Ethna H. Showing weight and food curves and calories per kilogram weight. The patient entered the hospital July 9, aged one day; weight, 1360 gm.; condition fair. Discharged October 5; aged eighty-eight days; weight, 2512 gm. Showed a steady increase after 137 calories was reached and continued to grow steadily until 220 was fed. The growth averaged only 13 gm. daily, being lower than several infants fed with a much lower energy quotient.

FIG. 118.—Joseph and Edward R. (twins). Age, three days. Birth weight: Edward, 1360 gm.; Joseph, 1190 gm.

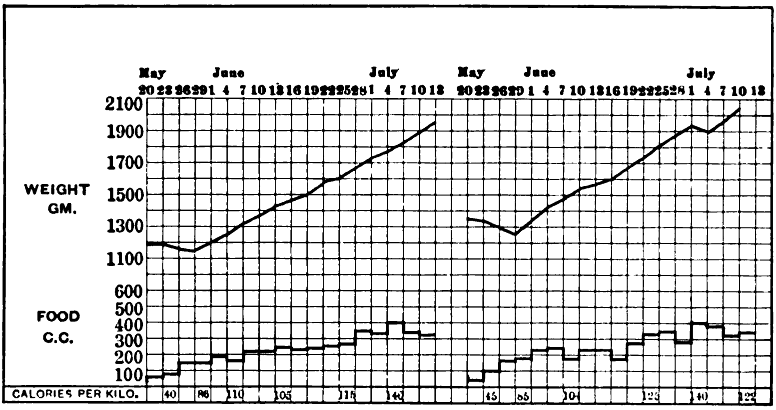


FIG. 119.—Baby Joseph R. and Baby Edward R. (twins). Born May 17, admitted May 20. Discharged July 13; age, sixty days. Mother died third day after labor.

Joseph R.—Age, three days; entrance weight, 1190 gm.; discharge weight, 1950 gm. Regained entrance weight on twelfth day in hospital, when fifteen days old. Initial loss = 40 gm.

AVERAGE DAILY GAIN IN GRAMS.

1	week	average	loss	per day	= 3.57	gm.;	average	33.5	cal.	per kilo.
2	"	"	gain	"	= 8.57	"	"	86.8	"	"
3	"	"	"	"	= 19.29	"	"	110.7	"	"
4	"	"	"	"	= 10.0	"	"	107.0	"	"
5	"	"	"	"	= 17.8	"	"	106.2	"	"
6	"	"	"	"	= 15.0	"	"	98.9	"	"
7	"	"	"	"	= 20.7	"	"	140.7	"	"
8	"	(6 days)	"	"	= 20.0	"	"	144.4	"	"

Edward R.—Age, three days; entrance weight, 1360 gm.; discharge weight, 2100 gm. Regained entrance weight fourteenth day in hospital, when seventeen days old. Initial loss = 80 gm.

AVERAGE DAILY GAIN IN GRAMS.

1	week	average	loss	per day	= 9.28	gm.;	average	32.8	cal.	per kilo.
2	"	"	gain	"	= 11.42	"	"	82.5	"	"
3	"	"	"	"	= 23.57	"	"	104.13	"	"
4	"	"	"	"	= 15.7	"	"	100.2	"	"
5	"	"	"	"	= 29.29	"	"	107.4	"	"
6	"	"	"	"	= 14.27	"	"	114.7	"	"
7	"	"	"	"	= 10.7	"	"	126.6	"	"
8	"	(6 days)	"	"	= 16.66	"	"	122.6	"	"

Fig. 120.—Baby Grace B. Taken at admittance. Birth weight, 1395 gm.

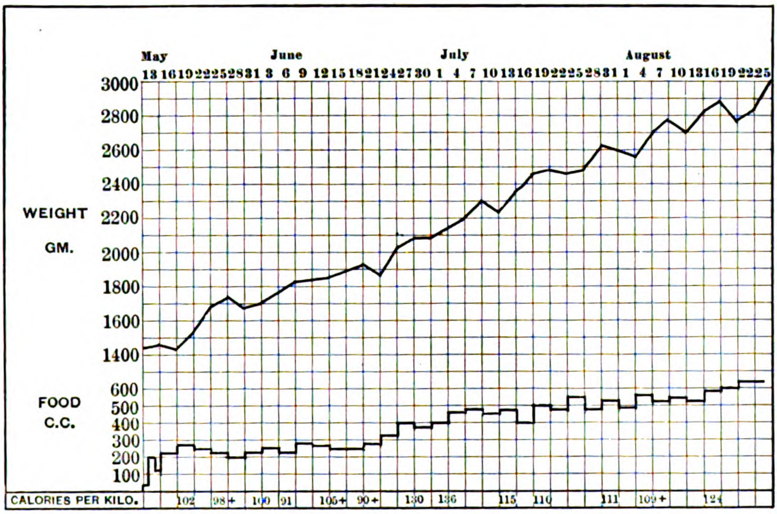


Fig. 121.—Baby Grace B. Weight and food curves and calories per kilogram weight. The patient entered May 13. Age, one day; weight, 1440 gm.; condition fair. Discharged August 27, aged one hundred and six days; weight, 2960 gm. Showed initial gain on 102 calories, followed by a loss when the same was reduced below 100; followed by a gain at 100, and a steady loss at 91; a moderate gain at 109; the loss was again repeated at 90.5 and was followed by a rapid gain at 130 to 137, averaging daily 24.5 gm., and a less rapid growth, with greater fluctuations, at 115 to 109.5, averaging 10 gm. daily and again rapidly rising with 124.

FIG. 122.—Baby Glenn. Age, two days. One of twins. Other twin died on first day.

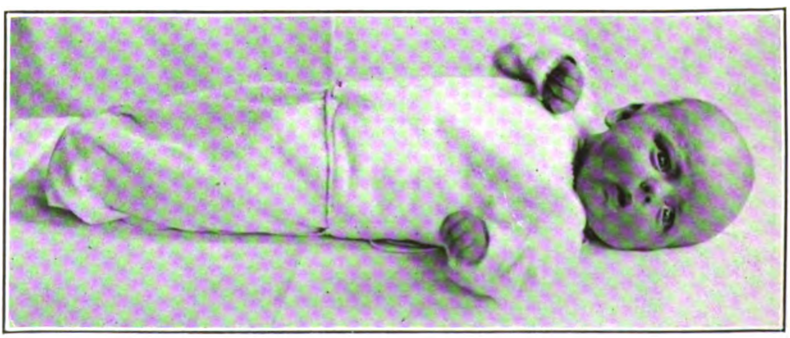


FIG. 123.—Baby Glenn. Age, one hundred and eight days.

FIG. 124.—Baby Glenn. Age, five years.

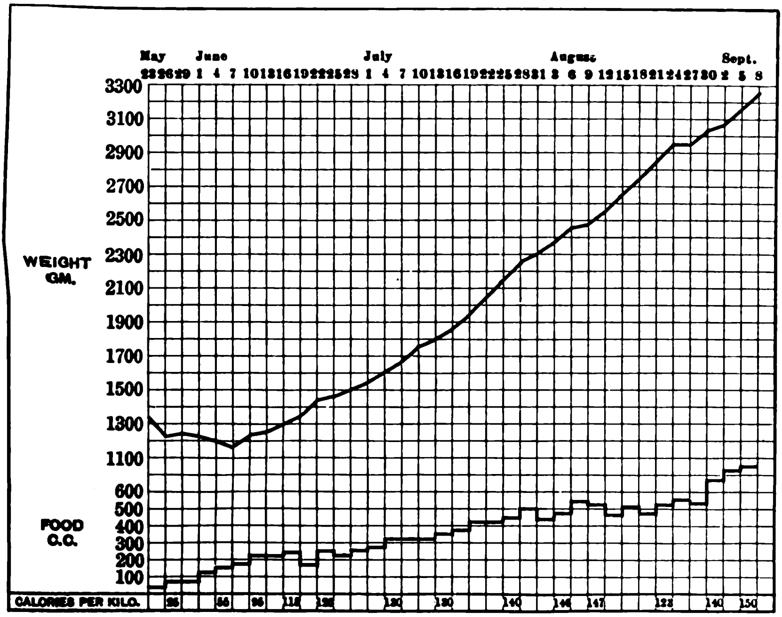


FIG. 125.—Baby Glenn. Weight and food curves and calories per kilogram. Admitted May 23; weight, 1340 gm. Discharged September 8; weight, 3245 gm.; age, one hundred and eight days. Regained entrance weight twenty-eighth day in hospital. Initial loss = 155 gm. Doubled birth weight in eighty-six days.

AVERAGE DAILY GAIN IN GRAMS.

1	week	average	loss	per day	= 1.4	gm.;	average	25.1 cal. per kilo.
2	"	"	"	"	= 1.4	"	"	55.8 " "
3	"	"	gain	"	= 4.18	"	"	94.33 " "
4	"	"	"	"	= 15.0	"	"	115.0 " "
5	"	"	"	"	= 15.7	"	"	112.3 " "
6	"	"	"	"	= 15.0	"	"	125.9 " "
7	"	"	"	"	= 23.5	"	"	104.9 " "
8	"	"	"	"	= 24.18	"	"	159.7 " "
9	"	"	"	"	= 32.1	"	"	146.5 " "
10	"	"	"	"	= 27.8	"	"	147.9 " "
11	"	"	"	"	= 16.4	"	"	123.2 " "
12	"	"	"	"	= 31.4	"	"	136.7 " "
13	"	"	"	"	= 31.4	"	"	141.9 " "
14	"	"	"	"	= 15.7	"	"	135.8 " "
15	"	(3 days)	"	"	= 15.0	"	"	132.7 " "
16	"	(7 days)	"	"	= 24.2	"	"	143.3 " "

FIG. 126.—Baby Ann C. Age, eighteen days.

FIG. 127.—Baby Ann C. Age, one hundred and thirty-six days.

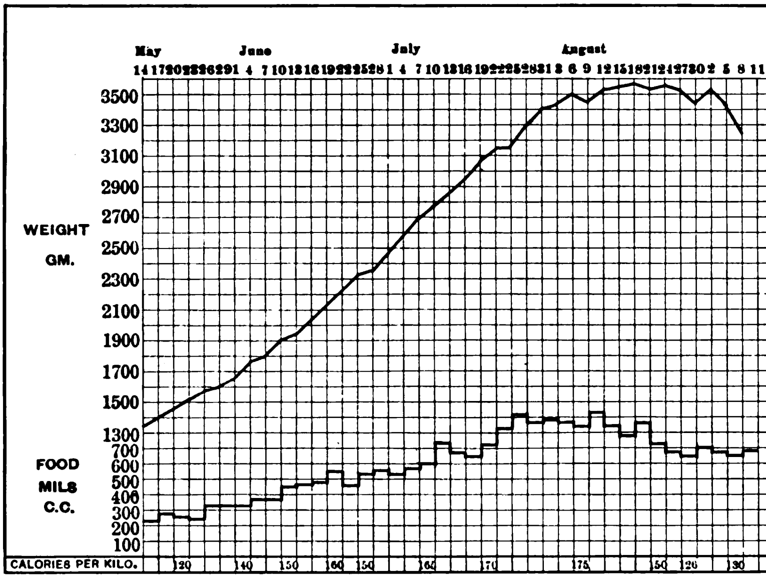


FIG. 128.—Baby Ann C. Weight and food curves and calories per kilogram. Born April 26, Admitted May 14; weight, 1340 gm. Discharged September 9; weight 3265 gm.; age, one hundred and thirty-six days. No initial loss.

AVERAGE DAILY GAIN IN GRAMS.

1	week	average	gain	per day	= 19.2	gm.;	average	120.0	cal.	per kilo.
2	"	"	"	"	= 11.4	"	"	123.0	"	"
3	"	"	"	"	= 24.1	"	"	122.7	"	"
4	"	"	"	"	= 25.7	"	"	143.0	"	"
5	"	"	"	"	= 27.0	"	"	159.0	"	"
6	"	"	"	"	= 31.4	"	"	166.0	"	"
7	"	"	"	"	= 22.8	"	"	135.0	"	"
8	"	"	"	"	= 30.4	"	"	119.6	"	"
9	"	"	"	"	= 42.8	"	"	140.5	"	"
10	"	"	"	"	= 23.5	"	"	139.0	"	"
11	"	"	"	"	= 27.9	"	"	180.0	"	"
12	"	"	"	"	= 16.4	"	"	162.5	"	"
13	"	"	"	"	= 10.0	"	"	140.6	"	"
14	"	"	"	"	= 0.7	"	"	140.0	"	"
15	"	"	loss	"	= 2.0	"	"	129.6	"	"
16	"	"	Standstill				"	125.0	"	"

ARTIFICIAL FEEDING.

There can be no comparison between the results to be expected in feeding premature infants on human milk and those to be obtained with artificial food. This is especially true of infants with a weight below 1500 gm. Therefore, if it becomes necessary to resort to artificial feeding, the selection of a food, its preparation, and its adaptation to the infant must all be given the most painstaking consideration. Many varieties of artificial diet have been suggested by many different authors, such as simple milk dilutions cream and top-milk mixtures, skimmed milk and buttermilk preparations, malt soup preparation, condensed and evaporated milk, etc. The results with these various diets are to a great degree dependent upon the physician's intimate understanding and directions for the use of the individual food (Fig. 129).

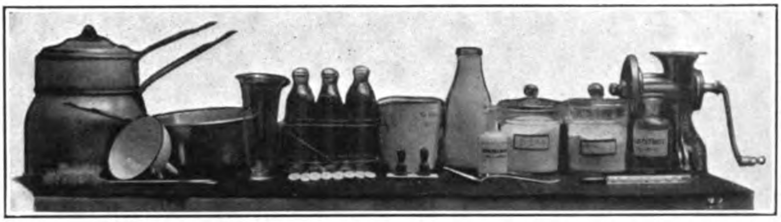


FIG. 129.—Utensils needed for artificial feeding: Double boiler (small), pan, funnel, bottle-brush, 250-cc (8-oz.) graduated glass or pitcher, six nursing bottles and rack, paper caps for bottles (sterile), nipples, milk, sugar, flour, milk of magnesia, citrate of soda, tablespoon, dairy thermometer, vegetable mill.

Quantity of Food.—It must be remembered that the figures quoted for the feeding on breast milk are the maximum that can be assimilated and are excessive quantities for artificial feeding in the first weeks of life because of greater difficulty in the digestion of cows' milk. These infants when artificially fed must at all times be closely watched for evidences of overfeeding, and the first evidence of digestive disturbances or of intercurrent infections should lead to the feeding of human milk whenever possible.

From the foregoing statement, it is quite evident that smaller and slower weight increase may be expected of the artificially fed.

Quality of Food.—As previously stated, opinions vary greatly as to the best food for artificial diet. Most clinicians have obtained the best results with the feeding of low-fat mixtures. Boiled milk, skimmed milk and buttermilk, with carbohydrates added, are among the best.

In feeding with buttermilk and skimmed milk with added carbohydrates, the fat-free mixtures must not be too long continued, otherwise the infants will suffer from fat inanition. By the end of the third week some whole boiled milk may be added or the lactic-acid milk should be prepared from partly skimmed milk. It is our routine to *boil all artificial food mixtures* for at least two minutes.

In feeding with a buttermilk high *carbohydrate mixture*, the caloric requirements are lower than in feeding with the full milk (chymogen) mixture with its *lower carbohydrate* content, because the energy for digestion and assimilation requirements are lower with high-carbohydrate low-fat feeding as compared with high-fat low-carbohydrate mixture.

BUTTERMILK AND SKIMMED MILK MIXTURES.

Buttermilk or skimmed milk	1000
Flour (dextrinized)	10
Sugar (cane)	40

The above being used for the first weeks.

Buttermilk or skimmed milk	1000
Flour (dextrinized)	15
Sugar (cane)	60

For later feedings.

Maltose-dextrin compounds can be substituted for the cane sugar if desired.

Directions for Preparation.—Add the flour to a few tablespoonfuls of buttermilk or skimmed milk and rub to a paste. Add buttermilk or skimmed milk to 1 liter.

Bring mixture to a boil and withdraw from the fire.
Add the sugar and bring to a boil for the second time.
This process should require about twenty minutes.
Make up to 1 liter with boiled water.
Keep on ice.

In the use of the buttermilk mixture it must be remembered that infants are not to be kept on this low-fat mixture for too long a time, addition of whole milk being indicated after the first few weeks, beginning with one-third whole milk and two-thirds skimmed; by the fourth week equal parts whole and skimmed milk should be used in the preparation of the lactic acid milk.

Chymogen Milk.

Requirements for Preparation.

Milk.
Single boiler, 1- or 2-quart.
Dairy thermometer.
Chymogen powder (Armour & Co.).
Teaspoon.
Egg beater.

Directions for Preparation.—Boil milk for five minutes over direct flame, cool to 104° F., and add one full teaspoonful of chymogen to each quart of milk, and stir for one-half minute. Let it come to a clabber by allowing it to stand for fifteen minutes, holding it as near 100° F. as possible by keeping it in a warm place; then beat it well until the curd is finely divided. Put it in individual feeding bottles and place on ice. Do not heat above 100° F. when preparing individual bottles for feeding, otherwise curds will clump and will not pass through the nipple. Reheating is best accomplished by placing the individual feeding bottle in a cup of warm water not over 115° F. and allowing it to stand for ten minutes. Replenish the warm water if necessary.

We have found this predigestion of boiled milk, by the addition of rennet (chymogen) assures the infant's stomach of a fine, flocculent curd, which is about the size of that of human milk. In beginning feedings with the above preparation it is usually diluted with 3 parts of water and increases in quality made as indicated, and the quantity increased as in the feeding of human milk. In feeding with the diluted predigested milk, 15 gm. (½ ounce) of lactose should be added to each liter during the first few days, and the amount gradually increased to 30 gm. (1 ounce). When chymogen is not available, whole milk boiled for five minutes may be used.

In the feeding of these food mixtures the relative caloric values of the mixtures as compared with breast milk must be borne in mind, otherwise inanition will be the result.

Human milk equals 700 calories per liter or 21 per ounce.

Skimmed milk or buttermilk mixture containing 10 gm. dextrinized flour and 40 gm. sugar per liter equals 16 calories per ounce or 525 per liter.

Chymogen milk (whole milk) equals 700 per liter or 21 per ounce.

Chymogen milk when diluted with 3 parts water and 15 gm. of sugar per liter equals 235 calories per liter or 7 per ounce.

Chymogen milk when diluted with equal parts water and 30 gm. of sugar per liter of mixture equals 470 per liter or 14 per ounce. Each individual ounce of sugar per liter of mixture increases its food value by 4 calories per ounce. Rarely should more than 2 ounce of sugar be added for each liter of the mixture, which represents an addition of 8 calories (6.5 per cent) of carbohydrate to that already contained in the milk. It is, therefore, of the greatest importance that the milk itself either skimmed, buttermilk or whole be increased gradually along with the sugar.

Amounts to be Fed.—Depending upon the weight and development of the infant, the tables as given for human milk feeding should be followed. It must be remembered that while the artificial diets recommended have a lesser caloric value per cc than human milk, they represent for most infants the maximum capacity for digestion and assimilation. Of necessity, lesser weight increases and slower progress are to be expected. The infants are paler, tissue turgor is lacking and they are less immune to infection.

Other Dietetic Requirements.—To counteract the effects of boiling, orange-juice feeding should be instituted by the third week, beginning with 0.5 cc (8 drops) and increasing 2 to 4 cc ($\frac{1}{2}$ to 1 dr.) daily by the eighth week, in order to avoid scurvy. Cod-liver oil as an antirachitic should be fed by the fourth week, beginning with 0.5 cc (8 drops) daily divided into two feedings and increased to 2 cc (30 drops) daily by the eighth week. It may be mixed with the orange juice. To counteract the low iron content of these diets, carbonate of iron in 0.03 gm. (gr. $\frac{1}{2}$) or citrate of iron and ammonia in 0.03 gm. (gr. $\frac{1}{2}$) once daily should be started by the fourth week. The latter may be prescribed in solution.

Mixed Diet.—These food additions apply for breast-fed as well as bottle-fed infants.

Fifth month, a little well-cooked cereal may be added to one of the meals (begin with one teaspoonful), adding it to the bottle of milk.

At the sixth month, infants readily take a broth and vegetable meal as a substitute for one of the milk feedings, in the form of a vegetable and meat soup. Begin with one ounce and follow by a second bottle containing the milk mixture with one ounce less than full feeding. Gradually replace an entire milk feeding.

Ninth month, a vegetable soup or a clear broth (chicken, lamb or veal), and toast or zwieback crumbs, with an additional portion of stewed fruits (apples, prunes) or a strained vegetable (spinach, carrots or turnips). The broth is usually given in the same quantity as the bottle, if given alone, or somewhat less if either the vegetable or fruit is given in addition.

Cereal.

Two tablespoons of cereal.

One-half pint of water and one-half pint of milk.

One pinch of salt.

Cook in double boiler for one hour.

Begin feeding one and a half tablespoonful, gradually increasing to two tablespoonfuls.

Add the cereal to the milk mixture or pour part of bottle over the cereal and feed with a spoon. Finish the meal with remainder of the bottle.

Toast or zwieback (one-half slice crumbed) at about the eighth month.

Vegetable Soup.

Lean lamb cut into small pieces ($\frac{1}{4}$ lb.).

One potato.

One carrot.

Two stalks of celery.
One tablespoonful of pearl barley.
Two tablespoonfuls of rice.
Two quarts of water.
One pinch of salt.

Finely divide the vegetables. Add the vegetables barley and rice to two quarts of water. Boil down to one quart, cooking three hours. Add pinch of salt. Pass through fine sieve.

Begin feeding one ounce, gradually increasing to eight ounces, cutting out an ounce of milk mixture for each ounce of soup given.

When less than a full feeding is fed finish the meal with sufficient milk mixture, from a second bottle, to make a full feeding.

CHAPTER IX.

INCUBATORS.

The History of Incubators.—The first records of the use of incubators are found in description of their employment for the hatching of eggs of fowls in Alexandria, and it is possible that it may have occurred to the Egyptians to apply this method to the new born. One cannot, however, assume this, for no author of that period has mentioned it. Hippocrates, in his writings of 460 B.C., makes the following statement: "No fetus coming into the world before the seventh month of pregnancy can be saved." We note that the literature of our day records only a limited number of exceptions to these conclusions that infants born before the end of the twenty-eighth week are viable.

Pasquad quotes a thesis of the Eleventh Century of the Republic wherein the author, Rudellet, writing on the vitality of infants, reports the following which we think well worth citing. He quotes from Baillet (*Decisions of Savants*, Paris, 1722) as follows: "Among the records of celebrated children Baillet reports that of Fortunio Liceri, whose mother gave birth to him long before the ordinary time during the fatigue and shocks of a sea voyage. This fetus was no larger than the palm of your hand, but his father who was a physician, having examined it, had carried it to the place which was to be the end of his voyage. There he had other physicians see it. They found that there was lacking nothing essential to life, and his father undertook to finish Nature's task and to work at the formation of the child with the same skill that men exhibited in hatching chickens in Egypt. He instructed a nurse in all that she had to do in the maintaining of exactly measured artificial heat and the requirements for his general care and feeding. He lived to be seventy-nine years of age and distinguished himself in science by a large number of works."

This is a tale the recording of which leads us to believe that use was made of the knowledge of the methods used at that age in incubating fowls. We will dispense with any remarks and will content ourselves with mentioning the fact, in citing the reflection of the historian himself: "One must admit," says the author in concluding the narrative of which we have just given the analysis, "that all which is unbelievable is not always false, and that probability is not always on the side of truth."

Little is recorded from 1722 to 1857, the time when Denucé described his incubator. Modern French writers attribute the origin of the first incubator for infants to Denucé, of Bordeaux, who in 1857 described his model which consisted of a double-walled tub which was to be filled with warm water at intervals (Fig. 130).

Clementovsky states that a somewhat similar contrivance was used by Rühl in St. Petersburg as early as 1835.

Credé, in 1866, published the results which he obtained with a similar apparatus which had been in use in his clinic in Leipzig since 1860, with the use of which he was able to lower his mortality by 18 per cent. This simple tub has been modified by some of the continental clinicians by putting it on a stand and providing it with a hose attachment for connection with a hot-water faucet (Fig. 131).

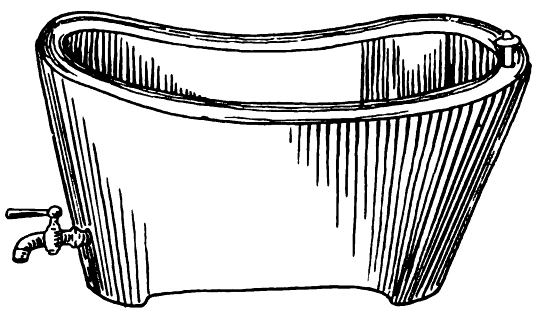


Fig. 130.—Warm tub with double wall jacket. First used by Denucé in 1857 and Credé in 1860.

In 1879 Winckel described a permanent bath in which the infant floated. This apparatus was an attempt at imitating intra-uterine conditions, but needless to say, because of the danger of drowning and infection, it proved unpopular. The water in this tub was kept between 36° and 38° C.

In 1680 Tarnier had an infant incubator constructed similar to those used as chicken incubators. This incubator was built for him by Odile Martin, director of the Paris Zoo, and was built of such size that it could hold several children; and was installed in the Maternity Hospital of Paris in 1881 (Fig. 132).

This is the first closed incubator which may be qualified as modern, for the perfected apparatus of our day differs from it only in detail.

This is the time that dates the principal work undertaken on incubator construction, and the most varied modifications have followed each other almost without interruption until our day.

The first important work on the results obtained by their use is an account by Auvard in 1883. In this interesting work the

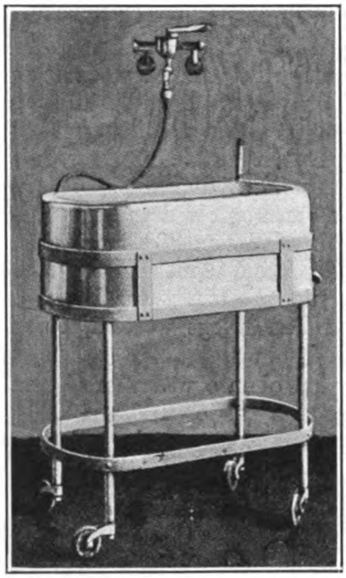


Fig. 131.—Modified warm tub.

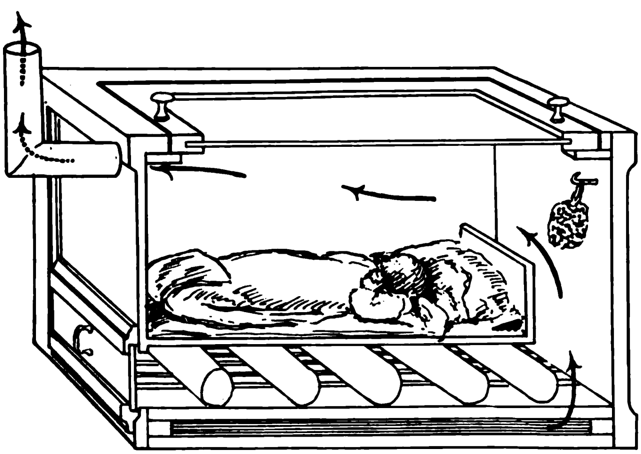


Fig. 132.—Tarnier incubator.

author gives the first statistics on the use of the incubator in the Maternity in this period under the scientific direction of Tarnier.

Berthod, an interne of Tarnier, in an excellent thesis (1887), continued this study, adding thereto some new data on the conditions indicating the use of an incubator for the new-born infant. His statistics are still most imposing, and they treat upon almost a thousand cases in which the incubator was used. Among the most influential of these was Budin.[11]

The monographs of Auvard and Berthod are the only two important early works treating of the study of incubators and the results of their use. Their work shows the importance of the prevention of hypothermia, and they lay particular stress upon the protection of the infant from the moment of its birth. It may be stated with justice that the early progress in the care of premature infants was to a great extent influenced by the interest of the French obstetricians in the care of these infants.

Since then a large number of authors have written on this subject, but it is rather to modify certain details or to propose new forms of apparatus than to give new rules.

From that time on until our day the incubator has undergone changes—some quite radical, while some have remained as rudimentary as when first originated.

Only those models demonstrating more important changes and improvements will be described.

Hearson introduced automatic temperature regulation within the incubator. His apparatus was so constructed as to set off an electric alarm clock when the maximum temperature desired was past. This apparatus was modified by Eustache who attempted to attach automatic gas or oil-heating apparatus to the so-called "thermostat nurse of Hearson."

In 1896 Diffre, of Montpelier, and Lion built metal incubators, providing for what they termed final perfection which provided for automatic heating through thermostat control, the heat being furnished through a hot-water system heated by an oil or gas stove at the side of the incubator. In this incubator refinements in ventilation and control of humidity were introduced.

A giant incubator was prepared by Prof. Pajot, in 1885, for use in his clinic, consisting of a large heated chamber, practically an oven; the congenitally feeble infants, entirely separated from their mothers, being fed and tended by wet-nurses.

Budin, in stating the disadvantage of the Pajot apparatus, said: "The wet-nurses were obliged to feed and tend the infants in this oven; and the mothers, separated from their infants, soon lost all interest in those whom they were unable to nurse and cherish. It is better by far to put the little one in an incubator by its mother's bedside."

Prof. Hutinel, of Paris, whose studies on the subject are of interest, constructed a couveuse composed of a boat-like vessel of enameled crockery whose bottom was replaced by a plate of galvanized sheet-iron pierced by holes. The plate served as a cover to a metal box, which contained three bowls filled with hot water. The top of the apparatus was closed by a heavy glass which could be raised to a desired degree by the aid of a screw, and which allowed the airing of the box. The water bowls were replenished every two or three hours to maintain the temperature. The crockery tub could be disinfected with ease by wiping it with a cloth saturated with bichloride of mercury solution, which was its best feature.

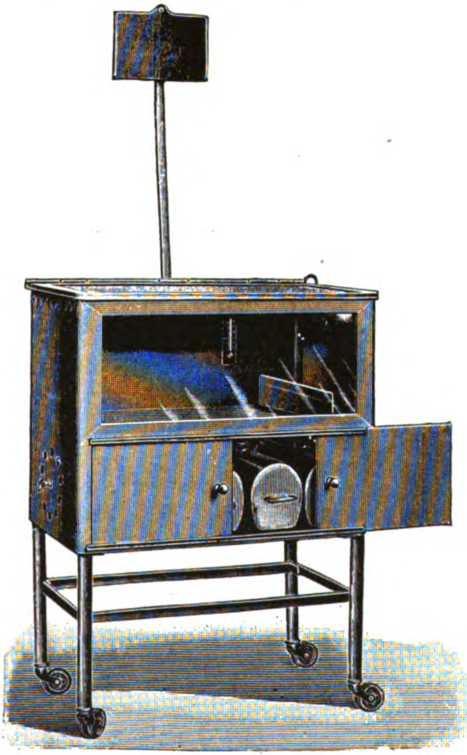


FIG. 133.—Finkelstein incubator.

Simple and cheap in its operation is Finkelstein's incubator. The essentials of its construction may be seen from the accompanying illustration (Fig. 133). The circular holes in the side walls of the box for inserting hot-water vessels also serve as inlets for the incoming air, while the used air escapes through the holes at the upper part near the cover.

Rommel's apparatus proved to be good and is at the same time like the latter easily carried from one place to another (Fig. 135). The chamber is 0.83 cubic meter large, enclosed on three sides by mirror glass, the corner being rounded to facilitate cleaning. The ventilating shaft permits the air to be renewed 100 to 120 times every hour. The humidity regulation is simple. The large supply of hot water of about 15 to 20 liters permits a pretty constant temperature, the fluctuations according to Rommel being less than 1°. For heating electric incandescent lamps are used.

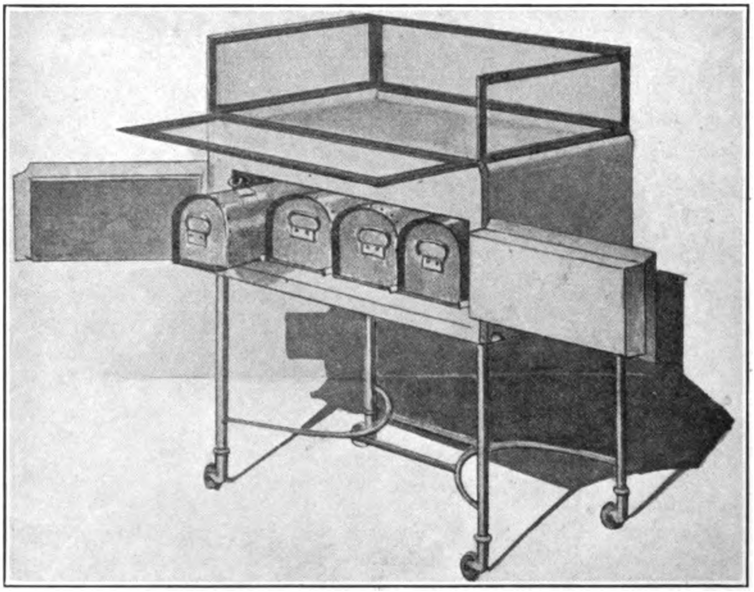


FIG. 134.—Reinach heated bed.

Moll's incubator (Fig. 138) distinguishes itself by the fact that the head of the infant remains outside the warm box and breathes the air of the room, this having a great advantage for respiration of debilitated infants, since, because of stronger respiratory stimulus, attacks of asphyxia may be more easily avoided.

To this class of incubators, which are all modifications of the origional Lion type, belong the models of Couney, DeLee and others now on the market in the United States (Fig. 136 and 137).

These models differ but slightly in principle, the chief variation being in the manner of heating and distributing the air and supplying moisture. They may be heated by gas or oil stoves situated at the side of the incubator, heating the air as it enters, or by a system of electric bulbs within the incubator. In the latter models the bulbs are usually located either in the floor or sides. The best models are those in which the heating system is modelled after that used in hot-water heating plants for houses. The temperature is automatically controlled by a thermostat.

A thermometer is fastened near the side window, so that it may be easily read, and a hygrometer is used to indicate the degree of moisture.

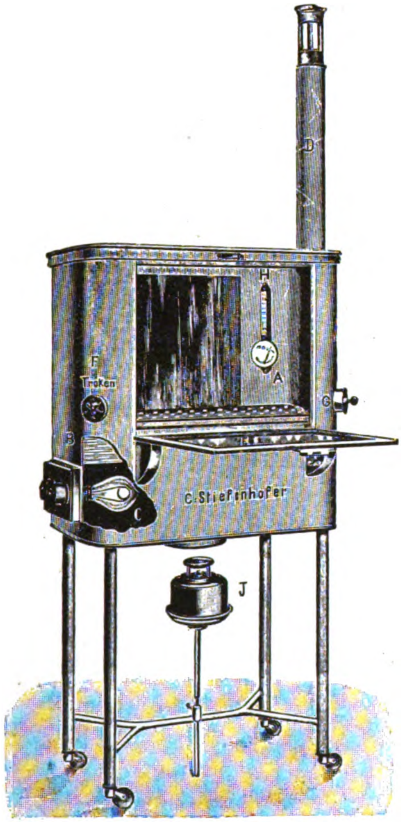


FIG. 135.—Rommel incubator.

This type of incubator has in the past ten years lost considerable of its early popularity, as is evidenced by a visit to most of the large hospitals. A great deal of this deserved unpopularity is due to the inability to ventilate them in the ward and the necessity for furnishing a trained attendant. To properly supply these incubators with a free current of air it is necessary to connect them so that they will receive a supply of air from outside of the building. To counteract the tendency to an insufficient air current in the absence of winds or when they are in the wrong direction an electric fan should be incased on the outside of the building in such posi-

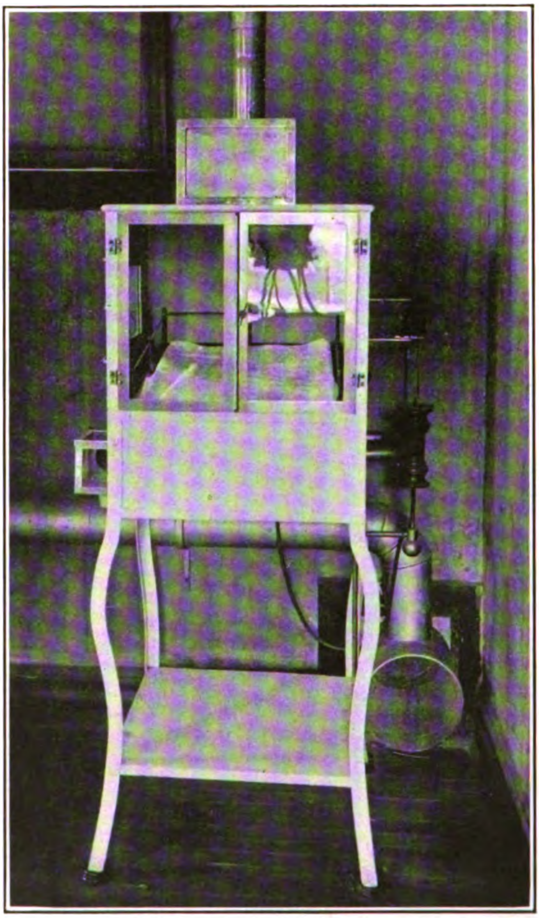


FIG. 136.—Lion-type incubator (Couney model). The fresh air is forced through a large air-shaft by an electric fan on the outside of the building.

tion that air may be blown directly through the incubator. This is difficult of arrangement when the incubators are located above the first floor of the building. When the station is located on the first floor it is necessary to avoid the dampness and dust of the street level, and this can be accomplished by installing a large funnel 15 or 20 feet above the ground level, some 24 to 36 inches in diameter, to which is attached a 10-inch stack which can be connected with a cage at its base in which the electric fan is installed. From this point the air is blown through the system of incubators.

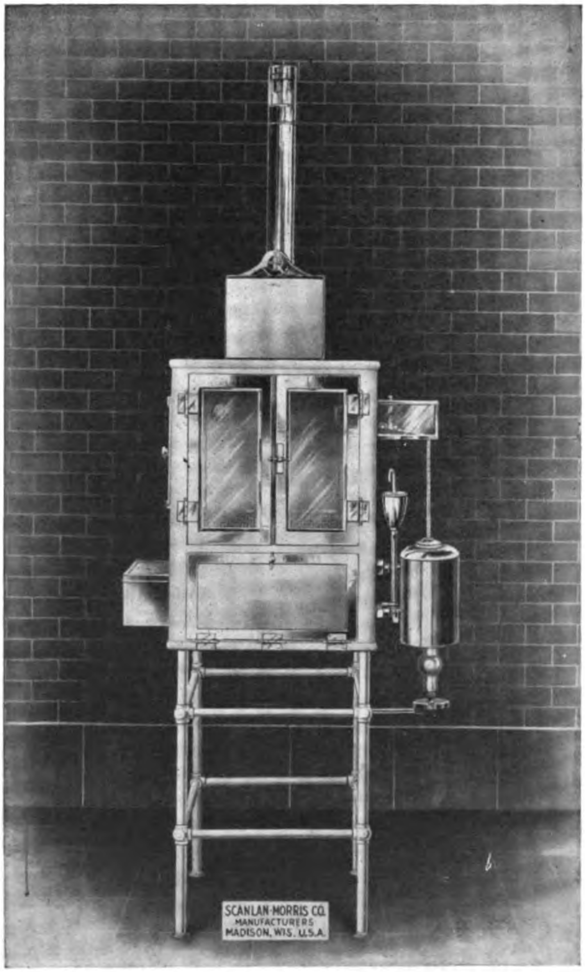


FIG. 137.—Lion-type incubator. (DeLee model.)

When such a considerable quantity of air is blown into the incubator system, it becomes necessary to filter it through several layers of cotton. This is best done at the side of the individual incubator.

The Cincinnati Hospital uses an electrically heated bassinette.

The temperature is regulated by a series of electric lamps under the mattress. The apparatus consists of a double wall frame, with

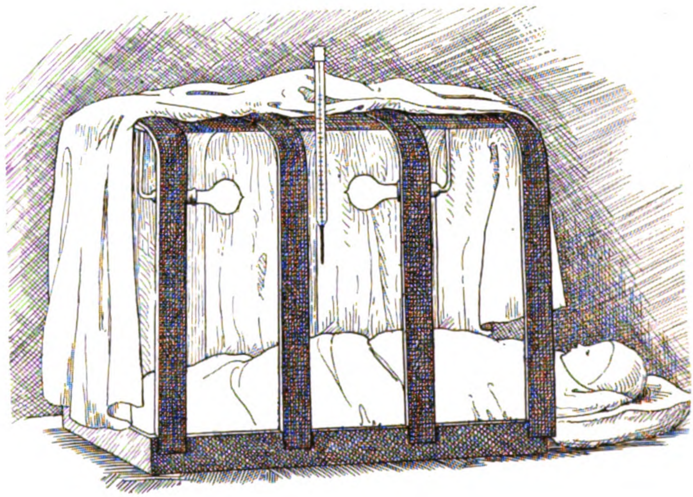


Fig. 138.—Moll heated bed.

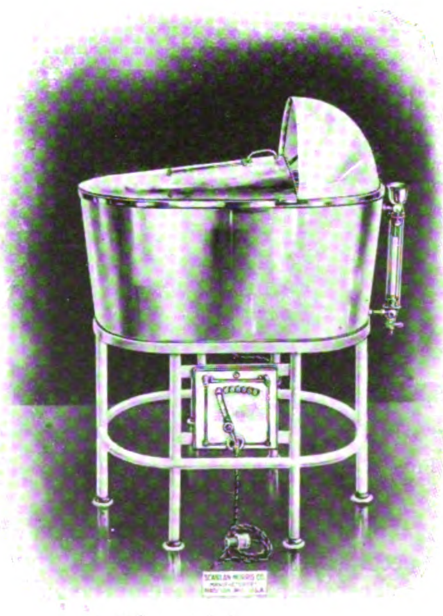


Fig. 139.—Hess water-jacketed infant bed.

hot air rising in this double wall and escaping through small holes near the top which can be opened or closed as may be required by a slide damper.

In 1914 the writer designed an electric heated water-jacketed infant bed.

It combines the double-wall water jacket with insulation to prevent external loss of heat, and electric heating by a large plate with rheostat control.

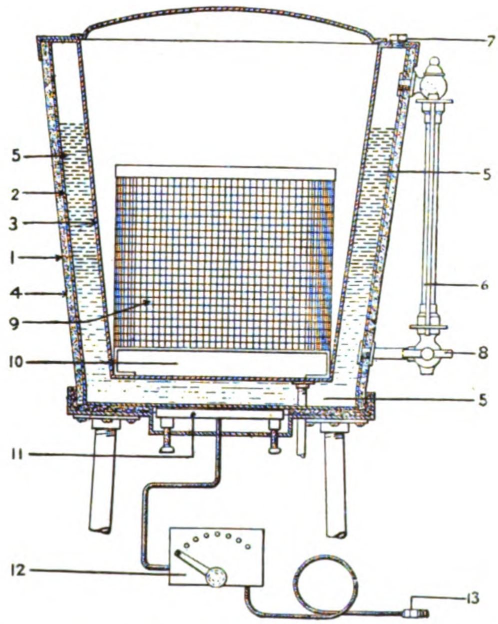


FIG. 140.—Cross-section of Hess heated bed. 1, Cooper wall covering asbestos layer; 2, asbestos layer insulating water-jacket; 3, 4, copper walls covering water-jacket; 5, water surrounding side and floor of bed; 6, water glass; 7, funnel for filling jacket; 8, cock for emptying jacket; 9, removable crib; 10, air space underneath crib; 11, electric heating plate; 12, rheostat; 13, electric plug.

For hospital and home equipment the bed answers many requirements, because of its simplicity of operation in any well-ventilated and moderately heated room.

It can be used for the care of premature infants, for the protection of the new-born full-term infant immediately after delivery and for infants suffering from hypothermia from other causes.

This bed fulfils the following needs of the infant: (1) *Safety.* The maximum temperature which can be obtained within the bed is about 110° F. when the lid and canopy are in place with a room temperature of 70° F. While such a temperature would be injurious if maintained for a long period of time, such surroundings if temporary can cause but little injury. (2) *Simplicity of operation.* It requires practically no attention unless there are extreme ranges of temperature within the ward, since the asbestos insulation prevents radiation from the outer surface of the bed and the heater holds the water at a constant temperature. (3) *Ventilation.* This apparatus assures the baby of an adequate supply of fresh air if placed in an ordinary room which is well ventilated. (4) *Humidity*

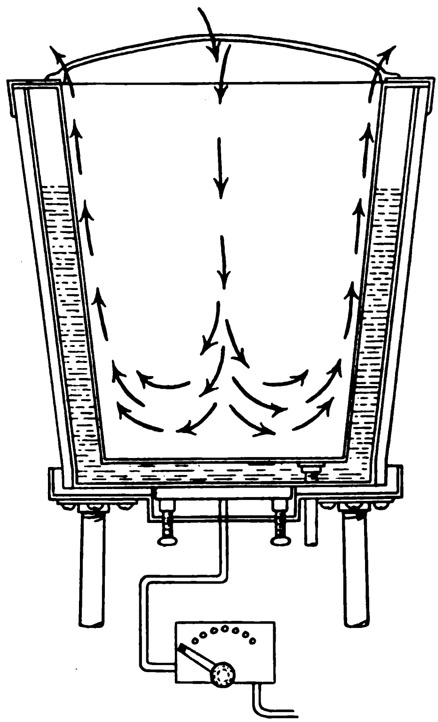


FIG. 141.—Cross-section of Hess bed showing direction of air currents.

is maintained at nearly the same degree as the surrounding air because of the almost constant change of air within the bed and moisture supplied by an evaporation pan beneath the crib. (5) *It is easily cleaned and disinfected.*

The construction of the bed is such that it can be used in an ordinary ward or room, giving the infant the advantage of a most perfect room ventilation.

The following suggestions will aid in the practical application of this bed for use in hospitals or the home.

A special room should be provided. This has a practical advantage as it impresses the nurses to consider this room as barriered. This will make a demand upon the nursing staff for observation of all of the rules of aseptic nursing.

This room should be supplied with an ample system of heating coils controlled by a thermostat for winter use, thereby facilitating the maintenance of a more or less stable temperature in the room which should, in so far as possible, range between 68 and 75° F.

The temperature within the room and bed should be read and charted at six-hour intervals, best at 6 A.M., 12 M., and 6 and 12 P.M. as the most likely time for maximum changes in the ward temperature.

Ventilation should be adequate but not excessive, and the room should be so constructed that the beds may be placed without the line of direct air currents. This is accomplished by having the ventilating windows and transoms on one side of the room, while the opposite side is built with non-ventilating windows or blank walls at either end.

Humidity in so far as the room is concerned will require little attention except at such time when considerable artificial heating is necessary. To supply the needed moisture during cold weather when ventilation of the room is more or less limited, a large evaporating pan should be in direct contact with the radiator coils. When these means fail to furnish the desired moisture, a wet sheet may be hung in the room and remoistened as indicated by the hygrometer.

In so far as possible the relative humidity should be kept at about 55 per cent. However, amounts less than this down to 45 per cent will usually cause little or no discomfort or retardation of progress. It has been our experience that with a good free ventilation through open transoms or windows when the temperature of the room does not exceed 80° F., the normal water content of the air is quite sufficient and little or no attempt at influencing the room humidity is necessary. However, this will not answer the purpose where a closed room is used.

When a special room cannot be provided the beds for well prematures should be kept in the nursery used for normal infants. They must never be brought into contact with infected infants because of the danger of crossed and mixed infections. Neither should infected prematures be placed among well new-born infants.

All infants should be removed from this room to the nursery once daily, so that it may be thoroughly ventilated and cleaned by the use of soap and water. Before replacing the infants the air should be reheated to remove excessive humidity.

CARE OF THE BED.

General Care.—Once daily the infant should be removed from the bed to allow of cleaning the interior with a damp cloth. This is best done at the time of renovating the room. The crib itself should also be wiped with a damp cloth. All linens should be changed at least once daily and at other times when soiled. Extra mattresses should be supplied so that they may be given an airing on alternate days and a thorough renovating as frequently as soiled. Renovation is imperative between cases. Mattresses should be protected by rubber sheeting. A thick pad, however, must be placed between the rubber sheeting and the infant.

The heating apparatus consists of a plate with a 6-inch surface in direct contact with the floor of the water jacket, and especially constructed to carry a maximum capacity of 300 Watts, which makes it impossible to heat the water above 155° F. and the interior of the bed above 110° F. at a room temperature of 70° F.

A rheostat with seven contacts is fastened to the standard. Six of them are graduated to take current varying from 25 Watts on contact 1 to 300 Watts on contact 6. The first contact shuts off the current.

For the protection of very frail infants a partial cover for the tub, 21½ inches in length, is provided to shield them more completely from outside air currents. It is provided with a thermometer, so that the temperature within the tub can be ascertained by the nurse at all times. Further, a brass nickel-plated frame covered by a removable linen cover is provided in the form of a hood. This can be set over the open space not covered by the metal lid in case of great air currents and extremely cold nights. This allows a free circulation of air to enter at the front of the canopy while at the same time preventing direct downpour of cold air onto the infant's head. The hood raises the temperature within the bed on an average of from 5° to 10° F., depending on the room temperature and current used, but does not interfere with perfect ventilation. The hood is made collapsible, and may be set at any angle desired, as may be indicated.

The *hood* is used in combination with the lid for very small or frail infants where a high temperature is desired or when the room temperature is more or less beyond our control, because of a defective heating system or extremely cold weather. Both are used when it is desired to heat this bed rapidly in an emergency.

The *removable metal lid*, which also holds the thermometer for temperature reading, is used alone for most cases, the length of time varying from a few days to several weeks. The hood and lid are both left off for the more mature cases and those being prepared for graduation from the heated bed to the nursery or home.

With the lid on it is rarely necessary to pass contact 4 of the rheostat to obtain a temperature of 90° F. in a room approximating a temperature of 70° F. When it is desired to heat the bed rapidly preparatory to its use, the rheostat may be set at point 6 with the hood and lid on until the bed is heated to the temperature that may be needed when it may be returned to points 2, 3 or 4, depending upon the fetal age and development of the infant.

As the infant develops it should be gradually prepared for graduation from the incubator by lowering the temperature of the bed by degrees to that of the room temperature. This may cover a period of several days or weeks. At this time the lid may be removed. We have found it of advantage to remove the lid of the bed when the infant has developed sufficiently to thrive in the room temperature of 75° F., after which the temperature of the bed with the lid off can be left a few degrees above the room temperature by advancing the rheostat by one or two points. The temperature of the bed is now measured by placing a thermometer alongside of the baby within the sleeping bag or under the blanket.

It is our custom to cover the infant when in the bed with a light sleeping bag or light woolen blanket, in order to more completely stabilize its body temperature, as our beds are kept in a well-ventilated room. The sleeping bag should either be fitted with a flap, which can be used as a hood or a small bonnet should be worn or the blanket should be so applied so that it can be used as a head cover. *Outer garments or covers should be applied loosely so as to allow of free movements of the extremities.*

In order to use the bed rationally it is necessary to have an idea of the effect of the various factors influencing the crib temperature. To this end the following observations are offered for the guidance of the attendant.

COMPARATIVE MEASUREMENTS OF TEMPERATURE IN HEATED BED UNDER DIFFERENT CONDITIONS.

The temperature as read from the lid thermometer and that of a thermometer placed alongside of the infant under a light blanket used as a cover will show variations which rarely exceed 1 to 3° F.

At a room temperature varying between 70° and 80° F. The lid temperature will approximate the following:

Lid and Canopy On.—10° F. above the room temperature when on contact 2; 15° F. above the room temperature when on contact 3; 20° F. above the room temperature when on contact 4; 25° F.

above the room temperature when on contact 5; 30° F. above the room temperature when on contact 6.

Lid On and Canopy Off.—5° to 10° F. above the room temperature when on contact 2; 10° F. above the room temperature when on contact 3; 15° F. above the room temperature when on contact 4; 20° F. above the room temperature when on contact 5; 25° F. above the room temperature when on contact 6.

Lid and Canopy Off.—The temperature alongside of the infant under its blanket will average from 5° to 10° F. higher than the room temperature on contacts 2, 3, 4, 5 and 6 with a room temperature between 70 to 80° F.

As in all other care of these infants individualization should be the watchword and only by a careful observation of weather and temperature changes can the best results be obtained.

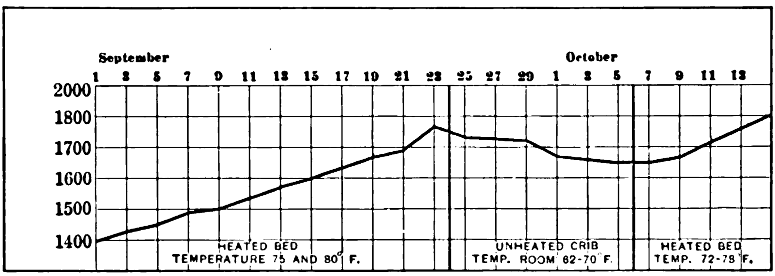


Fig. 142.—Showing variations in weight curve of an infant while in and out of a heated bed. The diet was unchanged between the dates September 19 and October 13.

The maintenance of desired temperature for a given case resolves itself into a very simple problem if the above facts relating to the recording of the lid thermometer is borne in mind, in that the only variable factors are the room temperature and air currents. The former in most hospital rooms will average from 65 to 75° F. throughout most of the day, and the ventilation of the room can easily be controlled. In most cases it is only necessary to change the rheostat one or two points at the extremes of the day, as at midnight when the temperature is likely to fall, and in the morning when the hospital temperature is again more uniform.

We require recording of the temperature of the room and bed at 6 A.M., 12 M., 6 P.M. and 12 P.M. In order to insure safety from extreme heat currents and extreme fluctuations in room temperature the point of the rheostat ward temperatures and humidity should also be recorded at these times (Fig. 88).

The degree of temperature to be maintained within the bed must of necessity vary with the individual infant and be dependent in part at least upon the infant's physical development. We rarely find it necessary to maintain a temperature above 90° F. for more than a limited number of hours even in extreme cases. In small infants it may be necessary to hold the temperature between 85 and 90° F. for several days. Most infants, after a few days, do best in a temperature ranging between 75° and 80° F., depending upon their development. An average of 76° to 78° F. will answer the latter needs of the better developed infants. It may be stated that moderate fluctuations of 3 to 5° F. in the temperature in the bed during the course of the day have little detrimental influence on the infant's progress. Marked fluctuations are extremely dangerous (Fig. 142).

Ventilation.—Ventilation within the bed is maintained automatically when the bed is heated. This is due to the fact that the air in the center of the bed is cooler than at the side walls, thereby causing the cooler air to pass into the bed at its center, then to flow to the floor, along the floor, to the side walls and then up and out at the sides. The direction of the air currents within the bed has a double advantage in that the infant receives the renewed fresh air for breathing while it is surrounded by the warmed air.

Humidity.—Excessive drying of the air is prevented by the constant circulation through the bed of the free air of the room and by evaporation from a flat basin, containing baked porous clay (as used in water filters), over which water is poured to allow of evaporation. This is placed on the floor of the bed immediately under the baby basket. Varying with the degrees of temperature to be maintained within the bed, it is necessary once daily to supply from 8 to 16 ounces of water to replace that lost through evaporation.

Dangers.—The dangers in the use of any heated bed which must at all times be avoided to insure success are:

1. *Overheating and Refrigeration.*—Reading and recording of the room temperature, the rheostat contact and the bed thermometer at regular intervals throughout the day will furnish the necessary data to avoid these dangers.

2. *Water Hunger.*—Fluids must be supplied to an amount not less than one-sixth to one-eighth of the infant's body weight every twenty-four hours as early as possible following birth. (See Feeding, p. 181.)

It is, therefore, necessary to control the temperature, ventilation and humidity of the bed, and to keep a careful supervision of the feeding, more particularly the fluid intake. Respiration must also be carefully watched in order to detect cyanosis and asphyxia sufficiently early to save the infant. This requires that these infants be observed day and night.

No attempt should be made to prevent heat loss entirely by keeping the air surrounding the infant at anywhere near its body temperature. Leaving an infant in such an environment would soon result in heat stagnation with resulting symptoms of heat stroke which is early evidenced by restlessness, rapid respiration and dry skin.

The bed temperature should be lowered gradually but steadily until it reaches 72° F. The best method of judging the infant's external temperature requirements is by taking the rectum temperature at stated intervals. The infant should be graduated from the incubator as soon as its general condition permits. It should then be kept in a clean, well-ventilated room, in which the temperature can be stabilized at about 70° F. The average time that a higher surrounding temperature will be indicated will vary between one and eight weeks and the hospital stay from two to ten weeks. "Mothering," in the form of exercise, and massage are essential to every premature once its physical condition permits handling. The same is true of needs for the strictest attention to its personal hygiene.

The infant should be discharged to its home as soon as possible for several reasons: (1) In a good home environment it will receive more individual care than in a general hospital; (2) the interest of the mother in the child must be maintained; (3) placing the infant at the breast is the best way of maintaining the mother's milk supply, if the breasts are still actively secreting; (4) in order to prevent "hospitalism" due to lack of "mothering" and a tendency to secondary infections.

The bed must be kept scrupulously clean.

The infant's bedding should be of such material that it can be destroyed when contaminated by vomit and excreta. Feathers are not practical. Untarred jute can be used for this purpose. The mattress should be covered by a heavy pad to prevent soiling.

All contact with infected cases and attendants must be avoided.

All visitors are best excluded.

The conservation of heat must be begun immediately after birth.

The infant must be properly dressed; its head as well as its body should be protected.

The body temperature of the infant should not be allowed to go lower than 97° nor above 98.6° F. Daily fluctuations greater than 1.5° F. are dangerous.

The general care and feeding should receive the most careful attention.

Above all else all care administered to the premature should tend to individualization.

HOME-MADE HEATED BEDS.

Emergency Equipment.—As many of the cases must be cared for in the home and in most instances without time or facilities to properly equip a nursery, every physician should have some definite ideas on the construction of a bed which will meet exigencies of the individual case. We have already spoken of the general care and equipment of a nursery unit in the home. A number of practical emergency beds have been described, the specifications of a few of which will be given more in detail.

A *small wash basket* well padded inside and outside by quilting, into which is fitted a removable platform about 4 inches above the padded floor of the basket, makes a fair emergency bed. Beneath the platform in the floor of the basket hot-water bottles or bags are placed which must be refilled from time to time. The removal of the bags for refilling, which should be three or more in number and which are to be filled at different times, is facilitated by cutting an opening along the lower outer wall of the basket through which the water bags can be removed at will without disturbing the infant. A box can be built for this purpose to even better advantage.

Whether a box or basket is used it must be provided with some form of cover for three-quarters of its upper surface. This may be accomplished by using a heavy blanket or building a lid to fit.

In such a bed the infant must be provided with proper clothes as previously described to prevent undue heat loss.

This bed should be kept in a well-ventilated warm room, the temperature of which should range between 68° and 72° F. if possible.

Brown[26] describes the following practical home-made heated bed:

Take a 24-inch wicker clothes basket and pad the bottom with non-absorbent cotton to a depth of 8 inches. On top of this cotton fit a sheet of oilcloth, sewing the edges through the sides of the basket. On the oilcloth lay a double layer of white flannel and on the flannel a napkin of absorbent cotton. Take half a dozen of 12-ounce citrate of magnesia bottles with wire and rubber corks and cover them with flannel. These bottles are filled with water at 110° F. and hung on the inside walls of this basket. A thermometer hung inside should register a temperature from 80° to 90° F. all the time. At night an oilcloth is spread over the foot half of the top of the basket.

Electric-heating pads, protected by copper jackets, have been in use by the writer over a period of several years, and offer a valuable means of meeting emergency requirements. They are also valuable for use in the home where the temperature cannot be well regulated after infants leave the hospital station. Electric-heating pads have lost their popularity through the danger of fire following short circuit due to broken wires, and through the poor quality of the thermostat attachments of some of the pads. To avoid the danger of fire from short circuits in electric-heating pads, a copper receptacle is used, 16 inches long, 13 inches wide and $1\frac{1}{4}$ inches high, into which a 12 x 15-inch heating pad is laid. To allow of a maximum radiation from the lid or upper surface of the same, the floor and sides are lined with asbestos sheeting, while the lid is not lined. The cord passes through a small rubber insulator at the side to prevent contact with the metal and injury to the cord. This simple device can be used temporarily in wards and homes where better facilities for the care of this class of infants are lacking. It is to be placed in the bottom of a basket or crib, under the mattress or pillow (Fig. 143).

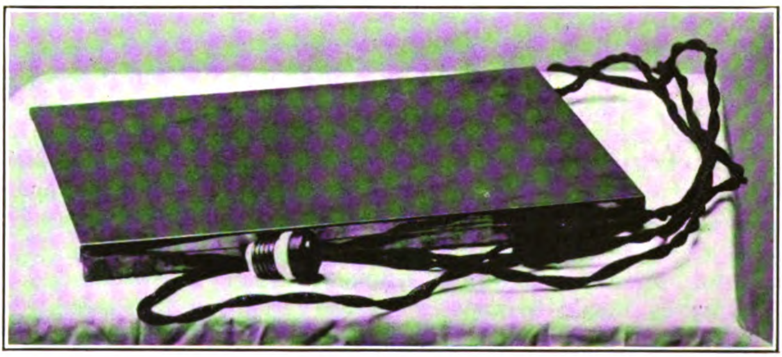


Fig. 143.—Copper receptacle containing pad.

Litzenberg[27] has described a practical bed for home or hospital use for which the specifications are as follows. A box 24 inches long, 20 inches high, 18 inches wide. Eight inches from the bottom is a false bottom dividing the box into two chambers, the heating apparatus being in the smaller lower chamber and the baby in the upper one. The false bottom is the support for the bed of the baby and does not cover the whole bottom of the box, a space of 4 inches being left at one end for the circulation of hot air. The top of the box may be fixed on hinges, or to slide, which is better. There is a pane of glass in the top so that the baby may be watched, and there are two ventilating holes near the end of the cover opposite the place where the hot air enters. An ordinary pillow is laid on the false bottom for the bed. The incubator is heated by bottles filled with very hot water and placed in the lower chamber through a small door in the side of the chamber. Fresh air enters this door, passes over the hot bottles, is heated and ascends by way of the 6-inch space at the end of the box to the baby's chamber and out through the ventilating holes in the top, giving a constant supply of warm fresh air. A thermometer is placed in the incubator beside the baby, or better beneath the first fold of the enveloping blanket.

By watching this thermometer a fairly constant temperature can be maintained by frequent filling of the bottles. This is the method usually advised for heating. He has further devised a hot-air radiator made of ordinary 3-inch eaves-spouting. A temperature not varying 2 to 3° F., he states, is easy to maintain. The heat from the chimney of an ordinary lamp enters the spout radiator through an elbow 1 inch or 2 above the chimney. This elbow curves upward toward the box, which it enters by way of a hole in one end of the chamber where the spout divides into two parts to give more radiating surface. These two branches unite at the other end of the box, and the warm air passes out through a hole in the end without entering the chamber in which the infant is placed. Thus, the products of combustion in the lamp do not enter to injure the baby. The air for the baby enters by the door in the side of the box described before, and is heated by the hot pipes and ascends to the baby. Over the discharging end of the radiator is a cap with a hole 1 inch in diameter. This discharge hole being very small, keeps the hot air from rushing through without radiating its heat. The box can easily be made collapsible so that the whole thing can be slipped under the seat of a buggy and be set up complete in less than five minutes.

Specifications.—Board 1 inch thick, 10 inches wide and 21 feet long. Cut six pieces 2 feet long and one piece 18 inches long. On four of the 2-foot pieces nail a small cleat, the full width of the board, 1 inch from each end. Eight inches from the edge of two of the 2-foot pieces nail a cleat parallel to the long way of the piece and on the same side of the piece as the small cleat. In the center of the 18-inch piece cut a hole 3.25 inches in diameter. Now set the pieces with the long cleat on edge. The cleats will face each other and be 8 inches from the floor. Place one of the 18-inch pieces with the hole in it against the end cleats of the two side pieces and fasten them there by means of two hooks screwed into the short edge of the side pieces, the hook fastening in a staple or ring in the 18-inch piece. Fasten the other end in the same manner and then place the radiator in the two holes at the end. Now lay two of the 18-inch pieces on the long cleat, and you have the false bottom or bed support. The other 2-foot pieces with the cleats are now put together with the two remaining 18-inch pieces with hooks arranged as described, and when put together they are placed on top of the first set and securely fastened, thus making a box 18 x 20 x 24 inches. There now remain two of the 2-foot pieces which are fastened together with several cleats to make a top. A hole about 8 x 10 inches is cut near one end of the top for a window for observing the child, and still nearer the end are cut two ventilating holes about 2 inches in diameter.

ROOM INCUBATORS.

The room incubator or so-called *giant incubator* claiming to have all the advantages of little incubators without their inconveniences, was constructed in Lyon, France, for the first time in 1886 by H. Colrat. It consisted of a room 12 feet long and 8 feet wide. Its two main features were an attempt to hold a constant temperature, and a system of aëration permitting of renewing the air. It was, no doubt, a good innovation at that time.

In 1900 Arnaud, of Turin, introduced the hot-air room, and it found followers in other cities.

The incubator chambers built by Escherich and Pfaundler in Graz and Vienna, Brauer in Marburg and Langstein in the Kaiserin Auguste-Victoria House in Berlin are all of the same type with added improvements. They are completely enclosed cells of glass and metal construction, having sufficient room for two or more infant beds, obtaining the air from outside and are provided with automatic regulation of gas heating, ventilation and humidification. Between the cells and the nursery room there is a small space providing against cooling of the infant when the door of the incubator is opened. It is possible to change the clothing of the infants, to bathe them and to feed them in the room (Fig. 144).

Several clinics in the United States have built such rooms, among them Washington University, of St. Louis, and Michael Reese Hospital, Chicago.[1]

[1] *Specification of Warm Room, Washington University, St. Louis.*—The fresh air from outside is driven in by an electric fan. It then passes over a system of steam coils enclosed in a closed steel cabinet, and is moistened by steam escaping through a small valve within the cabinet. Thermostat contact is used. The air makes a complete circuit of the heated chamber and passes into a closed shaft and enters the room through small registers located in the shaft. The used air leaves the room through the out-going shafts of the ventilating system. The room itself is insulated and the windows double. A thermometer and hygrometer are placed near a window and are visible from the corridor. A nurse records the room temperature and humidity on a chart every hour. To hold the room temperature at approximately 80° F. and humidity at 55° F., regular inspection is necessary because of the unsatisfactory working of the thermostat.

Specifications of Warm Room,. University of California, San Francisco.—The room is 9 by 11 feet, with an 11-foot ceiling. It accommodates five infants, the cribs being separated by glass partitions 4 feet high, extending 2½ feet out from the side wall. Entrance is through double doors so placed that the outer one is closed before

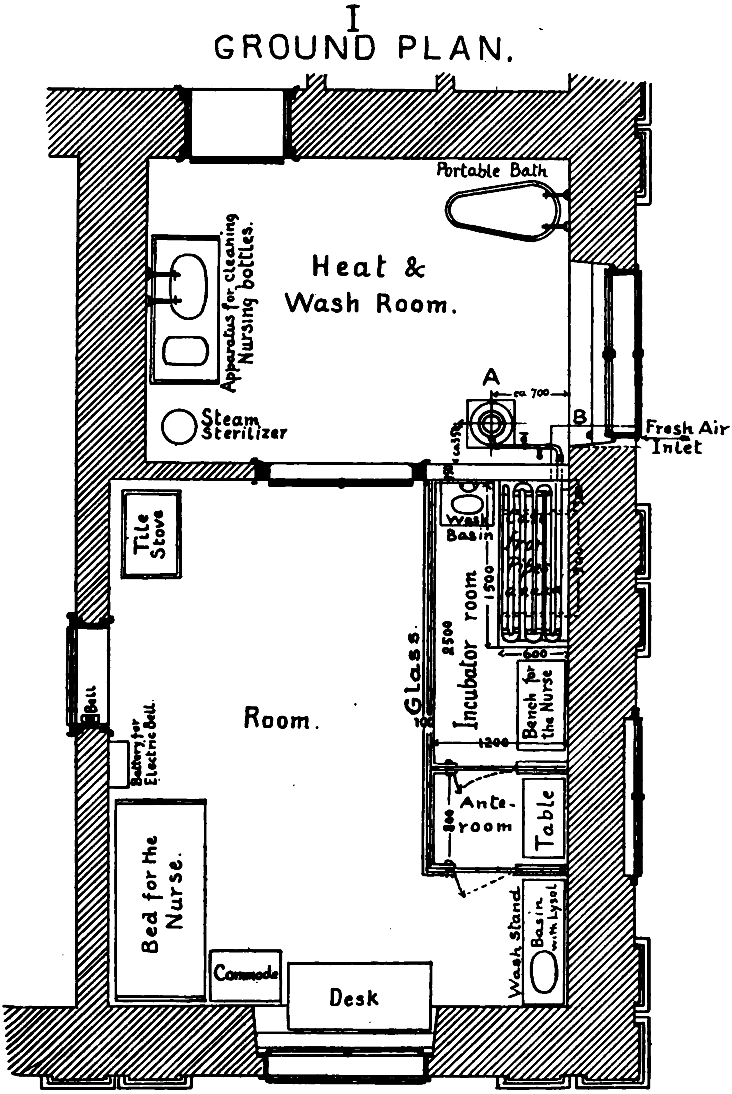


FIG. 144.—Incubator room. Escherich-Pfaundler system.

the inner one is opened. A large window at the opposite end admits ample light, and a closet is provided for gowns and supplies. Furniture consists of a dressing table, chair and scales. The ventilating system delivers 200 cubic feet of air per minute, thus affording a complete change of air every five minutes. A thermostat and hygrometer maintain constant temperature and humidity of the entering air. The room is kept at 80° F. The infants arrive from the delivery room and are placed beside the radiator, additional heat being furnished by hot-water bottles if

Some of the greater difficulties to be overcome are the automatic heat regulation, the cost of equipment and maintenance when only a small number of children are to be cared for, the distress caused the attendants when they are required to remain for a considerable period in the heated room, and most important the difficulty encountered in individualizing the care of premature infants of different ages and stages of development.

The disadvantages of the larger incubator room have led in many clinics to their being discarded, among others that at the Michael Reese Hospital. More practical is a room provided with special facilities for heating and ventilation which can be used in conjunction with individual heated beds.

In its primitive form an incubator room may be provided in a private home by heating the room to 75° to 80° F., at the same time making provision for moistening the air sufficiently by hanging wet clothes near the stoves or radiators. It is, of course, impossible to maintain a constant temperature and ventilation by such crude means, so that in conjunction with a more moderately

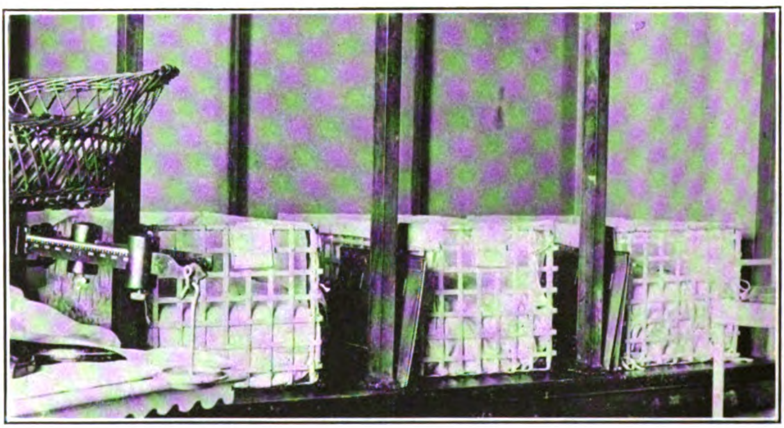


Fig. 145.—Heated room used as station for the care of premature infants (University of California, San Francisco, California). Showing individual cubicles, built on a shelf running across the room.

necessary. It is seldom necessary to keep them here longer than twenty-four hours, after which they maintain a fairly steady body temperature with the room at 80° F. and no additional heat in the crib.

Michael Reese Hospital Incubator Room.—The specifications of this room are as follows. It is 16½ feet long by 10 feet wide, with a plate-glass partition cutting off a vestibule 6 by 10, in which the nurse may stay out of the greater heat of the incubator room proper. The incubator room itself is a cube 10 feet each way, lined with cork, felt and asbestos, besides the other normal coverings. There is a double window with separate double transom, and exhaust fan and an intake fan.

heated room, 70° to 75° F., some type of individual bed for the further protection of the infant should be used.

A modification of the incubator room and doing away with some of its disadvantages has been installed in the Sloan Maternity Hospital, in New York, described by Dr. E. B. Cragin. It is possessed of many valuable features, such as filtered air, the absorption of air by an electric fan and the serial electric light heating. The disadvantages are to be found in the inability to individualize the infant care and the necessity for constant supervision (Fig.146).

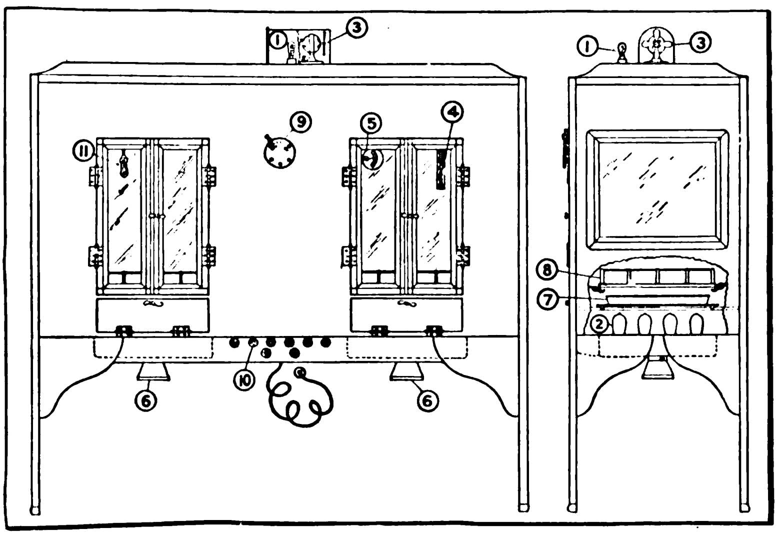


FIG. 146.—The Sloan Hospital incubator.

Selection of Method for Supplying Artificial Heat.—This must of necessity depend upon the facilities at hand. Every community should be supplied with the proper equipment for handling these infants. Such a station should be a part of every maternity department. In institutions more especially designed for the care of infants, a more elaborate station should be supplied and wet nurses should be available.

TRANSPORTATION INCUBATORS.

Probably the most important epoch in the life of the premature infant is that period between birth and the institution of some proper method for the prevention of refrigeration. It is the experience of all institutions receiving such infants that many of them are lost through carelessness in protecting them during the first hours after birth. The figures of Ylppö are illuminating on this point.

TEMPERATURE ON ADMISSION AND MORTALITY OF PREMATURE INFANTS.

	Temperature 37 to 35 degrees. Died within the first month. Per cent.	Temperature 28½ degrees. Died within the first month. Per cent.
Group I:		
600 to 1000 gm.	66.6	100.0
Group II:		
1001 to 1500 gm.	37.5	85.7
Group III:		
1501 to 2000 gm.	21.05	60.0
Group IV:		
2001 to 2500 gm.	5.88	20.0

By looking at the above table we may easily come to a one-sided conclusion that the mortality of the premature infants is in the first place influenced by the more or less severe initial cooling

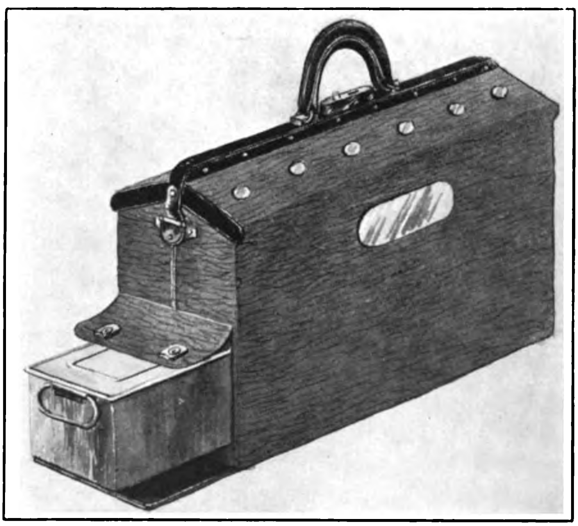


Fig. 147.—Obstetrical bag with false bottom designed by the author as a transportation incubator.

occurring after birth, and that therefore the mortality of the premature infants may be markedly reduced by painstaking care in preservation of heat.

A *simple transportation incubator* can be made by the employment of an ordinary obstetrical bag with a false bottom. Hot-water bags or bottles can be carried in the lower compartment, and the infant in the bag proper. It is only necessary to make a sufficient number of ½-inch holes beneath the handle for ventilation. These should be reinforced by a metal rim so that they cannot collapse and cut off the supply of air. The fresh-air supply can be controlled by a metal slide covering these holes or by using corks. Eight larger holes should be made in the floor of the satchel, so that the heat can pass from the lower compartment into the upper compartment. These are best made close to the edge at the ends, so that they will be less likely to be covered by the bedding (Fig. 147).

FIG. 148.—DeLee transportation incubator.

The *De Lee incubator ambulance* is a minature incubator with a circulating hot-water system heated from the outside by an alcohol lamp. It is well ventilated and lighted by electricity. It is 21 inches long, 11 inches wide and 11 inches high (Fig. 148).

Welde[25] has described a transportation incubator which is rather simple in construction (Figs. 149 and 150).

Heat is supplied by a thermophor or hot water bottles placed in lower compartment.

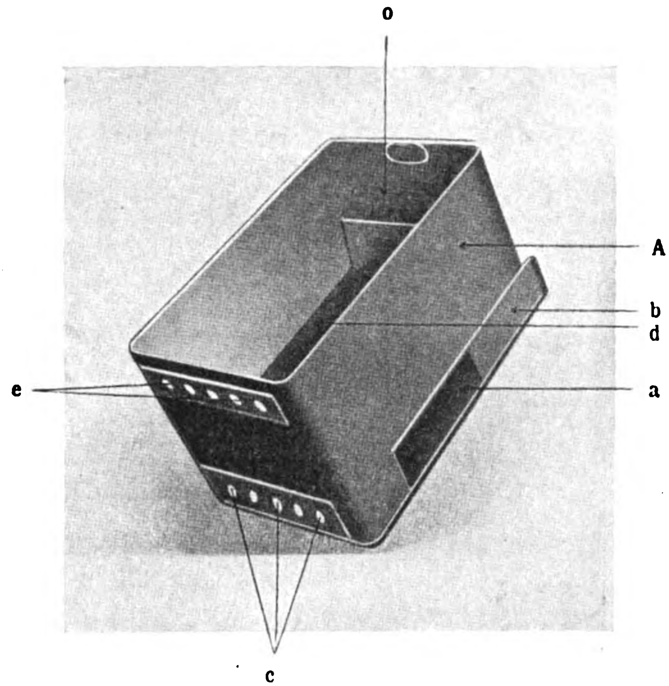


FIG. 149.—Inner case. *o*, air compartment; *A*, inner metal box; *b*, sliding door; *d*, removable upper wall of the double floor; *c*, lower air holes; *e*, upper air holes; *a*, double floor.

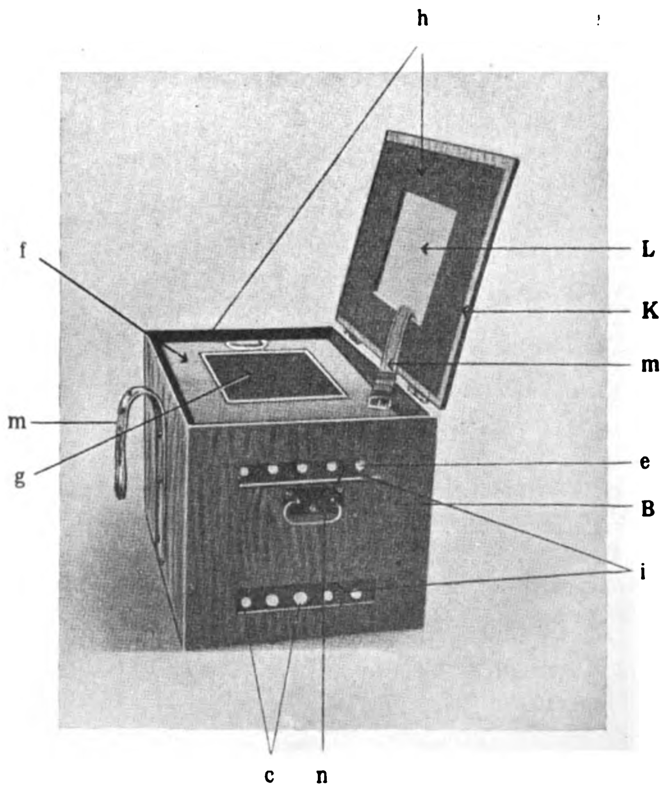


FIG. 150.—Outer case. *B*, outer wood case; *K*, lid of wooden case; *L*, glass window; *h*, felt lining; *m*, carrying strap; *e*, upper air holes; *n*, handles; *c*, lower air holes; *g*, windows in inner case; *f*, removable lid of inner case.

BIBLIOGRAPHY.

1. Denucé: Jour. de méd. de Bordeaux, December, 1857.
2. Clementovsky: Oesterr. Jahrb. f. Padiatrik, 1873, **3,** 30.
3. Pasquad: La couveuse artificielle chez les nouveau-nés, Thèse de Paris, 1899.
4. Credé: Arch. f. Gynäk., 1884, **24,** 128.
5. Winckel: Centralbl. f. Gynäk., 1882, Nr. 1 bis 3.
6. Tarnier: Sie wurde 1881 in der Maternite aufgestellt und wohl zuerst in einer Arbeit von Auvard (Arch. de Tocologie, October, 1883) beschrieben.
7. Auvard: Arch. de Tocologie, 1883, p. 577.
8. Berthod: La couveuse et le gavage a la maternite de Paris, Thèse de Paris, 1887.
9. Hearson: Zit. nach Czerny-Keller, **1,** 673.
10. Eustache: Jour. d sc. méd. de Lille, 1885.
11. Diffre: Montpelier méd., 1896.
12. Lion: Zit. nach Czerny-Keller, **1,** 673.
13. Pajot: Zit. nach Budin, Manuel pratique d'allaitement, Paris, 1905.
14. Budin: Le Nourrisson, Paris, 1900.
15. Hutinel and Delestre: Revue mens. des mal. de l'enfance, 1899, **17,** 529.
16. Finkelstein: Lehrbuch der Säuglingskrankheiten, Berlin, 1905, II Teil, s. 32.
17. Rommel: München. med. Wchnschr., 1900, Nr. 11.
18. Polanos: München. med. Wchnschr., 1903, Nr. 35, s. 1498.
19. Escherich and Pfaundler, L.: Mitt. d. Vereins d. Arzte in Steiermark, 1900, Nr. 3.
20. Colerat: Société des sciences médicales de Lyon, 1896.
21. Arnaud: La Sala incubatrice; Contributo allo studio della fisiopatologia dei neonati prematuri, Torino, 1900.
22. Cragin, E. B.: Jour. Am. Med. Assn., No. 11, **63,** 947.
23. Ylppö: Ztschr. f. Kinderheilkunde.
24. DeLee: Obstetrics for Nurses, W. B. Saunders Co., Philadelphia, 1919.
25. Welde: Jahrb. f. Kinderheilkunde, 1912, **75,** 551.
26. Brown, Alan: Arch. Pediat., No. 8, **34,** 609.
27. Litzenberg: J. Minnesota Med. Assn., Minneapolis, **28,** 87, 91, 1908.

PART III.

GENERAL DISEASES.

CHAPTER X.

DISEASES OF THE RESPIRATORY TRACT.

ASPHYXIA NEONATORUM.

ASPHYXIA is a condition produced by any interference with oxygenation of the blood. It may be present at birth or it may occur subsequent to that event. Asphyxia in the new born is characterized by an absence or feebleness of respiration which is accompanied by cardiac action, showing that life is present. Asphyxia occurring after birth is most frequently due to prematurity or to congenital weakness.

During intra-uterine life the wants of the fetus are supplied from the maternal blood stream through the placenta, oxygen being present in sufficient quantities so that respiration is unnecessary. Normally this state of *apnea* terminates at birth and respiration is established, in all probability, as a result of the decreasing supply of oxygen derived from the placental circulation, and of the increasing amount of carbon dioxide which is accumulating in the fetal blood, and upon which the stimulation of the medullary center depends, the fetus passing from a condition of apnea to one of *dyspnea*. At the same time the heart action is slowed and the blood-pressure raised, both the result of the carbon-dioxide stimulation. Since the respiratory center is only with difficulty affected in the premature, it is sluggish in responding to the increase of carbon dioxide, and if this increase is slow in appearance respiration may not be attempted at all. Cutaneous stimulation from extraneous influences in the outer world also plays a part in the establishment of primary respiration.

Etiology.—Asphyxia of the premature newborn may be due to any one of the many causes which interfere with the oxygen supply of the fetus either before or during labor. These causes may be listed as follows:

1. Abnormally strong and prolonged labor pains. Such lengthy and oft-repeated uterine contractions may interfere with the exchange of gases in the placenta or with the oxygen-laden umbilical blood stream.

2. Unequal pressure exerted by the uterus after the membranes have ruptured, if applied to the placenta or the cord, may prevent oxygen reaching the fetus.

3. Compression or tearing of the placenta.

4. Twisting or tearing of the cord or its compression while in the uterine cavity or when prolapsed.

5. Premature separation of the placenta, either complete or partial.

6. Slow labor the result of weak pains or contracted pelvis.

7. Premature respirations resulting from attempts at version or from the application of forceps. In this instance the aspiration of amniotic fluid or vaginal mucus usually forms the obstruction to respiration.

8. Maternal anemia or asphyxia from renal, cardiac or pulmonary affections, diseases of the blood, eclampsia or other forms of toxemia such as are produced by morphine, chloroform, etc.

In the extra-uterine variety of asphyxia the infant attempts respiration after birth but is unsuccessful. The reason for this failure may be due to the presence of mucus, blood or liquor amnii in the respiratory passages; to the presence of anomalies of the heart or lungs; to injuries of the skull; to the pressure from cerebral hemorrhage; or to inherent constitutional weakness or weakness of the respiratory muscles. In the premature infant the respiratory center is but insufficiently developed, the respiratory muscles are weak and the lungs are in a state bordering more or less closely upon fetal atelectasis. All of these factors favor the development of asphyxia, and the younger the fetal age of the infant at the time of birth, the more pronounced are these conditions, though it must be remembered that not all premature infants are debilitated (see Atelectasis).

Cerebral pressure from injuries of the skull or from intracranial hemorrhage causes anemia of the medulla and consequently prevents stimulation of the respiratory center with resulting lack of respiratory activity, or with stimulation of the vagus with excessive slowing of the pulse, which interferes with the exchange of gases through the placenta or the lungs.

Our present belief is that the asphyxia occurring immediately after birth is due to oxygen deficiency and to paralysis of the respiratory center by overloading the blood with carbon dioxide. The presence of atelectasis and pulmonary congestion and edema favors the development of this state, which is so frequent in prematures and leads to general acidosis. Ylppö demonstrated in living premature infants alkalinity of the blood lower than that ever found in the blood of adults. Conditions are thus favorable for excessive acidification of the organism, not only by carbon dioxide but also by the other acid products of metabolism. Because of the abnormal reaction of the blood the irritability of the respiratory center is early reduced, leading to asphyxial attacks. In addition, it must be borne in mind that the frequency of cerebral and spinal hemorrhages in the smaller prematures will explain asphyxial attacks occurring in the first two or three months of life. Finally, traumatic lesions of the respiratory center may, in themselves, lead to disturbances in respiration and to interference with oxygen intake.

Morbid Anatomy.—Examination of the body of a premature new-born infant, dead of asphyxia, shows besides the evidences of prematurity, marked congestion of the internal organs. The right heart, sinuses of the dura and the great vessels are filled with blood. The brain and the organs in the thoracic and abdominal cavities are congested and edematous. Small hemorrhages are found in the pleura, pericardium, peritoneum, liver, kidneys, adrenals and retina. Occasionally effusions are seen in the serous cavities. In the lung areas of aërated tissue are seen along with areas of atelectasis, and the trachea and bronchi may be found filled with mucus or amniotic fluid. Edema of the extremities and scrotum may be present. Extravasations of blood are found in the skin and mucous membranes as well as in the internal organs.

Symptoms.—The strong premature infant at birth behaves much as does the full-term healthy child; it breathes deeply, utters a more or less vigorous cry, and the skin which at first is of a purplish hue rapidly becomes pink. If asphyxia exists two sets of symptoms may present themselves, depending upon the variety of asphyxia, *asphyxia livida* or *asphyxia pallida.*

In asphyxia livida or asphyxia of the first degree the skin has a reddish-blue or bluish tinge, the face is swollen, the eyes protrude somewhat and the conjunctivæ are injected. The extremities remain passive though the muscles retain their tonicity or are even hypertonic; the heart beats strongly and the apex-beat is often apparent to the eye; the vessels of the cord are filled with blood and pulsate; the respiratory efforts may be absent or shallow and infrequent. These infants can be roused and made to cry, respirations being established after suitable measures of resuscitation have been used.

In asphyxia pallida, or asphyxia of the second degree, the vasomotor center is overstimulated by the excess of carbon dioxide in the blood and this overstimulation causes contraction of the peripheral vessels with venous engorgement of the deeper vessels, thus further overloading the heart. The face is of a waxy pallor, the visible mucous surfaces are cyanosed, the muscle tone is lost and the extremities hang lax. The reflex irritability is lost; there is no attempt at respiration or at the most very feeble efforts; the pulsations of the heart are weak and either fast or slow, and the pulsations in the cord are absent or only weakly perceptible. The distinguishing feature that separates this condition from asphyxia livida is the lack of muscle tone in the pallid form, these infants having a corpse-like appearance and only the presence of the heart action and the few feeble respiratory gasps show that the infant is not dead.

Further Course.—If an asphyxiated infant is revived it frequently remains somewhat apathetic, cries very little and does not nurse well, requiring artificial aid in obtaining nourishment. In the stronger infants, however, this condition tends to clear up, so that in a few days the cry is vigorous, the movements active and the ability to nurse is good. In the weakling, whether premature or full-term, such improvement is much slower. The poorly developed respiratory mechanism results in superficial and irregular breathing and the existence of areas of atelectasis tends to delay development of the lung. These weak infants may have breathed spontaneously at birth though not enough to have dilated the alveoli of the lungs to a sufficient degree and as a result repeated attacks of cyanosis occur. These attacks of cyanosis are accompanied by a condition of apnea which lasts a moment or longer, during which the infant ceases to breathe entirely. These attacks appear without warning and may be very frequent in the weaker infants during the first two weeks of life, and are evidently the result of lowered irritability of the respiratory center. The outlook for the infant in these spells is not good, despite the fact that treatment is undertaken, because they are an indication of inherent weakness in the individual. In those cases which are to recover, these attacks of cyanosis become less and less severe and less frequent.

The after-life of these infants may be affected to some extent as the persistence of a degree of atelectasis renders them less resistant to infection.

Sequelæ.—Cerebral symptoms that develop later are not at all infrequent in children asphyxiated at birth and probably depend upon cerebral sclerosis secondary to minute intracranial hemorrhages. Developmental cerebral anomalies or injuries may, however, be primary causes of asphyxia and may later be evidenced by motor and psychic disturbances.

Diagnosis.—Asphyxia must be differentiated from hemorrhage of meningeal or cerebral origin occurring during prolonged or abnormal labor or after the application of forceps. The symptoms of a slight hemorrhage resemble those of asphyxia, the breathing being very superficial with frequent lapses into stupor. Convulsions occasionally occur and the pulse may be slow or fast. Continued slow pulse with the occurrence of coma and convulsions speak strongly for a cerebral hemorrhage, especially after a prolonged labor or the application of forceps. The differentiation is extremely difficult during the first days of life in premature and weak infants and death frequently results before the etiological factor is ascertained. Delmas[1] recommends lumbar puncture as a diagnostic and therapeutic measure.

Prognosis.—The outlook for strong prematures suffering from asphyxia livida is good, the majority recovering under proper treatment. In the weaklings it is always grave. In asphyxia pallida the prognosis is bad, the infant invariably succumbing if left to itself. If the heart action improves while attempts at resuscitation are being made it is a favorable sign. Endeavors to revive the infant should be kept up until the heart ceases to beat. At all times undue violence must be avoided, all attempts at resuscitation being applied gently and at regular intervals to avoid visceral injury. If cerebral hemorrhage is combined with asphyxia the outlook is very poor.

The cause of death in asphyxia may be a recurrence of the asphyxial attacks, lowered irritability of the respiratory center, atelectasis of the lung or blocking of the air passages by inspired foreign matter or cardiac failure.

Treatment.—The treatment of asphyxia is concerned with clearing the respiratory passages and supplying oxygen to the tissues. In the milder cases the finger is gently introduced into the pharynx, or the throat stroked downward, while the child is held in an inverted position, sufficient to clear out the obstruction to respiration. In the cases of asphyxia livida there is usually mucus in the trachea or bronchi, and this can frequently be removed sufficiently to allow of respiratory activity by inverting the infant and introducing a catheter as far as the upper opening of the larynx. Only in the larger infants is it possible to pass the catheter into the larynx. Suction is made with the lips and the mucus is drawn into the catheter. Occasionally it is necessary to repeat this maneuver several times. The dangers of a syphilitic infection are to be remembered.

Once the passages are cleared of mucus the reflex stimulation

[1] Le Progrès médical, 1912, **40**, 88–89.

of respiration by external irritation is attempted. In the milder cases the back and buttocks of the suspended child are gently slapped, cool (90° F.) water is sprinkled over the body, or the latter is rubbed with a warm cloth. In the severer cases the child is immersed in hot water at a temperature of 40.5° C. (105° F.) for a few minutes and then in a cool bath for an instant. The warm bath relieves the vasoconstrictor spasm and the overloaded heart, the blood being brought to the surface. Weak mustard baths, warm enemata and careful compression of the chest are all advocated.

In the severest cases cutaneous stimulation is not sufficient and it becomes necessary to resort to artificial respiration.

Insufflation has dangers, especially for the premature infant whose pulmonary tissue is very delicate. If the lung is torn emphysema follows and only a slight tear is necessary because of the very poorly developed state of the elastic tissue in the lung of the premature. On this account it is best to use some method by which the amount of air to be forced into the lungs may be measured. The capacity of the lungs being about 30 cc, the use of a thin rubber bulb of a capacity smaller than this would obviate the risk of tearing the lung tissue. The difficulty of entering the trachea of these small premature infants must be kept in mind.

The choice of the method to be used in inducing artificial respiration depends upon the severity of the asphyxia. There is no use wasting time in spanking the back or making traction on the tongue in the severer cases. In the lighter forms the simpler measures usually suffice, but in asphyxia pallida more energetic measures must be practised. First the air passages are cleared and then Prochownik's method is used for thirty seconds. If this is unsuccessful the tracheal catheter is inserted with great care and the lungs dilated with air.

The treatment of secondary asphyxial attacks consists in the use of warm baths, oxygen insufflations and artificial respiration. The oxygen tank should be kept at the side of the infant's bed and either continuous or intermittent showers of oxygen given in the attempt to ward off cyanotic attacks (see Cyanosis).

The intracutaneous injection of oxygen with an aspirator has been recommended in the treatment of asphyxia by Delmas.[1] He advises injecting from 30 to 60 cc beneath the skin, from which region it is readily absorbed with beneficial effect. In the opinion of the author such injections, because of the considerable trauma and shock, might result disastrously in the treatment of premature infants.

Reanimation of asphyxiated infants by the insufflation method

[1] La médecine infantile, 1912, **16**, 210.

of Meltzer and Auer is, according to Plauchu, quite practicable and efficient. In this method a current of air, directed as far as the tracheal bifurcation through a small catheter, ventilates the lungs sufficiently to oxygenate the blood even if no respiratory movements occur. The necessary apparatus consists of a rubber bulb, a small mercury manometer and a No. 12 (French scale) rubber catheter. A rod of soft copper is placed in the lumen of the catheter to give it the proper shape for introduction and the catheter itself is marked with transverse lines at 8, 10 and 12 cm. from the tip, indicating the distance from the mouth to the bifurcation of the trachea in a 2000-, 3000- and 4000-gm. child, respectively.

The method of the procedure is as follows: With the little finger or a small gauze sponge in the hold of a forceps any mucus in the infant's throat is removed and the child is then wrapped in a blanket and placed with the neck slightly overextended. The index finger of the left hand is introduced as far as the upper border of the larynx, finding the soft opening of the glottis. The catheter is introduced by the right hand between the tongue and the palmar surface of the left index finger into the laryngeal opening. When it has reached the proper distance the copper rod is removed, the insufflation apparatus attached and air injected with the bulb the pressure not exceeding 10 or 15 mm. of mercury.

The insufflation may be continued as long as needed. Soon the child appears less relaxed and the heart tones become stronger and more regular and respiratory movements begin.

In infants weighing under 2000 gm. the larynx and trachea are passed only with great difficulty because of their small diameter, and the dangers of secondary infection due to trauma of the tissue is great.

The use of the pulmotor or lung motor, several modifications of which are on the market, is not to be recommended in treating the asphyxia of premature infants, because of the danger of rupture of the delicate pulmonary tissue.

CYANOSIS.

Of all functions of the premature infant, that of respiration is usually the least developed at birth, evidencing to a marked degree the general lack of development of the central nervous system. Failure on the part of the respiratory apparatus to respond in a sufficient manner to the needs of the infant is the most frequent cause of symptoms of the gravest nature in these weaklings and indeed not seldom of death itself.

The underlying factors in the production of cyanosis may be divided into inherent and extraneous. The *inherent* causes of cyanosis are:

1. Lack of development of the central nervous system, especially of the respiratory center.

2. Weakness of the general musculature and softness of the ribs.

3. Persistence of fetal atelectasis which tends to delay development of the lungs.

4. Congenital malformations of the heart or great vessels or myocardial asthenia.

5. Malformations of the respiratory tract or of the diaphragm.

6. Diseases or compression of the air passages.

7. Injuries of the skull or cerebral hemorrhage.

8. Obstruction to nasal breathing.

9. A birth weight below 1200 gm. These infants almost invariably suffer from attacks of cyanosis.

10. Cooling of the body is given as a cause by Budin, but many infants have a temperature of 95° F. or even 93° F. without the occurrence of cyanosis.

11. Elevation of the body temperature to more than 102° F. is given by Zahorsky as a cause.

In the premature infant the causes among the above which are chiefly operative in the production of the characteristic attacks of cyanosis are the weak respiratory muscles, the softness of the ribs, the underdevelopment of the centers of respiration and the presence of fetal atelectasis.

Involvement of the heart is ordinarily of secondary occurrence, the diminished amount of oxygen in the blood resulting in a slowing and weakening of the heart's action. The atelectasis which is so frequently present, tends to hinder the closure of the foramen ovale and the ductus Botalli and these defects in turn predispose to cyanosis.

The *extraneous* causes include:

1. The aspiration of food or vomitus into the larynx or trachea. The lack of development of the pharyngeal and laryngeal reflexes is responsible for the food reaching the air passages and the lack of reflex cough prevents its being ejected. Pneumonia not infrequently follows the aspiration of such foreign particles.

2. Distention of the stomach from overfeeding. This is one of the most common causes of cyanosis and death in premature infants. This leads to interference with the action of the diaphragm.

3. Meteorism, due to gastric and intestinal stasis.

4. Attempts at drinking are often followed by cyanosis, either the direct result of the mechanical prevention of respiration or secondarily through the lessened oxygen content of the blood,

resulting in a lack of stimulation of the respiratory centers (von Reuss).

5. Undernourishment is strongly advanced by Budin as a causative of cyanosis, and he has shown that with increased feeding these attacks stop.

6. An insufficient supply of water.

7. The occurrence of a local or general infection.

Symptoms.— Oftentimes, without apparent cause, attacks of cyanosis appear with frequency during the first few weeks of the life of the premature or weakly infant. Usually without warning the respirations, which have previously been superficial and irregular, become still weaker and then cease entirely for a minute or longer, somewhat resembling the Cheyne-Stokes' type of breathing. Accompanying the apnea is a deep cyanosis which gradually disappears as breathing is resumed. Not infrequently, if immediate steps to restore the respiratory activity to something like the normal are not taken, the infant dies; in other cases breathing is spontaneously resumed and the attack passes off, leaving the infant more or less prostrated. Care must be taken in pronouncing it dead before examination for heart sounds. In a few hours or days cyanosis recurs, the attacks gradually increasing in length and severity despite treatment, until death occurs; or they become less frequent until they cease entirely.

Occasionally the attacks are preceded or accompanied by convulsions. Generalized edema sometimes develops.

Diagnosis.—From congenital cyanosis due to other causes, or acute affections of the respiratory tract with cyanosis, these attacks are differentiated by the history or other evidence of premature birth, and the frequently accompanying cyanotic edema, the respiratory weakness, absence of the normal vesicular breathing, particularly over the bases and the tendency to a subnormal temperature.

Prognosis.—The prognosis of cyanosis in the premature infant varies directly with the severity of the attacks which in turn are more or less directly dependent upon the fetal age and physiological development, the ability of the infant to maintain its body temperature, the quality of the food and the ease with which the infant digests it.

In no other condition to which these infants are subject is the previous training and experience of the attending nurse in the care and handling of this class of cases, of such vast importance.

Treatment.—A premature infant must be carefully watched for signs of cyanosis, otherwise it may be found dead in bed. Should an attack occur while the child is being fed, the proceeding must be stopped and efforts made to restore respiration. The first thing to do is to ascertain if there is any obstruction in the upper respiratory passages. Should inspired food or vomitus be present, an effort must be made to dislodge these particles. Inserting the little finger into the pharynx while the child is in an inverted position, often serves to clear out the respiratory tube, and then slight cutaneous stimulation by pinching, friction or gentle slapping is often enough to reinitiate breathing.

Again, exhaustion of the infant may be solely responsible for the cyanosis. In these cases artificial respiration should be tried, the chest being rhythmically pressed upon, or one of the other methods of artificial respiration may be tried. Simple compression of the chest may be tried without removal from the incubator or bed, though removal will be found more serviceable generally.

The use of oxygen is of value in quickly reducing the degree of asphyxia after breathing is once established, although it will not of itself restore that function. A tank should be kept by the infant's bed and any sign of approaching asphyxia should be the indication for the generous shower of oxygen. The continued use of the gas when properly applied is advocated as a valuable measure in the checking of attacks. About 80 to 100 bubbles of oxygen gas from a partially protected mask should escape in close proximity to the infant's mouth.

Aromatic spirits of ammonia in one-half to two drop doses, diluted, is of value, and nitroglycerin, one drop of a 1:1000 solution may be placed on the tongue. The use of camphor, caffein, atropin or other respiratory stimulants hypodermically does not offer much practical help.

Sprinkling the baby with cool water will occasionally stimulate respiration and as this means is always at hand it should be kept in mind.

Infants suffering from repeated attacks of cyanosis should be immersed in a hot bath at a temperature of 102° to 105° F., and subjected to gentle friction, more especially along the spinal column. The infant may be kept in the bath for from a few seconds to several minutes, when it should again be placed in its warmed bed, avoiding all chilling. The efficiency of the bath may be increased by the addition of a teaspoonful of mustard to the gallon of water. Care should be taken to prevent aspiration of the bath water, or its entrance into the eyes, and the danger of infection of the umbilical cord, although not great must be borne in mind. The bath may be repeated as indicated.

In our own experience the warm mustard bath has proven one of the most satisfactory means of overcoming prolonged attacks. It is quite evident that the facilities for preparing the bath must be prearranged and great care taken to keep it at an even temperature throughout the immersion. To facilitate handling and to prevent undue manipulation during the cyanotic attacks the infant should be wrapped in a blanket.

It cannot be too strongly emphasized that the manipulations used to relieve the cyanosis should be the minimum necessary to accomplish the result as cyanotic infants react poorly to trauma. After an attack is over the infant should be placed in a warm bed or bath in order to overcome the tendency to a reduction of temperature by the previous manipulations. Afterward it is also necessary to supervise carefully the feeding in order that two things may be accomplished: (1) That the occurrence of further attacks of cyanosis due to mechanical obstruction by food may be prevented; and (2) that the nutrition of these weaklings may be immediately bettered and thus the cyanosis indirectly controlled.

The prevention of cyanosis may be aided in several ways. The too rapid taking of food or distention of the stomach by overfeeding must be avoided (see Feedings). Underfeeding in cases where too frequent feeding is undesirable can be avoided by catheter feeding at longer intervals, although the maximum food quantities must be carefully ascertained by starting with minimum feedings, carefully increased according to the infant's tolerance. Catheter feeding is not well borne by all infants and may occasionally in itself induce cyanosis. The strength of the infant should be built up as rapidly as possible, and the temperature of the body should be maintained by the use of the heated bed inasmuch as a lowering of the body temperature not only favors the development of cyanotic attacks, but makes them more severe when they do occur. The use of oxygen may be of value.

Insufficient supply of fluids should be avoided by the administration of water where the fluid intake is less than one-sixth of the body weight during the twenty-four hours.

Meteorism may be relieved by small quantities of low saline enemata, part of which may be left in the rectum to good advantage where the fluid intake per mouth is insufficient to meet the body requirements.

Gastric lavage must occasionally be resorted to as a means of last resort in overdistention of the stomach with paresis of its walls and should be performed with the infant's head at a lower level than the body to prevent aspiration of stomach contents, as passage of the tube very frequently results in vomiting. This procedure is always associated with great danger during a cyanotic attack. Occasionally the gas can be relieved by simple passage of the catheter into the stomach with slight pressure from without over the epigastric region.

DISEASES OF THE NASAL PASSAGES.

THE anatomy of the nasal passages of the new-born infant is such that comparatively small degrees of swelling or accumulations of mucus are sufficient to lead to obstruction of nasal respiration, thereby interfering with the act of nursing. When during sleep the tongue falls backward, thus occluding the passage between the pillars, attacks of cyanosis and dyspnea may result.

A nasal discharge present at birth or developing within the first two or three weeks of life should lead to a search for evidence of congenital lues. When the syphilitic infection is sufficiently virulent to cause premature labor the external manifestations usually appear early.

Other sources of infection of the nasal mucosa can be found in the passage of the child through the maternal birth canal, from the bath water or by direct transmission from an individual suffering from a similar infection. The organisms which may be concerned include the various pyogenic bacteria, the pneumococcus, colon bacillus, influenza bacillus and, less frequently, the gonococcus. The diphtheria bacillus is frequently seen as a cause in institutional infants.

Obstruction of the posterior nares is occasionally seen in the new-born premature, the opening being closed by either a membranous or a bony partition. When bilateral it favors respiratory obstruction and may be the direct cause of attacks of asphyxia and cyanosis. Nasal infections may threaten the infant by extension to the lower respiratory passages, while generalized septic processes may have their origin in a nasal infection.

Treatment.—The *prophylaxis* of nasal infections requires that if the mother is suffering from any infection of the respiratory tract every effort should be made to prevent infection of the offspring. Coughing or direct breathing into the infant's face should be avoided and care taken that infectious material is not carried from one to the other on the hands, or by means of infected articles. The same precautions must be taken in case an attendant is the one infected. A vaginal discharge from the mother at the time of delivery requires that the infant's nose should be cleaned thoroughly but carefully with a cotton pledget after birth. Lowered resistance due to chilling of the infant is an important etiological factor and must be avoided.

It may become necessary to remove crust formation with instillations of normal salt or weak alkaline solutions. This must be carefully performed to avoid forcing the infection into the Eustachian tube and air passages, small quantities only being used. Pledgets of cotton saturated with 1:1000 solution of adrenalin chloride if placed within the nostril will temporarily relieve the nasal swelling. As curative agents some of the organic silver salts in weak solutions may be mentioned. The use of an ointment of the yellow oxide of mercury (ung. hydrarg. ox. flav.) of 0.5 or 1 per cent strength will be found of value. A portion the size of a small pea should be introduced into the anterior nares and the nostril then gently massaged in order to force the ointment as far into the nose as possible. In cases of syphilitic or diphtheritic infections specific treatment must be instituted.

The breast-feeding of these infants with rhinitis offers some difficulty because of the interference with respiration which accompanies obstructions of the nose. Nursing at the breast is likely to be a difficult matter under the most ideal circumstances when the infant is as weak as many prematures are, and if added to this is an inability to breathe while sucking and swallowing. The difficulties are so great at times, even in infants approaching maturity, that it becomes necessary to feed expressed milk per catheter. This method of food administration must be instituted before the infant shows the results of inanition.

CONGENITAL STRIDORS.

Congenital Laryngeal Stridor.—In the premature infant the presence of a stridor may go unnoticed for several days because of the weak inspiratory effort, in contradistinction to the full-term infant in which it is usually interpreted in the first days of life. It must, therefore, be expected that the croaking or crowing sound will be much more feeble than is usually heard in these cases. The stridor usually disappears when the infant is deeply asleep, which in the premature is the greater part of its day. Unless there is a considerable stenosis, the infant shows no distress and cyanosis is absent. During intense crying and in the presence of cyanotic attacks, signs of obstruction may become evident. It is often difficult to make an exact diagnosis in these cases because of the dangers of direct transillumination of the larynx in these small infants and the diagnosis is often dependent on the ability of the clinician to exclude other causes of inspiratory dyspnea. Two cases examined by the author at autopsy have in both instances shown similar findings, in that there was a marked narrowing of the lumen of the larynx with thickening of the aryepiglottic folds and deformity of the epiglottis. Nervous disturbances due to arrested development in the cortical centers with resulting disturbed coördination of the act of respiration may occasionally be a causative factor. Arrest of development affecting the center for the recurrent nerve may also be another factor.

Treatment.—There is usually a spontaneous functional correction. The prophylactic care should consist in the prevention of respiratory infections.

Stridor Thymicus.—The frequency of true thymic enlargement with direct tracheal pressure has undoubtedly been exaggerated by incomplete diagnosis. The most frequent sign proving stenosis of the upper air passages is the presence of suprasternal retraction. In the premature the tendency of the entire chest wall to collapse with each inspiration may be mistaken for this sign and easily lead to an error in diagnosis. The author has seen two such cases which were verified by palpation of a soft tumor mass in the fossa

Fig. 151.—Specimen of thymus gland weighing 40 gm., and resulting in thymic death.

jugularis during expiration as well as by percussion with flatness to the right and left of the manubrium and substantiated by roentgen-ray findings. In both cases the stridor developed shortly after birth and disappeared spontaneously with diminution in size of the thymus gland, both infants making an uneventful recovery. The author has also seen a case of congenital thymus stridor in a luetic infant which died on the sixth day. At autopsy the thymus gland weighed 40 gm. and was the seat of numerous miliary abscesses.

Prognosis.—The prognosis varies with the cause of enlargement. The benign forms which disappear spontaneously undoubtedly belong to the vascular type. While the number of sudden deaths due to causes associated either directly or indirectly with the thymus gland are less frequent than one would be led to believe from a review of the literature, they do occur and must be given proper consideration. These deaths may be due to mechanical compression of the trachea by an enlarged gland either due to a true hypertrophy or hemorrhage within the gland, or death may be caused by hypersecretion of the gland. Syphilitic changes in the thymus with miliary abscess formation has already been described as a cause of death under Thymic Stridor.

Treatment.—An expectant attitude should be adopted in the absence of marked signs of stenosis. In the presence of congenital lues, specific treatment should be instituted. The only other form of treatment which offers any degree of encouragement is that of roentgen-ray exposure in the hope of creating rapid involution, with the development of moderate fibrosis. Friedlander[1] describes prompt results, stating that dyspnea is lessened even after the first treatment.

It is self-evident that the exposure of premature infants to the roentgen ray, unless carefully guarded, may be disastrous not alone in the too rapid atrophy of the thymus gland which is so necessary to the growing organism, but also to the thyroid and other parenchymatous organs as well as the danger of skin irritation.

In our wards at Michael Reese Hospital, Dr. R. A. Arens makes use of the following treatment:

8 inch spark gap.
3 mm. aluminum filter.
10 inch S.T.D. (Skin Target Distance).
5 M.A. (milliampères).
4 minutes exposure.

The treatment is guided entirely by the clinical course. Frequently one or two treatments are sufficient.

Stridor from Other Causes.—These are most commonly due to congenital enlargement of the thyroid gland which is usually of the vascular type and disappears spontaneously without treatment. Congenital tracheal stenosis, deformities of the mouth, congenital tumors of the mouth and acute inflammatory conditions of the upper respiratory passages may be further causes.

SUFFOCATION FROM EXTERNAL CAUSES.

Death from suffocation due to external causes such as faulty position (infant on face), obstruction of breathing by clothing or

[1] Am. Jour. Dis. Child., **6**, 38.

overlying on the part of the parent have been responsible for the loss of many premature and weakly infants. These have often been described as instances of thymic death. Death from these causes is far less common in full-term, robust new-born infants, as the latter possess the ability to change the position of the head when threatened with suffocation.

AFFECTIONS OF THE BRONCHI AND OF THE LUNGS.

1. Congenital Anomalies.

Fetal Bronchiectasis.—Fetal bronchiectasis is a rare condition of the new born which affects the whole or only part of one lung. *Universal bronchiectasis* is the result of hydremic degeneration of an entire bronchus, the lung structure being replaced by cystic formations which contain a serous fluid in which are found ciliated epithelium and nuclei.

The *teleangiectatic bronchiectasis* is characterized by the formation either of individual cysts or less often of multilocular sacs, the walls of the cysts being lined with several layers of cuboidal epithelium.

A third variety known as *atelectatic bronchiectasis* is due usually to lack of development of certain portions of the lung which later become cirrhotic from pressure from a bronchus. (Birnbaum.[1])

Hypoplasia and Hyperplasia.—These malformations are due either to lack of sufficient development or to excessive development. In *hypoplasia* a small airless structure is found in place of one lung. Since the healthy lung in such cases usually grows into the empty half of the thoracic cavity, deformity results, the thoracic wall not developing well over the healthy lung. The same is true of primary hypertrophy, which consists either in abnormal size or in formation of supernumerary lobes (Birnbaum.)

Diagnosis.—On account of the equalizing growth of the healthy lung the diagnosis is possible only in the presence of deadening of the sounds over one-half of the thorax. This is much more important in the new born than in older children, since in the latter the above-mentioned physical finding is much more significant of an infiltration or exudation (von Reuss.[2]) Roentgen-ray studies are of assistance in localizing the lesion although they may not determine the type of lesion.

Bronchiectasis in the new born is not accompanied by any distinctive symptoms. In the premature their existence increases the respiratory handicap under which these infants labor, and if

[1] Congenital Diseases of the Fetus, Springer, Berlin, 1909.
[2] Diseases of the New Born, Springer, Berlin, 1914.

they are extensive, death with symptoms of asphyxia usually occurs soon after birth. The occurrence of inflammatory complications makes the outlook still graver.

Atelectasis.—Atelectasis is also spoken of as acquired asphyxia though it may be congenital as it is a persistence of the fetal state in all or in part of the lung. In the congenital variety the lung is not entirely expanded at birth, while in the acquired form collapse of the previously expanded lung occurs. The congenital variety is seen chiefly in the premature and debilitated, either due to a developmental anomaly or insufficient strength on the part of the respiratory muscles to inflate the lungs. The acquired form is most frequently due to obstruction of the bronchi or alveoli by intrathoracic exudates, diaphragmatic hernias and deformities of the spinal column.

Atelectasis is to a degree physiological during the first few days after birth, gradually disappearing with increasing strength. When associated with asphyxia at birth, it is often overcome entirely by the means used to revive the infant.

In the weak the methods used are not enough to cause complete expansion of the lung and collapsed areas persist, the soft and yielding thoracic wall and poorly developed respiratory muscles of the premature both favoring the non-expansion.

The cyanosis which is so frequently seen in those suffering from atelectasis may be directly due to the aspiration of food into the larynx, the absence of the pharyngeal and laryngeal reflexes favoring this. Mechanical interference with respiration during the act of drinking may also result in cyanotic attacks; interference with the action of the diaphragm through overdistention of the stomach (Birk[1]) and according to Budin[2] underfeeding, may both be responsible for cyanosis in the premature. (See Cyanosis.)

Pulmonary atelectasis also occurs after cerebral hemorrhage, due to injury to the respiratory center, and is characterized by small respiratory excursions and slight exchange of gases. In the premature the irritability of the respiratory center is low *a priori*, while in those suffering from natal asphyxia it is lowered by the asphyxia.

Pathology.—The anterior portions of the lungs are most frequently the portions expanded, the paravertebral parts being atelectatic. Peiser[3] showed that in organs hardened *in situ* the central portion near the hilus was also atelectatic, while the apices and borders were usually expanded, the expanded portions often being emphysematous (Holt[4]). When death occurred early a

[1] Leitfaden der Säuglingskrankheiten, Marcus and Webers, Bahn, 1914.
[2] The Nursling, Caxton Pub. Co., London, 1907.
[3] Jahrb. f. Kinderh., 1908, **67**, 589.
[4] Diseases of Infancy and Childhood, D. Appleton & Co., New York, 1913.

large portion of the lung was usually not inflated. The left lung is usually more atelectatic than the right. The involved parts are rich in blood and thus form sites of predilection for inflammatory processes.

Hemorrhages and edema frequently complicate this condition, which is made worse by the deficient heart action. These hemorrhages are chiefly in the region of the hilus. In vessel injuries of lesser degree there is no bleeding, only edematous extravasation.

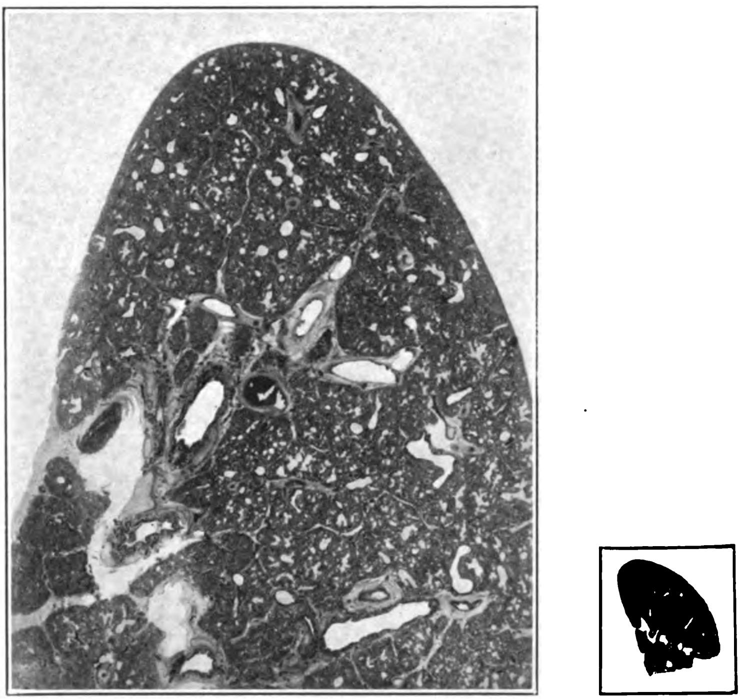


FIG. 152.—Congenital atelectasia. Four-fifths normal size and magnification of 6 diameters.

The atelectatic lung is of brownish-red color, does not crepitate, is very vascular and shows the lobular outline on the surface. Usually both lungs are affected to the same degree. The heart frequently shows the presence of a patent foramen ovale or other congenital lesion, the liver and spleen are often congested and the latter may be enlarged (Fig. 152).

Symptoms.—Very frequently the subjects of atelectasis give a history of asphyxia at birth; in others there may have been nothing to attract attention to the lungs. Some are noticeably quiet, cry weakly, sleep much and their voices are feeble. The temperature is usually below the normal; occasionally there is some edema of the extremities or slight puffiness of the face, while the breathing is shallow and often irregular. The gain in weight is slight or absent, and the children remain small and delicate with poor circulation. At any time there may develop attacks of cyanosis, which occur without warning and which may be fatal in a few hours, often being preceded by convulsions. These attacks may occur as late as ten or twelve weeks after birth.

Physical Signs.—*Inspection.*—The breathing is shallow, often irregular and at times almost ceases.

Palpation.—This is negative unless râles are plentiful, when fremitus may be felt. Vocal fremitus is absent.

Percussion.—There is usually resonance over the entire chest and only posteriorly may diminished resonance be demonstrable. The collapsed areas are surrounded by areas which are overdistended with air and thus resonance is not much interfered with. Small areas of collapse give no dullness at all. If only one lung is involved a difference can usually be made out.

Auscultation.—The breath sounds are very feeble and the expiratory sound in particular may be nearly inaudible. The sounds may be rather harsher than normal, but are rarely bronchial in character. The most marked physical sign is the presence of crepitant râles, the so-called atelectatic crepitation, which are best heard usually over the bases when the infant, by flagellation or otherwise, is induced to take a deep inspiration.

Diagnosis.—The diagnosis of atelectasis is to be made more from the symptoms, the shallow breathing, the stupor, the asphyxial attacks and the debilitated condition of the infant than from the physical signs which are likely to be ambiguous and not well defined.

If the respiratory efforts of the infant are sufficient to supply the needed amount of oxygen the dangers from asphyxia disappear and only the inflammatory complications which may arise in the uninflated lung threaten its well being. Any atelectatic area may become inflated (bronchopneumonic) and thus areas of collapse and bronchopneumonia may be present in the same lung. Pneumonia in an atelectatic lung is not easy of recognition. The presence of crepitant and subcrepitant râles, impaired resonance and the absence of respiratory sounds, accompanied by dyspnea and ineffectual cough, all speak of an inflammatory condition. The percussion note may be vesiculo-tympanitic and auscultatory signs of consolidation, such as bronchial breathing and bronchophony may be inaudible because of the diminished respiratory excursion,

Differential Diagnosis.—A number of conditions must be considered in the differentiation of atelectasis, the more important of which are the following:

General debility with quantitative and qualitative lack of development attended with impaired respiratory cardiac and digestive functions. This is commonly associated with lack of development of the thoracic wall and a tendency to collapse on the part of the costal cartilages, and a poorly developed respiratory musculature.

Cerebral injury associated with hemorrhage is one of the most difficult pathological conditions to differentiate, because of the tendency toward involvement of the respiratory centers, more especially in basilar hemorrhages. A careful inquiry should be made for a history of opisthotonos and clonic contractions of the extremities or facial muscles.

Hyperplasia of the thymus and occasionally the thyroid gland, with associated stridulous respiration, retraction of the diaphragm and local physical findings must be differentiated. When the chin is brought down upon the chest respiration becomes more difficult and, in turn, is made easier if the head is bent back.

Aspiration of foreign matter or food with lack of expulsitory effort resulting in cyanosis may lead to error in diagnosis.

Underfeeding, with secondary asphyxia.

Congenital diaphragmatic hernia.

The differential diagnosis of this condition is based on the fact that the abdominal organs containing air enter the pleural cavity, thus giving rise to physical signs of pneumothorax. In addition, the following signs are presented: Respiratory movements on the affected side are absent or less marked than normal, and there is usually bulging of the thoracic wall on the same side; pectoral fremitus is slight or absent and the percussion note is deep and loud and in some cases tympanitic. Not infrequently succussion sounds can be elicited. The normal breath sounds are absent over the affected area and the heart is found displaced to the right. Moreover, these findings change with a change in the position of the patient. From the foregoing it will be seen that the findings of percussion and auscultation are very important but variable, as they depend entirely on the amount of air or semisolid material contained in the abdominal organs present in the pleural cavity.

Radiographic examination is of special value in differentiating atelectasis pulmonum, hyperplasia of the thymus and diaphragmatic hernia.

Prognosis.—This depends upon the degree of atelectasis which in turn usually depends upon the degree of debility of the child. When accompanied by attacks of asphyxia and cyanosis which appear with frequency during the first two weeks of life, the outlook is bad, despite the institution of proper treatment, as these attacks commonly result fatally. In favorable cases they become less frequent and finally cease. Pneumonia in atelectatic areas often leads to a fatal issue.

Infants who have suffered from congenital atelectasis may remain in delicate health for a long time, although many ultimately recover completely.

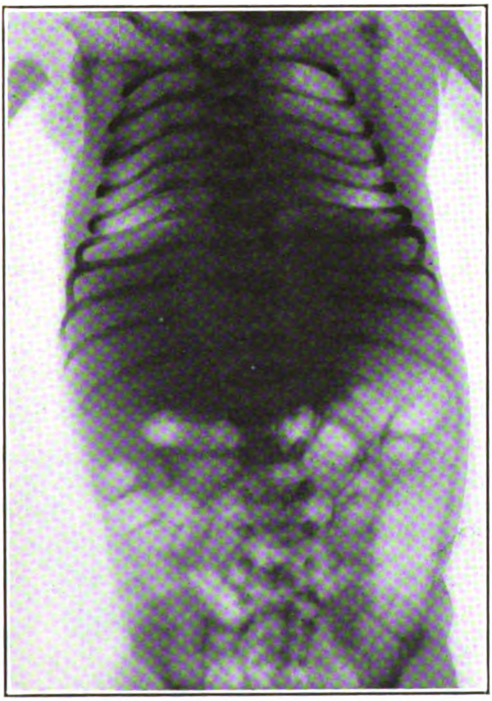


FIG. 153.—Diffuse congenital atelectasis.

Treatment.—The physical condition of these weaklings is oftentimes so precarious that undue roughness in the application of restorative measures can work infinitely more harm than they may do good, and so it must be remembered that the less the manipulation necessary to overcome the cyanotic attacks, the less is the danger of injuring the infant at this critical time either by overstimulation mechanically or by medication. The object of treatment is directed toward the expansion of the lungs through deep breathing. This is done by crying, and if the child does not cry strongly every day, it should be made to do so. In the mild cases cutaneous stimulation is sufficient, the child being very gently spanked thrice daily for fifteen or twenty times, thus tending to expand the collapsed portions of lung and to expel mucus from the bronchi. The mustard bath is made by adding one tablespoon of powdered mustard to one gallon of water at a temperature of 100° to 105° F. Alternate immersions in warm water with a

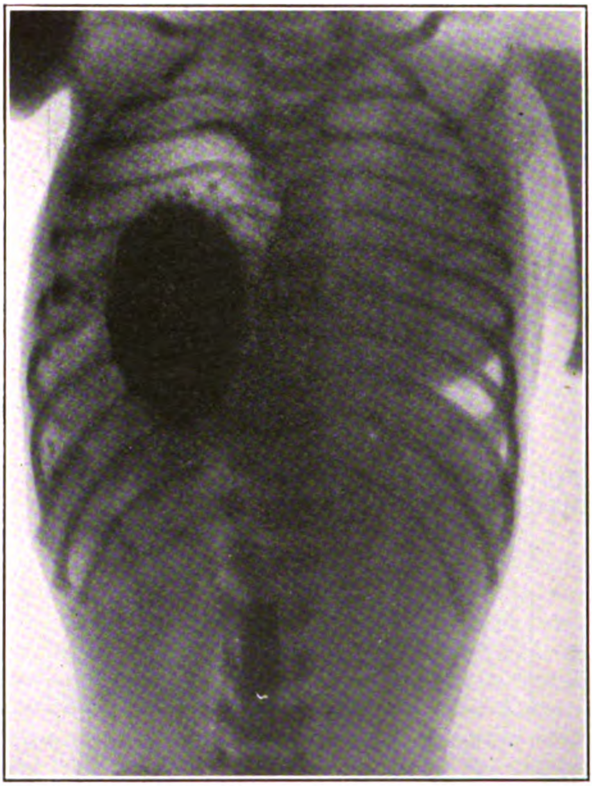


Fig. 154.—Incomplete diaphragmatic hernia (case of Dr. Irving Stein). Roentgenogram taken three and six hours after ingestion of bismuth. Stomach and bowel in chest.

temperature of 104° F. and cool water of 95° F. may be tried, always beginning and ending with the warm immersions. These may be repeated at intervals as indicated by the physical condition of the infant. The objects of the bath are the diversion of the blood from the lungs to the cutaneous vessels, and expansion of the collapsed areas. Expansion of the collapsed lungs is much easier during the first few days, the difficulty of doing this increasing proportionally with the length of time elapsing since birth.

The infant should not be allowed to lie quietly in one position, but its position must be changed frequently and the child picked up several times a day. Particularly where many infants are housed with but little individual attention atelectasis is seen most

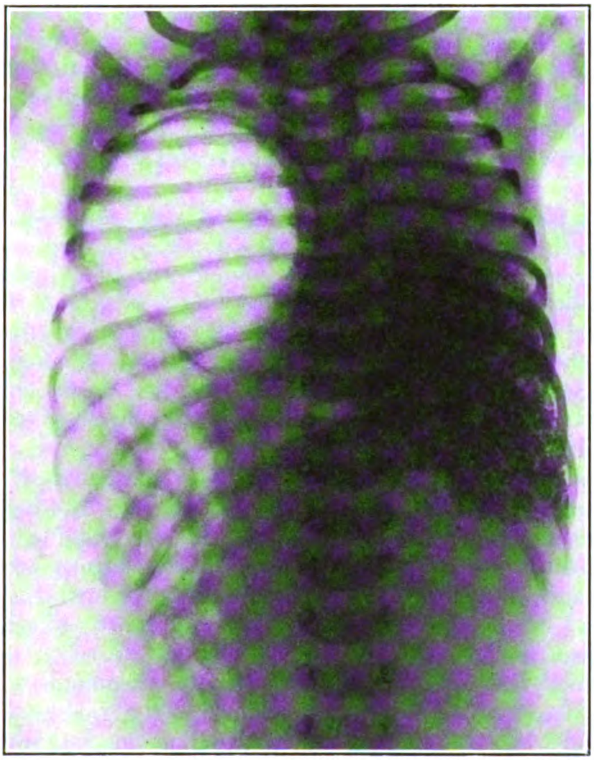


Fig. 155.—Incomplete diaphragmatic hernia (case of Dr. Irving Stein). Roentgenogram taken soon after death with postmortem injection of bismuth in the bronchi. Only lower lobe of right lung admitted the bismuth emulsion. The gas distention of the stomach and bowels here beautifully portrays the extent of eventration.

frequently. The further treatment should be similar to that advised for attacks of cyanosis.

As the temperature is so often subnormal, these children must be kept warm, either by being surrounded with hot-water bottles or else kept in some form of heated bed. The feeding of these children is an important problem (see chapter on Feeding). It is essential to increase the general nutrition in order to increase the function of the respiratory center and muscles. Aside from this, it is improbable that increased feeding as recommended by Budin is of any direct value as a therapeutic measure.

During attacks of asphyxia, oxygen inhalations are recommended, and are valuable when the infant can be made to inspire, a tank being kept in close proximity to the infant's bed. Other measures of resuscitation mentioned under asphyxia (see Cyanosis)—cutaneous stimulation and artificial respiration, or even the use of forcible means of inflating the lung with a catheter in the trachea—may be necessary but their danger must not be underestimated. The use of drugs hypodermatically, such as camphor, caffeine, atropin, etc., is not of much value. Aromatic spirits of ammonia in one-half to three-drop doses, well diluted, is worth trying.

CONGESTION AND INFLAMMATORY CHANGES OF THE LUNGS.

Congestion of the Lungs.—Congestion of the posterior lower portions of the lung most commonly results from long-continued rest without change of position, congenital or other anomalies of circulation. At first there may be extravasations of serum or blood in the alveoli and later, especially in the greater degree of congestion, tissue infiltration.

Clinically, the condition is manifested by disturbances of respiration, shallow breathing and asphyxial attacks. The impairment of resonance and auscultatory findings may be confounded with those of atelectasis or inflammations. The findings are usually bilateral and in dependent parts; these facts aid in the differential diagnosis. They develop post partum thereby differing from atelectasis and are primarily associated with fever.

Congenital Pneumonia.—That congenital pneumonia may exist seems to be well substantiated, although the number of cases reported in which the infection was hematogenous and transmitted by way of the placenta is small.

Infection of the fetus may also occur through infected amniotic fluid before labor. However, when it is the result of the aspiration of infectious material during the passage through the birth canal, these cases must be classed as extra-uterine pneumonia.

Post-natal Pneumonia.—**Etiology.**—Bronchitis during the first few days of life may be the result of aspiration of infectious material, or it may accompany a general septic infection. The fact that the vaginal secretion always contains microörganisms offers every opportunity for infection, should aspiration occur during the infant's passage through the birth canal. Infection of the bronchi may reach the child from an infected mother or attendant, or from a third person through the agency of feeding utensils, spoons, or other articles. Infections in the upper air passages may spread by direct extension to the deeper structure of the respiratory passages and there occasion a bronchitis or a bronchopneumonia.

Atelectatic areas, so common in the lungs of the premature or weakling, and the frequency of aspiration of food or vomitus in the debilitated favors the occurrence of pneumonic inflammation. The richness of the atelectatic portions of lung in blood and tissue fluids make them a most favorable medium for the multiplication of the invading bacteria.

The organisms found in the bronchopneumonias of early life are the pneumococcus and staphylococcus most commonly, less frequently the Bacillus coli, the streptococcus and the influenza bacillus.

Meyer[1] emphasizes the fact that the "grippe" with respiratory involvement may cause a surprisingly extensive infection.

Pathology.—In the majority of cases both lungs are involved, the parts most frequently affected being the lower posterior portions. The principal lesion is an inflammation of the walls of the bronchi, and the walls of the alveoli surrounding the bronchi. Microscopically before section there is often no visible evidence of consolidation, and seemingly all of the lung can be inflated. The walls of the bronchi and alveoli are thickened and infiltrated with round cells. The involved alveoli are filled with an exudate which is at first composed of desquamated epithelial cells and later of leukocytes. On section there are seen grayish-red or yellowish-gray areas which correspond to the cut bronchi and the surrounding peribronchitis. From the cut bronchi the fluid contents exude, composed of epithelium, pus cells and mucus. Many of the smaller bronchi become occluded by the excessive exudate and collapse of the contributory alveoli follows. The collapsed portions are depressed beneath the surface of the surrounding lung and are of a beefy-red color.

In some cases, particularly in those instances where the streptococcus is the causative organism, the inflammation may be of a hemorrhagic nature. In these cases the bloodvessels of the affected areas are deeply congested, the lung tissue is studded with small hemorrhagic patches whose size varies from that of the head of a pin to several centimeters in diameter, the latter being true infarcts. They are distinguished from the zone of congestion that surrounds them by their projecting above the surrounding tissues, their dark color and their durability. On section they are of triangular shape with the apex more or less deeply in the lung substance. They

[1] Ueber den Hospitalismus der Säuglinge, Berlin, 1913.

are seen particularly in the lower lobes. The mucous membrane of the large and small bronchi is the seat of a catarrhal inflammation with round-cell infiltration.

Death in the case of these prematures and weaklings is not always the result of the virulence of the invading organism but may be attributed rather to mechanical phenomena secondary to the involvement of the lung. Because of the excessive amount of exudate and intraparenchymatous hemorrhage the alveoli are filled with fluid, the bronchial ramifications are obstructed and the gaseous exchange limited or prevented almost entirely. Pneumonia, therefore, and particularly hemorrhagic bronchopneumonia kills the premature by asphyxia. In other instances death is the result of a true toxemia.

For contrast and comparison we have the recent investigations of Ylppö[1] who found that typical bronchopneumonic changes were very rarely observed in very young prematures.

The following table shows the frequency of lobular pneumonia in his series:

FREQUENCY OF BRONCHOPNEUMONIA IN PREMATURES.

Weight.		Death of the age of:					
		1 day.	2 days.	3 days.	4 to 15 days.	1 mo.	Older.
Under 1000 gm.	Number of sections.	14	9	2	6	2	1
	Bronchopneumonia in these	0	0	1	2	2	1
1000 to 1500 gm.	Number of sections.	24	18	9	6	10	14
	Bronchopneumonia in these	0	0	1	3	8	12
1501 to 2000 gm.	Number of sections.	10	1	8	3	3	16
	Bronchopneumonia in these	0	0	0	2	1	11
2001 to 2500 gm.	Number of sections.	2	1	1	1	2	12
	Bronchopneumonia in these	0	0	0	1	0	3

Ylppö's histological investigations showed that the bronchopneumonic areas in prematures were not at all as frequent as the bronchopneumonic areas in the full terms in the first days of life. Hess Thaysen[2] stated that in newly born infants dying in the first

[1] Ztschr. f. Kinderheilk., 1919, **20**, 212.
[2] Jahrb. f. Kinderheilk., 1914, **79**, 140.

three days of life there could be demonstrated small bronchopneumonic areas in 42 per cent of the cases. These changes are not due to aërogenous infection but to the aspiration of infected material from the mother during birth. Hochheim[1] showed that the vaginal secretion and amniotic fluid was aspirated and demonstrated the presence of foreign bodies, as squamous epithelium, fatty bodies, meconium and lanugo hairs in the lung alveoli.

Symptoms.—The onset is most often insidious in the weakly new born. At first there is noticed possibly a slight nasal discharge and a cough of varying severity. Soon increased frequency of respiration makes its appearance accompanied by dilatation of the alæ nasi. The cough in the more mature becomes worse and the respiration increases to 60 or 80 per minute. Now and again slight attacks of cyanosis occur, which in the severe cases are correspondingly more marked. There is great restlessness in older infants with inability to sleep, and the cyanosis becomes continuous. Convulsions occur with more or less frequency, while the temperature may be slightly elevated or may be subnormal even in the severest cases. There is a marked loss of weight, the stools become dyspeptic, and greenish with mucus and undigested particles. The prostration may be extreme.

There is often a singular lack of symptoms and the disease may go unrecognized. The respirations range from 40 to 60 or even 80 to 100 per minute, but are usually not labored, the pulse-rate is increased to 140, 160 or may be uncountable; the cough may be absent entirely, and there is often apathy and even deep stupor. The course in these infants is usually acute, either immediate improvement or death occurring.

The severity of these early symptoms is to be explained either on the basis of the sudden intense congestion of the small alveoli interfering with the bronchopulmonary apparatus almost as much as does consolidation, or their severity may be due to the intensity of the infection.

Physical Signs.—The usual physical findings of a bronchitis or bronchopneumonia are often lacking or only suggested in the pulmonary inflammation of the premature and debilitated, especially when the involved areas are small in size. This is due to the fact that the respiratory efforts are weak and their amplitude small. In addition it is often the atelectatic portion of the lung which is involved and if this is situated centrally the air may fail to gain access to it.

On inspection there is seen more or less marked dyspnea, with inspiratory retraction of the lower ribs; the face may be pale or

[1] Path. Anat. Arbeiten, Berlin, 1903 (Hirschwald).

cyanosed; cough if present is frequent, short and non-productive. Palpation may reveal nothing. Evidence of consolidation such as increased resistance may be entirely lacking.

Percussion may give indication of consolidation if impaired resonance or slight dulness is demonstrable, but this occurs only in the presence of massive involvement. Occasionally the note over the whole posterior chest may tend toward the tympanitic.

Auscultation usually offers the most reliable findings. The breath sounds are often entirely absent over collapsed areas or the respiratory sounds are weak and possibly higher pitched than normal. In other instances the breathing is exaggerated, and bronchial in character. Probably the most characteristic finding of pneumonia in these infants is the occurrence of fine sibilant or moist râles. These are often heard behind over the lower lobes and are the most distinctive sign of the disease. The voice sounds are, as a rule, unchanged.

Diagnosis.—Pneumonia may, in these infants, be easily confounded with atelectasis. If the premature infant is strong and possesses a loud cry, congenital atelectasis may be excluded; in the weak the latter condition is most commonly present and the physical signs of pneumonia are absent. It must be remembered that the two conditions may exist side by side in the same infant. It may be necessary to make the infant cry or breathe deeply by mechanical irritation in order to bring out the various abnormal sounds. A careful study of the history from birth may be of great assistance.

Prognosis.—If the inflammation complicates infection in the upper air passages, such as rhinitis or bronchitis, the outlook is better than in primary pulmonary infections. Mixed infections with the influenza bacillus, staphylococci and streptococci, offer a more serious prognosis than primary pneumococcus. Involvement of a large portion of both lungs or an extremely weakened condition of the infant, both militate strongly against recovery. The younger the infant the shorter the intra-uterine life the higher the mortality and when hypothermia exists death usually occurs soon. Cases which run their course with little or no temperature are usually fatal, probably because they occur in infants who are very feeble, of low vitality, with limited resistance to infection.

Treatment.—*Prophylaxis.*—The prevention of pneumonia in the premature requires that the little weaklings shall be protected against infection from every source. The lungs are most frequently the site of bacterial invasion, as there the organism finds a most favorable medium for its growth. In the adult there exist at the entrance to the respiratory tract defensive agents capable of stopping the invading bacteria but in the premature these defenses are absolutely rudimentary and consequently offer but slight impediment to the entrance of pathogenic germs (Delestre[1]).

The transmission of respiratory infections occurs by means of infected hands or other objects or through the medium of air. No one suffering from any infection of the nasal or respiratory passages should handle the infant. If the mother is affected with a coryza or bronchitis she should take care that her hands are not contaminated with nasal or bronchial secretions and that she does not breathe, or especially cough, in the face of the infant. A mask must be worn by the nurse or mother if she has a respiratory tract infection. In institutions where many babies are taken care of by one nurse, the hands of the attendant should be washed before the handling of each baby. Isolation of the premature should be practised if respiratory affections exist among the other members of the family, or in the common wards if the infant is in an institution. Only if the attendants are thoroughly trained in the principles of aseptic nursing is it safe to leave the infant in close proximity to others suffering from respiratory affections. These of all infections are hardest to prevent. All utensils should be individual and should be sterilized before use; feeders, spoons, glasses, nipples, bottles, stomach tubes, etc., must all be boiled before they touch the child or its food. The French insist on the restricted use of the incubator in the management of premature and weak infants when the closed type is used, and believe that their success in the handling of prematures depends upon the fact that they remove them from the closed incubator as soon as the body temperature reaches 37° C, and their vitality permits. As soon as these babies can be removed from the incubators they are kept in large, well-ventilated rooms, which are not overheated. They should be given the benefit of open air and sunshine as their development warrants. In favor of the open-air treatment is the fact that most of the late deaths occur during bad weather. The mortality drops with improved atmospheric conditions.

General Treatment.—The treatment of pneumonia is preëminently that of watchful expectancy, and overtreatment must be avoided, as these feeble infants are unable to withstand overmanipulation or stimulation. As a rule, pneumonia in robust infants is an acute self-limited disease, but in premature infants the course is apt to be somewhat subacute without the tendency to limitation. The indications in the treatment are to support the heart and conserve the strength. The feeding problem is difficult at any time in the premature and during an attack of pneumonia it becomes doubly difficult.

[1] Étude sur les infect. des prématures, Thèse de Paris, 1901.

The hygiene of pneumonia requires that the child receive plenty of fresh air, and to insure this in an incubator of the closed type is difficult. The use of the open type in part overcomes this difficulty. The position of the child should be changed frequently in order to obviate any tendency to hypostasis. If the sick infant has been housed in a closed incubator with questionable ventilation it should be removed to an open, well-heated room, and placed in a properly warmed crib or incubator bed. The prime indication is for the promotion of elimination and sufficient administration of inert fluid. Stimulation of the respiratory tract is best accomplished by mild counterirritation to the chest and the use of hot applications to the extremities. The use of drugs such as cardiac and respiratory stimulants is not to be regarded with favor but strychnine sulphate in $\frac{1}{500}$ grain (0.00012 gm.) doses, or atropine sulphate in $\frac{1}{2000}$ to $\frac{1}{3000}$ grain (0.00006 to 0.00002 gm.) doses given hypodermically may be of some help. The use of whisky or brandy is permissible in quantities varying from 3 to 10 drops every two or three hours depending upon the indications. Aromatic spirits of ammonia in 1 to 5-drop doses is one of the best stimulants at our command. Both the whisky and the ammonia should be given well diluted in at least 8 parts of water. In cases of emergency, of sudden heart failure or of weakness accompanying a sudden fall in temperature, the use of camphor-in-oil 2 to 10 minims to the dose given hypodermically will be found to be a rapidly acting, reliable heart and respiratory stimulant.

If the infant shows a marked rise in temperature the use of hydrotherapy may be considered. Temperatures up to 103° F. are well borne, and do not require interference. As a general thing the temperature tends to remain subnormal in these weaklings and cool or even tepid baths must be avoided and instead warm or hot mustard baths resorted to. Even if there is an excessive amount of fever, should it be accompanied by a cold surface, feeble pulse and shallow respirations, cold is contraindicated. The best hydrotherapeutic measure used for the reduction of an unduly high temperature is the tepid pack. The use of cold baths or packs is probably never justified in the premature or weak infant. The temperature of the tepid bath may range from 100° to 105° F., depending upon the condition of the child.

The treatment of *attacks of collapse with cyanosis,* which are so frequent in the atelectatic prematures, should be prompt. The infant should be immediately placed in a mustard bath (one teaspoonful of powdered mustard mixed with one gallon of tepid water being of sufficient strength), of about 102° to 106° F. together with gentle massage. Respiratory and cardiac stimulants may be needed. Oxygen should be administered continuously.

Disturbance of the nervous system, occasionally so prominent in older and stronger children, is not marked in the premature during a pneumonic process. When present mild hydrotherapy offers the best results.

The use of the coal-tar products is contraindicated.

The diet is an extremely important part of the treatment of pneumonia and will be considered under "The Feeding of the Premature."

Frequent changes at regular intervals of the infant's position in its bed are imperative to successful care of the pneumonias in the premature.

CHAPTER XI.

DISEASES OF THE GASTRO-INTESTINAL TRACT.

A. DISEASES OF THE ORAL CAVITY.

Sprue (*Thrush, Soor or Mycotic Stomatitis*).—**Etiology.**—Premature infants, weaklings and more especially those suffering from nutritional disturbances are subject to this affection. It occurs only where a lesion of the mucous membrane is present. The abrasions of the epithelium may, however, be very slight and in the premature is usually caused by wiping out of the mouth or through other mechanical injury. The source of infection is very commonly from the nipple. However, it may be carried into the mouth through utensils or soiled pledgets.

Symptoms.—The importance of thrush is probably always secondary and its significance above all symptomatic. It is improbable that thrush itself may cause a general serious infection. It may be an indicator of a serious general affection or an essentially lowered resistance; not infrequently it is seen in those apparently in good health. In the premature it sometimes invades the esophagus, and it has been described as invading the blood stream. In the more robust premature infants it is usually seen as small white punctiform and flat eruptions on the tongue, gums and inside of the cheeks. In infants with lowered vitality it may assume the form of extensive membrane covering the whole buccal cavity. The latter is especially true where it accompanies septic diseases. In the severe forms it is also frequently associated with Bednar's aphthæ.

Usually the most serious symptom is the inclination on the part of the infant to refuse its food. However, it may be associated with vomiting and as has been stated, is frequently a complicating factor in the severe nutritional disturbances.

Prognosis.—While thrush is usually curable within a week in the full-term infant, in the premature, unless the treatment is very carefully undertaken, the traumatism in the course of local applications may cause new local lesions which become readily infected, thus frequently prolonging the course of the disease.

Treatment.—*Prophylaxis.*—Thrush being due to lack of cleanliness and trauma, these two factors should by all means be avoided, and every effort made to avoid trauma of the mucous membrane in the first care of the mouth of the new born. It is not contagious and if the proper prophylactic means are observed in the daily routine, it should not be spread from one infant to the other. In the breast-fed the mother's nipples must be washed with a saturated solution of boric acid and moistened with one-half strength alcohol, which should be allowed to evaporate from the nipples before nursing. In the bottle-fed the nipples and bottles should be carefully boiled after each nursing, and only such nipples should be used as can be completely everted so that both the inside and the outside can be thoroughly cleansed, following which they should be preserved in a borax solution of one ounce to a pint of water. The nurse should use every precaution in the care of the hands, dress and all objects which may be carried between the cribs.

Local Treatment.—Every form of local treatment must be carefully and gently applied so as not to abrade the sensitive mucous membrane. Gently sponging the mouth with a solution of borax, 10 grains to 1 ounce of boiled water (this is preferable to boric acid), using a very soft pledget of cotton on the finger if the mouth is not too small, otherwise on a swabbing stick or toothpick after each feeding, will usually cure the disease. Traumatism of the tender mucous membranes must be avoided. The solution may also be used as an irrigation by allowing it to come gently in contact with the infected surfaces, including the tongue if involved, the infant being turned on its side so that the solution will flow out of the mouth. A small sucker containing equal parts of borax and sodium bicarbonate may be placed into the mouth of larger infants for a few minutes four or five times daily. In severe and persistent cases it may be necessary once or twice daily to gently paint the mucous membrane with a one-fourth of 1 per cent solution of silver nitrate. Following the application of the silver nitrate 2 drops of olive oil or castor oil can be used in the mouth to allay the irritation. Mixtures of honey and borax as well as all sugar preparations should be avoided.

Internal Treatment.—Where the infant refuses to nurse it may be necessary to resort to feeding with spoon, medicine dropper, Breck feeder or even to gavage. Every effort should be made to improve the general health of the premature infant by proper feeding, cleanliness and good hygienic surroundings.

Various Types of Stomatitis.—The term "stomatitis" is applied to inflammations of the mucous membrane of the mouth. In the full-term infant three types are usually described: the catarrhal, the aphthous and the ulcerative. The classification in the premature is far less distinct than in the full term. The types as most commonly seen are the traumatic ulcerations, usually involving the palate. They may be very slight and superficial or by secondary infection become serious lesions. The simple traumatic patches are usually seen as yellowish, superficial lesions often covered by a slimy membranous film which can be easily removed, such removal being followed by small punctate hemorrhages. They are usually seen from the second to the fourth day after birth, decreasing in intensity and showing a tendency to rapid healing with proper care, usually disappearing within a week. Even in the premature this variety is usually harmless, except insofar as it interferes with nursing. However, the dangers of secondary infections must never be overlooked.

Etiology.—Although the cause may vary it is usually trauma of the mucous membrane through cleansing of the mouth. This is especially true of the handling of asphyxiated and cyanotic premature infants and follows injury due to mechanical removal of mucous from the mouth. More serious lesions over the pterygoid processes which have been described as Bednar's aphthæ and which are usually due to more intense trauma of the mucous membrane in the cleansing of the pharynx, may lead to more serious complications. Similar ulcerations may be found in other areas where a thin mucous membrane is in close contact with the hard bony structure.

Syphilitic stomatitis is not uncommon in infants suffering from congenital syphilis. The ulcerations are more commonly seen about the lips unless secondary to trauma.

Gonorrheal stomatitis is a rare condition. The tongue, palate and gingival folds are the seat of small whitish deposits, usually appearing on a non-inflammatory base. It is rarely manifest before the fifth or sixth day after birth. After one or two days the patches assume a yellowish color and become elevated above the surrounding tissue.

Prognosis.—The tendency to secondary infections and deeper ulcerations should always lead to a guarded prognosis, because of the influence on the future health of the infant and the difficulties of feeding.

Gonorrheal stomatitis, while usually healing without unfavorable results in the full-term infant, when properly treated, is always a serious complication in the premature.

Treatment.—*Prophylactic.*—This should consist of the avoidance of all trauma at birth and the absolute prohibition of subsequent mechanical cleansing of the mouth, unless there are special indications. The latter is entirely superfluous when the proper care is taken in the preparation and administration of the infant's food. Infants suffering with ulcerative stomatitis should be isolated to impress the attendants with the dangers of spreading the infection by careless handling.

Curative.—The curative treatment is the same as that described under thrush and even greater care should be taken in the application of local treatment. The deeper ulcerations in the mouth can be treated to advantage with small quantities of peroxide of hydrogen or 1 per cent potassium chlorate solution, or careful application of 1 or 2 per cent of nitrate of silver solution. In all cases, however, in the premature infant the attendant should not become overzealous in the administration of local treatment, because of the dangers of further traumatizing the sensitive mucous membrane. The feeding offers the same difficulties as in the severer cases of thrush necessitating hand feeding of expressed milk in most cases.

In syphilitic and gonorrheal stomatitis the local measures are the same as for the other varieties of stomatitis. The general measures for the former are such as are described under the treatment of congenital lues.

Cancrum Oris (Noma).—**Etiology.**—No single microörganism has proved to be the cause of noma. Spirillæ and fusiform bacilli have been found (Weaver and Tunnicliff[1]) not only in the necrotic tissue, but in the surrounding healthy parts. Whether these organisms represent the primary cause of the lesion or only secondary invaders is not known. In other instances the Bacillus diphtheriæ alone has been found.

Symptoms.—The site of the disease is usually the inner side of one or both cheeks. The gangrenous process usually begins as a small inflamed, infiltrated area in the mucous membrane. Localized destruction of tissue follows, and this process extends with great rapidity until the tissue sloughs away in masses.

Prognosis.—The disease usually occurs in weakly, marantic infants, who die from exhaustion and sepsis within ten days or two weeks from the onset of the disease. Hemorrhage is rarely a complication. The disease is usually fatal even under the best management.

Treatment.—Treatment at best is very unsatisfactory. The procedure followed in ulcerative stomatitis together with the use of surgical measures affords the best possibilities. Nicoll[2] reported a case which had resulted in recovery following the intravenous injection of salvarsan.

B. DISEASES OF THE GASTRO-INTESTINAL TRACT.

In a consideration of this very important section as relating to the premature infant, we must recognize: (1) The possibility

[1] Jour. Inf. Dis., 1907, **4**, 8.
[2] Arch. Pediat., 1911, **28**, 912.

of congenital malformations and other prenatal factors which might have an important bearing on the function of the digestive organs; (2) the lack of proper physiological development necessarily present in the prematurely born, the importance of which varies inversely with the fetal age; (3) postnatal pathological conditions, developing in the gastro-intestinal tract; and (4) the importance of systemic infections in their influence on the processes of metabolism.

When the great importance of the interdependence of the second and third factors is recognized even in the absence of any congenital anomalies, we at once realize the marked tendency toward the development of disturbances involving the nutrition and well-being of the entire organism. The use of the term "nutritional disturbances" rather than that of "digestive disturbances" is beyond any question more generally applicable to the premature than to any other stage of life, as in these individuals the rapid development of general nutritional disorders is the rule following even moderate causes.

It must also be borne in mind that all factors which affect the general well-being of the premature infant, such as exposure and infection, have an almost direct effect upon gastric and intestinal functions. The very important relationship between the fetal age of the infant and the quality of the food and the method of its administration will be emphasized in the chapter on "Feeding."

The subject of gastro-intestinal disturbances in the premature infant offers a far more complex problem than do those of the new-born full-term infant. As previously stated, they require a consideration of possible developmental defects, constitutional anomalies, a low grade of immunity to infection, and a general lack of physical and functional development. The last two often lead to inability to take and assimilate the required food. Further complications are due to the rejection of the food or the development of gastro-intestinal irritation upon the slightest indiscretion in feeding. All of these have an important bearing upon adequate digestion, resorption from the intestinal tract and the further intermediary functions.

It cannot be too strongly emphasized that the immediate institution of the proper hygiene, and the establishment of the proper prophylaxis toward the prevention of nutritional disturbance by the early administration of human milk whenever possible, are absolutely necessary to avoid disaster. It cannot be disputed that a great number of premature infants die, not because their organs lack that degree of maturity necessary to proper functions, but because of early neglect, either through lack of adequate facilities or ignorance of exact methods of feeding and care.

Our acquaintance with the tendency to the rapid development of marasmus in premature infants leads us to give great consideration to the development of even the slightest nutritional disturbances. It should become the rule to give even moderate disturbances the same consideration that is given to athrepsia (marasmus) in the older infant, which is always regarded as making the feeding of human milk imperative. As this also entails the feeding of minimal amounts of food the body temperature must necessarily in part be conserved by artificial heat.

I. Functional Insufficiencies of the Gastro-intestinal Tract Wholly or in Part Dependent on Lack of Development: (*a*) **Difficult Nursing.**—The causes of difficult nursing are to be found either on the part of the infant or mother or both.

The Infant.—Various factors may enter which may make nursing difficult or even impossible. Some of these will be treated under the chapter on "Methods of Feeding" (page 171). Of the malformations, those offering the greatest difficulty are cleft palate, hare-lip and nasal deformities due to lack of cartilaginous development. The tendency to sleep constantly is often very perplexing. General weakness and lack of muscular development in the poorly developed premature are not infrequently sufficient to make nursing impossible. Infections of the mouth resulting in thrush, stomatitis and ulcerative processes are always of serious import. All conditions interfering with proper respiratory functions, whether due to lack of development, such as atelectasis or pulmonary infections interfere with the proper taking of food. These are but a few of the many complications which may be cited as impeding proper nursing.

The Mother.—On the part of the mother the various pathological conditions of the nipples and breast must be given due consideration.

(*b*) **Anorexia.**—Premature infants born in the seventh and eighth months rarely show a disposition to feed spontaneously during the first days of life, and in a large proportion of cases we are forced to administer the food without the infant's taking active part. Only a small portion of the infants weighing between 1000 and 1500 gm. are able to nurse without assistance, and very few of them, unassisted, are able to suck with sufficient strength to take food from either the breast or the bottle. A very interesting fact which we have noted in premature infants weighing under 1500 gm., and occasionally in even larger infants, is a tendency to at least attempt to nurse spontaneously during the first two or three days of life, during which they, however, receive very little food. This period is followed usually about the third day by a marked somnolence, during which they show little or no inclination to nurse. This is usually associated with a rather rapid loss in body weight due to underfeeding. At later periods not infrequently infants show a repugnance toward food, which may follow periods of overfeeding or be seen in the course of the gastro-intestinal or systemic infections. At whatever stage of the infant's development anorexia is seen it must be given the gravest consideration and every attempt made to administer food sufficient to meet the demands of the organism. Gavage must be resorted to if the less drastic methods of feeding are unavailing. It has been our experience that occasionally the omission of one or two feedings with the administration of a one-half strength physiological salt solution per mouth will result in the further stimulation of the appetite by producing thirst.

During this period the fluids should be given in sufficient quantity to meet the infant's needs, about one-sixth to one-fifth of the body weight daily. The addition of one to three drops of brandy is often a beneficial stimulant.

(*c*) **Inanition Fever.**—Unquestionably hyperpyrexia as seen in the first days of life and during the time when these infants are receiving a minimum of food need not necessarily be due to inanition. Many are undoubtedly due to infection or toxic products which enter the circulation through the gastro-intestinal tract. The products of decomposition as seen during the period of change from the meconial flora to the milk flora can undoubtedly give rise to hyperpyrexia, as is also true of the toxic products formed by decomposition of milk. The effect of products absorbed from the intestinal tract on the parenteral cells, as well as the by-products due to the rapid changes seen in the body tissues, may, any and all of them, following their absorption, give rise to increase in body temperature. Occasionally one sees cases of hyperpyrexia in the premature and in the new born in whom the high temperature cannot be due to the surrounding artificial heat, and who make a rapid recovery without after-effect by simply increasing the fluid intake of milk or water. The most striking cases that are seen are those in which water insufficient to meet the body needs has been given.

Treatment.—This consists in the administration of fluids equal to at least one-sixth of the body weight of the infant in twenty-four hours and the administration of food as per the rules on "Feeding of Premature Infants during the First Ten Days of Life." Of equal importance is the prevention of overheating by application of excessive external heat. When doubt arises as to the causative factor in so-called "inanition fever" a small dose of castor oil (5 to 10 minims) together with a colonic flushing with a saline solution should be given in addition to the increase in the amount of water and human milk administered.

(*d*) **Vomiting.**—In general the vomiting in the premature is of greater or lesser importance depending upon its intensity, and the result upon the general state of nutrition. Vomiting must be considered only as a symptom and not primarily as a disease, and again as a symptom which in its development is influenced by many factors peculiar to the premature infant. The relatively vertical position of the stomach in the sixth, seventh and eighth months, as described under the "Physiology of the Premature Infant," is a factor of considerable importance, as is also the poorly developed sphincter at the cardia.

Of equal importance is the fact that most of these infants are fed mechanically in amounts theoretically correct for their weights and ages. But these same quantities may not agree with Nature's idea of sufficiency, thereby leading to a rapid overfilling of the stomach through catheter and other mechanical means of feeding. Again the tendency toward abdominal distention and the frequent handling and manipulation of the infant all tend to promote regurgitation.

Vomiting persisting beyond the first or second week, even when varying in frequency and intensity, is likely to result in a considerable degree of undernourishment. There is also the added danger in the case of premature infants with a minimal development of reflex irritability, that due to the lack of proper response on the part of the laryngeal reflexes, the regurgitated food may be aspirated with resulting sequelæ, such as cyanotic spells, asphyxia or even pulmonary infection.

Because of the great danger of underfeeding in the presence of small food intake there is the gravest danger of weight losses, with the consequent development of inanition.

Etiology.—Previous to the taking of food the infant may vomit the various fluids such as liquor amnii and blood, which may have been swallowed during labor. These may easily be recognized by their character. Following the intake of fluid many factors must be considered, such as atresias in the digestive tract, excessive feedings, which may be due to too free nursing from an easily secreting breast by the older premature infants, or too rapid feeding of large quantities mechanically given to the smaller infants.

The dangers of compression of the abdomen due to improper holding or excessive handling of the infant are usually overcome by feeding in the bed. The tendency to habitual vomiting is not uncommon in the first months of life in the premature. This is not infrequently due to a general state of nervousness or a neuropathic constitution. According to Alfred F. Hess, this can easily be demonstrated by passing a catheter and exciting the pharyngeal, cardiac and pyloric reflexes, which in the normal child are but slightly developed, while in the neuropathic individual the passage of the catheter is easily noted by the reflex manifestations following its passage. Undoubtedly many of these cases are true instances of pylorospasm. That true cases of pyloric stenosis may occur has been proven beyond doubt. The fact that these infants not infrequently vomit quantities larger than a single feeding should not lead to the diagnosis of a hypertrophic stenosis, as is proven by the fact that the stools usually contain a considerable amount of food residue.

The toxic vomiting as seen in the infants of eclamptic mothers and in the presence of sepsis, as well as hematemesis, will receive further consideration in the discussions on these topics. Vomiting may at any time become of serious moment and should always be given proper consideration. The relative loss of food as foretold by the scale, by weighing before and after feeding, and after vomiting, the weight curve and careful observation of the stools will give the best indications for therapeutic interference.

Treatment.—In the majority of cases there is no indication for active treatment. The occasional or even more or less regular "spilling" in the presence of a normal gain in weight and general well-being need receive little or no attention. However, when vomiting is persistent and is attended by stationary weight, which is equivalent to loss in weight in older individuals, or when it is associated with nausea or is expulsive in character or contains bile, blood and other matter foreign to the normal stomach content, it should receive prompt and careful attention. No set rules for treatment can be prescribed, as the etiological factors in each and every case must be considered individually. The following general principles of treatment will in a great number of cases prove sufficient:

1. The infant should be subjected to a minimum of handling. It should, whenever possible, be fed without being removed from its bed, or where handling is necessary, all violence should be avoided.

2. The recumbent position with the head and shoulders slightly elevated assist in overcoming the tendency to regurgitation in the presence of a weak sphincter at the cardia.

3. Regulation of feedings is primarily indicated. This should cover first the number of feedings and the interval between feedings, and the amount of the individual meal. Not infrequently an infant who is receiving quantities too great for the stomach capacity will cease vomiting upon simple reduction of the size of the individual meal. Again it may be necessary to decrease the number of feedings, thereby lengthening the intervals between feedings. Furthermore large meals at long intervals may be replaced by

small meals at short intervals with a very beneficial result. Where the infant is nursing at the breast, simply reducing the time allowed for nursing in many instances will accomplish the desired end. The same may be true as regards the slower administration of the individual feedings in the bottle-fed.

4. In infants nursing directly at the breast where the shortening of the period of nursing is insufficient to control the vomiting, drawing the milk by expression or by breast pump and feeding a measured quantity which can be retained, either by hand or catheter, is often successful.

It is customary to start such feedings by giving 2 to 10 gm. at short intervals, 10 to 12 if bottle fed, or 6 to 8 if fed by catheter, in twenty-four hours, preferably, although not necessarily, of freshly drawn milk, following this by gradually increasing quantities, and as soon as the proper quantity for growth is retained, lengthening the intervals, returning to direct nursing when the infant's general condition allows of the same. In the more severe cases the human milk may be boiled or a skimmed human milk should be used.

Under these conditions the milk supply should be protected through the emptying of the breasts by expression to prevent their drying up.

The question of even temporary starvation in the premature infant is one of serious import and should only be practised after the most careful consideration, because of the rapidly developing apathy in this class of infants. The presence of so-called "hunger stools," consisting of a brownish, stringy mucous substance with little or no food residue, is as a danger signal of almost equal importance with loss in weight.

If the above suggestions fail to accomplish the desired end, rather than to institute a starvation diet we prefer to empty the stomach by careful lavage, using a weak sodium bicarbonate or saline solution, and before withdrawing the catheter placing a small feeding of human milk into the stomach. Lavage is practised not so much with the idea of removing any decomposed food content, but because of the sedative action on the mucosa. The dangers of gastric irritation from repeated introduction of the catheter in careless hands must, however, always be remembered, also the dangers of promoting cyanotic spells, through the careless deposit of fluids in the pharynx and larynx. In the infants artificially fed the problem is far more serious, and offers for its solution greater difficulties unless human milk can be obtained. In our own experience a well-boiled milk, in which the casein has been precipitated as a fine flocculent curd by the addition of rennet, has given the best results when human milk was not to be obtained (see Preparation of Chymogen Milk). Diluting the milk thus prepared before feeding, or skimming before boiling may also be of benefit. Lactic acid milk mixtures may be used.

(*e*) **Gastric and Intestinal Indigestion and Distention.**—These may be of very serious consequence in the premature through interference with the respiratory and cardiac functions, and the precipitation of cyanotic attacks. Although frequently following relative *overfeeding* this need not necessarily be the case. Most of the factors which result in vomiting also predispose to indigestion and distention. *Abdominal distention* is exceedingly troublesome in the premature infant, but does not necessarily imply that indigestion is present. It may result in restlessness, vomiting, colic, borborygmus, increased respirations and cardiac action, hypothermia, cold extremities and not infrequently cyanosis.

While we have seen abdominal distention result in hypothermia it is equally true that when hypothermia is present it is almost invariably associated with impaired digestion, and a tendency to cyanosis and syncope. Owing to the tendency to abdominal distention there is the danger of *underfeeding* due to low-food tolerance. Excessive external heat whether from the use of simple heating devices or the more complex incubators often cause increased body temperature and result in impaired digestion. I have not infrequently seen death result from a relative overfeeding, due to attempts to feed infants food sufficient for their needs; but even more frequently do we see grave catastrophes from underfeeding in the same class, with rapidly developing syncope due to inanition.

Indigestion may be followed by an increase in the number of stools, and they become green and foamy and contain curds. The inability of the infant to handle foods sufficient for its maintenance without the development of functional derangement is a very grave deficiency, directly dependent upon the fetal age and factors predisposing to prematurity in the individual case. Less dangerous are the cases due to absolute overfeeding following early correction in the errors of diet, as are also the cases following indiscretions on the part of the wet nurse or mother. In the first few weeks of life severe indigestion is often fatal. This is especially true in artificially fed infants. Improper hygienic surroundings such as poor ventilation, oppressive humidity and lack of personal cleanliness may be factors in the development of indigestion.

The role of intestinal and systemic infections will be considered under their respective headings.

Treatment.—In the treatment correction of dietetic errors is most essential, and this is especially true in the artificially fed premature infants. It should always be remembered that the correction of the mild forms of indigestion are the life-saving measures. Severe indigestion has a high mortality. Stimulation of peristalsis and thereby emptying of the intestinal tract is usually accomplished by a low-pressure saline enema of 1 or 2 ounces. In some instances the addition of 1 gm. of glycerin to an ounce of water is of great assistance. Warm baths with or without very gentle abdominal massage may aid in increasing the peristalsis. Where the symptoms are persistent 5 to 8 drops of castor oil, 1 to 5 grains of sodium phosphate, or 5 to 10 minims of milk of magnesia, together with 3 to 5 minims of essence of pepsin of good quality, after each feeding, can occasionally be administered with great benefit. But unless the hygiene and feeding of the infant are properly regulated little permanent good can be expected. Correction of dietetic errors in the breast-fed is best accomplished by reducing the size and lengthening the intervals between individual meals when the stomach is very irritable. However, decreasing the size of the feedings is always associated with more or less danger and where the same results can be secured by simply lengthening the intervals this offers the best solution. Starvation diet should under all circumstances be avoided, although it may be necessary in extreme cases to dilute the meals. While larger infants—those weighing in the neighborhood of 2000 gm.—will stand the reduction of the feedings to as low as 60 calories per kilogram, in the smaller infants 70 to 80 calories must be considered the danger zone for even a short period of time. In the artificially-fed every attempt should be made to obtain human milk and where this is impossible our best results have been obtained by feeding boiled milk, in which the curd has been finely precipitated. (See Artificial Feeding, page 199.)

(*f*) **Diarrhea.**—Constipation is the exception; loose stools or a tendency toward diarrhea in both the breast-fed and the artificially-fed premature infants is the rule. Therefore, frequency of bowel movements, especially in the breast-fed may be entirely physiological and unassociated with fever, vomiting and other evidences of gastro-intestinal disturbances. However, they may be due to contaminated food or intestinal infection, and every case should, therefore be carefully studied so that evidences of deep lesions may be immediately observed. In the infant nursed by its mother the colostrum will almost invariably result in frequent bowel movements. This is one of the prime reasons in the selection of wet nurses, who have passed at least two or more weeks of their puerperium. The early milk also has a tendency to be high in its carbohydrate and fat content, either of which may be factors in the causation of frequent stools. A gastro-intestinal infection may be unassociated with fever and may, therefore, go unrecognized and be of serious consequence. Changes in the mother's environment, when she is nursing her own infant, as is seen at the end of the puerperium when she leaves her bed and changes her mode of living, seem to have a very beneficial influence on the quality of milk secreted, and the mother and baby seem to adapt themselves more readily to each other. This beneficial change is, of course, not seen where the baby is fed by a wet-nurse.

At this point I desire especially to emphasize my experience with both mothers and wet-nurses who are given a too liberal diet. While the average mother can be allowed to select her own diet during the nursing period of a full-term infant, eliminating such foods as may cause colic, abdominal distention and diarrhea in the infant, in the case of the premature such liberties must under no circumstances be allowed as they may result in an early disaster, cyanosis and death due to abdominal disturbances. Therefore, every wet-nurse should be made to adhere strictly to the limitations of diet as prescribed under the section on "Diet of Wet-nurses" (page 121). So long as the infant is passing yellow stools of normal odor, without symptoms of indigestion and is gaining in weight, even though the stools may number five to eight daily, no alarm should be felt, and the diet should be sustained.

The change of the yellow stool to a green color shortly after passage is the normal process of oxidation. However, the green, frothy stool containing small white curds and considerable mucus should always be considered abnormal. Such stools usually lose their normal acid odor, cause excoriation of the buttocks, and are frequently associated with fever. This is not infrequently a finding in the breast-fed premature. In the treatment of such cases, while the care of the infant is of paramount importance, no less important is the careful regulation of the mother's surroundings, mode of living and diet and also her mental activity. Again every precaution must be taken in reducing the infant's diet, and the same dietetic measures which were instituted for the treatment of indigestion apply to every case of diarrhea with abnormal stools, as they are almost invariably attendant on an intestinal indigestion or infection.

Dehydration of the body tissues through excessive water losses must be met with sufficient water administration, so that a good working rule should lead one to administer at least one-sixth of the body weight daily in fluids, in all cases of diarrhea. Therefore, when the quantity of the meals is reduced, or the interval lengthened, water should be added to the feedings or be administered between. The normal infant's stool will form a water margin about the semi-solid mass about one-half to three-quarters of an inch in diameter. When more water than this is lost with each stool, the infant must be carefully weighed and its water losses noted so that they may be compensated.

Tarry stools are always due to the presence of blood, and abrasions of the intestinal mucous membrane are likely to lead to fatal infections and must therefore always be given serious consideration.

(*g*) **Constipation.**—The mechanical causes such as atresias in various parts of the intestinal tract, or an imperforate anus, must be considered as possible causes. As has been previously stated, diarrhea is far more common than constipation. This, however, does not mean that a sluggish lower bowel is uncommon in the premature. In fact the lack of power of the muscular wall and the minimal reaction of the mucous membrane to mechanical and chemical stimulation are both important etiological factors and are often associated with intestinal distention. The first evidence of this lack of response is often noted in the inability of the premature infant to evacuate the meconium which has accumulated in the lower bowel, and this may require mechanical removal by the aid of a small saline enema or further irritation with a soap or glycerin suppository. If these means fail, a single dose of 5 to 8 drops of castor oil may be administered without too great delay, as it is our rule to start feeding only after the first intestinal evacuation, so that the presence of an atresia may have been noted, and the meconium removed before it has become infected through bacterial ingestion. The next stage of the infant's existence which is associated with constipation is in the first few days of life when food ingestion is insufficient and below the caloric requirements of the infant. In such instances in the absence of other causes it may be considered as a certain symptom of underfeeding, as is the case at all times when "hunger stools" are present. Increasing the food judiciously removes the trouble.

As the infant's digestive function improves and it utilizes its food to the fullest advantage, constipation may result from the minimal amount of food residue. The best evidence of such a causative factor in the presence of sufficient feeding is the improvement in general condition and gain in weight. In fact, utilizing their food perfectly, they have a tendency to constipation so long as their food intake is not in excess of their required caloric needs. Therefore, feeding this class of infants moderately in excess of the normal caloric needs usually overcomes the constipation.

In the treatment where the ability to digest food is minimal, increasing the water intake is frequently beneficial. Even premature infants are creatures of habit, and where it is necessary to assist them in the evacuation of their bowels, this should be practised at a stated hour, once or twice daily, either through the use of a saline or oil enema, non-irritating suppositories, or what is better, the tip of a well-oiled catheter. Medication in the form of laxatives administered to the mother in the hope of influencing the character of the milk, as well as drugs administered directly to the infant are more or less dangerous agents and should be avoided whenever possible. Ten to 15 drops of paraffine oil, 5 to 10 minims of milk of magnesia or equal amounts of castor oil, may occasionally be administered, but only after attempts at correction by mechanical irritation.

(*h*) **Underfeeding.**—In order to consider this subject properly we must first take into consideration the types of infants with which we are dealing, that is: (1) Healthy premature infants, and (2) congenitally debilitated infants, either premature or full-term. We must again divide them into the classes of breast-fed and artificially-fed, and lastly, as to whether the underfeeding occurs during the first days of life during the period when we may expect normally stationary weight, or weight losses, or at later periods, during which we are more likely to see the development of the completed picture of marasmus.

Before entering upon the details of this subject several factors which tend toward the development of inanition and marasmus not directly dependent upon underfeeding must be considered: (1) The danger of an imperfectly developed digestive tract, which even in the presence of sufficient food may soon result in a metabolic bankruptcy. These infants rarely survive the first days of life. (2) Improper hygienic surroundings of the infant, of which one of the most important is the danger of overheating, thereby interfering with heat regulation and associated with excessive evaporation from the body surfaces. This is especially disastrous in its effects in the presence of decreased humidity. Both of these factors predispose to atrophy. Lessened immunity with the added dangers of local and general infections and the secondary nutritional disturbances are especially common in this class of infants, as are the tendencies toward repeated nutritional disturbances once they are established. All of these factors which may tend to impair the general nutrition of the infant will but serve to emphasize the need of careful observation. It will, therefore, be seen that underfeeding may be a primary affair, or may result secondarily following previous nutritional disturbances.

II. Underfeeding in the Healthy Breast-fed Premature Infant During the First Days of Life.—This represents the dangerous period through which most premature infants pass whose feeding is delayed, awaiting the secretion of milk from the mother's breasts. While the full-term infant may pass through this period with slight disadvantages to its future development, the life of the premature infant may be jeopardized beyond all hope of recovery.

It has been our experience that when feeding is too long delayed the infants, unless very carefully fed in minimal quantities, are subject to repeated digestive disturbances and secondary infection, the latter due probably to lowered resistance. In the premature infant this is true even though the infant's food be human milk. How much more important is the avoidance of long starvation in those who are to be fed artificially, can easily be surmised. Whereas the birth weight in the average premature infant is regained by the second or third week, in those who have suffered great initial weight losses through starvation the return to normal birth weight is greatly delayed. It is surprising, however, to note what minimal quantities of human milk alternated with water, with a total administration of one-eighth to one-tenth of the body weight of fluid in twenty-four hours, will tend to prevent great initial weight loss during the first few days. (See Section on "Feeding during the First Ten Days of Life.")

The removal to an institution supplied with wet-nurses or a wet-nurse in the home are the ideal remedies. Small quantities of milk obtained from other mothers to tide over the period of early lactation when secretion is delayed, as is not infrequently the case where labor is considerably before term, will prevent a critical condition. Where the prospects for human milk are much delayed, the anxiety of the family, due to the decrease in weight, may often be relieved by judicious artificial feeding, as suggested in the chapter on Feeding. It is always the part of wisdom to impress the family with the fact that stationary weight and fluctuating weight are to be expected for a much longer period in the premature than in the full-term infant, and that this stationary weight curve does not indicate a bad prognosis. Artificial feeding, if instituted should always be discontinued at the first opportunity.

In the congenitally debilitated infant, especially the premature, human milk is a practical necessity for a low mortality.

Atrophy and marasmus as seen after the first days or weeks of life are even more dangerous than the evidence of inanition during the first days of life, because they are almost invariably secondary to previous underfeeding, errors in diet, or gastro-intestinal and systemic infections, and improper hygienic conditions, which are unfortunately frequently neglected or overlooked, even when attempts to overcome the conditions are made. In all late cases of marasmus in the premature, while removal of the underlying factors is an absolute essential, the furnishing of the proper diet in the form of human milk is of equal importance.

III. Secondary Digestive and Nutritional Disturbances Accompanying Systemic Infections (Parenteral).—Just as digestive disturbances result in lessened immunity to infection, so do we find digestive troubles following the infections in the premature, such as infections of the skin, lungs, genito-urinary tract, ears and the general septic infections, which are of common occurrence in these individuals. These secondary conditions are also likely to run a more severe course than the primary nutritional disturbances.

Where it is possible to keep up the baby's nutrition by the proper administration of foods during the course of an infection, such children may be subject to little or no weight loss in the milder types. In more serious cases the food must be reduced both qualitatively and quantitatively. However, even in these, to avoid catastrophes, long-continued underfeeding or starvation must of necessity be avoided, and only in exceptional cases with resulting food intoxication must all food be withdrawn. Such cases furnish us with every indication for early feeding with human milk whenever possible. True alimentary intoxication is usually early recognized by the toxic symptoms—facial expression, rapid respiration, and marked drops in the weight curve. In these cases temporary complete withdrawal of food in the absence of severe infection results in disintoxication. In parenteral infections this is not the fact, and starvation only leads to a further reduction in fighting power and therefore should not be long continued. The further treatment of these cases is the same as that to be described under "Infection of the Gastro-intestinal Tract."

IV. Infections of the Gastro-intestinal Tract (Enteral).—No subject with which we have to deal in the care of premature infants calls for such mature judgment as the care and treatment of gastro-intestinal infections. In fact the entire hospital unit is more or less planned and constructed with the idea of prevention and isolation of these cases. Our thought should all be centered on their prevention, since once they become established their course is associated with the gravest dangers.

Infections of the intestinal tract are secondary in importance only to infections of the respiratory tract. It is well known that infection of either of these systems is likely to run rampant throughout hospital wards unless the individual cases are properly segregated at the outset. The infection may be spread through carelessness in handling the infant's utensils or lack in the care of the nose and mouth, through the nipples if nursing on the bottle, or by lack of asepsis in the care of the maternal breasts. Again it may be spread by the thermometer, unclean napkins, or it may be transmitted by flies and insects. The food is in all probability the most common source of transmission, and the milk may be infected either in the breasts themselves or in the handling. In the past before our wards were properly equipped to care for premature infants, doubt as to the advisability of instituting hospital treatment in preference to the home, even though the facilities for general care were limited, existed in our minds with good reason.

All intestinal disturbances must be considered serious because at the outset it is impossible to decide whether we are dealing with a simple indigestion or the first symptoms of an infectious diarrhea. Again it is quite difficult to determine whether we are dealing with the abnormal activities of the intestinal flora or with pathogenic bacteria or their metabolic products. It is also a well-known fact that the normal bacterial inhabitants of the bowels may under suitable circumstances either form toxic products or pass through the frail intestinal wall of the premature infant into the general circulation.

That we may have serious intestinal symptoms without infection is not to be denied. On the other hand, however, we have the findings of Schabort[1] who was able to isolate diplococci and staphylococci from the stools of every infant which he examined between the thirty-second and the ninety-sixth hour after birth. He found that the sooner the staphylococci appeared, the sooner the stools took on a dyspeptic character, and when the intestinal symptoms were at their height, these organisms dominated the bacterial flora. He believes that every infant has a staphylococcus enteritis in the first days of life. The etiological significance of these cocci is, however, questionable.

Von Reuss[2] states that he has not infrequently seen, usually toward the end of the first week of life, even temporarily, muco-hemorrhagic stools, dysenteric in character, which did not, however, impair the general health. He also states that the entrance of staphylococci into the oral cavity of the infant and from there into the intestine cannot be avoided, even with the most extreme care on the part of the attendants, because the cocci may come from the milk ducts or the genital canal of the mother. While the milder types of enteritis are brought about by the irritation of the bacteria or their products of decomposition of the normal intestinal content, they are distinguished with difficulty clinically from the forms of enteritis which are caused by pathogenic microörganisms.

Although in most instances the important element of time and lack of laboratory facilities precludes the making of a diagnosis as to the specific causative organism, in the light of the more modern work of Passini[3] in his studies of the new born, and Kendall and Day[4] and others in older infants, the results of their studies are equally applicable to infections in the premature. Passini found that with the introduction of human milk into the lower part of the intestinal tract the meconial flora consisting largely of the

[1] Monatschr. f. Geb. u. Gynäk., 1906, **24,** 29.
[2] Die Krankheiten des Neugeborenen, Berlin, 1914.
[3] A Study of the Anaërobic Intestinal Bacteria, Jahrb. f. Kinderh., 1911, **73,** 1911.
[4] Boston Med. and Surg. Jour., 1913, **169,** 753.

sporulating forms of the gas bacillus were changed to the fermentative, aërogenous forms, which were capable of forming irritative products which frequently resulted in an increase of stools more or less foamy, containing increased quantities of mucus. These stools are frequently seen during the first days of nursing and are the so-called "transitional stools." However, these products of food decomposition may reach a considerable degree of intensity and be associated with morbid manifestations (von Reuss).

Having thus described the mild types of irritation of the first group due to the ingestion of staphylococci and of the second group due to the transformation of the gas-forming flora of the meconium into the aërogenous organisms, as seen after the ingestion of milk, with the resultant irritation due to the fermentative action on the milk sugar, we come to the large class of cases which may be due to one of many types of organisms. Those due to streptococcus infections through the maternal circulation before birth (which in itself may be the cause of premature birth), or the secondary infections through the mother's milk, lochia or other products entering the gastro-intestinal tract by way of the mouth, are among the most virulent.

The Streptococcus enteritis may, therefore, make its appearance very early in life depending upon the time of infection. In premature infants it has a very high mortality. The various bacillary infections of the intestinal tract due to the colon bacillus, dysentery bacillus, typhoid, paratyphoid bacillus, etc., with the exception of the colon bacillus, are rarely seen during the first days of life, unless the mother is suffering with an infection due to one or the other organism. The clinical symptoms are dependent upon the fetal age and stage of development of the individual infant and upon the type and virulence of the infecting organism. The general health of the infant suffers in all cases. Weight has a tendency to become stationary or show a loss. The temperature curve varies directly with the influencing factors and the reaction on the part of the infant, in the milder types being dependent to a great degree on the absorption of the toxic products. Vomiting is an almost constant factor, and the stools which are at first frequent, containing food material when developing after the first few days of life, or meconium if the infection be very early, show mucus and often gas, depending upon the infecting organism, and in the severe cases sooner or later blood. Even the premature infant will give evidence of pain and tenesmus as seen by its facial distortion, its low whining cry, and drawing-up of the lower extremities. In the great majority of cases the abdomen is sunken rather than distended. The skin soon becomes dry. The body fat is burned up and collapse becomes imminent. The prognosis is in each case dependent upon the exciting factors, the degree of resistance, but in all cases, however mild, the outcome remains in doubt.

Treatment.—The therapy of these cases offers one of the most difficult problems confronting the pediatrician, because of the danger of marasmus following even short periods of starvation, and because of the limitations of dietetic treatment. Human milk is indeed the only food which can be given with any degree of safety, and this at the same time because of its high sugar content offers a splendid culture medium for the gas-forming organism. The frequent, somewhat foamy, mucous stools which are seen during the transitional period, that is, in the change from the meconial stools to the normal breast-fed stools as well as the frequent stools of similar formation as seen after this period, when unassociated with temperature, loss in weight, and other symptoms indicative of serious trouble, should not lead to dietetic changes, but breast nursing should be continued as long as there is an absence of all signs of beginning general disturbance. When the infant is feeding on the milk of its mother similar but physiological stools are very commonly seen during the period in which she is secreting colostrum and are not to be confused with intestinal infection. Evidence of more serious infection should lead to a short period of feeding on inert fluids of proper volume. This period of starvation in most cases should not be continued over six, or at the most, twelve hours, and should be followed by gradual feeding of smaller quantities of human milk than has been previously given. Where there is question as to the mother's milk being the source of infection, wherever possible, the milk should be obtained from another source, or the mother's milk should be sterilized before feeding. This latter is easily accomplished because in most cases premature infants are hand-fed during the first few days of life. These measures should be carried out until bacteriological examination is completed.

The intestinal tract may be cleansed by the administration of a single dose of castor oil, varying from 10 to 30 minims, depending upon the age of the infant and the conditions at hand. The large bowel may be emptied by irrigations of normal saline solution, which later may also be given as small repeated nutritive enemata after the intestinal tract is once thoroughly cleansed. Vomiting may be allayed by very careful gastric lavage, small quantities of saline solution being left in the stomach after the washing. Lavage must be rapidly and dexterously performed to avoid attacks of cyanosis. Brandy (1 to 5 minims), or aromatic spirits of ammonia (1 to 2 minims), at regular intervals are the best forms of stimulation for oral administration. Minimum doses of paregoric ($\frac{1}{2}$ to 2 minims) will frequently allay the intestinal peristalsis and relieve tenesmus, both of which add to the dangers of the intestinal infection. Intestinal antiseptics are to be avoided. Hypodermic stimulation must be very carefully given because of the dangers of toxic effects and of local irritation. Small doses of camphor in oil (1 to 5 minims) are the best. In the artificially fed infant every effort should be made to obtain human milk.

DISEASES OF THE LIVER AND BILE PASSAGES.

Icterus Neonatorum.—Two varieties must be distinguished—icterus simplex and icterus gravis.

While the simple form of icterus is, as a rule, a very benign condition usually running its course without severe systemic manifestations, the grave type is especially fatal in premature infants in whom it is usually a clinical manifestation of sepsis, syphilis, hemorrhagic diathesis, or some grave form of liver insufficiency or bile-passage obstruction.

Frequency.—The incidence of icterus neonatorum in premature infants varies with the observer from 15 to 100 per cent. This variation probably depends upon what the individual observer considers to be jaundice. If a yellowish tinge to the nose and cheeks is regarded as sufficient to make the diagnosis then the percentage will be high; should staining of the conjunctivæ only be taken as evidence the number will be low. The term "true icterus" can be applied only to those cases in which the yellow discoloration of the skin is caused by a staining of the bile pigments.

Pathology.—Autopsy in moderate cases shows that the intima of the arteries, the serous membranes and the various body fluids and the interstitial tissues are stained yellow, but the brain, the cord, liver, spleen and kidneys are usually only slightly stained, if at all. In severe types, however, deposition of bilirubin crystals may be found in the cells of the skin, in the capillaries and lymphatics and also in the renal pyramids, blood, adipose tissues, brain and other organs.

Etiology.—The recent work of A. Ylppö offers the most plausible solution for the occurrence of jaundice in the new born, and his experiments are well worth quoting in detail. His experiments were conducted through spectroscopic analysis of the blood for its bilirubin and biliverdin content. He found that biliary pigment secretion is small until the late fetal months are reached. Shortly before birth the secretion is rapidly increased, and this increase is intensified after birth.

This biliary pigment content of the blood increases up to the third to the tenth day and on the whole continues for a longer time in the premature than in the full term. He found that blood from the umbilical vessels averaged from 13,0—58,2·10⁻⁵ per 100 cc of blood and that this increased from the third to the tenth. In those cases which passed 125,0·10⁻⁵ gm. per 100 cc of blood, icterus developed, while it remained absent in those containing less than this amount. He also found that there was a direct parallel between the blood content of biliary pigment and the intensity of the icterus: Clinical manifestations due to the icterus were absent in the mild cases, while in the severe ones the cholemia resulted in somnolence. He found little evidence that syphilis, sepsis and traumata influence the development of the jaundice. From his studies he concluded that icterus neonatorum is hepatogenous in origin and is due to the fact that for some days after birth the liver continues to secrete bile into the blood stream by the same routes that this occurs in fetal life and that due to the fact that there is an intensified secretion of bile pigment shortly before and after birth, the blood content of bile is increased, and that these findings result in the development of the icterus when in excess. He therefore believes that it is a physiological process which, however, may become pathological when the blood content becomes excessive.

The earlier explanation of Knopfelmacher offers a closely related explanation. He describes two factors as concerned in production of jaundice in the new born, a hypersecretion of bile and a disturbance of excretion. The richness of the blood supply to the liver immediately after birth is responsible for a greatly increased production of bile at this time, while during the first few days of life there is only a rudimentary functioning secretory mechanism. Accordingly the tenacious and stagnated bile passes from the overfilled bile capillaries into the blood capillaries.

The increased viscosity of the bile during the first days of life is explained by Pacchioni as being due to the loss of water sustained by the infant at this time, leading to a greatly slowed biliary current with absorption into the blood and lymph stream.

A hematogenous origin of the bile in icterus neonatorum may be excluded by the experiments of Minkowski and Nyuyn, who demonstrated that the liver is essential for the formation of bile and that without this organ jaundice cannot be induced. The connection of icterus with a stasis of bile, the result of the closure of the ductus choledochus by meconium (Franck), by desquamated epithelium (Cruse), or by a plug of mucus (Virchow), is not supported by the facts; neither is Birch-Hirschfeld's theory of edema of the capsule of Glisson, Bouchut's theory of a hepatitis, or Epstein's explanation that the cause is a catarrh of the small bile ducts.

Freirichs explained the jaundice by a marked anemia and decrease in pressure in the liver capillaries, which in turn lead to a lessened pressure in the bile capillaries and an overflow of bile into the blood stream. The great degree of congestion of the hepatic capillaries at birth precludes this belief, however, while the fact that the blood-pressure is raised in asphyxia neonatorum, in which condition icterus is especially intense also nullifies this theory.

Symptoms.—In very mild cases the yellow color may appear only on the face, chest and back, the conjunctivæ being but faintly tinted and the urine and feces normal in appearance. In severer forms the urine may be high colored enough to stain the linen, and the jaundiced hue may extend to the arm and abdomen. Some infants present a yellowish discoloration of the whole body, with typical clay-colored stools. In most cases the jaundice has disappeared by the eighth or tenth day. It may persist for several weeks. In rare cases, after having much diminished, it reappears with renewed intensity. The liver and spleen are usually unchanged, however; in the severer types liver changes are the rule and it is usually found enlarged.

An early type often seen from six to twelve hours after birth is not infrequent in small prematures. These are usually severe cases and although not of the septic type are slow in disappearing.

While most of the simple cases are unassociated with gastrointestinal and febrile disturbances the severer types even of the simple form are associated with symptoms of indigestion which is always of grave import in the premature. They are also subject to febrile disturbances and are slow in overcoming their initial weight losses.

Diagnosis.—Icterus neonatorum being a physiological condition, it must be differentiated from jaundice due to causes other than a mere disturbance of interrelation between formation and excretion. There must be excluded septic, syphilitic and familial jaundice, that due to deformities or obliteration of the biliary passages, and three or four rarer conditions characterized by icterus.

Septic Jaundice.—Gessner believes that many instances of so-called benign icterus neonatorum are dependent upon umbilical infection, and DeLee agrees with him. Other cases are thought to be due to intestinal infection.

In these cases the child is ill, the temperature is elevated and the skin shows a marked degree of icterus, which in the severer cases becomes a bronzing. Hemorrhages are often present, sometimes a foul-smelling pus exudes from the umbilicus, there is anorexia and the abdomen may be distended and tender. Blood cultures sometimes reveal the causative organisms or they can be demonstrated in the septic foci. The outlook for these children is poor, the younger and less mature the infant, the less is its chance of recovery.

Syphilitic Jaundice.—This form of jaundice is usually present at birth or appears a few days later. It is generally rather intense and may persist, though sometimes it improves, only to recur again. Hemorrhages under the skin are not at all uncommon. It should be suspected if other signs of syphilis are present, especially a positive Wassermann reaction.

Family Acholuric Jaundice.—This is a chronic condition characterized by jaundice of long duration, the presence of bile pigments in the stools and their absence in the urine. The spleen is usually but not always enlarged, there is, as a rule, more or less anemia present and some enlargement of the liver. The affection is compatible with life but occasionally there occur slight rises in temperature with malaise, diarrhea, abdominal pain and an increase in icterus. This jaundice has been explained on the basis of a simple cholemia, biliary cirrhosis, splenomegalic jaundice, or it may be that all the conditions are but different stages of the same affection.

Prognosis.—It must be kept in mind that icterus neonatorum may be of prolonged duration and yet be only due to disturbance of bile secretion and excretion, and not dependent on malformation, sepsis or other disease. An increase in the intensity of the icterus during the second week should make one suspicious of some causative condition more serious than that responsible for a simple icterus neonatorum.

Treatment.—There is no treatment for simple icterus of the new born, nor is any needed though small doses of calomel with sodium bicarbonate and sodium phosphate have been recommended.

Affections of the Excretory Bile Ducts.—(*a*) **Stenoses and Atresias.**—The common, hepatic or cystic ducts, one or all, may be affected in congenital stenoses. The more common etiological factors are as follows: (1) One or more of the ducts and even the gall-bladder may be totally absent; (2) fetal inflammatory processes of obscure origin may result in atresias; (3) pathological development at the distal end of the common duct with a valve-like formation may result in atresia (similar formations may be present in the mucous membrane at other locations in the bile passages); (4) hereditary syphilis may result in a perihepatitis, or cholangitis; and (5) occlusion may be due to inspissated bile or concretions.

Symptoms.—Acholic stools are present where there is an involvement of the hepatic or common duct or both. This may or may not be evident in the meconium depending upon the causative factor. Progressive icterus is an almost constant finding. Urinary findings correspond to the degree of stenosis, and there is undigested fat in the stools when fat is contained in the infant's food.

Prognosis.—Death soon follows in the cases where the stenosis is the cause of premature birth. However, in other cases where it is simply a part of the general picture of congenital lues, or occlusion is less complete, the infants may survive for a considerable time, depending upon the degree of systemic involvement. Death is usually due to intercurrent infection, which is not uncommonly through the gastro-intestinal tract, or inanition due to lack of fat digestion or to cholemia. All cases of complete occlusion are fatal in the premature.

Treatment.—In all cases except those due to inspissated bile and lues the treatment would be surgical, but such interference is practically hopeless in this class of cases and is rarely to be advised in premature infants. The medical treatment of congenital syphilis is far from hopeless. A much poorer prognosis is offered in stenosis of the ducts than in those cases where jaundice is due to a hepatitis.

(*b*) **Gall Stones.**—Cholelithiasis due to fetal inflammatory processes has been described by Bland-Sutton[1] and Cautley states that they are more frequent during the fetal age and early infancy than at any other period of childhood.

(*c*) **Inflammations of the Bile Passages.**—Although they are rare they may be due to ascending infection, but are more commonly subacute conditions as seen in congenital syphilis.

Affections of the Hepatic Vessels.—Phlebitis and thrombosis of the portal vein may result from an ascending infection through the umbilical vessels.

Congenital lues may be associated with a periphlebitis of the portal vein, or its intrahepatic branches, or gummatous infiltration about the hepatic vessels. When the portal vein is the seat of considerable obstruction, ascites, gastric and intestinal hemorrhages, enlargement of the liver and splenic tumor usually result.

BIBLIOGRAPHY OF ICTERUS NEONATORUM.

Minkowski-Nyuyn: Arch. f. exper. Pathologie, **21**, 1.
Franck, J. Peter: De curandis hominum morbis Epitome, 1805, **55**, 183.
Cruse, P.: Arch. f. Kinderheilk., **1**, 353.
Virchow, R.: Gesammelte Abhandlungen, s. 847.
Birch-Hirschfeld, F. V.: Virchow's Arch., 1882, **87**, 1.
Bouchut (quoted from Marcel Delestre): Étude sur les infections chez le prématuré, Paris, 1901.
Epstein: Volkmann's Sammlung klin. Vorträge, 1880, Nr. 180.
Freirichs: Klin. d. Leberkrankh., 1858, p. 1.
Quincke: Arch. f. exper. Pathol. u. Pharmakol., 1885, **19**, 34; Virchow's Arch., 1884, p. 95.
Meckel, H.: Charité-Annalen, alte Folge 4, 1853.
DeLee: Principles and Practice of Obstetrics, Philadelphia, 1913.

[1] Gall Stones and Diseases of the Bile Ducts, 1911.

Knopfelmacher, W.: Jahrb. f. Kinderheilk., 1898, p. 47; 1908, p. 67.
Hess, A. F.: Am. Jour. Dis. Child., 1912, p. 304.
Abramow, S.: Virchow's Arch., **181**, 201.
Pacchioni: Rivista di clinica pediatr., 1911, **9**, 333.
Ylppö, A.: Ztschr. f. Kinderheilk., 1913, **9**, 208.

Affections of the Hepatic Parenchyma.—Although the liver is readily influenced by toxic and infectious products, which easily pass through the permeable gastro-intestinal wall, nevertheless, it is exceedingly difficult to recognize the part which this great organ plays.

Among the most common affections of the liver is the predisposition to icterus, which appears especially early in prematures. Besides the physiological jaundice, icterus may accompany a variety of disorders. In the later life we see different types of icterus, aside from obstruction of the gall ducts, as: Septic icterus, Winckel's disease, catarrhal icterus, toxic jaundice and acute atrophy.

Parenchymatous and fatty degeneration of the liver is present in all septic diseases. Degenerative liver processes are, however, seen where there is no focus of infection and these are probably due to toxins, which are absorbed from the placenta, as in the cases of infants born of eclamptic mothers, perishing shortly after birth. In some such instances there may be no gross change but microscopically a high-grade degeneration exists. In addition, as previously mentioned, subcapsular liver hemorrhages occur quite frequently. Ylppö[1] was able to demonstrate such lesions in almost 80 per cent of the prematures under 1000 gm. birth weight, and only in 5 per cent of those weighing between 2000 to 2500 gm. At times the hemorrhages may be very extensive and with rupture of the liver capsule result fatally. Parenchymatous liver hemorrhages may also be found, but they are small and are not of much significance.

In the literature one finds mention of cases of acute yellow atrophy of the liver with the finding of tyrosin and leucin in the urine, coagulation necrosis and hemorrhages into the parenchyma.

Septic icterus is characterized by a marked acute interstitial and parenchymatous hepatitis. In some cases there have been noted cyanosis, convulsions and digestive disturbances, which either disappeared in a few days or led to death. In these cases the liver symptoms are cloaked by those of general sepsis.

Icterus catarrhalis is often associated with duodenal catarrh and cholangitis, and is characterized by jaundice, acholic stools, bilirubinuria, and the prognosis is on the whole good.

[1] Pathologisch-anatomische Studien bei Frühgeburten, Ztschr. f. Kinderh., 1919, **20**, 329.

Cirrhotic processes in the liver are usually associated with congenital syphilis or deformities of the gall tracts. One sees a diffuse interstitial luetic hepatitis under the picture of hypertrophic cirrhosis. With anomalies of the gall ducts there is a biliary cirrhosis.

Congenital Tumors of the Liver.—These in themselves may be the cause of premature birth. However, most of the cases of malignant tumors described in the literature are those which have developed after birth in infants either born in seeming health at full-term, or those congenitally debilitated, but who did not give evidence of tumor formation until some time after birth; while the cases giving evidence of tumor formation at birth have been commonly angiomata or cystic degenerations.

DISEASES OF THE PERITONEUM.

Fetal Peritonitis.—Intra-uterine peritonitis is usually chronic in character, and in premature infants usually results in death shortly after birth, if not already the cause of still birth.

Etiology and Pathogenesis.—1. Malformations in the digestive tract with emptying of the contents into the peritoneal cavity. It is also quite possible that some of the malformations described previously may result from secondary changes due to fetal peritonitis.

2. Malformations of the genito-urinary tract may likewise cause fetal peritonitis owing to the extravasation of urine into the abdominal cavity.

3. Spontaneous rupture of any of the hollow abdominal viscera with extravasation of their contents may result in peritonitis.

4. Congenital syphilis is frequently associated with fetal peritonitis (Simpson.[1]) Maceration of the peritoneum, as frequently seen in still births in congenital lues, should not be mistaken for true peritonitis.

5. True congenital tuberculosis may be a causative factor and Mya[2] believes that the toxic bodies circulating in the blood of a tuberculous mother may in themselves cause peritonitis without the presence of the specific organisms.

6. Various septic infections may pass through the placental circulation into the fetal body and may among other lesions cause peritonitis.

Symptoms.—The symptoms depend upon the degree of peritoneal involvement and the nature of the cause. In living infants where the process is localized, there may be but few symptoms at birth,

[1] Cases of Intra-uterine Peritonitis, Zentralbl. f. Gynäk., 1877, p. 48.
[2] Monatsschrift f. Kinderh., 1906, **4**, 341.

but such a process usually results in the formation of adhesions and the development of intestinal obstruction in surviving infants. More commonly the process is generalized, the abdomen distended, containing more or less effusion with resulting dyspnea and cyanosis and the early development of ileus.

Prognosis.—Premature infants with fetal peritonitis rarely survive the first days of life and even the cases of localized peritonitis usually result in early death because of the inability of the individual to withstand surgical interference.

Acute Peritonitis.—**Etiology.**—It rarely occurs as a localized affection in the premature. The most common sources of infection are:

1. Hematogenous, either through general sepsis or local infection in some distant part.
2. Infections through the umbilical cord.
3. Infections through the intestinal canal either through rupture of the intestines due to trauma (this is usually located in the region of the sigmoid flexure), or the passage of bacteria into the peritoneal cavity, either through the uninjured intestinal wall, or through the inflamed, ulcerated or gangrenous bowel wall.

Symptoms.—Violent vomiting, abdominal distention with either diarrhea or obstipation, usually temperature, although it may remain subnormal, rapid respirations and pulse, not infrequently marked icterus, and early collapse are the usual findings. The diagnosis is often impossible before death owing to the rapid development of similar findings in the premature from other causes, unless there is evidence of transmission from some localized source of infection, as about the umbilicus.

Prognosis.—Entirely unfavorable.

HERNIA.

Congenital Diaphragmatic Herniæ.—These hernias are described as true and false. The true diaphragmatic hernias are covered by the peritoneum and there is no direct communication between the pleural and the abdominal cavities. In the latter or false type there is really an extrusion of the abdominal organ, and therefore a direct communication between the abdominal and pleural cavities. The latter are by far the more frequent type and the left side is more commonly involved than the right. The diagnosis offers considerable difficulty in the premature and because of the commonly associated cyanosis they are usually diagnosed as congenital atelectasis, a diagnosis which is not greatly in error as the lung on the side involved is not infrequently entirely undeveloped. In contradistinction to full-term infants who may live to considerable age, premature infants usually succumb during the first hours or days of life with symptoms of asphyxia and cyanosis, usually due to gastric or intestinal distention within the chest cavity (Figs. 154 and 155, pages 256 and 257).

Ventral (Lateral) and Lumbar Herniæ.—Etiology.—They are usually due to defects or arrested development of the lateral abdominal or lumbar muscles. They may, however, result from increased intra-abdominal pressure.

Prognosis.—This depends on the extent of the hernia and the general development of the infant. Three cases which I have seen in premature infants who survived resulted in spontaneous recovery.

Treatment.—During the first months of life treatment must necessarily be limited to abdominal bandages or adhesive strips.

Umbilical and Inguinal Herniæ.—Navel and inguinal herniæ are especially common in the premature. Ylppö[1] found herniæ of one or both varieties in 84 per cent of the premature infants with a birth weight of less than 1500 gm. before the third month.

UMBILICAL AND INGUINAL HERNIÆ (YLPPÖ).

Weight in grams.	No. of cases.	Hernia present.	Per cent.
1000	1	1	
1001 to 1500	50	42	84.0
1500 to 2000	81	31	38.3
2000 to 2500	73	15	20.5

Inguinal herniæ are rarely observed at birth, usually developing when the infant is several days or weeks old, following intra-abdominal distention or severe crying in stronger infants. They are most frequently bilateral and are fairly common. The testicles are often undescended, leaving a direct communication with the abdominal cavity which is followed by rupture of the processus vaginalis communis through the canal. This is rather interesting when we note that the processus vaginalis is open at birth in the majority of infants prematurely born. The tendency to meteorism, which so commonly exists, enhances the development of hernias under these conditions. While incarceration is rare, and reduction usually easy due to the elastic walls, strangulation does occasionally occur and is always dangerous if neglected.

Treatment.—Operative interference is usually out of the question, and we of necessity have recourse to conservative treatment. Steel trusses almost invariably cause trauma and erosion, with the dangers of infection which are of graver importance than the dangers of strangulation due to the hernia. Conservative treatment by the use of yarn trusses as first devised by Fiedler[2] or by

[1] Ztschrft. f. Kinderh., 1919, **24**, 1.
[2] Zentralbl. f. Chir., 1906, **33**, 1161. Deutsch. med. Wchnschr., 1907, p. 105.

the simple truss designed by Dr. Andrew A. Gour offers the simplest and best methods of treatment, and in the majority of cases results in spontaneous cure (Fig. 157).

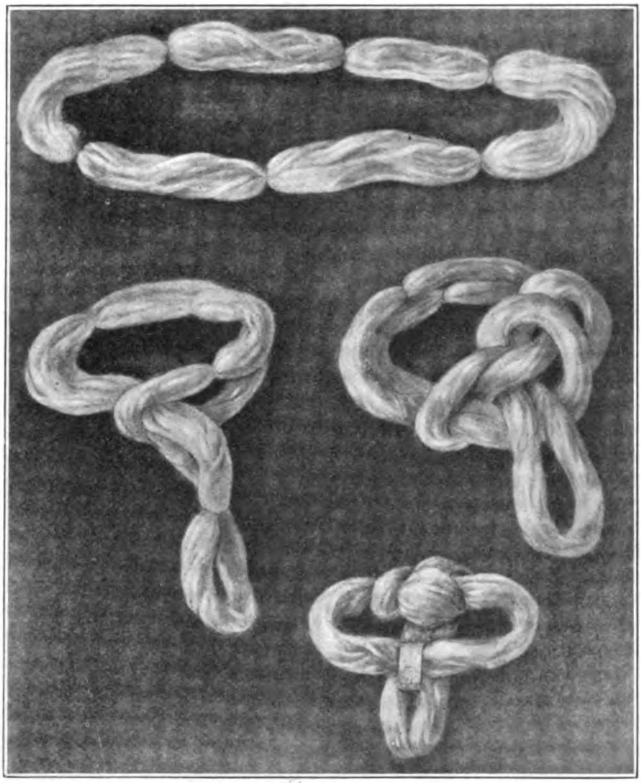


Fig. 156.—Illustrating the application in inguinal hernia. A pure wool, white yarn 4-ply is wound into a skein of fifteen to twenty strands, from 15 to 20 inches in length, depending upon the size of the infant to which it is to be applied. To prevent tangling it is knotted by a single strand at six points. A single loop is made which is passed around the body at the level of the crests of the iliac bones, with loop coming directly over the hernia. Fixing the loop at this point with the finger, the free end is now passed from above downward between the strands, making a snug knot which is fixed over the hernia. The free end is then passed between the thighs where it is fastened to the main loop over the back by tying with tape or by the use of a narrow rubber elastic to which snap fasteners are sewed. They are made to meet the needs of each individual case. Such a truss should be worn for two or three months or longer. The strand passing between the legs can be protected from excretæ by a cigarette made from oiled silk which can be slipped over the free end before fastening. Six skeins should be kept on hand. They can be washed in gasoline and soap and water. They should be stretched while drying.

The average mother or nurse can easily be taught to make the bandage from a good quality of poplin and of such size as will meet the infant's needs. It is usually necessary for the infant to wear the above improvised truss over a period of from one to three months.

FIG. 157.—Inguinal hernia bandage with small oval metal pad inserted on left side. (Dr. A. Gour.)

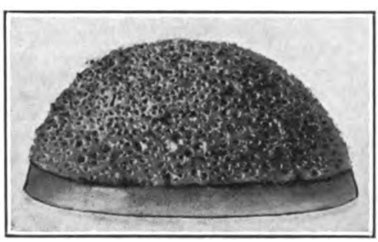


FIG. 158.—Pad designed by author for use with hernia bandage. An elliptical piece of fine pore rubber sponge is glued to a slightly larger piece of sole leather. The leather is perforated at four points so it can be stitched into the bandage. The rubber pad insures close approximation and elasticity to the bandage.

Umbilical herniæ are usually not extreme, most commonly developing during the first month increasing up to the third month. With proper care they often disappear completely by the end of the first year with the development of the recti and the other abdominal wall muscles.

This seems to be hastened when the child reaches the age of walking where the cases are not already healed. The chief factors in the causation of navel herniæ are the weak abdominal walls, the tendency toward rectus diastasis and the delayed healing and falling off of the cord, which averages eight to ten days in the premature as compared with five to seven in the full term. The diaphragmatic respirations are also a factor. Although we may have hernia of the umbilical cord proper, that is, true congenital umbilical hernia, the condition more commonly described as navel hernia is the acquired hernia of the umbilical ring due to deficient closure, resulting in the protrusion of the omentum, or the intestines or both through the ring resulting from increased intra-abdominal pressure. With proper conservative treatment operative interference is rarely necessary.

FIG. 159.—Umbilical hernia bandage; ½-inch cotton cigarette and strip of adhesive plaster.

The method as illustrated usually results in cure in from one to three months, and if a good grade of zinc oxide adhesive plaster is selected, there is usually little excoriation of the skin even in the premature, if the bandage is not applied until the umbilical wound is entirely healed and all granulation tissue has disappeared. It is, therefore, necessary to treat the umbilical wound by the open method until thoroughly dried. A small "cigarette-like" roll is made of cotton about ½ inch in length and from ⅛ to ¼ inch in diameter, depending upon the size of the hernia and the elasticity of the abdominal wall. This small cotton cigarette is then partially or entirely buried between the overlapping skin lateral to the hernia, and while it is being held by the operator (doctor, nurse or mother) a strip of adhesive plaster about 3 inches in length and $1\frac{1}{2}$ inches in width is applied directly over the umbilicus.

FIG. 160.—Umbilical hernia bandage. Cotton cigarette in place. The next step consists in burying the cotton by folding the skin over it.

FIG. 161.—Umbilical hernia bandage. Adhesive strap in place.

We have found the short strip of adhesive plaster preferable to the longer strips encircling the entire body, as it causes less irritation, allows greater motor activity on the part of the intestines, and is equally efficient in the treatment of these cases.

CHAPTER XII.

DISEASES OF THE URINARY TRACT.

SMALLER or larger *hemorrhages* in the kidney capsule are frequent, but extensive extravasations are rare. Minute hemorrhages on the renal surfaces are common. In the kidney substance hemorrhages are most commonly found at the apex of the pyramids and in the medulla. Besides this zone of predilection there is another at the junction of the cortex and the medulla. Here there are not always hemorrhages but markedly engorged vessels (venæ et arteriæ arciformes). Hemorrhages occur more often between the urinary tubules than in them.

Uric-acid infarcts are found as yellowish granules in the kidney pyramids of prematures, still-born or perishing after a few days of life. Hemorrhages are usually present also in the same regions. Bile pigment is precipitated in the kidney in the same areas in which there is the predilection for hemorrhage.

That *cylindruria and albuminuria* may be present without gross demonstrable pathological change was mentioned before. The transition from physiological to pathological albuminuria is not abrupt and the instance of severe albuminuria is infrequent.

Observations have been so few in the cases of *nephritis* in newly born prematures that an exact clinical picture has not been established. Quite frequently one finds parenchymatous or fatty degeneration of the kidneys following toxic or infectious conditions. Perhaps the most outspoken form of nephritis in the new born is the syphilitic. An interesting question is the influence of nephritis and eclampsia in the mother on the kidneys of the infant. One commonly sees cases where the infant is unaffected and the urine retains its normal character, sometimes even when the premature shows eclamptic symptoms. At times there may be the findings of a well-marked hemorrhagic nephritis which clears up within a few weeks. Infrequently the infant may show congenital edema and ascites. The presence of "hydrops fetus universalis" has been shown to have some relation to the presence of nephritis during pregnancy, with well-marked renal pathology, causing still birth or premature birth with death in a few days.

Shrunken kidneys have been demonstrated in the infant following chronic nephritis in the mother.

A relatively large portion of the cases of nephritis in the new born have been ascribed to infectious processes. Thus Mensi[1] examined 17 nephritic infants, ten to fourteen days old, and based the condition on infections secondary to the diseases of the respiratory and alimentary tracts.

ECLAMPSIA NEONATORUM.

The analogue of classical eclampsia in the mother is very seldom seen in newly born infants. These may show no untoward symptoms or may be prematurely born dead, or if alive succumb in a few days from degeneration of the organs, hemorrhages or nephritis. Convulsions in infants born of eclamptic mothers are quite rare. Esch,[2] in 1910, was able to collect only 32 cases from the literature and his own experience. The convulsions appear in the first days of life, sometimes a few minutes after birth, usually before the end of the second day. Involvement of the eye muscles is usually first noted, then cyanosis appears, followed by tonic and clonic spasms of the body musculature. The convulsions last but a few seconds, sometimes several minutes. The severity of the eclampsia in the mother seems to have no influence on the frequency of appearance of convulsions in the infant. If the children survive the first few days the prognosis is relatively good. The treatment is to force fluids by mouth, per rectum, subcutaneously or intravenously, in order to dilute the circulating toxins.

We have experienced severe toxemia, as evidenced by stupor and other nervous manifestations, in both premature and full-term infants fed on eclamptic and nephritic mothers' early breast milk. To avoid this catastrophe it has become our rule to examine the infants very carefully for toxic symptoms and in their presence to feed all such prematures human milk obtained from healthy women, during the first days or weeks of life.

PYELOCYSTITIS.

The appearance of an infectious process in the urinary tract, as pyelocystitis, is as possible in the newly born as in older nurslings. Although general infections appear relatively easily in the first days of life, nevertheless, clinical symptoms are often lacking. The presence of chills, fever and sweats, as noted in older children, is seldom observed in the first days of life, so that the diagnosis is only made by urinary examination, disclosing blood, albumin, pus cells in large numbers and not infrequently colon bacilli.

Pushing of fluids and the administration of potassium citrate to the point of positive alkalinization of the urine are the only therapeutic measures applicable to the premature.

[1] Rev. di clin. Ped., 1903, No. 8.

[2] Arch. f. Kinderh., 1909, **88**, 60.

CHAPTER XIII.

DISEASES OF THE NERVOUS SYSTEM.

MENTAL AND NERVOUS DISTURBANCES.

THE frequency of mental disturbances and other phenomena on the part of the central nervous system in premature infants has been variously estimated. Finkelstein states that mental disturbances and spastic phenomena are not more frequent in prematures than in full-term infants, but Ylppö strongly contests this statement. The attempt to express the frequency of permanent mental defects and other cerebral disturbances in percentages is only rarely possible before the end of the first year of life, with perhaps the exception of the typical Mongolian idiot. Demonstrable mental defects, either complete idiots or imbeciles, were found in 7.4 per cent of Ylppö's cases.

Mental defects in premature infants are frequently accompanied by other symptoms on the part of the central nervous system. The most common are the spastic paraplegias and diplegias. These are present in prematures with demonstrable mental defects in at least 75 per cent of all cases. However, mental development may be complete in the presence of spasticity of the extremities dependent upon cerebral irritation. In most instances this is secondary to intracranial hemorrhage. Paraplegia or diplegia was present in 3.1 per cent of all Ylppö's cases. These figures would certainly be much higher, had all the prematures remained alive, since most of the infants suffering from injury to the brain die very early. The cerebral affections occur the more frequently, the smaller the infant at birth.

In our experience mental disturbances and defects on the part of the central nervous system have been confined largely to those infants who survived from among the class of so-called *weaklings*. These are the infants who have suffered from intra-uterine disease or congenital malformations, traumata at birth, or postpartum dietetic errors and infection. Among the more mature that are normal for their fetal age the prognosis for a full mental development is good.

Treatment.—In the postmortem examination of infants dying of cerebral hemorrhage, Rodda[1] found over 50 per cent followed

[1] Am. Jour. Dis. Child., 1920, **19**, 268.

non-instrumental deliveries and many followed normal and easy births. In these cases the blood was found slightly or not at all coagulated. Cerebral hemorrhage was by far the most frequent cause of death in the new born in his group of cases. In many cases at postmortem, no torn veins were found in the cerebrum or cerebellum to account for the hemorrhage, and multiple hemorrhages were found in portions of the body where it was inconceivable that they could be explained by trauma. Over 25 per cent of all infants dying of cerebral hemorrhage showed this picture of multiple hemorrhages. An analysis of cases reported in the literature deepened the conclusion that these hemorrhages were due to factors other than trauma. Further study led to the conclusion that there was a disturbance in the coagulation time of the blood in the new born. It was found that the average coagulation time in the new born was seven minutes. In icterus, melena, jaundice, syphilis and non-traumatic cerebral hemorrhage, the coagulation time of the blood was prolonged. In melena it might be delayed to ninety minutes. The subcutaneous injection of normal blood was effective in cases in which there was delayed or slow bleeding.

The further treatment in those cases with a diagnosis of intracranial hemorrhage is symptomatic and expectant. There is always the possibility that there may be spontaneous cure. The infant must be kept quiet and warm. For the motor hyperirritability and convulsions narcotics may be employed, before all chloral hydrate (0.12 to 0.5 gm. per day per rectum), also bromides (0.25 to 1 gm. per day) or calcium lactate (1 to 2 gm.) or calcium bromide (0.3 to 0.5 gm. per mouth) per day.

Where the infants do not swallow well, feedings must be given per catheter or subcutaneous infusions must be used for emergency.

Lumbar puncture, although primarily a diagnostic measure, may have a beneficial therapeutic action. It is of diagnostic value when the hemorrhage is below the tentorium.

In the full term, cranial decompression when employed early has yielded favorable results, however little can be expected from such surgical interference in the premature.

Schulze's swingings and other violent measures for artificial respiration are distinctly contraindicated in the treatment of asphyxia in the premature.

For the paraplegias and diplegias, corrective measures should be undertaken early in order to prevent marked deformities. Massage and active and passive movements should be practised regularly beginning to advantage in the first year.

Muscle training in walking, climbing and other activities should be instituted under the supervision of a trained assistant.

Orthopedic appliances are frequently indicated.

Surgical procedures may be necessary later.

Another group is made up of premature infants with more or less serious mental defects in whom typical epilepsy gradually develops, often of the Jacksonian type. It is very difficult and often impossible to make a differential diagnosis between epilepsy and spasmophilia in the first attacks, and especially in those cases where the convulsions appear very early. Fortunately, as a general rule, the epileptic convulsions do not occur in the first year of life in prematures, while on the other hand electric hyperirritability and spasmophilic convulsions are quite frequent in this period of life. This makes the differential diagnosis somewhat easier.

On the other hand, however, in connection with febrile diseases of later life, convulsions occur very frequently in premature infants. Only the further course of the disease will show whether the convulsions are of epileptic or spasmophilic nature.

HYDROCEPHALUS; MEGACEPHALUS.

True congenital hydrocephalus is usually of the internal type with enlarged ventricles. The external form is very rare. Megacephalus must be differentiated from hydrocephalus, the two often being confused in the premature, as previously mentioned in the discussion of Pathology and Rachitis (pp. 104, 336). Internal hydrocephalus results from a transudation or exudation. Obstruction to the outflow may be the cause as in the case of intracranial hemorrhage or cerebellar cysts. However, most of the cases are probably due to an intra-uterine serous meningitis or meningoencephalitis of unknown origin. Syphilis is frequently the cause of congenital hydrocephalus,

The inflammatory process bringing about hydrocephalus may be at end by the time of completion of pregnancy, but usually persists thereafter. Most of the infants show enlargement of the head soon after birth or the enlargement becomes apparent at a later period. When the process begins early, intra-uterine, it may bring about a marked retardation in brain development. The head need not necessarily be enlarged; indeed the head may be small as in a microcephalic. The brain in these cases is really a large cyst. Often the skull is enlarged at birth, and it may hinder labor to such an extent that perforation or puncture of the head becomes necessary.

Where the head has the classic hydrocephalic configuration the diagnosis is, of course, easy. In many instances there are also

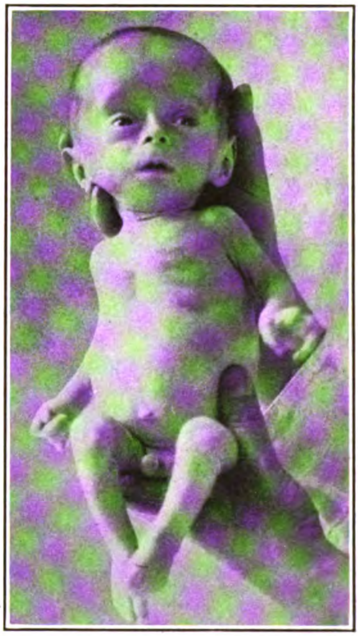


Fig. 162

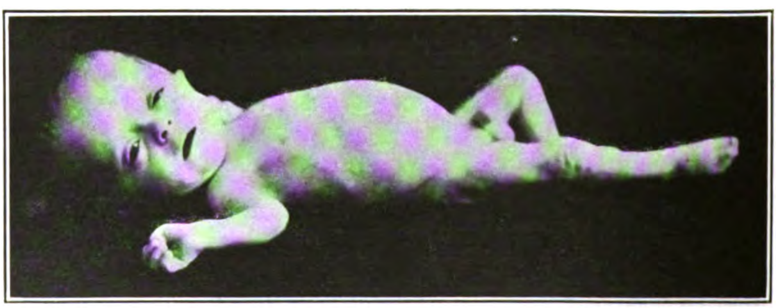


Fig. 163

Figs. 162 and 163.—Megacephalus. Baby P. H. at four months. Baby P. H. at six months.

the following symptoms at birth: Hypertonus and spasms of the muscles, increased reflexes, convulsions, psychic disturbances and apathy.

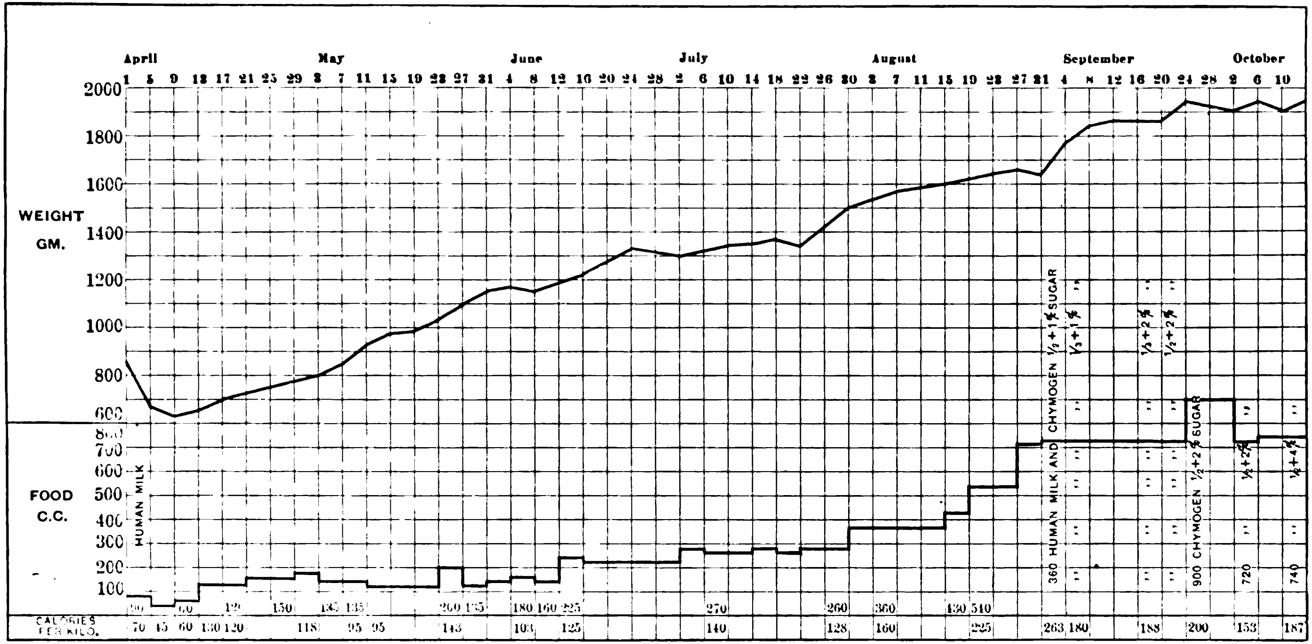


FIG. 164.—Baby P. H. Weight and food curves and caloric intake. Estimated fetal age, 32 weeks. One of triplets. Second died shortly after birth. Third born macerated. Born April 1, admitted April 1. Discharged, October 12; age, one hundred and ninety-three days. Birth weight, 840 gm.; lowest weight, 645 gm.; doubled birth weight in one hundred and fifty-three days; tripled lowest weight in one hundred and seventy-five days. (From service of Dr. L. E. Frankethal, Michael Reese Hospital.)

MEASUREMENTS.

Age, 23 days.				
Dates	April 23	May 13	June 20	August 28
Weight	725 gm.	922 gm.	1200 gm.	1720 gm.
Length	31.0 cm.	36.5 cm.	38.0 cm.	43.5 cm.
Occipito-frontal	..	8.75 "	9.5 "	11.0 "
Diameters:				
Biparietal	7.5 "	8.0 "	8.75 "	10.5 "
Bitemporal	6.5 "	7.5 "	7.0 "	9.75 "
Occipito-mental	7.0 "	10.0 "	11.5 "	12.5 "
Suboccipito bregmatic	6.5 "	6.0 "	8.0 "	9.25 "
Circumference:				
Occipto-frontal	..	27.0 "	30.75 "	35.0 "
Occipito-mental	..	28.0 "	33.0 "	37.0 "
Shoulders	..	20.0 "	25.75 "	28.0 "
Hips	..	16.5 "	20.0 "	22.0 "

Where the characteristic head is not seen and only slight enlargement of the fontanelle areas is noted, the diagnosis is difficult. Intracranial hemorrhage and meningitis must be ruled out. Lumbar or ventricular puncture is of great assistance.

FIG. 165.—Hydrocephalus. First signs when infant was four weeks old.

The prognosis is usually difficult to make early. The only early therapeutic measure is lumbar or ventricular puncture with drawing off of cerebrospinal fluid. Late surgical interference may be indicated.

Fig. 166.—Oxycephalus (Tower skull). Usually associated with other congenital defects and stigmata of degeneration. The skull is dome shape with bulging temporal regions. The deformity was present at birth. It is generally associated with exophthalmos, proptosis and frequently with other ocular abnormalities. Some children are mentally normal. Others subnormal.

The term *megacephalus* is applied to the conditions in which the head develops out of proportion to the other body measurements and length. It is characterized by an abnormally large head, with a relatively larger brain. This condition is a characteristic finding in a high percentage of infants prematurely born and is seen in inverse proportion to the fetal age and birth weight. Rosenstern,[1] in a series of sixty-one prematures observed over a period of at least three months, noted megacephalus in forty-four. He concluded that the lower the birth weight of the premature the more likely is megacephalus to develop.

RELATION OF BODY WEIGHT TO MEGACEPHALUS (ROSENSTERN)

Birth weight.	No. cases.	Present.				Absent.
		Severe.	Moderate.	Mild.	Total.	
to 1000 gm. . .	1	0	1	0	1	0
1001 " 1500 " . .	12	5	6	1	12	0
1501 " 2000 " . .	21	3	14	1	18	3
2001 " 2500 " . .	27	1	9	3	13	14
Total	61				44	17

[1] Rosenstern, J.: Ztschr. f. Kinderh., 1922, **22**, 129.

It usually occurs before the age at which rachitic changes are noted in the long bones and chest. However, as rickets occurs much earlier in premature infants than in full-term infants, I do not believe that we are at present in a position to dissociate these two conditions. There is therefore great probability that the same etiological factors underlying the development of megacephalus in the first months may be the cause of rachitic manifestations in the bones and other organs at later periods.

TIME OF OCCURRENCE OF MEGACEPHALUS (ROSENSTERN).

Age in months.	Cases.	Age in months.	Cases.
1	9	6	0
2	11	7	1
3	11	8	1
4	0	9–11	0
5	4	12	1

It is most frequently first seen during the second and third months of life and reaches its maximum between the sixth and eighth months. It then gradually becomes less manifest. There is usually an increased spinal fluid pressure in which it resembles hydrocephalus. The brain, on section, is found to be abnormally large but in true cases there is a complete absence of hydrocephalus.

Associated with the large skull and wide-open fontanelles and sutures, exophthalmos is frequently seen. The latter probably results from the lack of skull capacity, the eyes being protruded, with prominent cornea and, not infrequently, dilated pupils. Further characteristics of the head are a broad face, and mouth, nose and eyes which appear closely set together: the nose is stumpy and small and rises but little above the face; the tongue is often large and protruded.

ENCEPHALITIS.

The subject of encephalitis of the premature and full-term new born is still very much in the dark. The etiology is obscure and a clinical picture for the encephalitic processes has not yet been described.

Encephalitis interstitialis congenita was described by Virchow,[1] with changes in the medullary substance of the cerebrum, as a diffuse infiltration with fatty granular cells. Later, other observers declared that this was not pathological (Jastrowitz,[2] Limbeck[3]). Brain defects (porencephaly) have been linked with congenital

[1] Virchow's Arch., 1867, **38**, 129.
[2] Arch. f. Psych., 1870, vol. **2** and 1871, vol. **3**.
[3] Prague Ztschr. f. Heilk., 1885, **7**, 87.

encephalitis. Septic encephalitis is either a metastatic condition or a meningo-encephalitis, the difference between the two being almost impossible to define. The medullary substance shows clumps of bacteria and leukocytes, and later there appears a suppurative inflammation on the brain substance. Not infrequently in prematures the meningo-encephalitis is a distinctly luetic process (see Syphilis in Prematures, p. 330).

MENINGITIS.

The meningitic processes are as little understood as the encephalitic. They may be acute or chronic. Serous meningitis which is not well understood is supposed to be intimately related to congenital hydrocephalus. Pachymeningitis hemorrhagica interna seems to be a luetic process entirely (see Syphilis of Prematures, p. 330).

Purulent meningitis follows suppurative conditions in the middle ear, accessory nasal sinuses or is metastatic. Sometimes one sees typical meningeal symptoms as: Convulsions, rigidity of the neck, hypertonus, protruding fontanelles. However, meningitis may be present without any characteristic signs. The infants are flaccid, exhausted, and dried out. The diagnosis is verified by lumbar puncture. Fever is often absent or only present terminally.

The prognosis is absolutely poor. Death usually follows in twenty-four hours, but some linger eight to fourteen days. The inception of the process is difficult to fix because of the uncertainty of the symptoms.

Sinus thrombosis following middle-ear infections or phlebitis after navel infections sometimes are responsible for the meningitis.

Epidemic cerebrospinal meningitis is not an uncommon complication in premature infants during the first year. A spinal puncture should be made in every case showing marked evidence of cerebral irritation. In positive cases serum should first be administered intravenously through the longitudinal sinus, because of the tendency to generalization of the infection in this class of infants. Intraspinal administration of serum must always be made by the gravity method after withdrawal of as much fluid as is to be administered.

Finally we find among the prematures a number of idiots that have to be classified as "degenerative idiots." These are the infants that at birth already show stigmata of Mongolism or other malformations. These children are prematurely born with special frequency, and it follows therefore that a considerable number of children with Mongolian idiocy are prematures. After all, it is a known fact that children with various congenital malformations, be they congenital bone diseases, bone anomalies, congenital heart disease, malformations of the brain or spinal cord, etc., are born in an immature condition. This circumstance, as previously mentioned, is the reason that prematures have been very generally but erroneously regarded as congenitally inferior.

SPASMOPHILIC CONVULSIONS.

With reference to spasmophilic convulsions we must not regard them as purely functional convulsions; on the contrary the readiness with which they occur and their frequency in premature infants speaks very strongly for organic lesions, probably most frequently among these being cerebral hemorrhage occurring during labor. Naturally certain extra-uterine noxæ, as anemia and rachitis, are of importance as determining factors that make the spasmophilic disturbances manifest. Numerous roentgenological examinations of the long bones of premature infants have shown that the rachitic changes are not confined to the skull, but that the other bones are also early affected, as early as the second and third months of life. (See p. 347.)

CHAPTER XIV.

SEPSIS.

THE term sepsis may be defined as an invasion of the system by pyogenic cocci or other equivalent organisms. These bacteria may attain entrance through various atria and may spread by means of the blood stream or may remain at the point of invasion and from there discharge the products of their activity into the blood of the infant.

Bacteriology.—The bacteria occupying the first place among those causing sepsis are the pyogenic cocci, the streptococcus and staphylococcus, both albus and aureus. With them may be included the pneumococcus and colon bacillus. The colon group includes the paracolon and paratyphoid varieties. Of other bacteria there are found more rarely the Bacillus pyocyaneus, Bacterium lactis aërogenes, Bacillus enteritidis (Gartner), proteus bacillus, the gonococcus, the influenza bacillus and the meningococcus. Infection with Treponema pallidum is treated as a specific disease.

Blood examination during life and immediately after death in cases of sepsis in the premature gave the following results:[1]

Blood culture positive	15
Blood culture negative	4
Percentage positive	75.3 per cent
Streptococcus	6 times
Colon bacillus	5 "
Staphylococcus	1 "
Pneumococcus	1 "
Influenza bacillus	1 "
Colon bacillus and influenza bacillus	1 "

Ylppö,[2] in a small series of prematures, found:

BACTERIA IN BLOOD OF PREMATURES

Age, days.	No. of cases.	Bacteria found.
0 to 1	8	0
2 to 3	7	2
4 to 15	14	10

[1] Delestre: Infections chez le prématuré, Paris, 1901.

[2] Pathologisch-anatomische Studien bei Frühgeburten, Ztschr. f. Kinderh., 1919, **20**, 371–372.

In 70 per cent of the infants perishing between the fourth and fifteenth day of life, Ylppö was able to demonstrate bacteria in the blood. In 12 cases the following organisms were noted:

Bacillus coli	6 times
Staphylococcus	3 "
Streptococcus	1 "
Not identified	2 "

The frequency of Bacillus coli, he believes, speaks for an internal basis for the infection.

The Time of Infection.—This may be either intra-uterine, intrapartum or postpartum (extra-uterine). *Intra-uterine* infection may occur either through the placenta, or by way of the liquor amnii. Should the mother be suffering from a septic infection, the causative organisms may pass through the injured placental wall, which ordinarily is sufficient to exclude bacteria from the fetus.

Infection of the liquor amnii may occur before or after the rupture of the membranes. Before rupture the infection may occur by contiguity of tissue, the organisms coming from the peritoneal cavity, rectum or bladder. Infection through the vaginal canal with unruptured membranes probably does not occur, the cervical opening being obstructed by what Delestre[1] calls the "gelatinous stopper of pregnancy." Vaginal infection, therefore, usually stops beneath the internal os. But once the membranes rupture, infection can occur by the ascension of bacteria from the vagina. This, however, is uncommon.

Our knowledge of infection through the liquor amnii is more definite. Lehmann[2] states that bacteria can pass through uninjured membrane and reports cases in support of this statement.

Intrapartum infections occur during the passage of the infant through the maternal birth canal. Local infection occurs first and this may be followed by general sepsis. The atrium of infection may be the mouth, the digestive tract, the lungs after aspiration of infected vaginal mucus or amniotic fluid, or wounds of the skin. The eyes especially are subject to infection at the time of birth, but fortunately infection there remains local.

Post partum, the most important sources of entrance of infection are the umbilicus, the skin, the gastro-intestinal tract and the respiratory apparatus. As compared with intra-uterine and intrapartum infections those of extra-uterine origin are much the most important.

Umbilical infections through the physiological wound made at the time the cord is severed are the most frequent of all infections

[1] Thèse, A Study of the Infections of the New Born, Paris, 1901.
[2] Thèse, De l'infection amniotique et de ses consequences pour 18 enfants, Paris, 1899.

after birth and this forms the most common portal of entry for sepsis. At birth the most important structures found in the umbilical cord are the two umbilical arteries which conduct the blood from the fetus to the placenta, and which arise from the common iliac arteries, and the umbilical vein which carries blood from the placenta to the inferior vena cava *via* the left branch of the portal vein and the ductus venosus arantii. Immediately after birth the cord is ligated and cut, there remaining a stump a few centimeters in length which undergoes desiccation during the first few days of life and which separates on the fourth to the ninth day with a slight inflammatory reaction. In the premature and debilitated the falling off of the cord and subsequent cicatrization of the base often occurs later. Ordinarily the base is covered by epithelium by the end of the third week or a little before, but infection of the umbilical wound may occur at any time up to the moment of healing, and is especially common in prematures.

Of second importance as a gateway for the entrance of pathogenic bacteria is the *skin.* The frequent abrasions which occur during birth, erosions from too severe efforts at mechanical cleansing, the pemphigus lesions and the intertrigo so common in the neglected weaklings, all form portals of entry for bacteria. In the premature especially the skin is delicate, lacking the horny layer which in the better developed tends to prevent the occurrence of abrasions. Furunculosis and abscess formation are often the precursors of a general infection.

The *respiratory tract* is a frequent means of entry through the occurrence of a simple or suppurative rhinitis, otitis media, bronchial infection with epithelial necrosis or bronchopneumonic inflammation. It must be remembered that pulmonary inflammations are prone to develop secondarily in sepsis and unless evidence of pulmonary affection can be shown early in the course of sepsis, it may be difficult to say whether it was primary or secondary.

Of nearly equal importance is the *gastro-intestinal* tract as an atrium of infection. The buccal mucosa may be the seat of mucous patches, of Bednar's aphthæ on the palate, of thrush, of stomatitis, or gingivitis, of ulcerations from too vigorous cleansing, or of abrasions due to the passage of the tracheal catheter. The intestinal wall of the premature and even the full-term newly born weaklings may be permeable to bacteria which cannot pass through the intestinal wall of better developed infants.

We believe that though the gastro-intestinal tract is frequently the seat of ulceration in the stage of atrophy in infants, a condition more rapidly developing in the prematures than in full-term infants, and therefore offering numerous portals of entry for systemic infection, every attempt should be made to exclude all other atria before accepting the gastro-intestinal tract as the source of infection.

Genito-urinary infections are of importance as the source of sepsis in the premature newborn.

Susceptibility.—The premature is especially receptive to infection with the organisms of sepsis, seemingly possessing an extremely low resistance. The organs in which the leucocytes are formed are but imperfectly developed and the leucocytes themselves are deficient in phagocytic power; other organs are incomplete, the individual cells are immature and the lymph glands are of little importance in these infants, and fail to enlarge in the presence of infection.

The frequent subnormal temperature of these weaklings encourages this ease of infection, experimental evidence showing that to lower the temperature of an organ is to lower its resistance, and diminish phagocytic activity and the bactericidal energy of the blood. Without doubt there is also a deficient formation of antibodies in these premature infants (Pfaundler.[1])

Artificially-fed prematures possess a relatively greater susceptibility to septic infection than do breast-fed infants, a fact which may in part be explained by the fact that human milk is rich in protective substances in contradistinction to cow's milk.

In sepsis the difference between the infants born of healthy parents and those of diseased parents is marked. The healthy premature is formed of young cells, full of vitality and only requiring growth to perfect themselves, and capable to some degree of resisting the organisms of infection with which they are continually surrounded. The others are already affected by the toxemia of the parental disease, or are themselves directly involved, and thus their cells have their vitality reduced and so offer a medium already prepared for infection.

The frequency of sepsis among the new born is today very much less than it was in preaseptic days. Proper care of the hands and the conduct of labor, sterilization of instruments and dressings, has greatly reduced the incidence of this condition. The fact that infants tend more often to become septic in a hospital or asylum than in the home is to be accounted for by the greater frequency of infecting organisms in the former, where many sick are congregated, and by the fact that one attendant often cares for several infants in the same hospital or ward (Meyer[2]).

General Manifestations.—The onset of sepsis may occur at any time during the first days of life or the infant may be born with an infection present. The course varies, some almost without any

[1] Die Antikörperübertragung von Mutter auf Kind, Arch. f. Kinderh., 1908, **47,** 260; 1908, **48,** 245.

[2] Hospitalismus, Berlin, 1913; Ges. f. Gynäk., 1911.

symptoms which can be interpreted as involving any one set of organs, death occurring suddenly after collapse.

Local symptoms, if present, are dependent upon the situation of the primary infection or of secondary metastatic foci, while the general symptoms are those of a septicemia.

The *septic fever* in the premature infant does not possess those characteristics found in older children. The center for heat regulation lacks stability and the reaction to toxic influences is slight. The more robust infants may show a rise of temperature which may reach 105° F. or even higher, and which may run a more or less regular course. In those born considerably before term, and in the weaklings there may be little or no temperature reaction, in fact in these latter a subnormal temperature is the rule. Chills do not occur in these weaklings.

Loss of weight is likely to be rapid and great, depending upon the ability to take food and the degree of intestinal involvement, being due to disintegration of tissue, to loss of water, and to inability to take food and fluids. The pulse-rate is rapid and the quality is usually poor. Respirations are often irregular. Cerebral symptoms are common during the final stages, the infant has a prostrated appearance and is apathetic. The cry becomes more feeble and the movements less frequent than usual. The skin loses its turgor; anemia becomes evident and the skin color becomes grayish or, if icterus exists, yellowish. Occasionally there is a cyanotic tinge to the entire body surface. Hemorrhages are very common during the course of sepsis, occurring from the mouth, bowel, navel or into the skin.

Skin.—Icterus is a very frequent finding in the first few days or weeks of life and is especially frequent in premature infants and in the victims of sepsis. Particularly with umbilical infections is the icterus of marked degree. Edema of the feet and legs occasionally occurs and expecially in premature infants scleredema, or even sclerema, may occur toward the end of the disease. Hemorrhages into the skin are common in sepsis, being seen over the trunk and extremities, usually as petechiæ. In some instances they may be purpuric, or effusive in character. Pemphigus-like blebs with bloody contents are a frequent complication.

Inflammation of the *umbilical vessels* is a frequent primary process in a general sepsis. Most often the umbilical arteries are involved, and less frequently the vein. The amount of involvement varies, occasionally extending just a short distance within the abdominal wall, sometimes the entire length of the vessel, in which latter instance the thickened vessel cannot infrequently be palpated through the abdominal wall. Septic thrombi or pus may be present in the umbilical vessels, and pus can often be squeezed out from the stump of the cord. Inflammation of the umbilicus or of the abdominal wall in its immediate neighborhood may be present.

Omphalitis alone is sometimes seen. The usual termination of this infection is in abscess formation, but occasionally an inflammation of an erysipelatous character spreads to the abdominal wall (Holt[1]).

Nervous symptoms are many. They may depend solely upon the toxemia, or be due to an intercurrent meningitis, encephalitis or edema of the meninges. Most often the infant lies quietly in a stuporous condition, at other times there are restlessness, tremors, spasms, jactitation, dilated pupils, bulging fontanel, spasticity of the muscles with rigidity of the neck, and in cases of meningitis and encephalitis, paralyses.

Gastro-intestinal manifestations are practically always present. In the *mouth* are seen ulcers, fissures, stomatitis and purulent inflammations of the salivary glands. Not infrequently sepsis will run its course with clinical pictures of dyspepsia with secondary anhydremic intoxication, with vomiting and diarrhea as marked symptoms. The vomiting and diarrhea are manifestations of the toxemia, emesis being frequently cerebral in origin. The mesenteric glands are infiltrated and the gastric and intestinal mucosa are the seat of hemorrhages and frequently show evidence of inflammation.

Peritonitis is a rather frequent complication, either general or local. Oftentimes it is accompanied by an umbilical inflammation. Many cases are purulent, fluid being present. Adhesions of intestinal coils to each other or to the abdominal wall occur. The symptoms of this condition are abdominal distention and rigidity with tenderness, vomiting, umbilical protrusion, thoracic respiration and flexion of the thighs. Diagnosis of the condition is not at all easy as the presence of fluid is difficult to demonstrate. Probably the finding of greatest value in these infants is abdominal tenderness.

The *spleen* is usually enlarged. The *liver* shows evidence of an acute hepatitis, and not infrequently there are multiple foci of suppuration.

Involvement of the *circulatory apparatus* in sepsis does occur but is not very frequent. Pericarditis is commoner than endocarditis. The former usually arises by extension from the pleura.

The myocardium is frequently the seat of parenchymatous degeneration and hemorrhage.

The *respiratory organs* are involved very frequently in the picture of sepsis. Pneumonia is the most frequent lesion met with, and as usual in the weakling or premature, is difficult of diagnosis, especially when the process in the lung is not extensive, with lesions

[1] Diseases of Infancy and Childhood, D. Appleton and Company, New York, 1913.

small and multiple. The lungs show areas of bronchopneumonia, areas of atelectasis, alveolar fatty degeneration, hemorrhages into the alveolar walls of multiple abscesses. Effusion into the pleura is uncommon.

Rapid respiration and cyanosis are about the only symptoms which are seen in these cases of pneumonia. Occasionally the rapidity of breathing may occasion the belief that the lungs are the seat of a pneumonic process, when its presence is only the result of severe intoxication.

The *kidneys* usually show parenchymatous degeneration and hemorrhagic nephritis, with occasional necrosis of the epithelium and pyelitis. The albumin which is found in the urine is either the result of the action of the absorbed toxins on the kidneys or is the expression of the nephritis or pyelitis. In nephritis there will be found hyalin, epithelial or granular casts, and in pyelitis, pus cells and epithelium.

Bones and Joint Inflammations.—Rarely the bones are involved in an osteomyelitis and the joints are sometimes the seat of acute suppuration, usually several being involved at the same time. Immobility and swelling over the involved joints are the common symptoms seen. Pain is present and crepitus can be elicited when epiphysial separation has occurred.

Unfortunately the *blood* is of little value in completing the diagnosis, because of the usual absence of leucocytosis. A positive diagnosis is possible by finding the causative organism in the blood. The difficulties to be met in making blood cultures in premature infants must be remembered. The longitudinal sinus is the best source for obtaining blood.

Course.—In the premature the course is usually acute. Often the first symptom is loss of appetite; the child refuses to take the breast, or if artificially fed, it vomits. Convulsions may usher in the condition, followed by icterus which increases in intensity and soon is accompanied by diarrhea. Cyanosis may next make its appearance, the accompanying dyspnea being hard to detect because of the slight amplitude of the respiratory movements. It is sometimes revealed by movements of the alæ nasi or by an increased frequency of respiration, or by change in the respiratory rhythm, consisting of short inspirations followed by relatively long expirations. Occasionally the respiration is slow, feeble and superficial, because of the impermeability of the lungs involved by atelectasis.

Some cases of sepsis prove fatal in a few hours; the younger the infant and the weaker the condition at birth, the shorter the course as a rule. Symptomless sepsis is frequent in the premature.

Prognosis.—Septic infection in the very young is a fatal disease and the more immature the infant, the worse the outlook. In the lesser degrees it offers a grave prognosis and in the severer forms it is practically always fatal. Involvement of a large number of organs makes the lethal outcome almost certain.

Prophylaxis.—Since the treatment of sepsis in the premature new born offers so little, it becomes of prime importance to prevent the development of the disease, and sepsis may be considered as preventable. The vulnerability of the new born and particularly of the premature new born, who is deficient in vital functions, to the invading organisms of disease is notable, and the fact that sepsis occurs particularly in institutions makes the care of these infants of great importance.

Infection which reaches the child before birth is beyond our control, but subsequent to that time very much may be done to prevent the disease. The care of the umbilical wound is of great importance; instruments used in dividing the cord, the cord tape and dressings must all be aseptic. In hospitals the infant should be kept in a separate room from the mother, and the same attendant should not look after both mother and infant. The hands of the attendant and of the mother when she handles the child must be cleansed thoroughly before the child is touched. The nurse should wash her hands after the care of an infant before passing to another in the nursery. All articles which come into contact with the infant's mouth—nipples, feeders, spoons, gavage tubes, etc., must be sterilized before use. All utensils should, so far as possible, be individual. The mouth of the infant must not be traumatized and all rough handling or other body trauma must be avoided. The breasts of the nursing mother must be washed thoroughly before each nursing and protected between the nursing periods by covering them with thin, clean gauze.

Strict asepsis during delivery will do a great deal toward reducing birth infection to the smallest amount, while care in internal examinations before delivery will do much toward lessening the infections of the amniotic fluid. Lochial secretions can become the source of infections and their care is important. They should be disposed of at once.

In private families where there is not sufficient help and one person must attend to mother and child, the infant must be taken care of first, and the mother later.

To facilitate cleanliness the new-born infant should be given a a daily warm sponge, unless very weak, and the diaper should be changed frequently to prevent the development of intertrigo. The use of a dusting powder in the skin folds often acts as an irritant.

The room in which the infant spends its time should be kept at a temperature warm enough to meet the needs of its individual development if it is hypothermic. The air of its room should always be kept pure and fresh and light freely admitted. The clothing of the infant should be warm enough, but not too heavy, being suited to the surrounding temperature and to the individual needs of the child. It should not fit so tightly as to prevent movement of the arms and legs.

Only the greatest cleanliness of the skin and umbilicus will prevent infection. The falling-off of the cord and the subsequent cicatrization is, as a rule, delayed in prematures, and infection is favored. Wet compresses not infrequently macerate the delicate skin and so dry or alcoholic dressings are advised, best without dusting powder which is likely to cake and prevent absorption of the exudate.

The existence of an angina, rhinitis, bronchitis or any other form of infection, in the mother or nurse, make the separation of the infant from the mother or a change of nurses imperative. Masks must be worn by all infected individuals coming in contact with the infant.

Active Treatment.—This promises very little, as we possess no specific and our efforts must be directed chiefly toward the treatment of individual symptoms, as they arise. If abscesses occur they must be opened and drained. The strength must be supported by judicious breast-milk feeding if this be possible, and by the use of stimulants in 1 to 5-drop doses of brandy or whisky every two hours. In collapse stimulation must be resorted to, the most useful being camphor-in-oil, 1 to 3 minims hypodermically. Spiritus ammoniæ aromaticus, 1 to 3 minims by mouth, well diluted, every three or four hours is often of benefit. Infusion of digitalis or digalen in minimum doses may be used to support a failing heart.

Fluids should be pushed by mouth in the endeavor to dilute the circulating poison. Gavage feeding should be instituted without too prolonged delay. The use of saline transfusion has found great favor in recent years. Seven-tenths of 1 per cent sodium chloride solution may be injected subcutaneously beneath the breasts or into the loose areolar tissue of the interscapular region in quantities of $\frac{1}{2}$ to 2 ounces (15 to 60 cc) and repeated if indicated. The danger of infection must be remembered. Great elevations of temperature, if present, are to be controlled by tepid baths but care must be taken to avoid collapse. Often, these premature infants do not react to infection with temperature, and in such cases warm baths are indicated. Mustard baths or mustard compresses are of value in collapse.

CHAPTER XV.

SYPHILIS.

Among the most important factors producing *premature birth* syphilis ranks high. It is even more frequently the cause of intra-uterine fetal death. The greater the severity of the infection, the greater is the likelihood of still birth; they represent an overwhelming of the fetus by the spirochetes. Infants who show signs of syphilis at birth have a very high mortality percentage and in the case of those prematurely born, almost all die. The prognosis is much better in those developing clinical evidence one or more weeks after birth.

Jeans[1] found that (in his out-patient department):

"From 10 to 20 per cent of adult males and about 10 per cent of married women are syphilitic and a minimum of 10 per cent of marriages involve a syphilitic individual.

"Seventy-five per cent of all the offspring in a syphilitic family are infected.

"In a syphilitic family 30 per cent of the pregnancies terminate in death at or before term, a waste three times greater than is found in non-syphilitic families.

"Thirty per cent of all the living births in a syphilitic family die in infancy, as compared to a normal rate of 15 per cent in the patients coming under his observation.

"About 5 per cent of our infant population is syphilitic.

"According to St. Louis vital statistics, 3.5 per cent of all infant deaths are ascribed to lues."

Premature infants do not necessarily show symptoms of lues at birth. In fact, in the majority of cases syphilis becomes manifest only after a latent period and this may vary from one week to one or more months. The later the development of the manifestations the more likely is the infant to be viable. Cutaneous manifestations are usually preceded by coryza, splenic enlargement and retarded progress. While some of the infants, and this applies more especially to the later pregnancies of syphilitic mothers, may be well nourished at birth, more often the earlier pregnancies present a characteristic picture, even in the absence of specific cutaneous manifestations. The skin is flabby and wrinkled and

[1] Am. Jour. Syph., St. Louis, 1919, No. 1, vol. 3.

the facial expression senile—approximating the picture of extreme marasmus or athrepsia in older infants. This class usually perish shortly after birth and the postmortem examination reveals marked luetic, visceral changes.

Infants born with luetic eruptions usually evidence a more or less marked degree of visceral change and they run a much more serious course and give a worse prognosis. However, even in the premature the appearance of the cutaneous lesions need not necessarily be associated with marasmus. This is more especially true in cases unassociated with deep-seated visceral changes, hence the clinical picture of lues is enormously variable and all transitions occur from the serious generalized syphilis up to the case involving a single organ or set of organs. When lesions are present at birth, one or more of the following are usually in evidence: Coryza (snuffles), bullæ on the hands and feet and splenic tumor.

Mucous Membranes.—Coryza is most often the first symptom. In its onset it resembles an ordinary cold but is soon characterized by its severity and chronicity. The discharge is profuse, becomes mucopurulent and often tinged with blood. Nasal obstruction results from the formation of crusts. Mouth breathing follows and nursing becomes difficult. Pharyngitis and laryngitis are usually associated with a resulting characteristic hoarseness and aphonia.

Mucous patches and ulcerations develop on the mucous membranes and at the mucocutaneous surfaces, especially at the mouth, anus, vulva and scrotum.

Skin Eruptions.—When not present at birth the skin eruptions usually follow the development of the coryza but they need not necessarily be preceded by it.

The most common lesions, and which are very rarely seen at birth, are of two types, a diffuse more or less generalized skin infiltration. The skin becomes thickened and infiltrated and loses its elasticity and often after a short period the superficial layers crack. The skin in greater part has a waxy appearance with interspersed inflamed areas, more especially at the points of fissuring. This characteristic skin change may involve the entire body or appear in isolated areas, of which latter the face and extremities, more especially the hands and feet, are more likely to be the seat of changes. About the face, the region of the mouth, nose and eyelids are the sites of predilection, with frequently resulting rhagades in these regions. A massive involvement of the face results in a mask-like appearance. Following fissuring, there frequently results an exudate with later crust formation. When the scalp is involved alopecia usually results and the same may be true when the eyelids are deeply infiltrated.

The soles of the feet and palms of the hands usually present a diffuse infiltration and appear firm and shiny, sometimes more reddish or bluish red, at other times a copper-red or brown.

A true paronychia, which is often accompanied by complete destruction of the nails, is an almost constant complication in this type of skin lesions.

The surface is either smooth or shows fissures in the uppermost horny layers of the epidermis, which occasionally sloughs in large, lamellous scales.

In the second type of rash lesions which are more circumscript are noted. These lesions assume more nearly the characteristics

Fig. 167

Fig. 168

Figs. 167 and 168.—Congenital syphilis. Secondary lesions on face, body and hands and feet. Lesions first appeared during fourth week.

of the skin manifestations in acquired syphilis. The most frequent type of lesions are macules usually circular and slightly elevated, averaging 2 to 5 mm. in size. The face and the extensor surfaces of the upper and lower extremities and more especially the hands and feet are usually involved and they may cover the entire body, but more often the chest and abdomen escape. At first red, they soon become darker and assume a coppery hue. More elevated papules similar in character and without an inflammatory base may be interspersed among the macules. A squamous eruption is frequently seen upon the palms and soles and small masses of scales may appear upon the surface of the macules. The eruption may develop abruptly but more frequently it increases progressively during a period of from one to three weeks and under vigorous treatment disappears rapidly except for the remaining pigmentation.

In the most severe types the bullous or pemphigoid lesions may be superimposed upon the macular squamous syphilides or they may be primary. They may lead to deeper ulcerations of the skin with secondary infection and are always a source of danger to others because of the likelihood of the presence of spirochetes in the lesions. The possibility of the confusion of these lesions with non-specific pemphigoid lesions which are of not infrequent occurrence in obstetrical wards should be remembered as the latter are especially prone to affect the premature. This latter type of pemphigus neonatorum is probably a staphylococcus infection. Linear fissures and mucous patches are among the most charac-

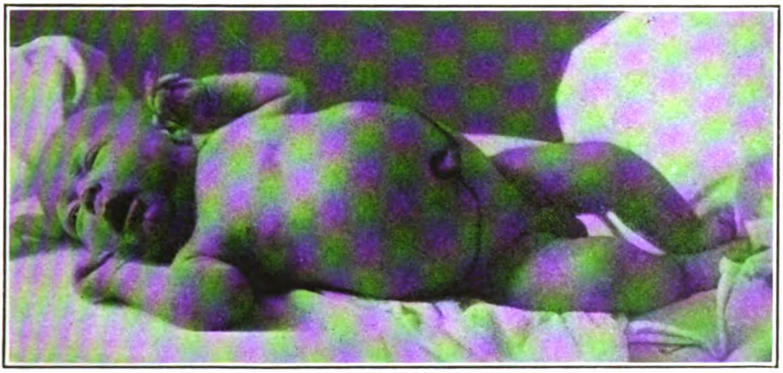


Fig. 169.—Congenital syphilis. Baby A. Fissures about mouth. Large liver and spleen. Six weeks later.

teristic features. On account of the fragility of the skin these rhagades easily occur, especially on the lips, nose, about the anus and less frequently about the eyelids. The healing of these lesions usually results in radiating cicatrices which result in the very characteristic "purse-string" deformity.

Umbilical Cord.—The umbilical cord often heals slowly and the stump has a tendency to become purulent and there is a tendency toward infiltration about the umbilicus. A more or less deep-seated ulcer may result which heals slowly unless constitutional treatment is instituted or mercurials are applied locally. Hemorrhage from the stump and secondary infection, with resulting syphilis hemorrhagica neonatorum are likely to result.

Lymph Glands.—Only exceptionally do they present a characteristic generalized enlargement in the new-born syphilitic premature.

In untreated cases the lymph glands become palpable and this may be due to luetic infection or, again, isolated groups of glands may become involved through secondary infection and go on to suppuration. The small size of the glands makes them difficult of palpation, more especially in well-nourished infants.

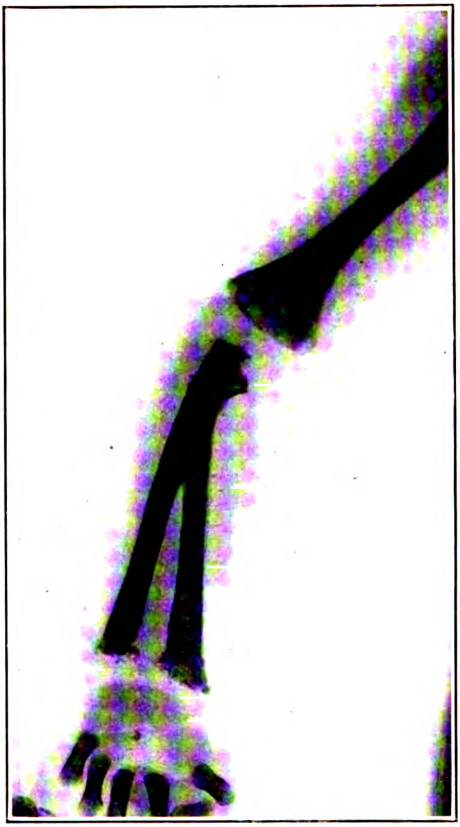


FIG. 170.—Osteochondritis syphilitica.

Osseous System.—Osteochondritis syphilitica ranks next in importance to the skin and mucous membrane lesions and splenic tumor in the diagnosis of syphilis. Pathological changes are most frequently seen in the long bones, the junction of the epiphysis with the diaphysis being the seat of predilection.

These lesions are usually bilateral although occasionally only a single lesion can be defined in the living. Involvement of the joints is far less common. While the long cylindrical bones are the seat of the lesions which can most easily be defined clinically, any of the bones may be the seat of a diffuse involvement of the bony structures or a periostitis. Such lesions are more commonly found in cases resulting in early fetal death (Fig. 170).

Roentgenography offers one of the best diagnostic methods for syphilis in the fetus and new born. Shipley and his co-workers[1] found evidence of syphilis in the osseous system of 25 per cent of 100 white fetuses ranging from six months of intra-uterine life to nearly term. Fifteen of these showed advanced luetic osteochondritis. The bone lesions in syphilitic new-born infants present characteristic lesions when there is sufficient involvement to be evidenced in the roentgenographic plates. While any of the bones may suffer, those most commonly involved and easiest of study are the lower end of the femur, upper and lower ends of the tibia, radius and ulna and the metacarpals. In their studies they found that the fetal type of reaction and the changes before birth were to a large extent confined to the epiphyseo-diaphyseal region, at which points there develops an abnormal arrangement and distribution of osseous tissue. After birth the periosteal reaction begins, possibly because of the increased demands made on this tissue by the increased muscular activity, and in young infants this may be the most marked skeletal lesion. The most characteristic lesions described by them were the following:

"The beginning of the process as shown by the roentgen-ray picture is an intensification of the shadow cast by the bone at the epiphyseal line. This line becomes much broader and more homogeneous and seems to form a cap on the ends of the trabeculæ of the spongiosa (Fig. 172). This is significant of the beginning of abnormally heavy calcification of the provisional calcified zone. It must be remembered that while the provisional zone of calcification in the cartilage of the normal embryonic bone is, relatively speaking, very narrow, in many cases only one or two cells deep, in the syphilitic bone the calcified cartilage may show on section a width of from 0.5 to 1.5 mm.

"In other bones, in which the osteochondritis is further advanced, it can be seen that on the marrow side of the intensified shadow of the provisional zone there is a band-like area where the shadow is less intense than in the rest of the bone (Fig. 170), giving an appearance of diminished density to the region of the epiphyseal line.

"Bones may also be seen in which the dense shadow at the epiphyseal end of the bone is broken by the presence of one or more small

[1] Shipley, P. G., Pearson, J. W., Weech, A. A., and Greene, C. H.: Bull. Johns Hopkins Hosp., March, 1921, p. 75.

FIG. 171.—Hand and forearm of human fetus to show extreme excessive calcification of the provisional area with irregular prolongation of the provisional calcified zone into the area of proliferative cartilage. Note the presence of the same lesions in the metacarpals and phalanges. (Shipley.)

FIG. 172.—Radius and ulna from human fetus showing beginning resorption of the area of intense calcification at the epiphyseo-diaphyseal junction. Resorption shown by areas of decreased density of shadow, each resorptive area surrounding a small nucleus of persistent trabecular tissue. (Shipley.)

FIG. 173.—Roentgen-ray picture of syphilitic osteochondritis of the bones of the hand and forearm of a human fetus showing a zone of rarefaction between two lines of abnormal calcification. Note the lesion in the phalanges and metacarpals. (Shipley.)

FIG. 174.—Syphilitic periostitis of both bones of the forearm. Note the longitudinal striation of the thick periosteal shadow which is nearly in contact with the shafts of the bone. (Shipley.)

areas of rarefaction so as to give an appearance of irregular density to the end of the bone (Fig. 173).

"At other times the bone appears to end in a double line, so that two lines of heavily calcified tissue are seen, separated each from one another by a zone in which lime salts are less heavily deposited. This zone is a region which histological preparations show to contain a great deal of delicate granulation tissue. This picture becomes more and more intensified as growth goes on. The areas of dense shadow and the fine clearer band between them grow wider and the surfaces bounding them become more and more irregular and jagged until the end of the bone has an irregular appearance (Fig. 171). During the course of the disease the calci-

FIG. 175.—Distal end of radius and ulna. This plate shows intense calcification of the provisional zone with resorption areas on the marrow side of the epiphyseal line. Both bones show syphilitic periostitis and there is separation of the cortex from the spongiosa in the ulna. (Shipley.)

fication of the infected areas is not only abnormally heavy but also most irregular, so that the epiphyseal border of the shadow cast by the bone has a notched, saw-toothed or serrated appearance (Fig. 175).

"Periostitis, when it occurs near term in the severe cases of lues, may be present throughout the length of the bone or only at the extremities. It is shown in roentgen-ray plates by a more or less wide, almost homogeneous, shadow or with longitudinal striations separated from the external surface of the cortex by a narrow clear area which bounds the bone (Fig. 174).

"One other feature of these pictures appears worth noting. It may be seen that in many luetic bones the cortex is separated from the spongiosa by a very narrow clear zone which gives the cone of spongy bone the appearance of being suspended unattached within the cortical cavity (Fig. 175). In the roentgen-ray picture the trabeculæ of the syphilitic bone appear to be finer than those of the normal bone.

"Two other conditions which are encountered in children may give roentgen-ray pictures which closely resemble, and in some cases are identical with, the pictures described above. Scurvy and rickets, when the latter disease is healing under the influence of cod-liver oil therapy, may be difficult or impossible to differentiate by roentgenographic means from osteal syphilis of the fetal type. Fortunately, however, in the early weeks of life neither of these conditions need be seriously considered in diagnosticating hereditary lues, since there is no good evidence to show that fetal rickets ever occurs and it is agreed that scorbutus is rare before the sixth month of life has been reached."

Liver.—It may be stated that not less than 50 per cent of prematurely born, syphilitic infants show a distinct enlargement of the liver. A fair percentage of the cases show a marked increase in size and consistency to such an extent that the abdominal distention in its upper half is visible to the naked eye. These latter cases are usually associated with marked jaundice, dilated veins and an impairment of hepatic function and a high mortality. Because of the relatively large liver of the premature new born normally present, difficulty may be experienced in the diagnosis of a moderate increase in size due to syphilis. It is also to be remembered that many other factors predisposing to premature birth have a direct influence on the size of the liver. The characteristic pathological findings are interstitial infiltration of the connective tissues between the acini and about the vessels. Small gummata, often the seat of central necrosis are more exceptionally found. The frequent involvement of the liver is readily explained by the peculiarity of the fetal circulation. The placental blood passing through the portal circulation by way of the umbilical veins, conveys the spirochetes into the liver substance.

Spleen.—Enlargement of the spleen, while usually moderate, is one of the most important confirmatory signs but is in itself of lesser diagnostic importance than the skin and mucous membrane lesions. An easily palpable and hard splenic tumor in the first three months of life, that is, before the advent of rickets in the premature, should always be looked upon with suspicion. The enlargement is usually due to hyperplasia of the pulp, with occasional presence of foci of myeloid cells. Cellular infiltration of the interstitial tissue may be present.

Respiratory System.—The lesions of the nasal mucous membranes have been described. Frequently there is a chronic catarrhal laryngitis and perichondritis, with involvement of the epiglottis. In the fetus and in infants dying soon after birth the so-called "pneumonia alba" or "white pneumonia" is often present. In these cases a considerable portion of the pulmonary tissue appears whitish-gray, airless and smooth on section, due to cellular infiltration of the interstitial tissue, filling of the alveoli and bronchi with degenerated epithelium and proliferation of the intima of the vessel walls. Not infrequently the pleura is the seat of small gumma-like nodular infiltrations. Massive involvement of the lungs is rarely compatible with life. Because of their lowered vitality, syphilitic infants are subject to secondary bronchial and pulmonary infection, pneumonia being a frequent cause of death.

Circulatory System.—Most characteristic lesions are found in the small bloodvessels and careful examination shows the presence of spirochetes in the vessel walls. These findings are most easily demonstrated in the parenchymatous organs. The characteristic lesions following such involvement are those of coagulation necrosis, with secondary hemorrhages, following rupture of the vessel walls. These lesions may result in more or less generalized or local hemorrhagic skin lesions and those from the various mucous membranes. Intracranial lesions frequently result from degeneration of the bloodvessels, even in the absence of trauma.

Digestive System.—Chronic catarrhal pharyngitis is a common early symptom which may later be followed by ulcerations of the pharynx, tonsils and fauces. Only rarely is the stomach involved and the lesions of the intestines which are also infrequent are usually seen as hyperplasia of the solitary follicles and Peyer's patches which may become necrotic and result in hemorrhages. Scattered areas of necrosis not associated with the lymphoid tissue, but due directly to bloodvessel degeneration may be found throughout the intestines. Peritonitis is a more frequent finding in the still born than in viable infants. It may be of the acute type but in most instances it is of the chronic type and may result in formation of adhesions. Localized or generalized ascites may result.

The *pancreas, thymus gland, suprarenal bodies* and *thyroid gland* occasionally exhibit interstitial inflammation, gummata or other syphilitic manifestations. Small cystic formations are frequently found in the thymus gland, usually varying in size from 1 to 5 mm. It is a question whether they are due to arrest of development or necrosis. Purulent material with which they are filled contains spirochetes.

Kidneys.—While all types of nephritis have been described, those of greatest importance are the interstitial and hemorrhagic. Interstitial nephritis is a serious complication because of the danger of late secondary contraction. It is frequently overlooked because of the absence of marked urinary findings. The hemorrhagic types are usually associated with hemorrhages from some of the other mucous membranes. The dangers of overmedication with arsenic and mercury preparations, in the presence of kidney lesions must not be overlooked. Hecker[1] states that he has been able to demonstrate microscopical changes in 90 per cent of his autopsies.

Nervous System.—Involvement of the brain and its meninges is more frequent than that of the cord. The most frequent lesions in the still born and those dying shortly after birth is a meningo-encephalitis, involving the pia and cortex. The pia is infiltrated and covered by an exudate composed of plasma cells and lymphocytes. Similar areas are seen in the cortex and the medulla may be involved. The most frequent lesion in viable infants is a meningitis serosa interna and externa, which is not usually noted until after the first few weeks of life. It may develop acutely or insidiously and usually results in a more or less marked hydrocephalus. Because of the late development in some infants and the early appearance of rickets in the premature it should not be confused with megacephalus so commonly seen in the latter. Pachymeningitis hemorrhagica less frequently seen than the former, usually develops after the first few weeks of life. Intracranial hemorrhages are probably a more frequent cause of extra-uterine death than is commonly supposed. Gummatous meningitis and ependymitis are among the rare lesions. Increased intracranial pressure, as evidenced by increased tension over the fontanelles, and which is usually accompanied by hyperexcitability on the part of the infant, should lead to a lumbar puncture for diagnostic purposes. Increased pressure and an increase in the number of lymphocytes in the spinal fluid are always suggestive but not positive evidence. A Wassermann and Lange reaction when positive may usually be considered as conclusive evidence. When these reactions are negative, in the presence of other positive signs, a careful search should be made for spirochetes.

Any of the lesions of the central nervous system may result in retarded mental and physical development.

Eyes.—The most frequent lesions are choroiditis, optic neuritis, iritis and parenchymatous keratitis. They are of frequent occurrence in the still born and may develop in the first weeks of life.

Ears.—The organs of hearing are occasionally involved by lesions which may be described as specific. The most common is an involvement of the eighth nerve. Involvement of the organs of

[1] Beitrag zur Histologie and Pathologie der kongenitalen Syphilis sowie zur normalen Anatomie des Fötus und neugeborenen, Deutsch. Arch. f. klin. Med., 1898, **61**, 1.

the internal ear early in life is difficult of proof. Otitis media as usually seen is due to a secondary infection.

Laboratory Diagnosis.—Whenever there is a suspicion of the presence of syphilis during pregnancy the blood of both parents should be examined so as to give both the mother and fetus the benefit of treatment. This will be given further consideration.

In cases in which the diagnosis has not been made before labor and the possibility of syphilis exists the *placenta should be examined histologically* and the *placental cord blood* should be examined for a Wassermann reaction. It is estimated that about 50 per cent of placentæ will show more or less diffuse lesions upon microscopical examination. Jeans and Cooke[1] found that 57 per cent of their syphilitic infants gave a positive Wassermann reaction on their cord blood. They found that in every instance in which the placenta was noted as showing syphilitic changes the infant was later found to have syphilis. While a *positive Wassermann reaction* may be regarded as nearly specific, a negative reaction must not be regarded as indicating an absence of the disease when made during the first days or weeks of life, as a large group of infants show little or no tendency to give a positive Wassermann before the end of the second month of life. In fact, some of them do not react before the end of the third or fourth month. Negative findings in the presence of lesions or suspicion of infection on the part of the mother should, therefore, lead to an examination of maternal and paternal blood. The variability in the reaction of a new born to the Wassermann test is best evidenced by the report of one positive and one negative reaction in each of a pair of twins by DeBuys[2] and Gerstenberger.[3]

The blood taken from the infant during the first week or two of life shows a somewhat higher average of positive serum reaction than examination of placental blood taken from the same cases. In the premature the blood can be taken from the longitudinal sinus, a scalp vein or by a small incision in the heel. The application of the *luetin test* offers serious objection in premature infants, because of danger of secondary infection. In full-term infants it averages a higher percentage of positives than the Wassermann.

Demonstration of spirochetæ in the open skin lesions and bullæ as well as from the scraping of the mucous membrane ulcerations, makes the diagnosis absolute when the Treponema pallidum is found.

While spinal and ventricular punctures are to be avoided when possible in premature infants, examination of the cerebrospinal fluid

[1] Trans. Am. Pediat. Soc., 1920, vol. **32**.
[2] Jour. Obst. and Dis. Women and Child., January, 1913, p. 65.
[3] Personal communication.

may be necessary when other findings are negative in the presence of possible clinical nervous-system syphilis. About 25 per cent of new-born infants will show spinal fluid changes of sufficient importance to have a diagnostic value. These changes consist of a positive Wassermann, which when present, is usually associated with a definite albumin and globulin increase. More often the cell count reveals a moderate pleocytosis.

Prophylaxis.—It is almost needless to say that luetic individuals should not be permitted to marry. With the improved methods of laboratory diagnosis of today—luetin, Lange and Wassermann tests—it is now possible in a relatively high percentage of cases to discover if an individual has a latent or active syphilis. When there is the slightest suspicion of a specific infection during pregnancy, the mother should be treated intensively. This offers the only hope of preventing a similar infection of the infant with its consequences, or of ameliorating the condition. It is noteworthy that women with luetic histories do much better if under treatment during pregnancy, so that prematurity and still birth may often be avoided. In the absence of specific therapy the child, instead of being born healthy, may show active syphilitic manifestations or develop them later.

As in the prophylaxis of any infection of infancy, extreme care must be exercised with reference to the sterilization of feeding and bathing utensils and clothing.

Nursing.—Whenever a mother bears an infant evidencing lues, if she is at all able, she should nurse her infant. It seems well established today that the mother is syphilitic, whether or not her history is positive, and even in the absence of clinical manifestations. The older controversies as to the possibility of infection of the mother by the child and *vice versa* consequently do not enter into consideration. Where an adequate supply of milk is present it is of the utmost importance that the premature infant be suckled. If the mother objects to nursing her infant at the breast because of nasal and mouth lesions a shield may be used or the milk expressed and hand fed.

Where the mother is unable to nurse her child a wet-nurse should not be employed to suckle the child at her own breasts because of the obvious danger of infection of the nurse. Expressed milk is, of course very desirable.

Active Treatment.—In syphilis neonatorum, which so frequently is associated with serious visceral changes and so commonly affects children born prematurely, the prognosis is in general serious. Very commonly the infants with serious forms of pemphigus, even with early instituted treatment and with human milk feedings, die in the first days or weeks of life. An essentially better prognosis is offered by the cases with maculo-papular or papulo-bullous syphilides provided always that the internal organs are not seriously damaged.

As soon as the diagnosis is made certain, antiluetic treatment should be immediately instituted. Healthy infants and those free from symptoms but born from luetic parents should be treated prophylactically.

Certain facts already enumerated in the general care of premature infants should be especially emphasized in the care of this same class of infants born of syphilitic parents, even though they show no manifestations at the time of their birth. Practically all of them show more or less evidence of malnutrition and, therefore, in this class of infants, as in no other, is breast-feeding indicated. Every effort should be made to stimulate the breast-milk supply on the part of the mother because of the difficulty encountered in obtaining a sufficient supply from other sources, as well as the danger to a healthy wet-nurse.

In the presence of a syphilitic history or positive laboratory findings in the parents, or the findings of clinical manifestations in the infant a vigorous course of treatment should be instituted without regard to the presence or absence of a Wassermann reaction. In every case in which treatment is instituted the fetal age and general condition of the infant must be taken into consideration and the effect of medication, whether mercurial or arsenic preparations, carefully noted. Early dosage with each form of medication should, therefore, be small, however, maximum administration for the given infant should be attained as early as possible.

Mercury Therapy.—Three routes of administration deserve consideration: Oral, external and intravenous.

In the treatment of older infants and children many clinicians of large experience advocate the use of the arsenic preparations as of first importance and while mercurial preparations are considered as absolutely necessary to effect a cure, they are given a secondary place. In view of this tendency it is well that we recall our earlier good results in the treatment of congenital syphilis before the discovery of these newer preparations. It is our belief that mercury should rank first in the treatment of syphilis in the premature and that arsenic therapy should rank second in importance: (1) Because of the lesser danger, and (2) because of the rapid improvement which may be expected in a large majority of the cases. However, the dangers of overmedication, both by mouth and injection with mercury must also not be overlooked. These are usually evidenced by a lack of progress on the part of the infant, diarrhea and evidences of hepatitis and nephritis.

For *internal* use the favorite preparations are hydrargyrum cum creta, 0.005 to 0.03 gm. ($\frac{1}{10}$ to $\frac{1}{2}$ gr.), or hydrargyrum iodidum flavum in doses of 0.002 to 0.005 gm. ($\frac{1}{20}$ to $\frac{1}{10}$ gr.) three times daily.

It is well to begin with small doses, preferably of the former and increase rapidly to the maximum dose in the absence of diarrhea. In the presence of diarrhea the dose should be reduced. The *intramuscular* treatment must be administered with even greater forethought. For this purpose 0.0005 gm. ($\frac{1}{100}$ gr.) of bichloride in 0.2 cm. (3 mm.) of distilled water or oil, for each kilogram of body weight ($2\frac{1}{5}$ pounds) are recommended. The injections are to be given once or twice weekly and should be made deep into the muscle. The gluteal muscles offer one of the best sites for injections. The skin surface should be sterilized with a not too concentrated tincture of iodine and the injection is made deep into the muscle by the use of a short needle, preferably of about a 20 gauge (for oil) and $\frac{1}{2}$ to $\frac{3}{4}$ inch in length. Care should be used so that none will be deposited in the subcutaneous tissues. A course of four to eight injections, covering a period of four weeks, is recommended, these to be followed by a rest period of four weeks, during which arsenic injections are given. The oral administration of mercury should be continued throughout this period. Sublimate baths may be successfully used in all moist forms, especially in all exanthemata associated with vesicle formation—0.2 gm. (3 gr.) for a bath of about 4 liters (1 gallon) of water.

Inunctions are applicable in infants in whom the skin is not too sensitive and are one of the best forms of treatment. In the presence of local skin irritation it becomes necessary to stop this form of treatment. The danger of overmedication with mercury must, however, be borne in mind. Mercurial ointment is especially valuable for local application to ulcerated lesions and may be applied to the deeper seated lesions of the hands and feet by the use of mittens and stockings. For these purposes the official mercurial ointment should be mixed with 2 parts of lanolin. In the more mature infants it should be carefully rubbed into the abdominal wall, axillæ or thighs and the same site used only at infrequent intervals in order that cutaneous irritation be avoided. For a local lesion 2 per cent of yellow oxide of mercury ointment will do. In the presence of snuffles a 1 per cent yellow oxide of mercury ointment should be used. The ointment is introduced into each nostril directly from a small compressible tube. It may be necessary to carefully remove any excessive secretions with a pledget of cotton or by washing with a normal salt solution before applying the ointment.

It is advisable to continue mercurial treatment for at least a year, decreasing the dose in the second six months, and repeating three months of such therapy during the second and third years, even in the absence of symptoms. As in treating older infants, there should be short periods when treatment is discontinued.

Arsenic Therapy.—It is indicated in most cases as an adjunct to mercurial treatment. Neoarsphenamine is the preparation of choice for use with the premature because of the fact that it can be administered in more concentrated solution, its greater solubility and the lack of necessity for neutralization. It can be administered intravenously in water or by intramuscular injections in a bland oil.

The average dose is 0.01 gm. for each kilogram of body weight. The dose should be diluted with 2 cc of sterile, freshly distilled water for intravenous use. It is advisable to give one-half of this quantity per kilogram for the first treatment. A course of four intramuscular, or, when possible, preferably intravenous injections are given at weekly intervals to be followed by a rest period of four weeks when the treatment is to be repeated. During the period of administration of neoarsphenamine the mercurial injections should be discontinued but the oral administration continued.

Complications following the intramuscular injection of neoarsphenamine, such as abscesses and infiltrations can, to a large degree, be avoided by the use of special needles, which permit the solution to be injected deep into the muscle. After injection, the needle is rapidly withdrawn and a cotton pledget is pressed firmly over the site of injection for a few minutes.

For intravenous administration the best sites are scalp veins or the external jugular vein. For administration into the latter site the infant should lie with the shoulders elevated and the head extended and rotated. Only in very exceptional cases should cutting down on a vein be practised. The longitudinal sinus route for arsenic injection is not to be considered because of the danger of passing through the sinus and extravasating the preparation over the brain tissue.

The general plan of treatment should, therefore, be as follows: One of the mercury preparations should be administered in suitable doses three times daily per mouth, and once or twice weekly during the first four weeks an intramuscular injection of one of the mercurial preparations should be given. During the second month the oral administration should be continued but the mercurial injection should be replaced by neoarsphenamine, preferably intravenously, once each week. Mercurial ointment as inunctions or local applications are to be used when indicated.

This plan of treatment should be continued throughout the first year, in the absence of toxic symptoms and at least three months of treatment should be given during the second and third years. Treatment should be continued for at least six months after all evidence of activity has disappeared. This includes a negative Wassermann. At no time should a negative Wassermann in early infancy be considered as sufficient evidence to interfere with the general course of treatment as outlined.

CHAPTER XVI.

TUBERCULOSIS IN PREMATURES.

THE recorded cases of tuberculous affections during the first weeks of life are unusually rare, and their clinical symptoms, even when anatomically demonstrable changes are present, ordinarily are not to any extent characteristic. While in comparison with the acquired tuberculosis, the congenital form is almost a rarity, nevertheless numerous authentic instances are on record.

M. Pehu and J. Chalier[1] have collected 51 cases from the literature, the authenticity of which has been established. While some of these cases have resulted in premature birth, the majority have been born at full term; and although some of the latter have been well developed, most of them have suffered from congenital debility.

Planchu and Devin[2] describe 39 premature infants born of tuberculous mothers. They believe that the morbidity and mortality is greater in infants born prematurely from tuberculous mothers than the average for those born prematurely of other causes.

While infants born at full term of tuberculous mothers may occasionally be well developed, the majority nevertheless, if infected with tuberculosis before leaving the uterine cavity, show marked congenital debility. As a case in point in evidence for the possibility of good development, may be cited the infant of H. Rollet,[3] which died forty-eight hours after birth, but in whom large caseous areas were found in the bronchial glands, lungs, liver and spleen. The mother of this child died eighteen days post partum from miliary tuberculosis, and on examination it was found that the uterus still contained placental remnants from which numerous tubercle bacilli were obtained.

In cases of intra-uterine infection the tubercle bacilli penetrate into the body of the infant, either by way of the placental blood or by the swallowing of liquor amnii. It is impossible for the embryo to become infected unless the mother be tuberculous.

The transmission of the bacilli from the mother to the infant

[1] Heredity in Tuberculosis, Arch. de méd. des enf., 1915, **18**, 1.
[2] Le Prémature de Mère tuberculeuse, Lyon méd., 1911, **116**, 72.
[3] Ueber intra-uterine miliare tuberculose, Wien. klin. Wchnschr., 1913, No. 31, **26**, 1274–1275.

can occur at any time during pregnancy. This may result from bacilli carried in the fetal circulation, from various parts of the mother's body, or through organisms found in placental lesions. The normal placenta is usually conceived to be a filter impermeable to bacteria. Presumption for the passage of tubercle bacilli from the blood of the mother to that of the infant is a lesion of this filter. Tubercle bacilli can pass into the blood stream of the infant only when a communication has been established between the intervillous spaces and the bloodvessels of the chorionic villi, or when liquor amnii becomes infected with the organisms. Therefore, the bacilli infecting the fetus must come either from a tuberculous placenta or from the circulating blood. The transmission of bacilli into the blood of the infant takes place when a bloodvessel of the villus becomes eroded or ruptured.

Tuberculous changes in the decidua vera or in the chorionic covering of the placenta may result in infection of the liquor amnii by breaking through the amnion, and also in intestinal infection with eventual general systemic distribution.

The intra-uterine infections above described may lead to advanced tuberculous processes at birth. Such infants are usually born premature or show great debility. Not infrequently the infant is infected through the transmission of the organisms during birth, when in the separation of the placenta the bloodvessels of the villi become ruptured, and thereby passage to the blood of the infant, either from the tuberculous foci of the placenta, or from the maternal blood, is made possible. In these latter cases no specific changes are found in the organs at birth, and these infants are likely to be well developed.

Intrapartum infection may take place through swallowing or more rarely through inhalation during the passage of the child through the birth canal.

The infection may take place after birth (acquired tuberculosis). This occurs either by way of the respiratory tract through inhalation, or by way of the digestive tract, or through other portals of entrance, far less common.

It is of the greatest importance from the clinical point of view to separate the infants who are born with tuberculous organic changes from those who are born without such pathology. The new-born infant in this situation is in the stage of incubation for tuberculosis.

Unfortunately such clinical distinction is usually impossible because of the absence of pathognomonic symptoms, and the failure of specific tests during the first weeks of life. While the cutaneous and intracutaneous reactions are rarely seen before the fourth week of life, a few cases have been described. Among these is that of Zarlf,[1] who reported a positive von Pirquet reaction in a seventeen-day old infant, which was still living at the time of the report, six weeks after birth. In the discussion of this case von Pirquet remarked that this was the earliest age at which a positive reaction had been reported to his knowledge, and that he believed it to be proof of the congenital origin of the case, as his conception was that at least four weeks must pass after the time of infection before a positive tuberculin reaction may be obtained.

It should be remembered that prematurity and congenital debility on the part of infants born of tuberculous mothers does not necessarily mean that the child is suffering either from congenital or hereditary tuberculosis. It should not be forgotten that infants infected with tuberculosis, in whom there are only minor or no tuberculous lesions, may be born at full term, seemingly robust.

Etiology.—The frequency of tuberculosis as an etiological factor in premature births or general debility of full-term infants must be considered: (1) From the standpoint of the effect of tuberculosis on the entire organism of the mother; (2) its influence on the generative organs of the mother; (3) its effect on the general development of the fetus; (4) of a systemic infection of the fetus; (5) from the viewpoint of the results as they affect the future development of the infant, which may be born at full term, without manifest evidence of congenital debility.

1. *Effect of Tuberculosis on the Entire Organism of the Mother.*—While numerous authentic cases of congenital tuberculosis are now on record, by far the majority of infants born of tuberculous mothers do not show evidence of systemic tuberculosis at autopsy, and in our own studies of such instances in the Cook County Hospital over a period of several years, the only well-authenticated case which has come under observation and which has proven to be one of general tuberculosis on the part of the infant, was reported by Grulee.[2] The infant died on the eleventh day after its birth, and at autopsy showed a generalized tuberculosis, affecting most markedly the abdominal organs and especially the periportal lymph glands, liver and spleen. The tuberculosis was miliary in type, but the stage of the tubercles suggested an intra-uterine infection. The mother was still living several months after the infant's death.

In contradistinction to this case we have had occasion to observe numerous instances in which infants born of tuberculous mothers have survived, and have either progressed more or less normally, or have died of infections other than tuberculosis—in whom at least tuberculosis could not be demonstrated at autopsy.

[1] Congenital Tuberculosis, Jahrb. f. Kinderh., 1913, No. 1, **67**, 95.
[2] Tuberculosis as a Disease of the New Born, Am. Jour. Dis. Child., 1915, **9**, 322.

2. *Effect on the Generative Organs of the Mother.*—Tuberculosis can be transmitted through the uterus, either through local lesions or without demonstrable lesions in the uterus and placenta. G. Luenberger[1] contributes records of two interesting cases of placental and congenital tuberculosis, which illustrate the abovementioned possibilities. In the first instance the mother died of tuberculous meningitis and miliary tuberculosis. Tubercle bacilli were found in the fetal liver and numerous miliary tubercles in the placenta. Injection of a small piece of liver extract and of the heart's blood of the fetus into a guinea-pig gave rise to pulmonary tuberculosis.

In the second instance the mother suffered from pulmonary tuberculosis, and aborted. Neither the fetus nor the placenta showed any tuberculous changes, but tubercle bacilli were found in the intervillous spaces of the placenta.

From the study of these two cases Luenberger draws these conclusions: When the mother suffers from acute miliary tuberculosis, there can develop numerous miliary tubercles in the placenta, and from these, tubercle bacilli can penetrate the fetal circulation. It is also true that without tuberculous changes in the placenta or membranes the bacilli can pass from mother to child, that is, during birth there can be sufficient injury to the chorionic vessels to allow the bacilli to pass from the intervillous spaces into the fetal circulation.

A. Dietrich[2] reported a case which suggests the possibility of congenital infection. A woman with general tuberculosis gave birth shortly before her death to a premature infant. Tubercle bacilli were demonstrated in the placenta. The baby was never in contact with the mother. It developed well for the first two months, when an abscess formed in the right groin. Following this there was loss in weight and rales in the chest. The child died in the third month. Autopsy showed many tubercles in the lungs, intestines and spleen, a few in the liver and a large lesion in the portal vein.

Tuberculosis of the placenta has been described by many observers. This is of importance in relation to tuberculosis of the fetus in proportion as the fetal or maternal portion of the placenta is involved. It is certain that in many cases only the maternal portion is infected, the fetal remaining uninfected.

3. *Effect on the General Development of the Fetus.*—In a consideration of this class of cases, theoretically it may be viewed from two standpoints: (1) That of general debility, without reference to a special predisposition to tuberculous infection; and (2) that of congenital predisposition to tuberculous infection. The question

[1] Contribution to Placental and Congenital Tuberculosis, Beiträge z. Geburtsh. u. Gynäk., 1909–1910, vol. 5.

[2] Congenital Tuberculosis, Berl. klin. Wchnschr., 1912, **19**, 877.

of the possibility of an inherited immunity against tuberculous infection is one which is open to great speculation, and we have not been able to satisfy ourselves that such an immunity may exist. That many of this class of infants seem to have a predisposition to tuberculous infection, which in all probability is, however, at least in great part due to their constant exposure and repeated infection with the organisms through contact with an infected mother, cannot be denied. This class of infants without really having tuberculosis often shows signs of malnutrition. Doubtless many of them have a diminished resistance to all infections and more especially to tuberculosis. They are below the average in development.

4. *Systemic Infection of the Fetus.*—If tuberculous changes are present in the body at the time of birth, if the infant is born alive, the disease leads to early death in the majority of cases, generally within the first week of life.

In a great number of cases in which tuberculosis is transmitted in utero, more especially in the last days of pregnancy, or intrapartum, the disease remains clinically latent during the first days of life, and may not become manifest for two or three months. The infection may, however, be entirely overcome. These cases may be described as the latent forms of tuberculosis. Of the 28 instances of congenital tuberculosis of which we have definite records at hand, 10 infants were born prematurely, and 2 of these were still births. Two of the living premature infants survived for three months, the other 6 living from one day to two months. Of the infants born at full term all died before the fifth month of life.

5. *Results as They Affect the Future Development of the Infant, Which May be Born at Full Term, Without Manifest Evidence of Congenital Debility.*—The future development of this class of cases is dependent upon their freedom from congenital infection, their protection against postnatal infection and their general resistance.

Symptoms.—Clinical data of tuberculosis of the new-born premature or full term are so scant that no conclusions can be drawn as to the symptomatology. The combination of enlargement of the spleen, high, irregular temperature and enlargement of the liver, together with tuberculosis in the mother is very suggestive. The infants are usually below weight at birth, pallid and may show a positive tuberculin reaction in the sixth to seventh week of life.

Treatment.—It is, of course, of the utmost importance that very careful hygienic and dietetic measures be instituted at the earliest opportunity. There are no specific cures or worth-while medicinal measures. The critical question is that of the advisability of nursing.

In general nursing should under all circumstances be forbidden in open pulmonary tuberculosis of the mother, and the same is advisable also in every active tuberculosis. The prohibition of nursing in these cases has for its purpose the removal of the infant from the coughing mother—from the tuberculous environment—and is done more because of the danger of inhalation tuberculosis than because of the possibility of an eventual transmission of the bacilli by the mother's milk. Marked tuberculosis of the mother should in all events be a contraindication against nursing for the benefit of both. In such a case it is the duty of the physician to do all in his power to accomplish the removal of the infant from the neighborhood of the mother as soon as possible, at least for the first months of life.

In the cases of mothers proven to be tuberculous, who show no manifest signs at the time of delivery and lactation, caution is necessary. When the removal of the infant from the mother encounters insurmountable opposition and the infant must remain at home, then it is more advisable in such cases not to endanger the infant any more by introducing artificial feeding but to put it to the breast. If in the mother there are neither clinically nor physically demonstrable tuberculous changes, and sputum examination is negative, and if the tuberculosis is not only latent, but also inactive and is confined to mild apex findings, then, when the infant remains with the mother, nursing should not only be recommended but strongly urged. If feeding by a wet-nurse is possible it is for all events and purposes the best method in doubtful cases. This should in justice to the wet-nurse be carried out by hand-feeding of expressed milk. The wet-nurse baby should not come in contact with the infected infant.

CHAPTER XVII.

EDEMA AND SCLEREDEMA IN PREMATURE INFANTS.

Besides asphyxia and hypothermia there is a tendency to edema in small premature infants. This occurs, sometimes during birth, but more frequently during the first days of life, as edema of the extremities and the genitalia. In contradistinction to the general view that these edemas and scleredema are to be regarded as sequelæ of subnormal temperature, it must be emphasized that these edematous conditions are not uncommon in small prematures, and that they may occur even in utero. In this connection attention may be called to *congenital general dropsy* and to other localized edemas, that have been observed by others in premature infants immediately after birth, or in the new born. (Ballantyne, Link, Kirk, Oswald, Chiari.)

Special forms of edema are *scleredema* and *sclerema*. It is not always possible to make sharp differentiations between these and other forms of edema. Scleredema is designated that form of edema in which the skin is hard and taut, while sclerema is that condition in which the skin is hard and dried out. Many authors emphasize that in an individual case the sclerema is not to be distinguished from scleredema, since they are only quantitative differences of the same process. Ylppö[1] believes that it depends entirely upon the water richness of the tissues, whether the skin feels pasty hard (scleredema) or wooden hard (sclerema).

Etiology.—As far as etiology is concerned we cannot make special differences. According to experience, the skin upon the external portion of the thigh, whenever edema of the feet is present, feels always somewhat tougher and harder (scleredemic), in comparison to soft edema of the genital region or of the inner surface of the thigh and leg. Because these differences in consistency are demonstrable in many premature infants a few hours after birth, we have to consider special anatomical conditions as factors responsible for their production. The younger the infant, the thinner is the fatty cushion. On the external surface of the thigh it is several millimeters thick even in the smallest prematures, while in other regions the subcutaneous fatty tissue is not well developed. The occurrence of hard edema on the external surface of the thigh with

[1] Ztschr. f. Kinderh., 1913, **24**, 53.

simultaneous occurrence of soft edema in other portions, forces upon us the conclusion that besides the water richness it also depends upon the richness of the subcutaneous fatty tissue, whether or not an edematous portion of the skin feels somewhat harder.

Now, new-born infants, and also prematures, whose bodies are especially rich in water, lose in weight during the first days of life, and thus it is easy to understand that the water content of the skin and of the subcutaneous fatty tissues gradually becomes less. According to Langer[1] and Knoepfelmacher,[2] the subcutaneous fat contains chiefly palmitic and stearic acids, and proportionately only a small quantity of oleic acid. The fat of the new-born infant is therefore even with ordinary body temperature somewhat harder than the fat of the adult, which is rich in oleic acid. The usual very high water content of the fatty tissue in the new-born infant makes it of normal softness during ordinary temperature. It is easy to understand that the oleic-acid-poor, fatty tissue begins to feel hard when the water disappears from the interstitial spaces of the fatty tissue.

Symptoms.—In small prematures that are observed carefully after birth, we may notice that the legs, and especially the feet and hands, may begin to swell in from five to seven hours after birth. These swellings often occur no matter whether the infant is transferred immediately after birth into a warming tub, or whether it shows subnormal temperature. In infants with subnormal temperature edema occurs more frequently and is more marked. If the child is put into a somewhat inclined position, so that the hands and legs hang down, then very soon cyanotic swelling may be observed in the dependent extremities. If we change the position and allow the head to be lower than the legs then the edema disappears in a few hours.

This simple experiment shows that the cause of the edema occurring in the premature infant during the first days or hours of life, may be looked for in circulatory weakness. Besides this, the high water content of the tissues and the ready permeability of the blood and lymph vessels in prematures is of great importance in this respect. In these infants edema occurs not only in the skin, or more properly in the subcutaneous tissues, but also in many other tissues. The marked tendency to hydrops of the cavities and the high-grade edematous swellings of the pelvic walls and brain coverings is also a manifestation of this general property of the body of the premature infant. It cannot be denied, of course, that hypothermia and initial cooling of the premature infant are of importance in the development of edema. If the cold easily

[1] Mathem.-naturw. Klasse, 1881, **84,** 94 (dritte Abtlg.).
[2] Jahrb. f. Kinderh., 1897, **45,** 177.

damages the small capillaries of an adult it does it even more easily in the premature infant, in whom the skin is rich in water. The water evaporation, by producing heat loss, favors the development of lesions of the capillaries. It is a mistake, however, to designate edema in premature infants simply as a sequel of hypothermia.

Treatment.—In the treatment of sclerema it is important to see first that the water intake is increased. It is understood that proper care must be taken of the temperature and other conditions. In general, the *prognosis* in sclerema of the premature infant is not as bad as has generally been supposed. If we succeed in preventing early the marked desiccation of the infant, then it is still possible to save the infant.

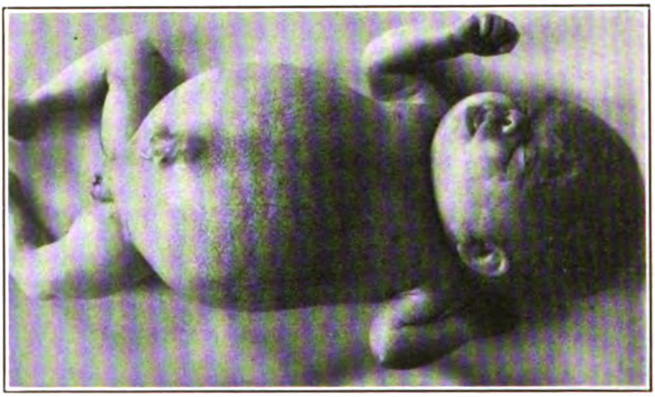


FIG. 176.—Case of erythroblastosis.

ERYTHROBLASTOSIS FETALIS.

Among the various forms of congenital dropsy, in which the infants are often prematurely born, erythroblastosis, first described by Schridde[1] and named by Rautmann,[2] is the least understood. Congenital generalized edema may be the result of cardiac anomalies and diseases, portal obstruction, syphilis of the liver, fetal peritonitis, abnormality of the D. venosus Arantii, deformities of the intestines and diseases of the kidneys. Schridde, in 1910, pointed out a form of congenital general dropsy with hydramnios associated with a pathological blood state.

The disorder is characterized by anasarca and fluid in the cavities,

[1] Die angeborene allgemeine Wassersucht, München. med. Wchnschr., 1910.

[2] Ueber Blutbildung bei fötaler allgemeiner Wassersucht, Ziegler's Beiträge, 1912, **54**.

hydramnios and enlargement of the liver and spleen. The latter two organs show the most marked changes, which consist of the accumulation, both inside and outside of the bloodvessels, of large numbers of erythroblasts and a smaller number of other marrow cells. The lymph follicles in the spleen are absent and the liver cells are crowded out. Accumulations of erythroblasts in small numbers may be found in the kidneys, adrenals and lymph glands. Erythroblasts appear in the blood in greatly increased numbers and they show very often mitotic processes. The heart is often hypertrophied.

Because of the presence of hemosiderin in the spleen and liver, Schridde was led to believe that the disease was due to a severe anemia with compensatory hematopoiesis having no relation to syphilis. Others have assumed that the extramedullary formation of blood corpuscles was due to some form of unknown toxic action. Chiari[3] described an infant in whom there was no blood pigment in the liver or spleen, and consequently no indications of any antecedent destruction of blood cells. Fischer,[4] in his examination of the older literature, came to the conclusion that many of the cases described as congenital leukemia were probably instances of erythroblastosis.

[3] Ein Beitrag zur Kenntnis der sogenannten fötalen Erythroblastose, Jahrb. f. Kinderh., 1914, **80**, 561.
[4] Die allgemeine angeborene Wassersucht, Deutsch. med. Wchnschr., 1912, No. 9.

CHAPTER XVIII.

DISEASES PECULIAR TO PREMATURE INFANTS.

RACHITIS IN PREMATURE INFANTS.

THE early appearance of rachitic manifestations in premature infants has been noted by many observers, especially associated with spasmophilia and anemia. Most prematurely born infants become rachitic—the lower the weight, the more certainly—and even human milk is not an absolute protection against this.

Huenekens[1] was able to collect 70 cases of prematures and twins, of which 58 developed definite signs of rachitis (82 per cent). The time of occurrence was interesting, inasmuch as of 33 cases seen for the first time at or before four months, 27, or 81 per cent, showed evidence of rachitis at that time. The first symptom usually noted was *craniotabes*, which in 3 instances was already present at six weeks. Langstein[2] observed it frequently in the third to the fourth month of life and not much less often was the tendency to *convulsions* (hyperirritability of the nervous system of these infants).

Ylppö[3] observed commonly a *megacephalus* in connection with rachitis of the skull, which often left marks permanent for all life. These have to be regarded as characteristics of the prematures and not, as unfortunately is often the case, as signs of special "constitutional degeneration." Along with this megacephalus with its somewhat large, plump skull, there is asymmetry, which is not congenital but is produced in a mechanical way by the pressure of the infant's head in the first months of life and the softness of the skull.

The narrow thorax with its more or less marked signs of rachitis may also be regarded as a peculiarity characteristic of the smallest prematures but not of those of greater weight. This is not to be confused with the early functional, funnel chest which can be demonstrated in the first weeks of life and is due to the softness of the ribs in the smaller prematures. This leads to further deformity of the thorax, as the marked contraction of the lower half, which is the result of the congenital softness of the ribs and the rachitic affections later developing. The constriction around the chest is best seen about the insertion of the diaphragm.

[1] Jour. Lancet, 1917, **37**, 804.
[2] Ztschr. f. Kinderh., 1916, **15**, 49.
[3] Ibid., 1919, **20**, 212.

The rachitic *rosary* is very prominent in prematures and is explained on the basis of the constant respiratory movements leading to deformities and marked enlargement of the epiphyses of the ribs.

The long cylindrical bones, however, only exceptionally show enlargements of the epiphyses in prematures, although rachitic

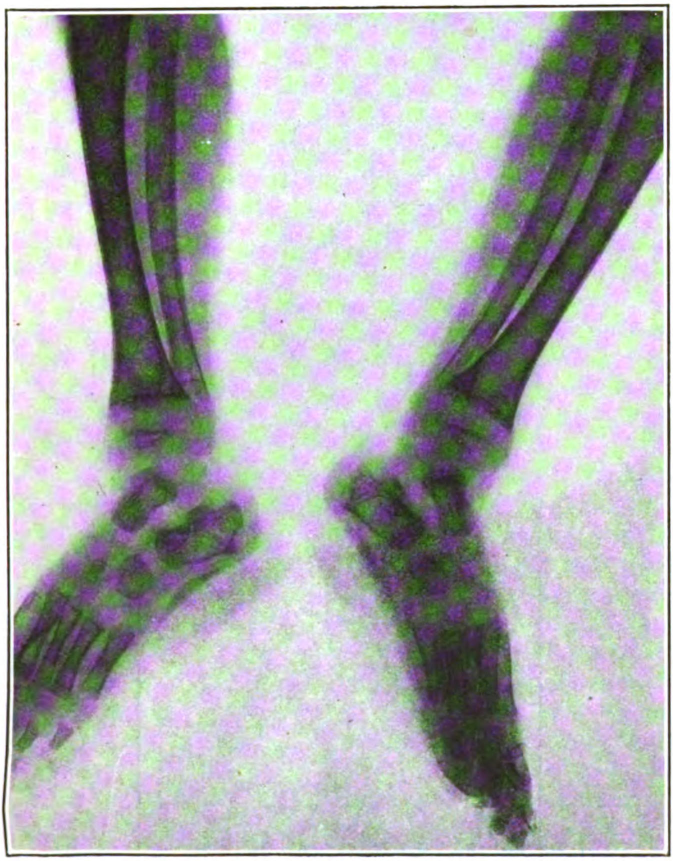


FIG. 177.—Rickets—first stage.

changes appear in these bones very early. The process in these bones results rather in bone absorption and fringing of the epiphyses than in marked proliferation, which is the rule in strong full-term rachitic infants. The explanation of this feature in prematures may be in the fact that the rachitis appearing very early is already at end by the time the infant learns to walk, whereas in the full-term infant the hyperplastic epiphyseal enlargement occurs as a compensatory process in the period when the lower extremities are called upon to support the weight of the body. In the absence of special rachitic curvatures and epiphyseal enlargements of the long bones, we cannot therefore exclude rachitis in

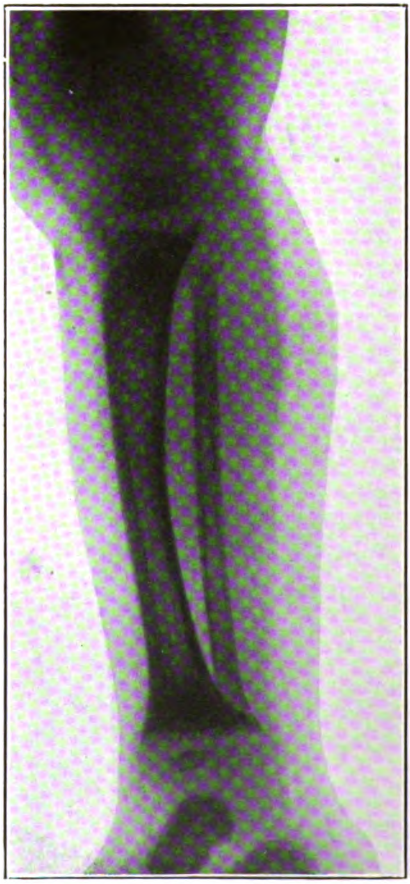


FIG. 178.—Rickets—second stage.

the premature. Histological examination shows a characteristic picture in the absence of marked external manifestations.

Etiology.—The etiology of rachitis in premature and full-term infants has been the subject of much discussion. Huenekens believes that the explanation may be found in that the chemical constitution of prematures is abnormal. Their salt content is far below normal. Birk[1] found that a four-months fetus contained 14 gm. of ash, at six months 30 gm. at nine months 100 gm., showing that two-thirds of the minerals were taken on during the last three months of fetal life. In the new born fully 75 per cent of this ash is made up of calcium and phosphate, the chief constituents of the bones. Huenekens believes, therefore, that the more premature the infant, the greater will be the deficiency of calcium and other minerals, so that by the third or fourth month of extra-uterine life the supply is entirely exhausted and rachitis results.

Underfeeding is another factor in the development of rickets in the premature. The low calcium content of human milk and the difficulty of metabolizing even this food in sufficient quantities to prevent drawing on the inherited supply may be an active factor. The *artificially fed* are especially prone to develop severe rickets. If the diet contains sufficient milk the tendency to develop the disease is less than when fed mainly on cereals and proprietary cereal foods with only small amounts of milk. A diminished calcium retention (negative calcium balance) exists in the florid stage of rickets, even though the intake is ample. A deficiency of calcium in the diet while important in itself is probably not the precipitating factor. It has been shown experimentally in puppies that a diet containing an abundance of calcium does not prevent rickets when the diet is deficient in other factors.

The average normal inorganic phosphorus concentration in the serum is about 5 mg. per 100 cc. Howland and Kramer[2] found that in all patients in the active stage of rickets the concentration of inorganic phosphorus in the blood serum was low and that in all children under two and a half years of age, in whom an inorganic phosphorus content of the serum of 3 mg. or less was found, active rickets was present. With the healing of the process in the bones that occurred after cod-liver oil medication, the phosphorus rose gradually to normal. These facts led them to consider the presence of a low percentage of inorganic phosphorus in the serum of a young child as nearly conclusive evidence of active rickets. They believe that there is constantly a marked and for the causation of the pathological lesions, an important deficiency in inorganic phosphorus. To this deficiency they ascribe the failure of calcium deposition.

The phosphorus content of the blood can be increased by feeding phosphorus per mouth. Marriott,[3] working with artificial blood, found that by small increases in the phosphorus content, a precipitate resembling in composition the salts of bone was formed.

[1] In Monatsschr. f. Kinderh., 1910, **1**, 644.
[2] Jour. Biol. Chem., 1920, **43**, 35.
[3] Report of Thirty-second Meeting of Am. Ped. Soc. Arch. Ped., 1920, vol. **37**,

Phemister[1] applied these experiments to children and noticed by roentgenogram studies that phosphorus affected the normal bones of children as it did Wegner's[2] animals and that the accumulation of calcium and overproduction of bone in the metaphysis continued for some time even after the administration of phosphorus was discontinued. He has more recently reported similar results in rachitic infants.

McCollum and his associates,[3,4] in a study of the effect on the growth and development in rats, came to the conclusion that the etiological factor is to be found in an improper dietetic regimen. Their experiments showed that the majority of young rats developed pathological conditions of the skeleton having a fundamental resemblance to rickets when fed upon diets low in both fat soluble vitamines and phosphorus. When they modified this diet so that the deficiency in phosphorus is compensated for by the addition of a complete salt mixture, containing the phosphate ion, the deficiency in fat soluble factors still existing, no pathological changes of a rachitic nature developed. They, therefore, concluded that a deficiency in this vitamine cannot be the sole cause of rickets. In summarizing, they state that the phosphate ion in the diet may be a determining influence for or against the development of rickets, but that these findings should not exclude the absence of fat soluble vitamine from consideration as an etiological factor in the production of rickets and kindred diseases, since the level of the blood phosphate is, in all probability, determined in part by the amount of fat soluble vitamine available for the needs of the organism.

Summarizing, it appears that rickets is a nutritional disturbance especially affecting the osseous and muscular system, with resulting lesions which prevent the bones and muscles from utilizing calcium, thus leading to a diminished retention of this element, although there is plenty of it in the food intake and in the blood. Phosphorus probably plays an intermediate rôle in influencing the formation and deposition of the lime salts in bone. Whether the diet plays its rôle by directly interfering with the calcium and phosphorus metabolism due to lack of an antirachitic factor or indirectly by causing an underlying nutritional disturbance is open to conjecture.

Hygiene is an important factor in that improper hygiene results in impaired metabolism with a resulting inability to properly utilize the dietetic constituents even when properly balanced.

[1] Effects of Phosphorus on Growing Normal and Diseased Bones. Jour. Am. Med. Assn., 1918, **70**, 1737.

[2] Virchow's Arch. f. Path. Anat., 1872, **55**, 9.

[3] McCollum, Simmonds, Parsons and Shipley: Jour. Biol. Chem., 1921, **45**, 333.

[4] Shipley and Park, McCollum and Simmonds: Johns Hopkins Hosp. Bull., 1921, **32**, 160.

Infections play a similar rôle. Impairment of the body functions also directly affects the glands of internal secretion with secondary disturbances following such dysfunction. Therefore, while an impairment of mineral metabolism precipitates the clinical symptoms, one or several of the secondary factors may have an important relation to the utilization of phosphorus and calcium.

Treatment.—Our therapy is along the same lines as in full-term children with special stress on the feeding of human milk. Fresh air and sunshine in the older children and the observation of careful hygiene for all are without doubt highly important.

Diet must receive very careful consideration. In the very young the ideal food is, of course, mother's milk. Where artificial feeding must be instituted the amount of cow's milk should be minimal, and cereals and vegetables started early. Orange juice diluted with water should be given in small amounts from the second or third months (one to four teaspoonfuls daily). After the first month the diluent in the milk mixtures should be a cereal water (one tablespoonful of whole barley or oatmeal to the quart of water—and not the dextrinized cereal flours). From the third month cereal should be fed. After the fifth month vegetable soups should be given, substituting an ounce of soup for an equal amount of bottle-feeding. By the sixth or seventh month a milk-feeding should be replaced by a vegetable-soup meal.

Cod-liver oil with phosphorus in a preparation containing 0.0003 gm. ($\frac{1}{200}$ gr.) to each 4 cc (1 dr.) oleum morrhuæ, is a most practical mixture and can be administered to most infants by the fourth to the sixth week, beginning with $\frac{1}{2}$ cc doses twice daily and increasing to 4 cc twice daily by the fifth month. The work of Schloss[1] has shown that the addition of a calcium salt to cod-liver oil with phosphorus further enhances the value of the mixture. Such a preparation is the tricalcium phosphate C. P. (10 per cent) in emulsion of cod-liver oil U. S. P.

ANEMIA OF PREMATURE INFANTS.

Closely associated with rachitis in premature infants is an anemia, which develops quite regularly and strikingly during the first three months of life. In our previous discussion of the physiology of the blood we noted from the work of Kunckel, Lichtenstein, Lande and others (p. 67, *et seq.*) that in contrast with full-term infants, in the premature there is a greater number of nucleated red blood corpuscles, a more frequent appearance of myeloblasts and myelocytes during the first days of life, a lesser development of absolute

[1] Zur Therapie der Rachitis, Jahrb. f. Kinderh., 1914, **79**, 194.

and relative leucocytosis, and a greater number of immature leucocyte forms. There is also a distinct and very early hemoglobin impoverishment of the blood, which reaches its maximum in about the third to the fourth month.

Etiology.—Kunckel[1] believed that this anemia appearing regularly in the first three months of life was physiological and was of the chlorotic type. His children improved in the second half year of life, but if infection was present any time the infants developed a severe secondary anemia much more readily than full-term infants. His opinion was that the anemia did not rest on an alimentary basis but was due to an insufficiency in hemoglobin metabolism, beside a deficient iron storage.

Pfaundler[2] felt that the anemia was closely related to a lack of fresh air and sunshine.

Lichtenstein[3] fixes the early anemia in the first three months of life as a hypoplastic condition resulting through insufficiency of the hematopoietic system. The later oligochromemia, after spontaneous retrogression of the oligocythemia, he considers as a sequel of the impoverished iron storage. He opposes the hypothesis of alimentary anemia of Czerny and Kleinschmidt. The theory of the harmful action of milk on the hematopoietic apparatus he asserts is disproved by the excellent results attending the feeding of human milk and the administration of small amounts of ferrous lactate.

Lande[4] is in accord with the opinions of Kunckel and Lichtenstein. As evidence in favor of the importance of iron storage he emphasizes the fact that eighth-month infants in the course of the second quarter year of life show a higher percentage of hemoglobin and erythrocytes than do seventh-month prematures.

The examination by Lande of the bone marrow in ten prematures disclosed no decisive picture except an insufficiency of the granulocyte system. Thus he disproves the theory that the basis of the anemia rests with a defective erythropoietic system.

Lichtenstein feels that there is no marked difference in the blood picture of artificially and breast-fed prematures. Examination of twenty-eight cases artificially fed, many of whom were born of nephritic, anemic and tuberculous mothers, showed no great differences in the blood picture from those breast-fed.

Symptoms.—The most marked symptom observed by Lande was pallor of the skin, which he saw with great regularity. It appeared especially early as a fore-runner of icterus, which in prematures is

[1] Ztschr. f. Kinderh., 1915, **13,** 101.
[2] Verhand. d. Ges. f. Kinderh., Breslau, 1904, **21,** 24.
[3] Svenska, LaKaresa As Kapets Handlingor, 1917, No. 4, **43.**
[4] Ztschr. f. Kinderh., 1918, **22,** 299.

constantly present. The question arises as to whether the anemia is promoted by the icterus or both icterus and anemia are not bound up with a third factor—the maturity of the infant.

One can differentiate various grades of pallor, which is earliest and most clearly seen in the face and well agrees in general with the degree of pathological blood change. The most marked form of anemia gives the infant a bluish, transparent appearance, or a waxen, yellowish color, somewhat akin to the infants with severe congenital syphilis or chronic pyelitis. The picture is accentuated by the outstanding bluish veins, especially prominent on the skull and abdomen. The ears are transparent with hardly the vestige of a rosy hue and the mucous membranes are very pale.

The pallor after open-air treatment has been noted to give way to a rosy hue, but only in a few cases is there a parallel permanent increase in hemoglobin. However, with the increase in hemoglobin and erythrocytes in the fourth to sixth months, the color simultaneously improves.

In Lande's series the appetite of the infants was in general satisfactory. There was no stupor, especially in the more anemic. There were no elevations of temperature, as described by some observers, present with the marked blood changes.

Marked glandular and splenic swelling was not observed by Kunckel or Lande, but Lichtenstein states that splenic tumor was present in two-thirds of his children.

Lichtenstein finds that the blood pictures in the well breast-fed prematures and those showing alimentary disturbances are both of the chlorotic type and differ mainly in degree. He also believes that the clinical picture described as pseudoleukemic anemia is a severe form of secondary anemia and is not a distinct clinical entity.

Treatment.—For the general and hygienic treatment of primary and secondary anemia, the suggestions made for general measures in the care of rachitis should be followed. The infants must above all be given the advantage of a good environment, plenty of fresh air and sunshine.

Iron therapy for the purpose of increasing the iron content of the tissues and the hemoglobin has met with individual success. It should be started early. Among the iron compounds to be recommended are ferri carbonas saccharatus 0.25 to 0.5 gm., ferri et ammonii citratis 0.06 to 0.12 gm. or ferri lactis 0.12 to 0.25 gm. one to three times daily.

Small doses of liquor potassii arsenitis 0.03 to 0.06 cc may be given one or two times daily for short periods. The infant should be observed carefully for evidence of arsenic intoxication.

In the presence of congenital syphilis, mercurial therapy is imperative and may be combined with the arsenic treatment to good advantage.

Lande suggests the use of intramuscular injections of normal human blood. The blood is drawn from the vein of a healthy adult with a Wassermann needle and allowed to flow into a flask containing small glass beads. It is shaken about five minutes and thus defibrinated and before injection is passed through a double thickness of sterile gauze. In individual cases the result may be very good, however, in a series of thirteen cases he was unable to demonstrate a marked increase in hemoglobin or red corpuscles.

It is of the greatest importance to bear in mind that, as in the case of rachitis, the treatment for anemia should be started early. It is our custom to begin the prophylactic treatment of both of these conditions in the first weeks of life.

SPASMOPHILIC DIATHESIS IN PREMATURE INFANTS.

TETANY.

Besides anemia and rachitis, spasmophilia is one of the most interesting clinical peculiarities of premature infants. The term

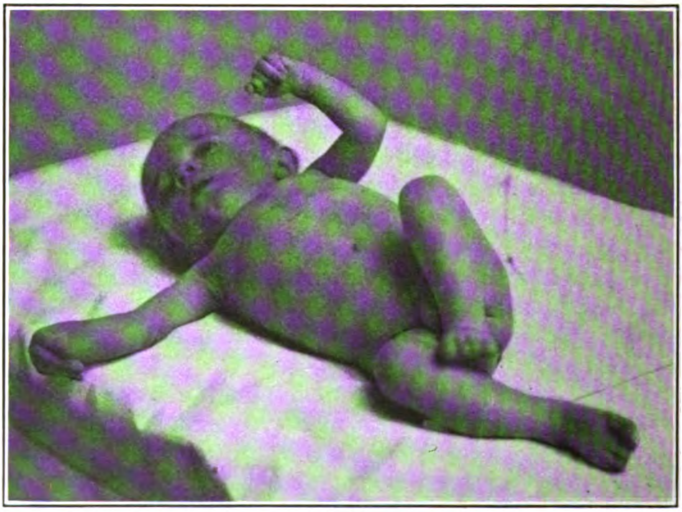


FIG. 179.—Spasmophilia. Infant in state of "tetany."

spasmophilia is used here in the sense of designating the manifestations occurring in the nursling, while tetany refers to the older child. Typical tetany with all its characteristic symptoms, the phenomena of Erb, Chvostek and Trousseau, carpopedal spasms, tonic and clonic convulsions and laryngospasm, has rarely been observed in the premature new born. While spasmophilia is by no means rare in artificially fed, full-term infants, it is found quite frequently in prematures, and not only in those artificially fed, but also in the infants fed on human milk. With breast-feeding, however, spasmophilic manifestations occur only exceptionally and lead to convulsions usually only in connection with infections.

Etiology and Symptoms.—There are other predisposing factors besides feeding in the development of spasmophilia in prematures. We may not call these factors constitutional, since in all probability they depend on the various noxæ of the extra-uterine life. These lead to a hyperirritability of the nervous system. The nervous system in all premature infants is extraordinarily lowered during the first weeks of life against all possible stimuli, including the electrical.

On the other hand, however, it is a fact that feeding plays a very important rôle in the development of spasmophilia and above all in the appearance of convulsions. Not uncommonly one observes prematures in whom the spasmophilic manifestations remain latent as long as breast-feeding is continued, but appear shortly after the institution of artificial feeding. Langstein[1] reported a case of twins in whom convulsions always appeared shortly after artificial feeding was added to human milk. There were individual differences between the twins in the ease with which the convulsions could be produced. In one infant they developed within seven to twelve days after the addition of artificial food, in the other within eighteen to twenty days.

If we systematically examine the electrical irritability in a large number of prematures we can determine that in infants fed on human milk the electrical irritability may increase to such an extent as to be C.O.C. less than 5 milliampères at the age of six to ten weeks. Rosenstern[2] studied the spasmophilic diathesis in premature infants and noted individual differences in the electrical hyperirritability, which appeared very early and frequently in breast-fed prematures. He was able to demonstrate spasmophilia in the form of electrical hyperirritability in 76 per cent of the prematures and debilitated infants that he examined.

In Ylppö's series the electrical hyperirritability was not as frequent. Among the 42 premature infants in whom he was able to determine the electrical reaction systematically during the first three to six months of life, only 15 infants (35 per cent) showed C.O.C. less than 5 milliampères. In 3 cases electrical hyper-

[1] Kassowitz, Festschrift, Berlin, 1912.
[2] Ztschr. f. Kinderh., 1913, 8, 171.

irritability was already present in the second month. One infant was on human milk-feeding and the other on mixed feeding. In the third month electrical hyperirritability appeared in 3 additional infants. It was most frequently present in the fourth month. From this data it seems that spasmophilia appears earlier in premature than in full-term infants.

Ylppö[1] also noted the especially interesting fact that the great tendency to electrical hyperirritability and convulsions, which he determined in many artificially fed premature infants, three to four months old, gradually disappeared in the fifth to sixth months without any treatment, while the feeding remained the same. In other children it often took months before the electrical hyperirritability disappeared.

In premature infants we frequently find very interesting deviations from the generally recognized symptoms of spasmophilia. It is not at all infrequent that the cardinal symptom of spasmophilia (Erb's symptom), the electrical hyperirritability of the peripheral nerves, may be absent, in spite of the manifest signs of the disorder. To know this is very important, because we know that there exist conditions in premature infants in which the electrical reaction remains increased for months, although no convulsions occur. This lack of electrical hyperirritability in spasmophilic convulsions in prematures exists not only after convulsions have taken place—which could easily be accounted for by exhaustion of the nervous system—but also before the appearance of convulsions.

In individual cases there may be pathologically increased electrical hyperirritability, even when the electrical reaction does not go below 5 milliampères for C.O.C. Rosenstern called attention to this fact and pointed out that the value below 5 milliampères for C.O.C., which is regarded as pathognomonic for the spasmophilic diathesis, was determined by Mann[2] and Thiemich[3] only for the age of eight weeks. From this it follows that this value is not to be regarded as a limit for younger infants, at least not for the younger prematures.

In the majority of cases the disappearance of the spasmophilic tendency in prematures occurs at the same time at which anemia and craniotabes begin to improve. Thus it becomes more and more apparent that the three symptoms, anemia, rachitis and spasmophilia are in a certain interrelationship. It may very well be that the same harmful factors that damage the activity of the hematopoietic organs in the first months of life and also the growth

[1] Ztschr. f. Kinderh., 1919, **24,** 1.
[2] Monatsschr. f. Psych. u. Neurol., 1900, **7,** 14.
[3] Jahrb. f. Kinderh., 1900, **51,** 99, 222.

and the normal calcification of the bones in such a high degree produce in some manner unknown to us changes in the nervous system.

Calcium Metabolism.—A calcium deficiency in the tissues has been demonstrated by numerous investigators, more especially in the brain and blood. The earlier investigations on the blood have more recently been confirmed by Howland and Marriott,[1] who found the calcium of the blood serum to be low in this condition, averaging 5.6 mg. per 100 cc of serum in a group of 18 cases, the lowest being 3.5 mg. per 100 cc of serum, the average normal amounts being 10 to 11 mg. per 100 cc. They found a normal calcium content in the serum in convulsive disorders due to other causes. These same authors found the magnesium content in the serum to be within normal limits even in the presence of active spasmophilia. The relation of calcium to the symptoms of spasmophilia has been studied extensively, especially its influence on the electric excitability. Physiologists have shown that certain mineral ions exert a specific effect on muscle-nerve irritability. Rosenstern[2] and Sedgwick[3] reduced the electric irritability in spasmophilic infants by administering large doses of calcium by mouth. Loeb's[4] findings indicate that Na and K increases the threshold for excitation, while Ca and Mg tend to decrease this. This muscle nerve irritability is the function of the quotient $\frac{Ca + Mg}{Na + K}$ as designated by Reiss.[5] During a diarrhea Holt[6] has demonstrated there is a much greater loss of Na and K than Ca and Mg in the stools. Diuresis and catharsis often cause an improvement in the spasmophilic symptoms. Consequently, there is much clinical and experimental evidence that spasmophilia is much influenced by the relationship between the Ca-Mg and Na-K group of ions.

Accidental removal of the parathyroid gland in humans and experimental excision of these glands in animals have both resulted in a tetany that resembles in its clinical manifestations the spasmophilia of infants. Following the animal experiments Howland and Marriott[7] have demonstrated a diminution in the calcium content of the blood. These findings have been verified by MacCallum and his co-workers,[8] who also found a decreased calcium content in the brain and an increased excretion.

1 Quarterly Jour. Med., 1917–1918, **11,** 289.
2 Jahrb. f. Kinderh., 1910, **72,** 154.
3 St. Paul Med. Jour., 1912, **14,** 497–519.
4 Oppenheimer's Handbuch der Biochemie.
5 Ztschr. f. Kinderh., 1911, **3,** 1.
6 Am. Jour. Dis. Child., 1915, **9,** 213.
7 Trans. Am. Ped. Soc., 1916, **28,** 200.
8 MacCallum and Voegtlein: Jour. Exp. Med., 1909, **11,** 118.

Greenwald,[1] in his experimental studies, found that the phosphorus excretion in the urine of his animals was greatly decreased (to as low as 8 per cent of the normal) shortly after operation. He also found an increase of the phosphorus content of the blood before the appearance of tetany. There was also a sodium and potassium retention. He believes that following the extirpation of the parathyroid there is a decreased excretion through the kidneys and an abnormal retention in the tissues of the alkali phosphates, which is followed by a decreased retention and an increased excretion through the kidneys as soon as the spasms develop.

There is, however, great question as to the relationship of parathyroid dysfunction and tetany in the infant. Pathological studies lead us to believe that parathyroid lesions in infantile tetany are the great exception. Parathyroid lesions have been described in patients who have shown no evidence during life of the pathognomonic findings of tetany.

In summarizing the pathogenesis we may state that a diminution of the calcium salts in all probability is the most important factor in the development of this condition. However, the possibility of an absolute or *relative* excess of the sodium and potassium salts, especially the phosphates, playing an important rôle cannot be overlooked. The relationship of disturbance in parathyroid functions to the diminution of calcium tissue content must be made the subject of further study before its importance can be fixed.

Diagnosis.—The differential diagnosis of spasmophilic convulsions in prematures is very difficult. Among the conditions to be considered are hydrocephalus, congenital syphilis and tuberculosis, epilepsy, infections, brain injuries, asphyxia and pulmonary atelectasis. Tetanus neonatorum is rarely seen today. Meningitis and encephalitis are the most important of the infectious processes, and the primary focus often is unknown. Perhaps the best test after careful history and physical examination is the determination of the electrical reactions.

It must not be forgotten that given an injured brain and a marked tendency to spasmophilia, this leads, in the premature, in the first place to convulsions and other manifest phenomena of this diathesis.

There are only a few cases in the literature where special attention has been devoted continuously from birth to the later years to the condition of the spasmophilic infants. Ylppö's material enabled him to fill this gap to a certain extent. He was able to show positively that spasmophilia in premature infants very frequently occurred after a preceding injury to the brain, and this

[1] Jour. Biol. Chem.., 1913, 14, 370.

injury rather than spasmophilia, causes the later brain changes. Spasmophilic convulsions may, however, produce extensive damage to the brain, and may result in various defects of intelligence and other cerebral disturbances.

Treatment.—The treatment of spasmophilia is largely prophylactic and embraces the therapy of rachitis and anemia (see p. 351). With the early institution and the continuation of these hygienic, dietetic and medicinal measures, the development of spasmophilic convulsions will be very unusual.

Fig. 180.—Spasmophilia—double fracture of both forearms following prolonged carpal spasm. Premature, aged six months.

If convulsions appear the infant must be kept absolutely quiet and warm. Narcotics are usually employed, the best being chloral hydrate 0.25 to 0.5 gm. per rectum and calcium bromide 0.5 to 1 gm. per day.

Lumbar puncture with the drawing-off of 5 to 15 cc of spinal fluid may give relief from repeated convulsions.

The use of general anesthesia and morphine derivatives to control convulsions is to be avoided except as measures of last resort.

Magnesium sulphate in sterile 8 per cent solution has been used in subcutaneous injections, 5 to 15 cc repeated once or twice within forty-eight hours, to control convulsions. Because of its marked depressive action on the nervous system, the infant must be very closely watched for collapse. Although this method has been much used in some clinics our experience has not warranted its use in preference to our preceding measures.

For the acute manifestations the calcium salts in maximal doses (preferably calcium lactate, 0.3 to 0.6 gm., three times daily, in solution or suspension) in our experience have been more valuable than the magnesium salts.

Administration of cod-liver oil and phosphorus or tricalcium phosphate in emulsion of cod-liver oil, as recommended in the treatment of rachitis, should be started at the same time and continued indefinitely, in doses varying from ½ to 4 cc twice daily, dependent upon the age and the indications.

Where feeding by mouth is difficult, catheter administration must be resorted to together with inert fluids per rectum. If human milk is not obtainable the best substitute is albumin milk, which is poor in whey and rich in calcium. Where stimulating treatment becomes necessary that which has been previously mentioned may be employed.

In all our measures extreme gentleness must be used, as any rough handling or violence, in case of asphyxia, has a very harmful influence.

PART IV.

THE OUTLOOK FOR THE PREMATURE.

CHAPTER XIX.

PROGNOSIS.

In estimating the outlook for an infant born before the natural termination of the normal period of pregnancy, one must consider the prenatal and the postnatal factors before arriving at a conclusion. Of prenatal influences the most important is: (1) The absolute age; (2) the physiological development and absence of constitutional anomalies; (3) transmitted parental conditions; (4) the presence of malformations. Of postnatal conditions the occurrence of any of the various diseases of the new born affects the prognosis unfavorably as a rule while the temperature and general behavior are of the utmost value in judging of its chances for life. In addition to these, the time at which the infant is received for treatment, and the character of the treatment it receives, go far in determining the probable outcome. While all factors must be taken into consideration, yet those of the most practical value relate to the child's behavior. Ability to nurse and swallow, coupled with strong muscular movements and a good cry, are the principal indications that the infant possesses a fair degree of vitality and resistance to disease, and that with proper care and nourishment it stands an excellent chance of resisting the enemies which threaten its existence during the first few weeks of its career, namely, cold and infection. At first doubtful, the prognosis becomes better as time passes in proportion to the care the child receives with respect to its hygiene and feeding. The secret of success in raising the premature lies in avoiding cold and infection, and in the proper selection of food as regards quality, quantity and method of administration.

There is not the slightest doubt but that the premature infant born of healthy parents, who is without congenital deformity and who survives the first few days of life, is entirely capable of complete and perfect development. The various factors that affect the outlook may be considered in detail.

Age.—The prognosis of the premature infant depends in the first place chiefly upon the actual (fetal) age, or in other words upon the length of time it has remained within the uterine nest (Pfaundler); the infant born before the twenty-seventh week of pregnancy having but a slight chance of living. Other things being equal, those who are not too young can be raised.

The influence of the age on the mortality is well shown by the figures of Potel:

Age.	No. of children.	Number dying.	Per cent.
6½ fetal months	56	45	80.4
7 "	131	76	58.1
7½ "	53	17	30.1
8 "	110	39	35.5

Sherman quotes the figures of several observers and includes those of his own experience in the Children's Hospital, Buffalo:

Incubator.	Tarnier Per cent.	Charles Per cent.	Sloane Hospital Per cent.	Gilbert Per cent.	Sherman Per cent.	Cook Per cent.
Saved at:						
6 months . .	30	10	..	20	0	17
6½ " . .	..	20	66	..	..	20
7 " . .	63	40	71	35	50	50
7½ " . .	..	75	89	..	66	
8 " . .	85	..	91	85	..	74
8½ " . .	95	..	..	..	100	

Sherman's table shows the fallacy of the popular belief that more children are saved at the seventh month than at the eighth. All things being equal the older the premature the better its chance of life.

Great confusion exists in a study of various statistics because of the misapplication of the term "months"; the latter should apply to lunar months (twenty-eight days) and not calendar months or better the age should be stated in days or weeks to avoid all confusion.

The Germans have usually considered one hundred and eighty-one days as the minimum period after which life may be sustained, while the French laws regard one hundred and eighty days of uterine life as necessary to viability. That one hundred and eighty days (six and one-half lunar months or nearly twenty-six weeks) are necessary to existence is disputed by many.

The exact age of an infant is not easy to determine. In fact, it is most difficult, due to the uncertainty as to the beginning of pregnancy. As previously stated, the statement of the mother as to her last menstrual period or as to the time that life was first felt are notoriously uncertain, and weight, length and other head and body measurements are uncertain factors in determining the degree of unripeness of the premature child. The most accurate method at hand today to determine the age of the premature infant is by roentgenograms of the skeleton, since the osseous development is more regular and offers more factors for consideration than determining the age based on length and other measurements (see "Skeletal Development," p. 101).

Weight.—This is a much less dependable factor than age in estimating the outlook for the premature child. All conditions being equal, a small older child has a better chance of living than a younger one who weighs more. Nevertheless, a decrease in the death-rate accompanies an increasing birth weight. The prognosis is better, on the face of it, in a child of 2000 gm., but on the other hand, the 2000-gm. child may have a poorer chance of life because of debility (Pfaundler).

Credé reported a mortality of 83 per cent for children weighing 1000 to 1500 gm. and 11 per cent for those of 2000 to 2500 gm. weight. Here the healthy and the debilitated prematures have not been descriminated between. Separating these two classes, as François did, one finds that of 81 children born of diseased parents, 30 to 37 per cent died, while of 386 apparently well premature babies, only 12.5 per cent died.

Carlini gives as the lowest figures compatible with viability, a weight of 1000 gm. and a length of 31 cm. These figures are high as attested by an examination of the literature, where several cases are on record as surviving with either a weight or a length smaller. (See list of smallest prematures saved.)

Sherman,[1] of Buffalo, published the following table showing the number of children saved according to weight in his institution:

Weight.	Percentage saved.
2 to 2½ pounds	25.0
2½ to 3 "	50.0
3 to 3½ "	42.8
3½ to 4 "	50.0
4 to 4½ "	75.0

[1] Sherman, D. H.: Buffalo Med. Jour., **44,** 653; New York Med. Jour., 1905, **82,** 272.

Cook's[1] results were as follows:

	No. of cases.	Percentage saved.
Under 1500 gm.	17	53
1500 to 2000 "	20	55
2000 to 2500 "	20	75
Over 2500 "	5	100

The smallest infant to survive in this series weighed 1250 gm. and was 38 cm. long. The initial loss was 130 gm.; it began to gain on the fourth day and had regained its birth weight on the fifteenth day. At the age of two months, which otherwise would have been at term, it weighed 2000 gm.

These figures indicate that the heavier the child at birth, the better its chances of surviving the first few weeks or months of life. What bearing the natal weight has on the future of the child we shall see later.

The smallest prematures recorded in the literature that were saved showed the following body weights and measurements:

Author.*	Weight, gm.	Length, cm.
Oberwarth[1]	500	
J. H. Hess[2] (71 days)	690	
(72 days)	740	
Oberwarth[3]	750	35.3
d'Outrepont[4]	750	37.0
Meyer[5]	750	
Roth[6]	750	31.0
Heller[7]	800	
	840	32.0
Ylppö	840	
L. E. Frankenthal	850	
Pfaundler[8]	860	35.5
Waegeli[9]	860	31.0
Klinker[10]	895	
Ahlfeld[11]	900	34.0
Pizzini[12]	900	30.0
Jardine[13]	907	
Villemain[14]	950	38.0
Maygrier and Scwab[15]	970	
Heiberg[16]	975	
Rommel[17]	980	
Ahlfeld[18]	980	37.0
Tissier[19]	990	31.0
Schmid[20]	1000	35.0
Kopp[21]	1000	
J. H. Hess[22]	1070	
Reber[23]	1120	

[1] Arch. Ped., 1921, **33**, 201.
* References will be found on page 376.

Martha and Augusta were two of triplets born of a Greek family at six and a half months, and were delivered by a midwife. The mother visited the children at the hospital on the fourth day after their birth and on the following, the fifth day, gave birth to a third, still-born fetus with a second placenta, and was again out on the ninth day. No less interesting were some of the deformities in the case of Baby Martha of the interesting group of triplets. She had but two fingers on one hand, and both knees and elbows were ankylosed in extension; in fact, there seemed to be an absence of the joint surfaces; while Baby Augusta had freedom of motion in all of her joints. Considering their prematurity, six and a half months, their weight at birth, 740 and 690 gm., respectively, together with the deformities in Baby Martha, it is surprising to find them surviving to seventy-two and seventy-one days, when both succumbed during attacks of cyanosis, due in all probability to overfeeding.

Because of Baby Augusta's better development, she was fed greater quantities from the start and although she did not have as great an initial fall in weight, both continued to lose until the twentieth day, Baby Augusta losing a total of 200 gm. and Baby Martha 230 gm. in this time. The records are rather incomplete as to the food given in Case II during this period. In Case I the estimates run from 65 to 89 calories per kilo. From the twentieth day on both infants showed almost stationary weight with food values below 120 and the greatest gain on an energy quotient between 130 and 140; and death in both cases with an energy quotient of over 200.

Temperature.—In order to correctly estimate the power of resistance of a premature infant it is necessary to consider the degree of depression of the temperature and with it the weight of the child. The figures of Budin show that the lower the temperature the more serious any further reduction will be, and the less the weight the more easily the child succumbs. In weaklings in whom the temperature was 32° C. or less (89.6° F.) the mortality was 98 per cent when they weighed 1500 gm. or less; 97.5 per cent when they weighed between 1500 and 2000 gm.; 75 per cent when they weighed more than 2000 gm. When the rectal temperature fluctuated between 32° and 33.5° C. (89.6° and 92.3° F.), the mortality of the first group was 97.3 per cent; of the second group 85.6 per cent; and of the third group, weighing 2000 gm. or over, 69.2 per cent. Thus it is necessary to consider both the weight and the degree of hypothermia. The most striking contrast is seen in comparing the figures of the Maternité and the Clinique Tarnier, Paris. To the former are often brought infants with a

FIG. 181.—Two of Greek triplets weighing 690 and 740 gm.

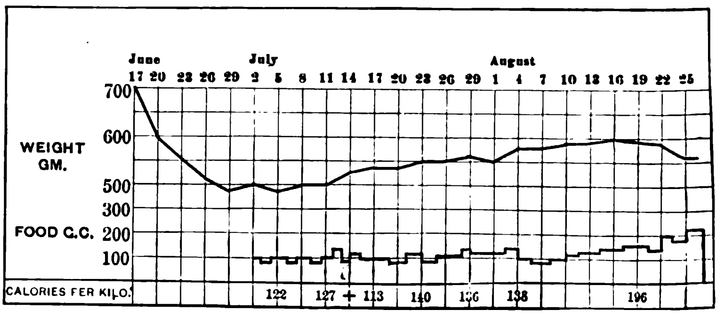


FIG. 182.—Weight and food curves of first of Greek triplets. Birth weight, 690 gm.

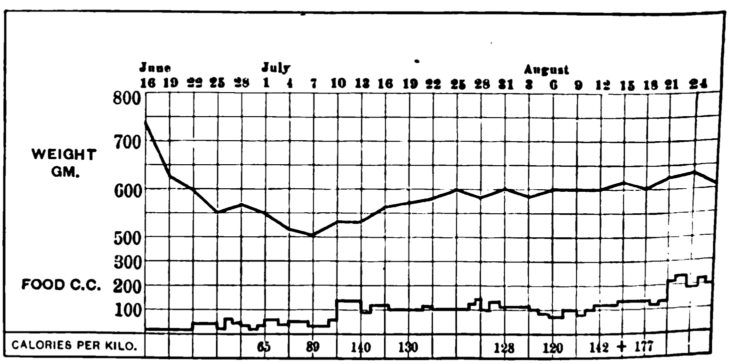


FIG. 183.—Weight and food curves of second of Greek triplets. Birth weight, 740 gm.

temperature lowered to 32°
these neglected weaklings of a
90 to 98 per cent. At the
taken to conserve the bod
infants of the same weight i
Sherman's experience is
10 babies having a rectal te
all but 2 died.
Porak and Durante esti
body temperature may s
follows:

Infants with weight l
Infants with weight 1
Infants with weight
Infants with weight

Ylppö disagrees with
that the mere cooling of
but that many of thes
the victims of birth inj
resulting in fatality.
Apert reported a pre
which lived. Ylppö s
in spite of a temperat
at birth, remained al
persist too long, w
especially of the lung
between mortality a
his material in the
Our experience h
temperature is soo
prognosis is grave.
rise of temperatu
abrupt rise afte
unfavorable. If
and remains the
its work properl
Body Measure
are of assistan

temperature lowered to 32° C. (89.6° F.); the mortality among these neglected weaklings of a weight of 2000 gm. or less ranged from 90 to 98 per cent. At the Clinique Tarnier every precaution is taken to conserve the body warmth and here the mortality of infants of the same weight is only 23 per cent.

Sherman's experience is comparable with that of Tarnier. Of 10 babies having a rectal temperature of 35.5° C. (96° F.) or less, all but 2 died.

Porak and Durante estimated the lowest degree to which the body temperature may sink, with reparation still possible, as follows:

Infants with weight less than 110 gm., 34° C. (93° F.)
Infants with weight 1100 to 1300 gm., 30° C. (86° F.)
Infants with weight 1300 to 1750 gm., 29° C. (84° F.)
Infants with weight above 1750 gm., 28° C. (82.4° F.)

Ylppö disagrees with the French observers in that his belief is that the mere cooling of the body surface does not result in death, but that many of these infants with subnormal temperature are the victims of birth injuries or brain hemorrhage, the latter factors resulting in fatality.

Apert reported a premature with a temperature of 30° C. (86° F.) which lived. Ylppö states that he has seen a series of infants who in spite of a temperature of only 27° to 28° C. (80.6° to 82.4° F.) at birth, remained alive. If the subnormal temperature does not persist too long, with resulting capillary damage and edema, especially of the lungs, recovery is possible. To show the relation between mortality and subnormal temperature, Ylppö summarizes his material in the table on page 368.

Our experience has been that unless the child with subnormal temperature is soon placed in surroundings more favorable, the prognosis is grave. If, when placed in an incubator, the resulting rise of temperature is retarded, it is an unfavorable sign. An abrupt rise after a previously stationary hypothermia is also unfavorable. If the body temperature rises to 37° C. (98.6° F.) and remains there, one may say that the nervous system is doing its work properly.

Body Measurements.—Other measurements beside those of weight are of assistance in estimating the viability of the premature.

TABLE SHOWING RELATION BETWEEN MORTALITY AND SUBNORMAL TEMPERATURE IN PREMATURES.

	37–35° C. (98.6–95° F.).			34.9–33° C. (95–91.4° F.).		
	No.	Death at: 5 days.	Death at: 1 month	No.	Death at: 5 days.	Death at: 1 month
Group I . . . . 600 to 1000 gm.	3	2 66.60%	..	12	8 66.60%	11 91.60%
Group II . . . . 1001 to 1500 gm.	40	10 25.00%	15 37.50%	46	15 32.82%	19 41.30%
Group III . . . . 1501 to 2000 gm.	76	7 9.12%	16 21.05%	40	3 7.50%	8 20.00%
Group IV . . . . 2001 to 2500 gm.	85	4 4.71%	5 5.88%	19	1 5.26%	5 26.32%
	32.9–31° C. (91°–87.8° F.).			30.9°–29° C. (87.6–84.4° F.).		
Group I . . . . 600 to 1000 gm.	5	4 80.00%	5 100.00%	6	5 83.30%	
Group II . . . . 1001 to 1500 gm.	27	7 25.90%	13 48.10%	12	7 58.30%	10 83.30%
Group III . . . . 1501 to 2000 gm.	20	5 25.00%	10 50.00%	12	2 16.60%	7 58.30%
Group IV . . . . 2001 to 2500 gm.	8	.. ..	2 25.00%	1		
	28.9°–27° C. (84.2°–80.6° F.).			26.9°–25° C. (80.4°–77° F.).		
Group I . . . . 600 to 1000 gm.	6	5 83.30%	6 100.00%	1	..	1 100.00%
Group II . . . . 1001 to 1500 gm.	7	3 42.80%	6 85.70%	1	..	1 100.00%
Group III . . . . 1501 to 2000 gm.	5	2 40.00%	3 60.00%			
Group IV . . . . 2001 to 2500 gm.	5	..	1 20.00%	1	1 100.00%	

Ostrcil gives figures which show a rapid lessening of mortality in infants weighing over 2000 gm. and of a length greater than 44 cm.:

Weight. Grams.	Viability. Per cent
1400	0
1500	0
1600	17
1700	27
1800	21
1900	33
2000	47
2100	50
2200	43
2300	49
2400	58
2500	54
2600	62
2700	59
2800	63

Length. Cm.	Viability. Per cent
40	21
41	20
42	25
43	28
44	51
45	50
46	55
47	58

Similarly Pfaundler demonstrated the decreasing mortality with increasing birth weight:

Age in fetal months.	Body weight.		Body length, cm.	Mortality in first weeks of life, per cent.	Surviving, per cent.
	Normal fetuses, gm.	Prematures, gm.			
6	1300	1000	35	95	5
6.5	....	1200	37	82	18
7	1800	1500	39	63	37
7.5	....	1800	42	42	58
8	2500	2200	45	20	80

The Cause of Labor.—Generally speaking, in those infants whose early birth depends upon the induction of labor, the outlook is better than when it results from spontaneous delivery. The following percentages are given as saved after induced labor:

Author.	Born alive per cent.	Saved, per cent.
Hahl	75.0	59.5
Raschkow	84.8	78.6
Heymann	..	71.2
Ahlfeld	..	90.9
Lorey	74.0	60.0
Hunziken	..	83.5
Ostrcil	..	56.9

That there are exceptions to the above statement cannot be doubted. For example, the occurrence of albuminuria may lead to the induction of labor, the child being not only premature but a weakling with low weight and vitality. On the other hand the infant born as a result of the shock attendant upon operative interference in non-suppurative appendicitis would in all probability possess excellent vitality. The artificial induction of premature labor with its associated trauma to the infant plays a very important part in the mortality. The foregoing figures are to be seriously questioned as there is no record of the birth weight, which in many instances was undoubtedly well above 3000 gm. and therefore not strictly applicable to the premature infant.

Prenatal Influences.—The health of the mother during the period of gestation is of the utmost importance in prognosticating the immediate future of the premature and the weak. The occurrence of syphilis, tuberculosis, alcoholism, eclampsia, nephritis, severe heart disease, or other conditions producing faulty nutrition of the fetus—all have their effect on the well-being of the infant. Of special importance is the occurrence of syphilis or nephritis. Though necessarily the age and weight of the child have a direct bearing upon its physiological development, yet the occurrence of constitutional diseases in the child is even of greater importance. Despite the greater age and the comparatively good development of a premature, the existence of a prenatal syphilitic infection or of an inherited predisposition to tuberculosis greatly jeopardizes the prognosis. If constitutionally well, the infant under 1000 gm. weight can live, providing sufficient attention is paid to the three conditions governing the survival of these infants. On the other hand, prematures or even full-term infants, the victims of parentally derived disease, often do not survive, regardless of the care they receive.

Francillon attempted to group the prematures in relation to the

Etiology.	No.	Not followed up	Followed through.	Death by: 5th day.	1 month.	1 year.	Over 1 year.
1. Diseases of the mother:							
(*a*) Lues	26	..	26	4 = 15.38%	10 = 38.46%	18 = 69.23%	19 = 73.08%
(*b*) Tuberculosis, spontaneous labor	9	3	6	.....	1 = 11.11%	2 = 22.22%	2 = 22.22%
Artificially induced	3	1	2	1 = 33.33%	1 = 33.33%	2 = 55.57%	2 = 66.67%
(*c*) Other infectious diseases (influenza, scarlatina, pneumonia, grippe)	7	1	6	1 = 14.29%	1 = 14.29%	2 = 28.57%	3 = 42.86%
(*d*) Eclampsia, spontaneous labor	12	2	10	.....	.....	4 = 33.33%	4 = 33.33%
Artificially induced	9	1	8	3 = 33.33%	3 = 33.33%	4 = 44.44%	4 = 44.44%
(*e*) Chronic or subacute albuminuria (nephritis), spontaneous	13	..	13	3 = 23.08%	5 = 38.46%	7 = 53.85%	8 = 61.54%
Artificially induced	3	..	3	1 = 33.33%	1 = 33.33%	2 = 66.67%	2 = 66.67%
(*f*) Heart disease, diabetes	13	1	12	3 = 23.08%	5 = 38.46%	5 = 38.46%	7 = 53.85%
2. Habitual familial premature birth	4	..	4	1 = 25.00%	1 = 25.00%	1 = 25.00%	1 = 25.00%
3. Premature birth because of anomalies or diseases of the sex organs—passages	3	..	3	.....	.....	2 = 66.67%	2 = 66.67%
(*a*) Small pelvis—Caesarean section	2	..	2	1 = 50.00%	1 = 50.00%	1 = 50.00%	1 = 50.00%
(*b*) Myoma uteri or other anomalies of the uterus	8	1	7	2 = 25.00%	2 = 25.00%	3 = 37.50%	4 = 50.00%
(*c*) Placenta prævia	9	..	9	3 = 33.33%	4 = 44.44%	4 = 44.44%	4 = 44.44%
(*d*) Diseases of the uterus, *i. e.*, adnexa, endometritis, gonorrhea	8	..	8	2 = 25.00%	5 = 62.50%	6 = 75.00%	6 = 75.00%
4. Premature birth following trauma, fall, blow, etc.	30	5	25	4 = 13.33%	5 = 16.66%	7 = 23.33%	7 = 23.33%
5. Twin pregnancy	128	12	116	20 = 15.63%	28 = 21.88%	43 = 33.59%	48 = 37.50%
6. Twin pregnancy	12	..	12	3 = 25.00%	4 = 33.33%	6 = 50.00%	8 = 66.67%
7. Unknown causes	369	43	326	66 = 17.89%	127 = 34.42%	178 = 48.24%	188 = 50.95%
	668	70	598	118 = 17.66%	204 = 30.54%	297 = 44.46%	320 = 53.53%

cause of the prematurity and to show the death-rate for each group. He considers as premature all infants born with a weight below 2900 gm. Of 2271 births, 832 were premature, a proportion of about 36 per cent. Of these 832 prematures the number born dead was 76, a still-born death-rate of 9.1 per cent. Of these 76 born dead, 59 died in utero. The rest died during labor either as the result of accident or of mutilating operations. Of 756 born alive, 39, or 5.1 per cent, died during their stay in the maternity department, that is, during the first three weeks of their existence. In grouping them according to the cause of the prematurity, Francillon finds that:

Because of obstetrical intervention: 6 out of 28 cases died (21.4 per cent).

Because of twins: 5 out of 49 cases died (14.2 per cent).

Because of albuminuria: 3 out of 23 cases died (13 per cent).

Because of syphilis: 8 out of 75 cases died (10.6 per cent).

Because of heart disease: 1 out of 13 cases died (7.7 per cent).

Because of unknown causes: 13 out of 499 cases died (2.7 per cent).

Ylppö, discussing the various factors which are concerned with the etiology and mortality of prematurity, presents the table on p. 371.

Deformities.—Certain deformities affect very materially the well-being of the premature child and not infrequently are important factors in the causation of labor before term. One of the most important compatible with life is cleft palate, either with or without hare-lip.

There are some features which are especially noteworthy. Of the 668 cases, more than half, 369, were due to unknown causes, which probably could have been explained by mild disorders or malpositions of the uterus. The prognosis in tuberculosis is much better than in lues—a mortality of 33.33 per cent in the former as contrasted with 73 per cent in the latter. Acute infections of the mother do not often appear in the premature, but are very important in bringing about premature delivery. Infants born of eclamptic and nephritic mothers have a very high mortality because of the fact that labor is shortened and often artificially induced, so that death most often results from the damage incidental to delivery. Diabetes and cardiac decompensation have a very deleterious effect on fetal development. The birth of twins is closely linked with prematurity and in Ylppö's series this class was 19.2 per cent of the total (128 of 668 cases).

Interference with the proper taking of nourishment complicates an already difficult problem, that of feeding, and impairs the child's chances of living. Of other deformities, atresias of the digestive tract are not uncommon and generally speaking offer an absolutely bad prognosis unless limited to the rectum and anus.

Illegitimacy.—Bakker paid attention to this phase of the birth of premature infants born in the Eppendorfer Hospital, Hamburg, from 1907 to 1912. Of one group weighing from 2000 to 2500 gm., 80 per cent of the legitimate children survived for at least one year, while of the illegitimate only 61.3 per cent lived that long. Thus the mortality in the illegitimate is seen to be almost twice as high as in the legitimate of the same weight. Of those weighing from 1500 to 2000 gm. the mortality among the legitimate was 30 per cent, among the illegitimate about 47 per cent. Of a group of 75 weighing from 1000 to 1500 gm. only 10 lived to leave the institution. Four of these were followed up, of which only one, a legitimate child, was alive at the end of the year.

Thus we see that the death-rate among the illegitimate born ranges from half again to twice as high, or even higher, than in the legitimate, depending upon the weight at birth. This difference is accounted for largely by the inferior care the illegitimate infant receives at the most critical period of its existence, the first few days after birth.

Infectious Diseases.—The secret of success in raising premature infants lies in three directions: (1) In the prevention of chilling of the body surface with the production of a subnormal temperature; (2) in the administration of the proper diet; (3) in the prophylaxis against infectious diseases.

Of the commoner infections *erysipelas* results fatally in the majority of cases. It is usually violent in its course in the very young and is frequently accompanied by signs of cardiac failure. The prognosis of *tetanus neonatorum*, fortunately now very rare, is generally unfavorable, even worse than with older children. In *ophthalmia neonatorum* the outlook is good when proper treatment is instituted sufficiently early. In sepsis the prognosis is bad, varying in direct proportion with the age and the immaturity of the infant attacked. The greater the number of organs involved the poorer the child's chances. In *gastro-intestinal* and other *visceral* hemorrhages as well as in other varieties of bleeding in the premature new born, the outlook depends upon the underlying cause or disease; sepsis, syphilis, asphyxia, etc.; but in general, it is grave, even more so as a rule than the underlying condition when uncomplicated.

Other Diseases of the New-born Premature.—*Icterus of the new born*, unless due to atresia of the biliary passages, offers a favorable prognosis and is not followed by complications. Recovery usually occurs from *moist gangrene of the cord* unless the infection spreads to adjacent parts. Only in the very weak are *umbilical ulcers* followed by extensive tissue destruction. *Inflammations of the umbilical cord*, usually seen in the very feeble, of necessity offer a poor prognosis. *Arteritis* has a comparatively favorable outlook, but *umbilical phlebitis* is almost invariably fatal.

General Conditions.—Of all prognostic signs, the study of the general condition of the premature infant offers the best evidence of the child's viability. If it cries strongly, exhibits vigorous movements, tends to stay awake and possesses well-developed ability to nurse, its viability may be considered as established and its opportunity for maintaining life good. On the other hand, if there is a tendency to deep sleep, to apathy, to asphyxia and cyanosis or to hypothermia, if the nursing ability is poor and there is difficulty in swallowing, the outlook is bad for the infant. Sometimes several days of observation are necessary in order to pass judgment upon the viability.

The condition of the turgor of prematurely born infants is of considerable importance as a prognostic sign. Absolutely flabby prematures with a poor turgor and a poor tonus prove to be lost in almost all cases. Prematures with a good turgor and a good tonus, even with a low weight, almost always live up to expectation. It is highly probable that the tissue turgor is conditioned by the mode in which the water is held. Where the water content is diminished the turgor decreases. The presence of water is closely connected with the presence of alkalies in the tissues and, therefore, it might be correct to state the hypothesis that the alkali deficiency of the prematurely born leads to a poor tissue turgor and therefore to inability to live (Langstein).

The greatest number of premature children die in the first few days of life. This is because of birth trauma, the unfinished condition of the organs, or the result of postpartum conditions or constitutional diseases or lack of facilities for proper care. At autopsy the cause of death is often not to be found, although the unripeness of the infant is evident.

Although 1000 gm. is accepted as nearly the low weight compatible with life, exceedingly small babies may live and thrive as is attested by the cases previously listed on page 44 (Physiology).

GENERAL MORTALITY.

A consideration of the preceding factors, prenatal and postnatal, forms the basis for the mortality statistics which have been compiled by Ylppö in a series of over a thousand premature infants.

The above figures are quite accurate to the first year. Beyond this it was difficult to follow the patients. However only about 70 of the series could be followed. Of the 668 prematures 320, or

	Number.	There perished.								Not followed up.
		On 1st day.	Up to 5th day.	In 1st month.	Up to 6th month.	In 1st year.	In 2d year.	In 3d year.	In 5th year.	
Total number . . .	668	62	120	206	275	301	315	319	320	70
		9.28%	17.96%	30.84%	41.17%	50.33%	52.67%	53.34%	53.51%	
600 to 1000 gm. . .	37	14	27	31	33	34				1
		37.84%	72.90%	83.80%	89.20%	94.40%				
1001 to 1500 " . .	183	28	57	88	111	114	117	...	118	8
		15.30%	31.10%	48.10%	60.60%	65.10%	66.80%	...	67.40%	
1501 to 2000 " . .	240	16	26	56	83	96	102	103	...	26
		6.70%	10.80%	23.30%	34.50%	44.90%	47.70%	48.10%		
2001 to 2500 " . .	208	4	10	31	48	58	62	65	...	35
		1.90%	4.80%	14.90%	23.10%	33.50%	35.80%	37.50%		

	Number.	There perished.								Not followed up.
		On 1st day.	Up to 5th day.	In 1st month.	Up to 6th month.	In 1st year.	In 2d year.	In 3d year.	In 5th year.	
Total number . . .	128	11	19	30	38	43	47	48	..	12
		8.59%	14.84%	23.43%	29.68%	37.07%	40.52%	41.38%		
600 to 1000 gm. . .	4	2	3	4	..	4				
		50.00%	75.00%	100.00%	..	100.00%				
1001 to 1500 " . .	36	5	12	16	18	19	20			
		13.89%	33.33%	44.44%	50.00%	52.78%	55.50%			
1501 to 2000 " . .	51	4	4	6	11	15	16	..	..	4
		7.84%	7.84%	11.76%	21.57%	31.91%	34.04%			
2001 to 2500 " . .	37	..	..	4	5	5	7	8	..	8
				10.81%	13.51%	13.51%	24.14%	27.59%		

53.5 per cent, died. About 50 per cent survived to one year. An interesting feature is the fact that the mortality after the first year fairly well approximates that of full-term children. In the first to the fifth day of life the greatest death-rate is noted and is linked with the damage to the infant in the course of labor.

Because of the fact that the etiological factors in the birth of twin prematures differs greatly from those of single birth (usual absence of infectious and constitutional disorders in the mother), Ylppö considers this class separately:

The table shows that at the end of the first year the total mortality was 37.07 per cent, considerably less than with single births.

REFERENCES.

1. Jahrb. f. Kinderh., n. f., **60**, 377 and short reports.
2. A Study of the Caloric Needs of Premature Infants, Am. Jour. Dis. Child., 1911, **2**, 302–314.
3. Oberwarth: Ergebn. d. inn. Med. u. Kinderh., 1911, **7**, 191.
4. d'Outrepont: Abhandlungen und Beiträge geburtshilflichen Inhalts, Part I, p. 167.
5. München. med. Wchnschr., 1912, No. 47, p. 2596.
6. Roth: Ztschr. f. Kinderh., 1913, **5**, 134.
7. Heller: München. med. Wchnschr., 1912, No. 47, p. 2596.
8. München. med. Wchnschr., 1907, No. 29, p. 1417.
9. Gynaecologia Helvetica, 1917, autumn edition, p. 199.
10. Allgemeine deutsche Hebammenzeitung, 1903, p. 289.
11. Lehrbuch der Geburtshilfe, 3d edition, 1903, p. 214.
12. L'arte ostetrica, November, 1908.
13. Cited by Mansell: British Med. Jour., 1902, **1**, 773.
14. Nouvelles Archives d'Obstetrique et de Gynecologie, 1895, Répertoire, No. 2, p. 50.
15. Bulletin de la Société Obstétr. de Paris, 1907, p. 216.
16. Monatsschr. f. Geb. u. Gynäk., **17**, 369.
17. München. med. Wchnschr., 1903, No. 37, p. 1603.
18. Lehrbuch. d. Geb., 1903, 3d edition, p. 214.
19. Zentralbl. f. Gynäk., 1912, No. 19, p. 626.
20. Allgem. Deutsche Hebammenzeitung, 1911, No. 11, p. 235.
21. Schmidt's Jahrbücher, **3**, 128.
22. Unreported case.
23. The Prematurely Born, Cor.-Bl. f. schweiz. Aerzte, Basel, 1918, No. 27, **48**, 897.

CHAPTER XX.

THE FUTURE OF THE PREMATURE INFANT.

THE early small, thin face with its mass of wrinkles in the presence of proper feeding soon becomes rounded out by the deposit of layers of fat, the skin becomes smoother and the face more nearly like that of a normal nursling. There, however, remains for a more or less indefinite period a prominence of the sucking cushions greater than that seen in the normal infant. The enlargement of the tongue may be noted until toward the end of the first year. The same may be true of exophthalmos. The small stumpy nose may also retain its characteristic appearance until the end of the first year. The "doll" type of face is also usually present until after the fourth or sixth month of life.

The infants often show the adenoid appearance, due to the small nose, with its tendency to the development of a posterior rhinitis, and the large tongue. This appearance is lost as the megacephalus disappears.

The other characteristic physical changes, which are evidenced by a short neck, a long, broad trunk, with a deeply seated navel and short legs, and which can usually be noted by the second to the fourth month of life, gradually disappear during the second year.

The question is often asked as to what is the ultimate outlook for prematurely born infants who live beyond the first year of life. It is desired to know (1) if they suffer from a higher mortality in early childhood than the full-term infant, and (2) are those that survive normal mentally and physically. In order to answer these questions in the proper way it is necessary to observe the children over a period of years. With institutional children this is often impossible and even in private practice difficult. Usually one must satisfy himself with comparisons at the end of the first year. For this comparison the full-term normal child is used as a basis, but as Pfaundler says, only those who have been similarly fed and raised under the same hygienic conditions can be fairly contrasted.

One observes with the premature as with the full-term child that the breast-fed infants raised among good home surroundings have a lower mortality than the same in institutions, and that the artificially fed have a greater mortality than the breast fed.

Ostrcil gives the statistics from the Prague Maternity on 1542 illegitimate prematures. The total mortality of these infants was 52.7 per cent. Of these cases 814 were followed for nine, ten and eleven years. Of this series 86.6 per cent are living, but these figures include those in whom there was no indication of syphilis, those who received breast milk after leaving the institution, and those weighing up to 2800 gm. Those under 2500 gm. weight and 45 cm. length, who left the institution alive and had, therefore, survived the first weeks, were 86 in number, of which 38 were boys and 48 girls. Of these 51 were alive at the end of the first year, 23 boys and 28 girls, a mortality of 40.7 per cent, or 39.5 per cent for males and 41.7 per cent for females.

Oberwarth's results are shown in tabulated form below. He followed for more than a year 12 infants who weighed less than 2000 gm. and who were, with 2 exceptions, illegitimate and raised under poor hygienic surroundings.

Entrance.		Later examination.			General physical development.	Mentality.
Length, cm.	Weight, gm.	Age.	Weight, gm.	Length, cm.		
46.0	1880	17 mos.	10,750	79	Good; has eight teeth, walked at fifteen mos.	Normal.
43.6	1890	17 "	10,250	77	Walks alone	Normal.
43.3	1960	24 "	9,700	75	Good; has twelve teeth; walked at fifteen mos.; has a congenital hip dislocation	Very good.
41.5	1460	30 "	9,750	77.6	Rachitis; anemia; congenital hip dislocation	Normal.
40.0	1750	40 "	10,900	80	Severe rachitis; not walking	Backward.
44.0	1820	54 "	15,000	95	Very good	Very good.
41.5	1710	60 "	10,900	86	Anemia; large head; convulsions	Good.
	1250	66 "	13,900	103	Flat occipital region	Fair.
44.2	1980	6.5 yrs.	14,400	101	Anemic	Nervous.
42.5	1710	6.5 "	17,000	106	Anemic for eleven mos.; now 100 per cent hemoglobin	Normal.
41.0	1500	6.8 "	16,700	109	Rachitic deformities	Backward.
45.5	1950	8.2 "	21,300	123	Good	Normal.

A comparison of these results with those attained with a similar group of full-term infants reflects with credit on the future development of the premature. The tendency to anemia and the results of rachitis, to both of which the premature is frequently subject, are not uncommonly seen in the early years of childhood. Whether the lack of resistance is a result of the shortening of the period of intra-uterine nutrition, or whether it is due to extra-uterine factors, more especially underfeeding and improper care during infancy cannot be stated, but we are inclined to believe that the former factor outweighs the latter.

According to Feer, many infants overcome their handicaps and make good progress, so that by the end of the second or third year their measurements are about the same as those of the normal child. Some, however, do not do as well as this, showing tendencies to rachitis, spasmophilia and especially anemia. The pallor developing toward the end of the first year depends in many instances upon the lack of iron deposits which are made in great part during the last few months of intra-uterine life; in other instances it depends upon a lack of development of the blood making organs.

Wallich and Fruhinsholz analyzed the previous history of older children and adults and also ascertained the later history of the prematurely born. Possibly the earliest instance of prematurity on record is that of the Professor at Padua who was born at the end of the seventh month and lived to be eighty. Other famous prematures include Newton, Rousseau, Voltaire, Cuvier, Victor Hugo, Lamartine and Renan.

The outlook for the future of the premature is shown to depend in a large measure upon the degree of development at birth, as evidenced chiefly by the weight. Of 17 infants weighing between 900 and 1500 gm., studied by Wallich and Fruhinsholz, 41.1 per cent developed into normal adults, a similar percentage were but slightly handicapped, while the balance were much below normal. Of the 25 weighing between 1500 and 2000 gm., 52 per cent were normal and 36 per cent slightly handicapped. Of the 36 between 2000 and 2500 gm., 75 per cent were normal and 22.2 per cent retarded. The last group comprised 65 weighing from 2500 to 3000 gm. of which 78.4 per cent were normal and 20 per cent somewhat under the normal.

The same authors traced back to birth the history of 180 children from Broca's surgical clinic and 620 inmates of the asylum for the epileptic and feeble-minded. Twelve per cent of the former and 8 per cent of the latter were known to be of premature birth. Thus we see that a large percentage of the prematurely born develop normally in both mind and body, while the balance exhibit varying degrees of inferiority, hernias, club feet, enuresis, pavor nocturnus, etc. These signs of degeneracy are seemingly the result of the prematurity and of the trauma sustained at the time of delivery.

The studies of Ylppö on the development of the premature from infancy to the school age led him to make certain generalizations.

The growth of premature infants (those with a weight below 2500 gm.) discloses a considerable derangement in the first three to five years of life. This discloses itself in that the weight, length, skull and thorax growth in almost all this class is slower. This retardation in growth is the more marked, the less the body weight and length.

Growth disturbances appear immediately after birth and are proportionately more marked in the first six to twelve extra-uterine months of life. At the age of two to four years there begins a gradual equalization, which in most instances ends at about five or six years. From this period on the curves of growth are parallel with those of full-term children. Only in the case of very small prematures with a birth weight of 1000 gm., the reparation does not seem to be completed by the age of five to six years. The growth in length is up to this time disturbed approximately to the same extent as the mass growth.

The chest, which is proportionately deficiently developed in prematures, also shows on the average, until the age of three years a retardation in growth. In the years following, the breast circumference, however, reaches practically the same value as in full-term children of a similar age. The cross-section of the chest of the premature approaches more the form of an ellipse than a circle. The cross-section area is in the smallest prematures strikingly small in comparison with the body length. The growth of the head is the least disturbed or retarded. This is explained by the fact that the brain growth in premature follows certain individual laws without depending, as a rule, on the development of the body.

The principal point of these growth disturbances in the premature is immaturity. The more immature an infant is born, the more deficient is the function of the various organs in extra-uterine life. Especially in the province of digestion are variations noted in prematures, because of poor utilization, particularly of fat and salts, a qualitative undernourishment results which favors the development of growth disturbances.

Besides these and other exogenous factors, certain endogenous factors probably play a passing but noteworthy part in the production of growth disturbances. All these defects, however, gradually disappear or are overcome, so that reparation is completed by the time the premature reaches the school age. From this time on the growth again turns back to the paths which have been designed for the hereditary body mass of the child.

Ylppö was able to follow up 89.52 per cent of his cases and thus compiled his figures for the mortality and future development of the premature.

INFANTS WITH A BIRTH WEIGHT UP TO 2500 GM. IN THEIR FIRST EIGHT YEARS.

Year of birth.	Total No.	Not followed up per cent.	Followed through.	In 1918 at end of year of life.	Of these still alive per cent.	Of these dead, per cent.
1918 . .	48	...	48	½	16 = 32.65	33 = 67.35
1917 . .	57	2 = 3.51	55	1	35 = 63.64	20 = 36.36
1916 . .	98	3 = 3.06	95	2	46 = 48.42	49 = 51.58
1915 . .	90	5 = 5.56	85	3	42 = 49.41	43 = 50.59
1914 . .	101	11 = 10.89	90	4	40 = 44.44	50 = 55.56
1913 . .	85	13 = 15.29	72	5	30 = 41.67	42 = 58.33
1912 . .	83	10 = 12.05	73	6	30 = 41.10	43 = 58.90
1911 . .	57	16 = 28.07	41	7	19 = 46.34	22 = 53.66
1910 . .	48	10 = 20.83	38	8	20 = 52.63	18 = 47.37
	668	70 = 10.48	598		278 = 46.49	320 = 53.51

These statistics show that only 40 to 45 per cent of the premature infants lived to the school age. Twin prematures showed a somewhat better average—50 per cent.

TWINS WITH A BIRTH WEIGHT UP TO 2500 GM. IN THEIR FIRST EIGHT YEARS.

Year of birth.	Total No.	Not followed up per cent.	Followed through.	In 1918 at end of year of life.	Of these still alive, per cent.	Of these dead, per cent.
1918 . .	9	...	9	½	5 = 55.56	4 = 44.44
1917 . .	14	...	14	1	10 = 71.43	4 = 28.57
1916 . .	26	...	26	2	15 = 57.69	11 = 42.31
1915 . .	15	...	15	3	7 = 46.67	8 = 53.33
1914 . .	18	5 = 27.78	13	4	7 = 53.85	6 = 46.15
1913 .	14	...	14	5	7 = 50.00	7 = 50.00
1912 . .	11	2 = 18.18	9	6	4 = 44.44	5 = 55.56
1911 .	11	3 = 27.27	8	7	7 = 87.50	1 = 12.50
1910 .	10	2 = 20.00	8	8	6 = 75.00	2 = 25.00
	128	12 = 9.38	116		68 = 58.62	48 = 41.38

In concluding it may be said that the future of the prematures who survive is on the whole good. They seem to be somewhat more subject to hydrocephalus and to psychic and nervous anomalies, such as enuresis and night terrors, and to anemia, rachitis and spasmophilia. Many are precocious, even original children. They tend to remain light in weight and short in length, but this is usually equalized by the time of entering school.

It is generally the case that in those infants who survive, most differences between the premature and the full-term child have disappeared by the time of puberty, and therefore every effort should be made to preserve all perfectly developed premature infants.

WALKING AND TALKING.

It is well known that in premature infants we may not expect the development of certain faculties, namely, speaking and walking, at the same time as in full-term infants. Wall states that his premature infants learned to talk seven and a half months later and learned to walk six months later than full-term children. He also reports that certain speech defects, as stuttering and stammering, occurred more frequently in his prematures. These differences, however, became equalized later on.

In general, the smaller the premature at birth, the greater is the delay in its learning to talk. It is rather an exception to the rule when infants that have been born weighing 1000 to 1500 gm. learn to talk before they are two years old. The following table shows when children of Ylppö's series learned to walk and to talk a few words:

The age and number of children when they were able to speak:

9 months to 1 year	3
1 year	9
1 year, 3 months	48
1 " 6 "	18
1 " 9 "	54
2 " 3 "	1
2 " 6 "	10
2 " 9 "	1
3 " 6 "	1
4 "	1
Unknown	37

The age and number of children when they started to walk:

9 months to 1 year	3
1 year	15
1 year, 3 months	46
1 " 6 "	52
1 " 9 "	28
2 "	25
2 " 3 "	8
2 " 6 "	4
2 " 9 "	2
3 "	4
3 " 3 "	1
4 "	1
Unknown	26

The statements as to the time at which the child spoke the first words, and when it started to walk vary widely in individual cases. Only intelligent mothers are able to make reliable statements pertaining thereto. On the other hand the delay in learning to talk and to walk depends in many cases not upon the docility or development of the infants, but upon the efforts of its mother or nurse.

From the preceding facts it follows that the small prematures learn the first sounds and the first words on an average of one year and six months. This occurs then about six months later than in full-term infants. The age at which the child learns to walk is about the same as that at which it learns to talk. This may be regarded as a proof that learning to walk depends in a healthy child upon its mental development.

CONSTITUTIONAL INFERIORITY.

The various lesions, either of traumatic nature, due to delivery itself or extra-uterine life, brought on by deficient resistance or deficient functional capacity of the different organs, result in various clinical symptoms, which have been designated under the collective

FIG. 184.—Infant born at thirty-six weeks. Birth weight, 1500 gm. Intense icterus, melena, double inguinal, lumbar and umbilical herniæ. Photograph taken at six months. Still showing evidence of megacephalus.

name "constitutional inferiority." Everything seems to point to the fact that this constitutional inferiority in the strict sense of the word does not occur in a much higher degree in premature infants than in full-term children, if we do not include the various gross anatomical malformations.

FIG. 185.—Same child, aged two and one-half years.

FIG. 186.—Same child, aged four and one-half years. Megacephalus has entirely disappeared.

We have reason to assume that many prematures who remain weaklings in their later life and show other signs of inferiority,

FIG. 187.—Infant born at thirty-four weeks. Complication, spastic paraplegia.

suffered from some constitutional anomaly, intra-uterine, or post-*natal* trauma, or were born in a state of physiological immaturity.

FIG. 188.—Child shown in Fig. 187, showing standing posture.

This view seems to be especially strengthened by the fact that the more premature and the smaller the infants come to the world, the more frequently they suffer with idiocy, Little's disease, serious anemias, rachitis and other diseases based upon the condition of deficient resistance.

The proportional diminution of various pathological symptoms with increasing birth weight would be difficult to understand in

Fig. 189.—Child shown in two previous illustrations, showing good results following tendon transplantation. Mental development in advance of age.

terms of congenital constitutional lesions. Also the frequent disturbances of growth in premature infants, especially during the first years of life, have some connection with this passing poor condition of the premature. Later, strikingly good reparation of the growth disturbances shows best that this state is not dependent upon congenital constitutional factors.

THE MENTAL DEVELOPMENT OF THE PREMATURE INFANT DURING EARLY CHILDHOOD.

In order to review this subject properly, it is necessary to divide premature infants into two large groups: (1) Prematures without pathological changes; and (2) those born with pathological changes due to constitutional diseases and congenital malformations. In the well-developed fetus which has not been damaged during the time of conception, and which is born at an age compatible with a physiological development necessary to meet its needs for life and which suffers no undue traumata during or following birth, a normal mental development may be expected. External influences will affect its mental growth as well as its physical development, therefore, it must be raised in a suitable environment and be judiciously fed. It may be stated that the longer the intra-uterine life of the fetus, the less the dangers of interference with its normal mental growth. It is quite natural to expect, therefore, that these immature infants are more subject to mental disturbances and defects than the full-term infant. Abnormalities in development need not be explained by anomalies in the embryo, but rather may be due to direct external traumata of a mechanical, dietetic and of an infectious nature. Thus, there remains no other choice than to make the intra-uterine and extra-uterine noxæ responsible for the frequent cerebral disturbances, be they connected with spastic states or idiocy, with or without spasms.

It is our experience that the majority of premature infants born after the thirty-second week into a proper environment without birth injuries, undergo a normal mental development. That these individuals are more subject to rickets, anemia and spasmophilia with their consequent effects on the nervous system is not to be forgotten. But all of these conditions are amenable to therapeutic procedures with only a limited after effect.

In the second group belong those suffering from constitutional diseases and congenital malformations. These individuals cannot be classified in groups as to their future development, but each one must be considered individually. While congenital lues usually leaves its mark in the full term, in the premature it is even more grave in its consequences. However, much can be expected from proper and early therapeutic measures. In those suffering from hemorrhages into the cerebrum and spinal cord, it is easy to understand that in the premature infant that has survived in spite of these lesions sequelæ may manifest themselves in later life. We would especially impress upon the physician the fact that not all infants with cerebral hemorrhages die in the first days of life but that many survive. Cerebral hemorrhage may not be suspected until late mental and physical signs develop.

The prognosis in this group must always be made with considerable reservations.

However, on the whole, it may be stated that mental development goes hand in hand with physical development. To this broad statement there are, however, many exceptions, and while we do see a number of these infants with good physical development who are of low-grade mentality, in our personal experience we have come in contact with a larger group of premature infants with a high grade of mental development, even to the point of precocity. They tend to remain light in weight and short in length, but this is usually equalized by the time of entering school.

INDEX OF AUTHORS.

In the preparation of this volume the following authorities were consulted.

GENERAL INDEX.

F

G

H