THOMAS MORGAN ROTCH, M. D.

PEDIATRICS

The Hygienic and Medical Treatment

OF

CHILDREN

BY

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INSTRUCTOR IN PEDIATRICS, HARVARD UNIVERSITY

PHYSICIAN IN CHIEF AT THE INFANTS' HOSPITAL

FOUNDED UPON THE TEACHINGS OF THOMAS MORGAN ROTCH, M. D.

VOLUME I

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To

CLARENCE JOHN BLAKE, M.D., O.M. (Vienna), F.A.C.S.

Professor of Otology (Emeritus), Harvard University

THIS VOLUME IS INSCRIBED

As a tribute to his high professional attainments and to his unfailing interest in the welfare of children, and in grateful remembrance of many acts of kindness

BY THE AUTHOR

PREFACE

The "Pediatrics" of Dr. Thomas Morgan Rotch was for many years the official text-book used in the Harvard Medical School. Professor Rotch originated many new ideas, and his book contained many contributions of permanent value in the teaching of pediatrics. The last edition was published in 1906, and is now entirely out of date. At the time of Dr. Rotch's death, he was contemplating the bringing out of a new edition.

It seemed a pity that the permanent and distinctive features of Dr. Rotch's teaching, which constitute the foundation of the teaching of pediatrics at Harvard, should not be preserved in written form. As much therefore of this teaching as has seemed to the author of permanent value, has been incorporated in the present work, with such revision as modern progress demands.

The work is not, however, designed to be merely a revision of Dr. Rotch's book. It is designed to be an entirely new modern text-book of the diseases of infancy and childhood.

Recent progress in the diagnosis and treatment of disease is becoming more and more centered upon the problems of etiology. For this reason, especial attention has been devoted to the discussion of the causes of the various diseased conditions of early life. Modern diagnosis concerns itself less with names than in former times, and consists in a recognition of the nature of the lesions and functional disturbances which constitute disease, and of their causes. Modern treatment concerns itself mainly with the prevention or removal of etiological factors.

Progress in knowledge is so rapid, that before a book on any subject in medicine is published, it is likely to be already out of date. One reason for this is that the writers of text-books hesitate to incorporate many new advances in research for fear that they will not stand the test of final proof. This leads to the exclusion of much material which does prove of final value, and justifies the criticism that the latest advances, the things which are being most widely discussed, are not to be found in the most recent text-books. The author has in this book attempted to obviate this difficulty, by means of a special subdivision under each disease, which, under the heading of Problems and Research, deals with the most recent advances in scientific medicine, and summarizes the problems awaiting future solution. The reader can thus easily distinguish the proven facts from the interesting and suggestive theories.

The author acknowledges his indebtedness to Dr. William W. Howell, and Miss Margaret Farquhar, Head Social Worker at the Infants' Hospital, for many valuable suggestions, to Dr. Joseph I. Grover for revision of manuscript, to Dr. John W. Hammond for assistance in collecting material for the division on gastro-intestinal diseases, to Dr. William Weston for supplying material for the article on pellagra, and to Dr. Percy Brown for selecting and collecting the roentgenograms.

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DIVISION I

THE NORMAL CHILD

I. INTRODUCTION

Pediatrics is a branch of medicine the importance of which as a special study has gradually come to be more and more generally recognized. It has become a specialty, partly from the same causes which have led to the great increase in specialization in all branches of medicine, but more particularly because of the great number of peculiarities inherent in the manifestations of disease in early life. From the very beginning, the diagnosis and treatment of disease in infants and young children is attended by difficulties not encountered with adult patients. These young patients can give no adequate description of their subjective symptoms, and the physician must rely on intimate and profound knowledge of the ways of young children, which can only be gained by long observation, if he will read aright the manifestations before him. Moreover, the most thorough knowledge of normal and abnormal conditions, if gained wholly from observation of adult patients, is of little value in children, because of the difference in the normal standard. Not only are all the standards by which deviations from the normal are recognized different from those of adults, but there is a different standard for each age. Manifestations which are normal at one age, are abnormal at another, and even the anatomic lesions of disease are seen to be modified in the various stages of the child's development. Young human beings must be regarded, throughout their early life, as incomplete. Growth is not merely increase in size, but it is a continuous process of anatomical and functional development, which is not completed until some time after the age of puberty. In early life, arrest, or retardation of the process of normal development may in itself constitute a disease picture; developmental conditions form an important division in the etiology of disease in childhood but play no part after adult life is reached.

The incompleteness of the child's development plays a still greater rôle in those actual diseases which are produced by conditions outside the body. It may be true that the same diseased conditions are seen in childhood and in adult life. But their manifestations are greatly modified by the incompleteness of the child's development, and often vary even with the different ages of childhood. Even the

reaction to treatment differs in the different stages of development. The rules laid down for the diagnosis and treatment of these diseases common both to childhood and to adult life, if gained by a study of adults, cannot hold for children.

There is further need for special study and training in pediatrics. Children, in addition to being liable to most of the diseases seen in adults, have also a group of diseases which are entirely their own. Here again, the fundamental cause lies in their uncompleted development, through which conditions acting upon their bodies from the outside world, which can have no injurious effect upon the completed adult, have a very injurious effect upon the young, undeveloped child. These young human beings, at each stage of development, have their own peculiarities of resistance or lack of resistance to these conditions. Their treatment must be modified at all times, to correspond to their age, and stage of development. The high rate of infant mortality is still one of the greatest problems of medicine. It is due primarily to no other cause than the lack of resistance to the conditions of its surroundings which exists in the undeveloped infant. Its existence is alone sufficient to point to the need for a special study of the pathology of early life.

The fundamental requisite in recognizing the abnormal, is a thorough knowledge of the normal. In pediatrics, a thorough understanding of normal conditions at every stage of development is essential. The distinction between infancy and childhood is neither so artificial nor so arbitrary as it seems at first. At about the age of two years, certain very important processes of anatomical and functional development become completed. The incompleteness of these functions in the first two years has been an important source of difficulty, and a cause of abnormality, which later are no longer seen. Hence we refer to the first two years as infancy, and to the later years as childhood. The distinctive line between childhood and adult life is much harder to draw. It is difficult to say at just what age normal development is finally completed, but the period is arbitrarily placed at the age of puberty. From birth to the age of puberty, then, is the period through which the development of the normal child must be studied.

II. THE INFANT AT TERM

A normal infant at birth has a reddened skin, and is covered thickly in many parts by the vernix caseosa, which is removed by the first bath. The description of the newborn infant will be given in two divisions. The first will be a description of a complete physical examination of a normal infant at birth, in the form in which physical examinations are usually recorded in case histories. The second division will be a description of certain features of the infant's internal structure, in the form in which they are recorded in records of post-mortem examinations. This form of description will afford the student a standard of comparison by which he may recognize at once the abnormal, both in physical examination, and in pathological anatomy.

THE PHYSICAL EXAMINATION

The baby appears well nourished, the body and limbs are well rounded, the cry is vigorous, the extremities are warm, and the grasp of the hands is strong and vigorous. The skin is usually clear, but may be somewhat mottled, and is some shade of delicate pink. The eyes are half open when the baby is awake, and are expressionless, of a dull grayish-blue color. The spine is very flexible, and can be twisted and bent at will in any direction. The neck appears short.

HEAD —The head appears large in proportion to the body, while the face is quite small in proportion to the cranium. The length and thickness of the hair is very variable. The cranium may be somewhat distorted by the pressure of birth, but these abnormal appearances pass away in a few months. An average circumference of the cranium at birth is 33 cm. (13 in.). The anterior fontanelle may be somewhat depressed immediately after birth, but is soon on a level with the bones. Its size is variable, but the measurements are usually 2 to 3 cm. in length, by about 2 cm. in width. The frontal suture is usually open in its upper part, and the posterior fontanelle, while open, is often obliterated by overlapping of the bones.

MOUTH AND THROAT.—The mucus membrane of the mouth is of a clear pink. The tongue is slightly coated, and comparatively dry. The gums do not completely meet. The soft palate runs backward almost horizontally, descending much less than in the adult. The uvula is rudimentary.

THE EAR.—The meatus passing inward, inclines downward, and the membranum tympani is almost horizontal, so that its inspection is difficult.

THORAX.—The thorax presents a very different appearance from that of the adult. It is much smaller in proportion to the head and abdomen, forming the upper and smaller portion of the egg-shaped trunk. Its whole shape presents a peculiar appearance, which is accentuated by the small shoulders. The sternum is relatively much smaller than that of the adult male, and its top is placed relatively higher, while the sides of the thorax are relatively shorter than in the adult. The ribs are more nearly horizontal, and their borders diverge relatively rapidly. The transverse diameter is shorter in proportion to the antero-posterior.

THYMUS.—The thymus is present and well developed at birth, but its outline cannot be clearly distinguished by percussion.

HEART.—The impulse is visible and palpable rather higher and nearer to the mammary line in the infant than in the adult. The entire position of the cardiac dulness is higher in proportion to the chest walls. The infant's heart is less covered by the lungs than is the adult's. The superficial dulness lies between the left border of the sternum and the mammary line, and the entire area can easily be covered by the tip of the finger used in percussing. The upper border, and the relative dulness are difficult to determine. The heart sounds are still largely of the fetal type, the diastolic pause being absent, and the first sound being much like the second. The rhythm is regular. Murmurs are frequently present at birth, without any abnormal significance; only their persistence should attract attention.

LUNGS.—The lungs are resonant to percussion, but at birth the resonance is less than in later life. The respiratory murmur is loud and harsh.

ABDOMEN.—The abdomen is large in proportion to the thorax. Its physical examination differs from that of the normal adult only in one important particular, namely, in the relatively large size of the liver. Its border is felt fully 2 cm. below the edge of the ribs in the right epigastric and hypochondriac regions, and its upper border of dulness encroaches on the resonance of the right lung to the extent of fully one rib and interspace. The dull area of the spleen is rarely perceptible, but when found corresponds to that of the adult. The border is not normally palpable. The bladder is an abdominal rather than a pelvic organ, but normally gives no dulness in a newborn infant.

TESTICLES.—The testicles are normally found in the scrotum.

LIMBS.—The limbs are well formed, and present no features of particular note in newborn infants. The grasp of the hands is remarkably strong. The feet appear flat, but this apparent flatness is due to a pad of fat tissue, and not to any flattening of the arch.

HEIGHT AND WEIGHT.—The height and weight are variable. The average height of a newborn male infant is 49.5 cm. (19¾ in.); in the female it is 48.5 cm. (19¼ in.). The weight is still more variable than the height. The average weight for males is about 3,520 grammes (7¾ pounds), and for females is about 3,290 grammes (7¼ pounds).

THE SPECIAL SENSES.—Although at birth the eye is anatomically perfect, visual perception is not developed. Hearing appears dull during the first few days of life. The sense of touch is well developed. No satisfactory conclusions can be drawn as to taste and smell.

The newborn infant passes very little urine. He does not usually perspire. He cannot cry tears. He can have a movement of the bowels, which consists of meconium. Meconium is inodorous, viscid, slightly acid, and of a brownish-black color. It consists of bile constituents and intestinal secretions, and contains mucus, epithelium, and fat drops from the vernix caseosa. At birth, it is sterile.

INTERNAL STRUCTURE

Among the features in the anatomy of the newborn infant which cannot be perceived by ordinary physical examination, only those will be described which present essential differences from those of adults.

BRAIN.—The brain of the newborn infant is proportionately very much larger than in the adult.

THE NASO-PHARYNX.—The nasal cavity is relatively long and shallow, and its respiratory portion is very narrow. The opening of the posterior nares is relatively very small. The naso-pharynx is simply a narrow passage running obliquely backward and downward from the constricted opening of the posterior nares. The lymphoid tissue on the posterior wall of the pharynx is well developed, and much richer in absorbents than are the faucial tonsils. The openings of the Eustachian tubes are opposite a higher part of the nose than in the adult, although their direction is more horizontal. The ends of their cartilages, which make such prominent folds in the adult, are not developed, so that these prominences do not exist.

TEETH.—There are at birth twenty embryo teeth, ten in each jaw, enveloped in their tooth sacs.

EAR.—The development of the ear in its several parts is very unequal at birth. The structures of the internal ear and of the tympanic cavity are fully formed at birth, while the external auditory meatus is very different in its development from that of later life. The mastoid antrum exists at birth, but the cells are wholly undeveloped.

THYMUS GLAND.—The thymus gland exists at birth, is well developed, and lies partly above, and partly in front of the heart.

HEART.—The anatomy of the heart at birth, and the changes in the circulation which take place shortly after birth, are so intimately connected with the subject of congenital cardiac disease, that their description will be postponed till that subject is discussed. In a baby dying immediately after birth, it would not be abnormal to find an open foramen ovale, or an open ductus arteriosus.

LUNGS.—The lungs at birth present a very notable difference from the lungs of older individuals. Their chief characteristic is their embryonic type. The alveoli are relatively small in size, and their number is small in proportion to the bronchioles. Their walls are relatively thick, and the connective-tissue stroma is in greater proportion. Blood vessels are relatively abundant, and play a more important rôle than the lymphatic absorbents. These conditions, while gradually diminishing, persist to a great extent throughout childhood.

KIDNEYS.—The kidneys and adrenals are of relatively large size in the newborn infant. The kidneys are markedly lobulated. A prenatal condition called the uric acid infarction exists normally in the kidneys at birth. This shows itself as an orange or light red deposit in the straight tubules, which cause these tubules to appear prominent on section of the organ. This deposit consists of urate of ammonia, amorphous urates, uric acid crystals, and epithelial cells. The adrenals quite cover the tops of the kidneys at birth.

STOMACH.—The stomach at birth is remarkably small, and more tubular than in the adult, the fundus being but slightly developed. It is consequently even more vertically placed, for it is the enlargement of the greater curvature which causes the later obliquity of the stomach's axis. Its capacity is about 25 to 30 c.c.

The Infant at Term

Fig. 1

Stomach, natural size. Infant three hours old.
Warren Museum, Harvard University.

THE INTESTINE.—The chief peculiarity of the intes
born infants, is that it is much less fixed than in adul
difference is most striking in the large intestine, particu
cecum, ascending colon, and sigmoid flexure, which s
siderable mesentery. The average length of the small
287 cm. (9 ft. 5 in.); of the large intestine it is 56 cm. (1
The total variation may amount to 61 cm. (2 ft.).

LYMPHATIC SYSTEM.—The lymphatic system is
oped and active at birth.

BONE MARROW.—At birth, and indeed, throughou
months of life, the bone marrow is red. The red color i
the numerous injected blood vessels, and is more intense
central portion of the bones.

III. NORMAL DEVELOPMENT

In following the normal development of the baby through the periods of infancy and childhood, those changes will be considered first which appear on physical examination. In a second division will be considered the development of internal structure.

NORMAL DEVELOPMENT AS SEEN ON PHYSICAL EXAMINATION

THE CORD.—By a process of disintegration the cord separates from the living tissues at the umbilicus, and falls off. This occurs at about the seventh or eighth day. The umbilical scar normally is always depressed.

THE SPINE.—The flexibility of the spine gradually becomes less as the infant grows older, although it always remains greater than that of the adult spine. There are at birth no natural curves in the spine except the sacral curve. As the dorsal curve is a permanent condition, part of the general curve of the body, it is the development of the cervical and lumbar curves which are of interest from the point of view of development. The cervical curve is produced by the pull of the muscles of the back of the neck, when the child begins to raise its head. The child usually begins to hold up its head, only the trunk being supported, during the fourth month. The cervical curve begins to appear at about this period, but is never more than a habitual position, as the convexity of the front of the neck can always be obliterated by changing the position of the head. The lumbar curve is supposed to be caused by the shortness of the ilio-femoral ligaments. When the child begins to stand, these ligaments tend to incline the body forward, and this tendency is corrected by the contractions of the muscles of the back, which straighten the line of the body by throwing forward the promontory of the sacrum. The lumbar curve is first observed when the child is one or two years old, but it is not continually present till some time later, and it can be obliterated until adult life is reached.

The surface anatomy of the spine in infancy and childhood presents marked differences from that of adults. In the infant, the back is rounded from side to side, the spine being the center of a more or less continuous curve. The spinous processes are relatively undeveloped, and the laminae overlap less, look more directly backward, and are nearer the surface. At three years of age the spine shows a great change which has been coming on since the age of

eighteen months. The spinous processes now stand out in a prominent row, and although the laminae are still near the surface, the back is flatter, and there is some appearance of a median furrow. At six or seven years the spine has made much greater progress toward the adult type, and shows no important differences during the remainder of childhood.

THE HEAD.—The head grows very rapidly, especially in infancy. The average circumference at birth is 33 cm. (13 inches). The growth is most rapid during the first year, the increase in circumference amounting to about 12 cm. In the second year the increase is 2.5 cm., and in the next three years it is 5 cm. After the age of five years, the increase in circumference is very slow, and only 2 or 3 cm. have been added when adult life is reached. The growth of the thorax is so rapid, that the proportion of the head to the thorax becomes equal during the first year. The table shows the relative and proportional growth of the head and thorax during childhood.

TABLE 1

Circumferences of Head and Thorax from Birth to Thirteen Years

AGE	HEAD		THORAX	
Birth	33 cm.	(13 inches)	31 cm.	(12½ inches)
5 weeks	38 cm.	(15 inches)	36 cm.	(14½ inches)
5 months	42 cm.	(16½ inches)	41 cm.	(16¼ inches)
9 months	45.5 cm.	(18 inches)	43 cm.	(17 inches)
1 year	45.5 cm.	(18 inches)	47.5 cm.	(18¾ inches)
2 years	48 cm.	(19 inches)	51 cm.	(20¼ inches)
3 "	51 cm.	(20⅛ inches)	55 cm.	(21¾ inches)
4 "	53 cm.	(21 inches)	54 cm.	(21¼ inches)
5 "	53 cm.	(21 inches)	54 cm.	(21¾ inches)
6 "	52 cm.	(20½ inches)	55 cm.	(21¾ inches)
7 "	54 cm.	(21¼ inches)	54 cm.	(21¼ inches)
8 "	53 cm.	(21 inches)	59 cm.	(23⅛ inches)
9 "	54 cm.	(21¼ inches)	61 cm.	(24 inches)
10 "	53 cm.	(21 inches)	62 cm.	(24½ inches)
11 "	56 cm.	(22⅛ inches)	63 cm.	(24¾ inches)
12 "	53.5 cm.	(21⅛ inches)	63 cm.	(24¾ inches)
13 "	54 cm.	(21¼ inches)	66 cm.	(26 inches)

The posterior fontanelle becomes imperceptible by the sixth week. The main sutures of the cranium are not usually ossified before the end of the sixth month. The anterior fontanelle appears to grow larger up to the ninth month, and probably grows with the head, although becoming relatively smaller. It appears to remain stationary from the ninth to the twelfth month, and then gradually becomes smaller. Its average time of closing is the nineteenth month, although variations in the time of closure extending from the fourteenth to the twenty-second months are not abnormal. An open fontanelle at the end of the second year may be considered abnormal.

The small proportion of the size of the face to that of the cranium

gradually grows less. The downward growth of the face is one of the important features of development. The table shows the proportions of the face to the cranium in the different periods of childhood.

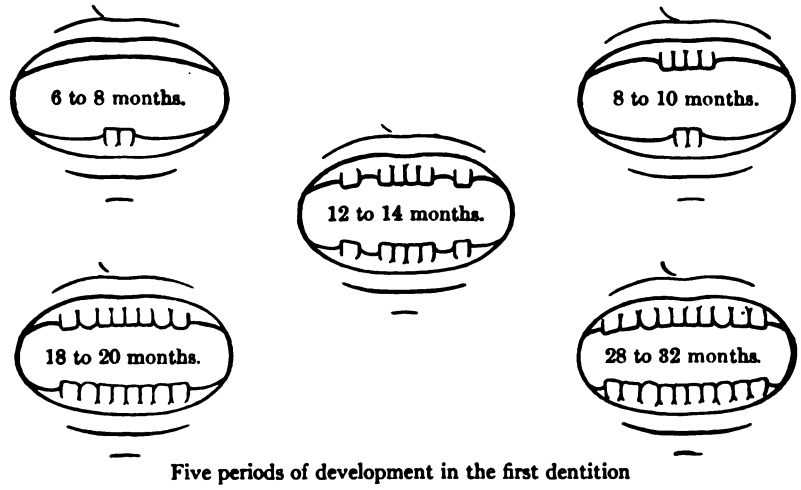
TABLE 2

Proportions of Face to Cranium

AGE	FACE		CRANIUM
Early infancy	1	to	8
2 years	1	to	6
5 "	1	to	4
10 "	1	to	3
Adult female	1	to	2½
Adult male	1	to	2

THE TEETH.—At birth, the twenty embryo teeth, enveloped in their tooth-sacs, are so enclosed in the alveolar processes of the jaws, that nothing but smooth mucus membrane is apparent on the gums above. When calcification of the neck of the tooth begins, elongation of the tooth follows, and the tooth is so enclosed that growth can only occur toward the gum, which forms the point of least resistance. This growth probably begins at birth. Pressure of the crown of the tooth causes atrophy of the gum, and finally the tooth pierces the mucus membrane. The various teeth appear at times dependent upon their order of development, which is fairly regular in a normal infant, although variations in the order and time of appearance of the teeth are so common, that they cannot be called abnormal. The first teeth usually appear from the sixth to the eighth month. Early appearance of the teeth is never a sign of disease, and children have been born with teeth. Delayed dentition is usually due to rachitis, but is seen in other conditions,

FIG. 2



Five periods of development in the first dentition

such as cretinism, and defective cerebral development. In many healthy infants, no teeth are seen before the tenth month, and dentition may even be delayed until the fourteenth month without other evidences of any abnormality. The order of appearance of the teeth is shown in the table.

TABLE 3

Temporary Teeth. First Dentition. Twenty in Number

	DENTAL PERIODS	GROUPS OF TEETH
I.	6 to 8 months	2 middle lower incisors
II.	8 to 10 months	4 upper incisors
III.	12 to 14 months	2 lateral lower incisors and 4 first molars
IV.	18 to 20 months	4 canines
V.	28 to 32 months	4 second molars
		20

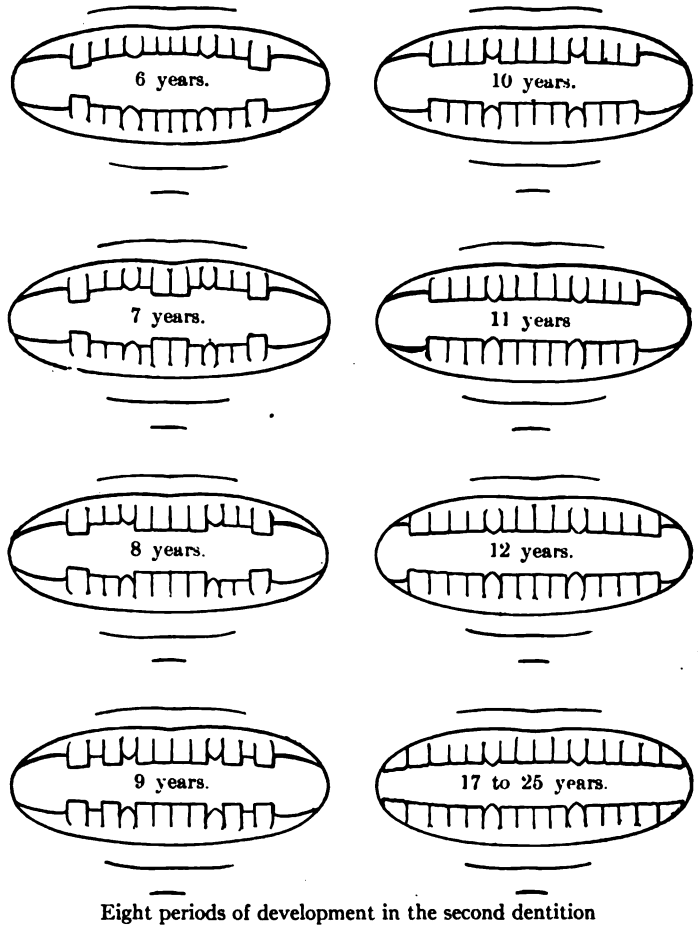
The second dentition begins at about the sixth year. The first of the permanent teeth to appear are called the sixth-year molars. They do not replace any of the temporary teeth, but the jaw having grown sufficiently to provide more space, they appear just back of the second molars of the first set. In the seventh and eighth years the permanent incisors replace those of the temporary set. In the ninth and tenth years the bicuspids replace the temporary molars. In the eleventh year the permanent cuspids (canines) replace the temporary, and in the twelfth year the four second molars appear. This completes the second dentition of childhood, twenty-eight teeth, the remaining four molars belonging to adult life.

TABLE 4

Permanent Teeth. Second Dentition. Thirty-two in Number

YEARS	GROUPS
6	4 first molars
7	4 middle incisors
8	4 lateral incisors
9	4 first bicuspids
10	4 second bicuspids
11	4 canines
12	4 second molars
17 to 25	4 third molars (wisdom-teeth)

FIG. 3



Eight periods of development in the second dentition

THE THORAX.—The thorax, which is insignificant at birth, grows rapidly. The measurements showing the rate of increase in the circumference of the thorax are seen in the table. The circumference of the thorax normally remains less or equal to that of the head throughout infancy. Not until the third year does the thorax begin to show a measurement greater than that of the head. With the growth in circumference, there occurs increase in the transverse diameter as compared with the antero-posterior, so that the thorax gradually assumes the elliptical shape which is characteristic of later childhood. At the same time, the peculiarities in the shape of the thorax which are so notable at birth, disappear, and at about the fifth year the infantile type of thorax is no longer evident.

THE THYMUS.—The thymus is most developed during the first two years of life. At no time, however, unless the organ is notably

enlarged, can its dulness be distinguished from that of the heart and great vessels.

THE HEART.—Most of the changes in the position of the cardiac impulse, and of the precordial dulness, which have been described as taking place during the development of the infant, are relative, and are due to peculiarities of the topography of the chest wall, rather than to changes in the position of the heart. That the cardiac impulse in infancy and early childhood is almost invariably found not in the fifth, but in the fourth interspace, is due to this cause. In early childhood the impulse may be found in either the fourth or the fifth interspaces, while in later childhood it is usually in the fifth. The relation of the impulse to the nipple presents similar variations. In infancy and early childhood it is usually outside the mammary line, and cannot be considered abnormal if less than 2 cm. beyond the nipple. In middle childhood, the impulse is in or near the mammary line. In later childhood it is more often seen inside the nipple, and should always be found in this position after the thirteenth year.

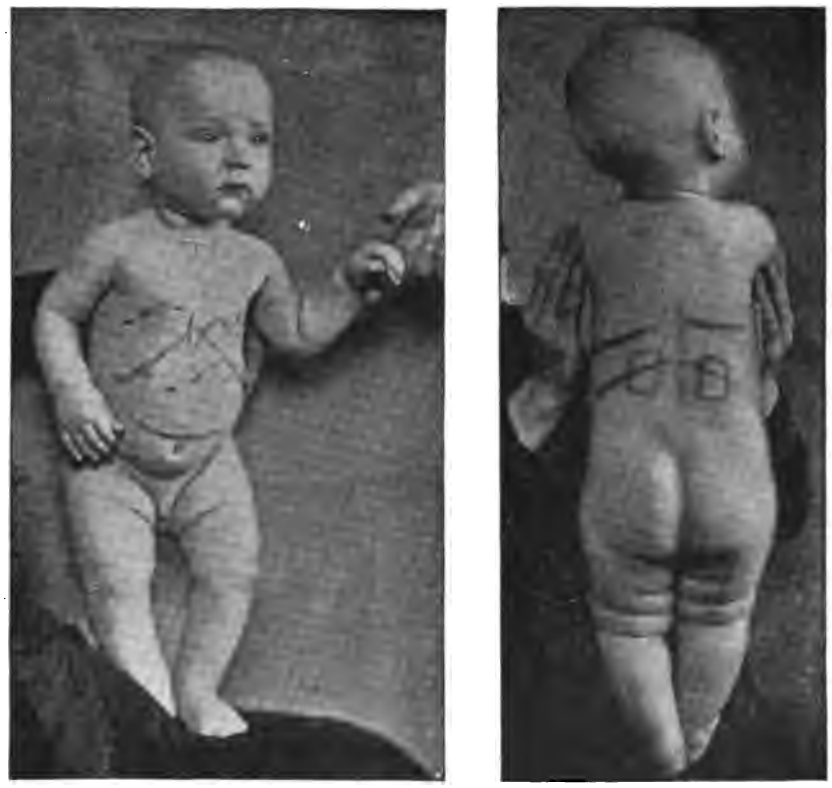
An important feature in the physical examination of the heart is seen in the position of the area of superficial or absolute cardiac dulness to percussion. In infancy and the latter part of childhood, the right border of absolute dulness is, as in the adult, at the left border of the sternum. In middle childhood it may encroach a little on the sternum, even as far as its middle. The relative or deep cardiac dulness is difficult to determine in young children. When found it is proportionately larger than in the adult. Its upper border is at the second interspace, or lower border of the second costal cartilage. On the left it extends to near the mammary line, corresponding to the position of the impulse at the various ages. On the right it follows the right parasternal line.

On auscultation, the diastolic pause remains slight or imperceptible throughout infancy, and the rapidity of the cardiac action is so great, that it is often difficult to distinguish the first from the second heart sound. Normally, however, after the first weeks of life, the first sound becomes louder than the second, and the cardiac cycle can be recognized from this. Murmurs persisting after the first week of life, are abnormal.

THE LUNGS.—The percussion note in childhood is more tympanitic than in the adult, especially under the clavicles, and in the interscapular region behind. There may even be cracked-pot resonance under the right clavicle, in healthy lungs. This tympany is due to the relatively large proportion of bronchial to alveolar air space, which persists throughout childhood, although in later childhood, the lung gradually approaches the adult type. On ausculta-

tion, the normal respiratory murmur differs entirely from that of adults. It is of a type generally described as "puerile." The breathing is rude, loud, and harsh, and expiration is heard better than in adults. It is often described as resembling the bronchial breathing of adults, but does not resemble it except in the terminology of description. The difference cannot be described, being one of quality, and can only be learned by experience and practice. It is gen-

FIG. 4

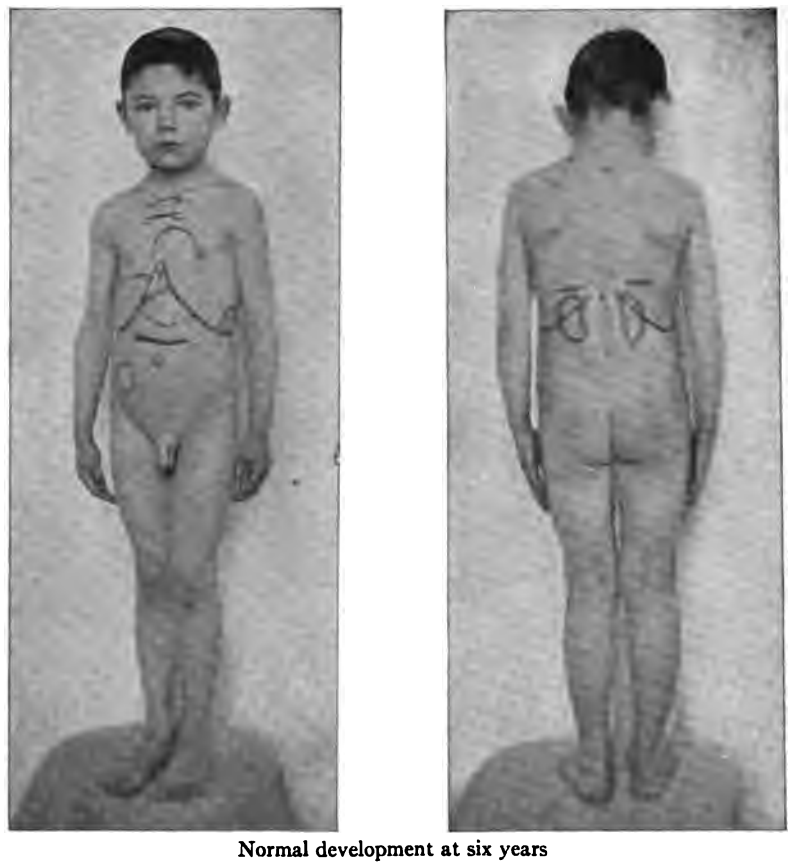


Normal infant seven months old

erally stated that this quality of the normal respiratory murmur of childhood is due to the fact that the sounds from the trachea and large bronchi are heard more plainly, because they are not transmitted through so thick a layer of lung and chest wall. This explanation does not seem to me at all satisfactory. If it were true, the sounds would have the quality of bronchial breathing, for we know that in both children and adults, we are hearing in bronchial breathing the sounds from the trachea and large bronchi, which are transmitted more easily through solidified lung. The normal respiratory murmur of children has not the so-called bronchial quality. I believe

its loud, harsh quality to be due to the greater size and number of the bronchioles in the child's lung. In later childhood, this peculiarity of the breathing gradually diminishes with the development of a greater proportion of alveolar space, as the lung approaches the adult type.

FIG. 5



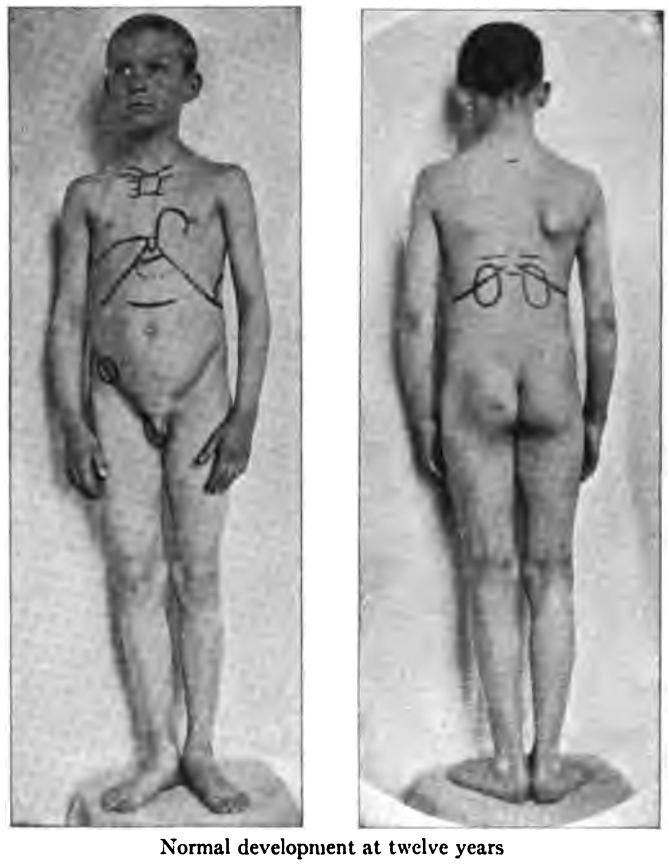
Normal development at six years

THE ABDOMEN.—Throughout infancy, the circumference of the abdomen remains about the same as that of the chest. After the age of two years, the enlargement of the chest causes the abdomen gradually to assume the adult proportion. The abdominal walls in childhood are thinner, and more easily relaxed, than in the adult.

THE LIVER.—The liver remains relatively large in infancy and early childhood. In the first two years, the edge can be easily felt about 2 cm. below the costal border in the mammary line. Later, the distance becomes somewhat less, but until the later years of childhood, the edge of the liver can still be felt below the costal border. Only in late childhood does the liver no longer descend

below the edge of the ribs in the mammary line, but even at this period, owing to the laxness of the abdominal walls, the edge of the liver can sometimes be felt by pushing the palpating fingers up under the ribs.

FIG. 6



Normal development at twelve years

THE SPLEEN.—The spleen presents no differences in size or position characteristic of childhood. It may sometimes be felt, by pushing the fingers up under the costal border, but normally is never felt below the costal border.

THE LIMBS.—The limbs in infancy are short in proportion to the trunk. Holt has found, from one hundred and fifty observations, that the length of the lower extremities at birth (measuring from the anterior superior spine of the ileum to the sole of the foot), is forty-three per cent. of the length of the body; at five years it is fifty-four per cent., and at sixteen years, sixty per cent. The pad of fat, which gives the infant the appearance of flat-foot, is, according to Dane, designed to support the arch of the foot until the mus-

cles are stronger. It is slowly absorbed, and by the fourth or fifth year, the foot presents the same appearance as in the adult.

MUSCULAR DEVELOPMENT.—At birth, the strongest muscular development is seen in the hands and forearms. Voluntary attempts to grasp objects are seen at about the beginning of the fourth month. The infant should be able to hold up its head with only the back supported, by the fourth month, though sometimes this is seen as early as the end of the second month. It begins to sit alone at between the seventh and ninth months, and creeps at ten months. Soon after this are seen the first attempts to stand, the infant trying to pull himself up with the aid of his nurse's skirts, or of pieces of furniture. It can usually stand at twelve months, and soon after, makes its first attempts at walking. There are wide variations in the age at which the normal infant learns to walk. Some infants learn to walk at twelve or thirteen months, and others, apparently normal, do not walk until they are seventeen or eighteen months old. The average age is fifteen months.

MENTAL DEVELOPMENT AND THE SENSES.—The eyes of the infant at birth are sensitive to light; by the end of the second or third week they will follow a bright light. The infant seldom smiles before the fifth or sixth week; he does not recognize objects before the sixth or eighth week. The muscles of the eye in early infancy act irregularly, and coördinate action is not seen until the third or fourth month, and sometimes incoördination is seen for a considerably longer period.

The hearing is undeveloped at birth, and for the first few days infants are deaf. The movements of respiration and deglutition cause the eustachian tubes to be cleared of mucus, air enters the middle ear, and the hearing gradually improves. After the first month it is very acute, the infant being easily disturbed by noises. Usually by the fifth month the infant will show distinct signs of distinguishing and interpreting particular sounds, and of recognizing individual voices. At this time it may very easily be frightened by loud or unusual noises.

The sense of touch, while present, is not very acute at birth, except in the lips and tongue. It develops rapidly, and by the end of the third month, is fairly acute all over the body. Sensibility to pain, while present in infancy, is dull compared with later childhood. The localization of sensory impressions is very imperfect throughout the first years of life.

Taste is said to be developed at birth, but I believe its development is poor during the earlier weeks. It is usually said to be acute throughout the greater part of infancy and childhood, this statement being based on the fact that the child easily detects changes in its

food. I believe that taste is only acutely developed in the case of the primary tastes of sweet, sour, bitter, and salt. The foods which it receives during the first year show variations only in these primary tastes. I believe that the infant does not perceive flavors, and that this inability to detect variations other than the simplest in the taste of its food persists through some of the earlier years of childhood. The infant is very sensitive to the feeling of various foods in the mouth, and it is this sensibility to the sensation of the food which is mistaken for a highly developed sense of taste. Young children do not usually object to the taste of castor oil, unless it has been suggested to them that castor oil has a bad taste.

The sense of smell has not been conclusively tested in infancy. This sense is probably present, but develops much more slowly than the other senses. Fine distinctions in odors are not detected until the late years of childhood.

SPEECH.—During the first year of its life the average infant uses its voice merely in crying to express its discomforts or desires. At about the end of the first year it usually begins to enunciate single words, and in the middle, or toward the end of the second year it learns to form short sentences. From this time on, the faculty of talking progresses rapidly, but children do not usually learn to talk connectedly till the third or fourth year. There is great variation in the time when the faculty of speech is acquired. Many children, who understand perfectly, make all their wants known by signs, and who show no other signs of delayed mental development, show a delay of from one to two years in every phase of the development of the speech faculty. Girls learn to talk earlier than boys.

DEVELOPMENT OF OTHER FUNCTIONS.—The function of the lachrymal glands does not usually develop till the baby is three or four months old. The time of its development is variable. Babies have been observed to cry tears as early as the first month.

The development of the function of the sweat glands is also very variable. It is occasionally seen developed at birth. Usually, however, perspiration is not seen till the third to the fifth week.

The salivary secretion is developed rather slowly in early infancy. There is not much saliva seen in the infant's mouth in the first three or four months of life.

THE URINE.—The function of the kidney begins quite early in fetal life, and the bladder has been found to be full of urine at birth. The urine is small in amount at birth, and during the first twenty-four hours it is not uncommon to find little or none passed. The urine which is first passed is usually dark, cloudy, and acid, and contains epithelial cells and urates and occasionally hyaline

casts; later it becomes clear, pale, straw-yellow, and usually of slightly acid reaction. Its specific gravity (1.010 at birth) falls in two or three days to 1.003, by about the fifteenth day is found to be 1.006, and rises from this time steadily till puberty. By the end of the first week and throughout childhood the amount of urine passed in twenty-four hours is relatively greater than in adult life. This in early infancy may be due to the preponderance of liquid food, but is in part the result of the infant's more active metabolism, for the urea is also found to be proportionately increased. According to Foster, the presence of uric and oxalic acid in unusual quantities is a frequent characteristic of the urine of children. It is also stated that the phosphates are deficient, being retained in the body for the purpose of building up the osseous system. The chlorides, sulphates and urinary pigments are less abundant than in the adult. The proportion of salts increases as soon as a mixed diet is given and closely approaches the normal proportion for adults. Indican is normally absent in breast fed-infants; in others it is usually absent unless there is a disturbance of digestion. The *uric acid infarction*, which has been referred to, and evidences of which may last for two or three weeks, consists of urate of ammonium (hedgehog crystals), amorphous urates mixed with uric acid crystals, and some epithelial cells. The variations in the amount of urine which has been computed to be passed during the early days of infancy and childhood are very great, as the amount in all probability depends very largely on the quantity of liquid ingested, and also upon the activity of the skin and bowels.

The difficulties in accurately measuring the amount of urine excreted by very young infants are such that few positive statements can be made as to the quantity. It is sufficient to say that it is about ninety cubic centimeters (three ounces) a day for the first few days, and then rises in amount very rapidly.

Reitz and Cruse state that during the first few days of life the urine contains more or less albumin, and that this disappears at about the seventh or eighth day, though sometimes, according to Carpenter's work, it may be detected for several weeks. Lesne and Merklen found the freezing point at birth 0° C., at one month −0.13° to −0.35° C., at two months −0.21° to −0.78° C. Sugar is occasionally found in the urine of healthy infants during the first two months.

The urine sediment of the young infant shows on microscopical examination, mucus, many epithelial cells, crystals of uric acid, urates, and calcium oxalate; also amorphous urates, occasionally a few hyaline casts, and rarely a granular cast. The urine of later infancy and childhood has no microscopical peculiarities.

INTESTINAL DISCHARGES.—The contents of the intestine continue to be mixed with meconium for three or four days or a week, the longer time being when the infant is weak and does not nurse well. After this time the infantile discharges, which have a characteristic appearance as distinguished from those of the older child, appear. When the nutrient is human milk, the discharges are of a golden-yellow color, smooth, unformed, of medium consistency, showing a large proportion of water, and sometimes changing on exposure to the air to a greenish yellow. If the baby is artificially fed, the movements are normally of a lighter yellow color, and of slightly firmer consistency. They as a rule contain undecomposed bile-pigment and bile-salts, while the older child's and the adult's discharges do not contain the bile undecomposed. The amount of fecal discharge in the first day of life is about forty-five grammes (one and one-half ounces), and increases in the following days to fifty grammes (one and two-thirds ounces). It consists of mucus, fat, epithelial remains, and a slight amount of albuminoid material. In early infancy there are from two to four discharges daily. As the child grows older there are two and finally one in the twenty-four hours. They do not lose their yellow color until amylaceous or albuminous food is given, when the different shades of brown begin to appear. They are not fully formed until something besides milk is swallowed. Starting at birth, with the sterile meconium, infection by the mouth and rectum quickly occurs, and in a short time many forms of bacteria may be found in the discharges.

TEMPERATURE.—The rectal temperature of the newly-born infant varies to a certain extent, lying between 99 and 100.5. Very soon after birth, as would be expected from the tax which is made on the infant by its surroundings, the temperature shows a slight fall, varying between 98 and 99.5. By the end of the first week, the normal temperature of the infant is regained, and during the first year, the temperature ranges from 99 to 100.5. In the second year, and during early childhood, the temperature is 98.5 to 100, and in later childhood, the normal temperature is that of the adult.

PULSE.—Immediately after birth, the pulse is often quite irregular, but soon becomes perfectly regular in rhythm. The small volume and force of the infant's pulse is proportionate to the small size of a baby as compared with an adult. The rate of the pulse presents the most extreme variations in early life, especially during the first year. It is much affected by nervous influences. The table shows the average pulse rate at the various ages for males; in girls the rate is apt to be somewhat higher.

TABLE 5

Pulse-Rate for Males

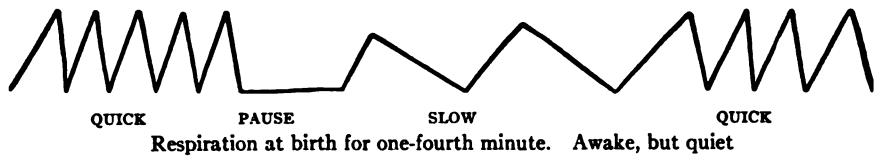
AGE	PULSE-BEATS PER MINUTE
Early weeks	120 to 140
Until 2d year	110
2 to 3 years	100
5 to 8 years	90

After the eighth year the pulse gradually acquires the adult rate.

RESPIRATION.—The rhythm of respiration in very young infants is so easily affected, varying with every disturbance, and even with changes of temperature, that a regular rate of respiration is only seen when the infant is asleep. At all other times the respiration is very irregular in rhythm, even when the child is quiet. It may be quite superficial for a few moments, and then quite deep. There are frequent pauses; at times one lung appears to be used exclusively for a brief period. A regular rhythm is not fully established till the child is two years old. The character of the respiration in early infancy is wholly diaphragmatic, and remains simply diaphragmatic until the sixth or seventh year. From this time on the costal element gradually becomes prominent.

The rate of respiration is also extremely variable. At birth it is usually from 35 to 45 per minute when the child is quiet. Until the third year it varies from 15 to 40 per minute, and from three to five years, the rate is 20 to 25 per minute.

FIG. 7



Respiration at birth for one-fourth minute. Awake, but quiet

HEIGHT.—The average height of the male infant at term is, according to a large number of measurements about 49.5 cm. (19¾ inches). Insufficient nourishment and improper food, especially as represented in rhachitic children, seem to retard the growth, while on the contrary, the various fevers seem to increase the activity of growth in length, while decreasing the total weight. In the first three or four months the growth is proportionately rapid to that in the latter part of the first year. In like manner the activity is greater in the first month than in the second, and in the second than in the third, becoming still less in the fourth, fifth and sixth months.

TABLE 6

The average increase for the first month is about 4.5 cm. (1¾ in.)
" " " " " second month is about 3.0 cm. (1½ in.)
" " " " " third to the fifteenth month is about 1 to 1.5 cm. (½ to ¾ in.)
" " " " " first year is about 20 cm. (8 in.)

TABLE 6—*Continued*

The average increase for the second year is about 9 cm. (3½ in.)
" " " " " third year is about 7.4 cm. (3 in.)
" " " " " fourth and fifth years is about 6.4 cm. (2⅝ in.)
" " " " " fifth to sixth year is about 6 cm. (2⅜ in.)

The height is about doubled in the first six years, and at fourteen years the final height has usually been attained to within about one-twelfth. The height at different ages in comparison with the weight will be shown in table. 8.

WEIGHT.—The normal infant manifests its growth and development most clearly by increasing in weight. All other signs of disturbance of normal development are not at once apparent, but manifest themselves slowly. Increase in weight, on the other hand, is a more or less continuous process of development, any disturbance of which manifests itself at once. Moreover, this growth in weight is, of all the processes of development, the one most easily disturbed by all kinds of adverse influences, and therefore is the most valuable and delicate index of health which we have. Of all the data which guide the physician in the care of infants, the records of the weights taken from time to time are the most important. The physician in the wards of an infants' hospital looks first at the weight curves depicted on the charts. In older children, the weight records are less important than in the first two years of life, but nevertheless afford valuable data throughout the period of growth.

The weight of the newborn infant is very variable, and can only be expressed by an average. It must be remembered that wherever any figure representing the normal is based on the method of averaging normal variations, a deviation from the normal figure in an individual case does not of itself constitute an abnormality. The limits of weight variation of full term infants at birth are not definitely known, but a birth weight of six pounds or over should be considered within normal limits; a birth weight of under six pounds is strongly suggestive of impaired vitality. The average birth weight for males is 7¾ pounds; for females it is 7¼ pounds.

During the first three or four days of life there is a loss of weight, which is usually designated as physiological. This initial loss of weight often amounts to 9 or 10 ounces. Not all of it is strictly physiological, but only that part of it, amounting to 4 or 5 ounces, which can be accounted for by the passage of meconium. The additional loss is nutritional in origin, and is due to an excess of tissue waste over nutriment. The secretion of the breasts is established rather slowly, and during the first days of life the infant obtains comparatively little food. The nutrition is probably further affected by the tax on the infant's vitality caused by the sudden change in its surroundings.

By the third or fourth day, when the milk secretion is established, the normal infant begins to gain continuously in weight. Failure to gain at this time is seen only when the milk secretion is established unusually slowly, when colostrum milk persists unduly, or when some other cause is present which actually produces a condition of disease. The initial weight is usually regained during the second week. If it is not regained by the third week, we must look for some cause disturbing normal development. In artificially fed infants, the initial weight is often not so rapidly regained as in the breast-fed.

During the first year of life, increase in weight is normally continuous, and comparatively rapid. The gain is most rapid during the first three months; in this period the average daily gain is almost two-thirds of an ounce. The gain is not, however, steady from day to day, a large gain on one day being often counterbalanced by a failure to gain on the next day. The rate of gain can best be judged by comparing the records from week to week. In the second three months the gain in weight is not quite so rapid. In the third quarter, the rate of gain is the slowest of any of the periods of the first year, and becomes slightly more rapid in the fourth quarter. The following table shows the average rate of gain in normal infants, in the four quarters of the first year:

TABLE 7

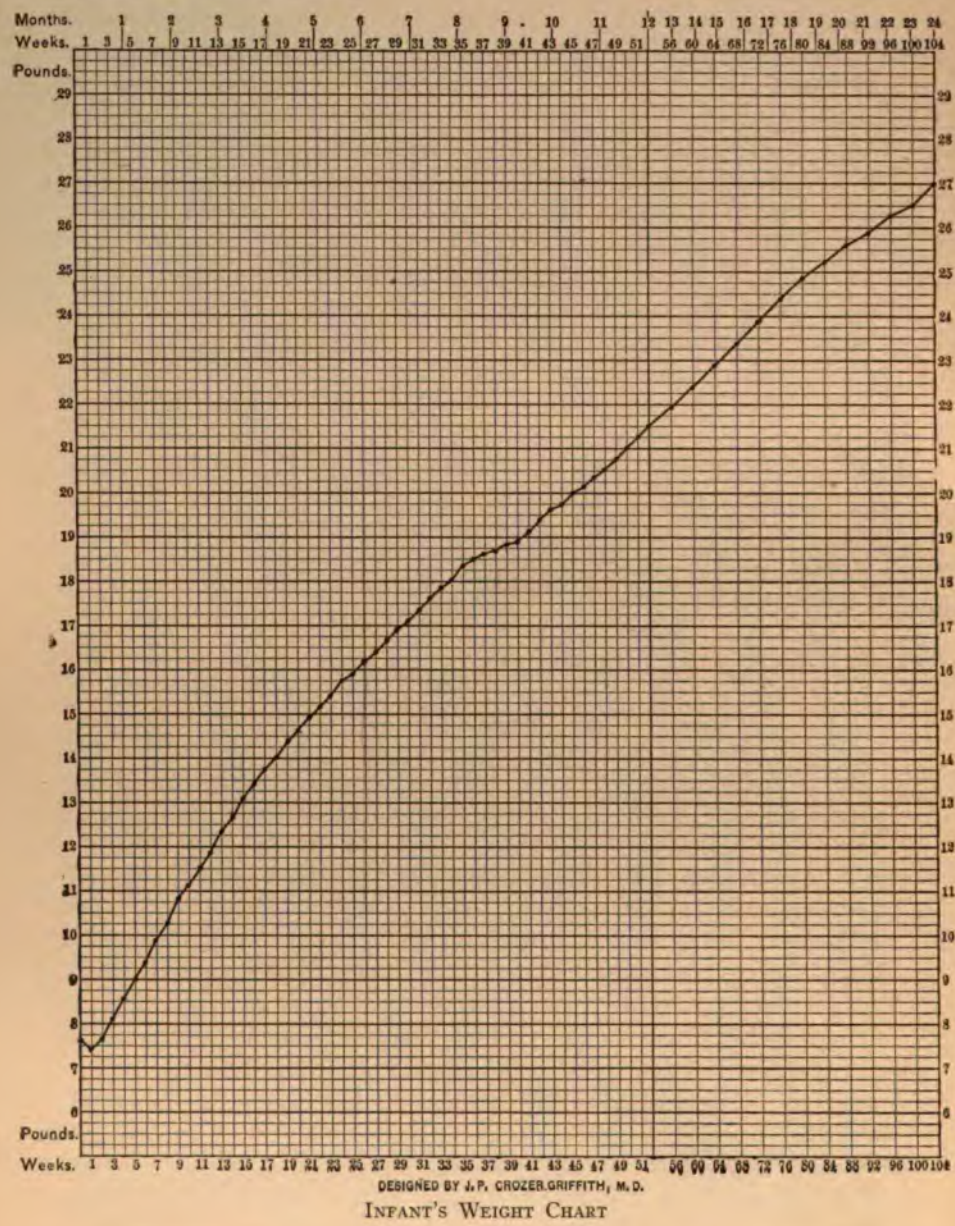
	TOTAL GAIN		WEEKLY GAIN	
	GRAMMES	POUNDS	GRAMMES	OUNCES
First three months	2400	5	180	6.2
Second three months	1920	4	148	5
Third three months	1200	2½	92	3.1
Fourth three months	1320	2¾	102	3.5

The figures for the weekly gain represent an average, and are rather larger than are often seen in babies who in every respect develop normally. One should not be worried as to the development of any baby whose weekly gain in weight was 4 ounces for the first three months, 3 ounces for the second three months, and 2 ounces for the last six months of the first year.

It is customary to picture the weight development of an infant in the form of a weight curve, such as that shown in the chart.

Such a chart represents the average rate of gain of a number of healthy babies. It must not be taken too literally in comparison with the weight curve of an individual baby. The rate of gain in individual babies is usually not so steadily continuous as in the ideal infant's weight chart. In many normal babies there are periods when the rate of gain is much less than the average, or in which there is even, for a time, no gain in weight. Such a period of slight gain or of stationary weight is particularly likely to be seen in the third quarter of the first year. In any child there are apt to be

CHART 1



INFANT'S WEIGHT CHART

periods when new processes of growth, such as dentition, or new functions, such as noticing, walking, or talking, are being rapidly developed. At such times the gain in weight is less pronounced. The weight chart is useful as a general guide for comparison in estimating

the development of the baby, but it represents an ideal condition, which is often not attained.

A further extension of such a method of comparison provides a weight index for estimating the weight development of any infant. This weight index is expressed by dividing the weight of the infant by the weight of the average healthy infant of the same age. If the quotient is multiplied by 100, the result expresses in per cent. the weight development of the baby. This is useful as a general guide, but again, must not be taken too literally as a criterion of normal development, on account of the factor of individual variation. It must also be remembered that babies who are above or below the average at birth, are apt to keep the same relation to the average throughout much of the first year. This is shown in the chart.

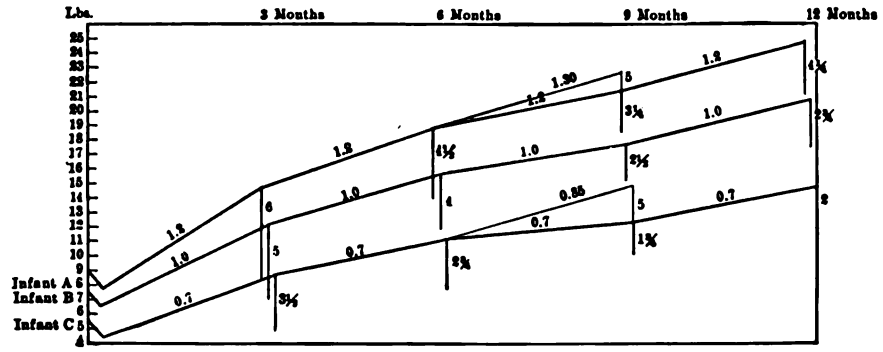


CHART 2

The middle line "B" represents the weight curve of the average healthy infant. This curve is taken as the constant by which to estimate the weight index of any infant. The perpendicular lines represent the number of pounds the child gains in the four quarters of the first year. The weight index of such an infant is 1.0, the weight development 100 per cent.

The upper curve "A" represents the theoretical curve of an infant weighing 9 pounds at birth, with a resulting weight index of 1.2. If such an infant were to maintain throughout the first year the advantage in weight development with which it starts out in life, its quarterly gains in pounds would be as are indicated in the perpendicular lines. These will be seen to vary considerably from those of the average healthy infant represented in curve "B."

In a similar manner curve "C" represents the theoretical weight curve of a $5\frac{1}{4}$ pound-baby, with a weight index of 0.7, and the perpendicular lines show the quarterly gains in pounds, which would be necessary to maintain throughout the first year the weight development with which it starts in life. These are seen to be considerably less than those of the average healthy infant.

Now, for example, if the infant C and infant A each gain from the sixth to the ninth month 5 pounds, instead of the theoretical gains expressed in the perpendicular lines, one will find by working out the above formula, that the weight index of infant C has been raised to 0.85, a gain of 15 per cent. in weight development; that the weight index of infant A has been raised to 1.30, a gain of 10 per cent. in weight development. In other words, although each infant has gained the same number of pounds in three months, the smaller infant C has gained proportionately 5 per cent. more in weight development than the larger infant A.

It is not to be supposed that every healthy infant weighing above normal at birth and having a weight development above 100 will maintain throughout the first year the same percentage of development with which it starts out in life; some will and others will not. There is in the majority of cases a tendency for the line of growth of these large infants to dip downward and gradually to approach the weight curve of the average child.

On the other hand there is a great tendency for the undersized infants, if properly fed, to grow, relatively to their initial weight development, faster than the average child, and their line of growth tends upward to meet that of the average infant. In judging the effects of feeding upon two such theoretical cases as "A" and "B" in the above example, it is obviously unfair to take the number of pounds gained in each quarter by each infant as the sole basis of comparison. A more exact and scientific method is to indicate their rate of growth in percentages of development. We may thus express with clearness and in easily understood terms their progress either in reference to each other or to the average healthy child.

The weight index, then, while not embodying any fixed law of growth may be used to express in definite percentages the variations in weight development of an infant which may occur as a result of natural growth or disease.

During the second year the gain in weight is much less constant and steady than in the first year. There is, of course, stationary weight, or loss of weight, with every illness. Also, even when there is no illness, and when the child is developing normally in every other respect, there are periods when the gain is very slight, or when the weight is stationary. Such periods are particularly seen in the summer months; many otherwise normal children, do not gain weight during an entire summer. The rate of growth is apt to be fastest in the autumn and early winter.

In the earlier years of childhood, the same conditions of weight development prevail as in the second year. In the later years of childhood the weight increase is slower, but rather more steadily constant.

The figures for the average weights and heights of normal children throughout infancy and childhood are shown in the table.

They afford a ready basis of comparison in estimating weight development, showing the relation of the individual child at all times to the average normal.

TABLE 8

Average Heights and Weights from Birth to Five Years, and of Boston School Boys and Girls, Irrespective of Nationality, from Five to Fourteen Years

BOYS				AGE	GIRLS			
HEIGHT		WEIGHT			HEIGHT		WEIGHT	
Centimetres	Inches	Kilogrammes	Pounds		Centimetres	Inches	Kilogrammes	Pounds
49.37	19.75	3.25	7.15	Birth	48.12	19.25	3.15	6.93
61.87	24.75	6.50	14.30	5 months	59.12	23.25	6.30	13.86
73.82	29.53	9.54	20.98	1 year	74.17	29.67	9.00	19.80
84.55	33.82	13.80	30.36	2 years	82.35	32.94	13.31	29.28
92.65	37.06	15.90	34.98	3 years	90.77	36.31	15.07	33.15
98.27	39.31	17.27	37.99	4 years	97.00	38.80	16.53	36.36
103.92	41.57	18.64	41.00	5 years	103.22	41.29	17.99	39.57
109.37	43.75	20.49	45.07	6 years	108.37	43.35	19.63	43.18
114.35	45.74	22.26	48.97	7 years	113.80	45.52	21.50	47.30
119.40	47.76	24.46	53.81	8 years	118.95	47.58	23.44	51.56
124.22	49.69	26.87	59.00	9 years	123.42	49.37	25.91	57.00
129.20	51.68	29.62	65.16	10 years	128.35	51.34	28.29	62.23
133.32	53.33	31.84	70.04	11 years	133.55	53.42	31.23	68.70
137.77	55.11	34.89	76.75	12 years	139.70	55.88	35.53	78.16
143.02	57.21	38.49	84.67	13 years	145.40	58.16	40.21	88.46
149.70	59.88	42.95	94.49	14 years	149.85	59.94	44.65	98.23

In remembering the course of normal weight development, useful figures are the following: An average child doubles its birth weight at five months, and trebles it at fifteen months. At five years its weight is double that seen at the end of the first year, and its weight at five years is doubled at twelve years.

DEVELOPMENT OF INTERNAL STRUCTURE

In order that the student of pediatrics may recognize certain pathologic conditions found on post-mortem examination, it is essential that he be familiar with the features of internal structure which are peculiar to early life. The development of those internal parts of the body which at birth show anatomic peculiarities will be traced.

The development of function, such as that of the digestive and urinary systems, is also of great importance to the student of pediatrics. This phase of development is, however, so closely connected with the various gastro-enteric and renal diseases, that its discussion will be postponed until these diseases are considered. Anatomic development only will be considered at present.

THE BRAIN.—The brain grows rapidly during the first seven years of life; after this age it increases very slowly in weight. The convolutions are not fully developed at birth, and are gradually perfected as the child grows older. One important anatomical condition in the brain of young subjects is that the dura mater is much more adherent to the skull than in later life. The subarachnoid space also, contains a larger amount of fluid.

THE EAR.—At birth there exists in the roof of the middle ear a distinct cleft between those portions of the tegmen tympani which are formed by the petrous and squamous bones. Through this cleft extends a small process of the dura. This cleft closes at about the beginning of the fifth month, becoming the petrosquamosal suture. It does not in early life, appear to play any rôle in permitting purulent processes to extend from the ear into the cranial cavity.

The cavity of the antrum is present at birth, but the mastoid process is very slightly developed, there being no mastoid cells. At the end of the first year, the mastoid begins to contain a few pneumatic cells. It becomes fully developed, resembling the adult mastoid, at three years.

The osseous meatus is not developed until about the fourth year. In introducing the ear speculum at a period previous to the fourth year, the ear should be drawn backward and downward, instead of backward and upward.

THE NASOPHARYNX.—The nasal cavity begins to increase in height directly after birth, and its growth is rapid until the beginning of dentition. After the completion of the first dentition, the growth is again rapid until the seventh year, during which period takes place the chief increase in breadth, and the growth of the olfactory portion. At the end of the seventh year, the cavity approaches the adult shape, although it still seems broad in proportion, and of course has not attained the adult size.

The change in the shape of the pharynx is very rapid during infancy. It attains the adult shape at about the age of three years.

There is little change in the position of the eustachian tubes up to the ninth month. After this, the openings are higher than the floor of the nasal cavity.

The pharyngeal tonsil increases rapidly after birth, and by the end of the third year has a length of eighteen millimeters.

THYMUS GLAND.—The thymus is most developed in the first two years of life, but it persists longer than was formerly taught. During its greatest development it is found in the neck as well as in the thorax, extending perhaps 2 cm. ($\frac{5}{8}$ inch) above the sternum. The thymus extends down the anterior mediastinum, lying on the pericardium in two long lobes on either side of the median line. The

extent of these lobes is very variable, and the two are not usually symmetrical. I have seen them, even in an infant, so developed that the longer nearly reached the lower end of the sternum; but it is very uncommon for it to reach the diaphragm. These prolongations become thinner as they descend. The thymus is a thick mass behind the first piece of the sternum, where it rests on the top of the heart against the great vessels concealing the innominate veins, more or less of the superior vena cava and the arch of the aorta, and extending back to the trachea. Lower down it extends on either side into the angle between the pericardium and the lungs, or rather pleurae.

The time of complete atrophy is extremely variable. I have seen it both present and absent in autopsies at every year from the third to puberty.

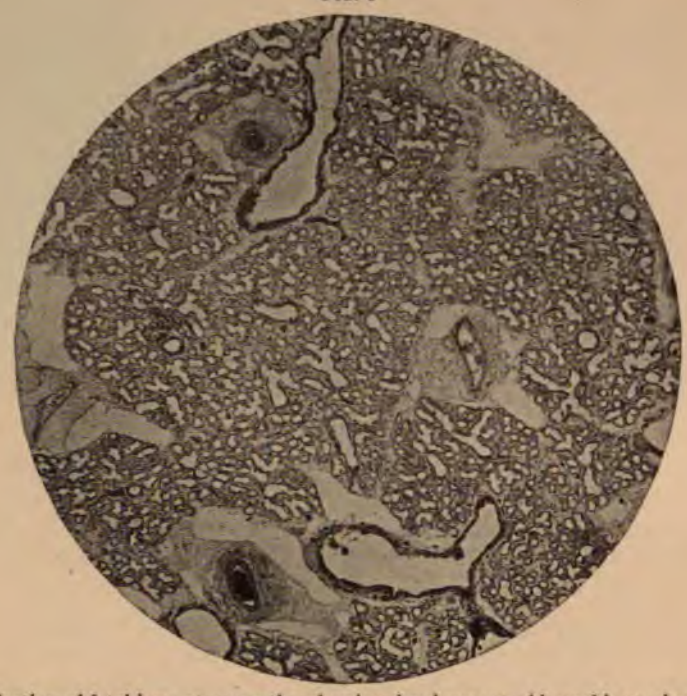
THE HEART.—At birth, the anatomy of the heart still shows the peculiarities required by the fetal circulation. Functionally, with the tying of the cord and the expansion of the lungs, the circulation of the blood immediately changes to the course seen in adults, although a small amount of blood may still pass through the foramen ovale and the ductus arteriosus. Anatomically the changes are of course not immediate, though they take place fairly rapidly after birth. The ductus venosus becomes a fibrous cord in from two to five days. While the intrauterine function of the eustachian valve ceases immediately, its remains can be found for an indefinite period. The foramen ovale usually is closed by the tenth day, although it is sometimes found open either as a whole, or in its upper portion, at any period of childhood, without any murmur or other physical sign. The ductus arteriosus normally becomes impervious in from four to ten days. The cavities of the umbilical vein, and of the upper parts of the umbilical arteries, are obliterated in from two to five days.

As soon as these changes from the fetal condition are completed, the anatomy of the heart differs in no essential particular from that of adults. Its growth is always more or less proportionate to the growth of the child.

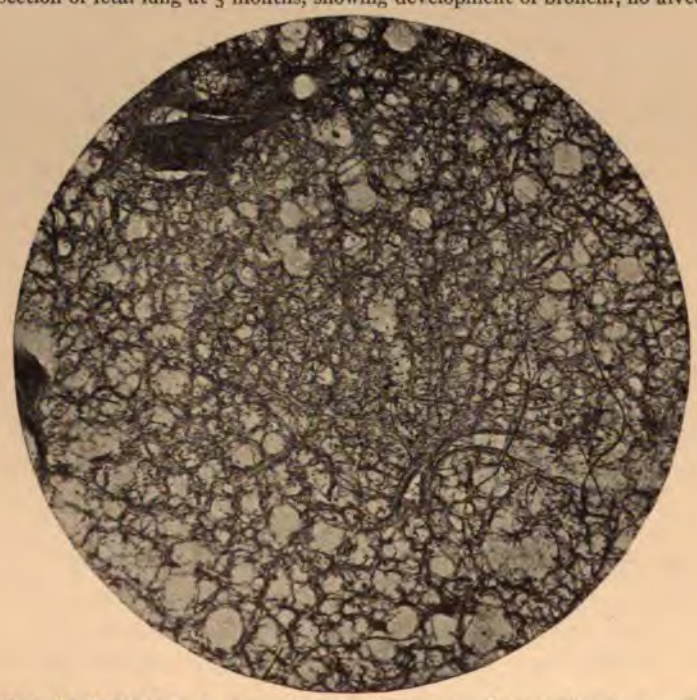
THE LUNGS.—The age at which the lungs reach their full expansion forward is very variable; it is certainly not earlier than the sixth year, and may be much later. There is, of course, a continuous increase in the relative size of the lungs to the heart, which keeps pace with the development of the thorax.

The marked anatomical peculiarities seen in the lung of the newborn infant persist almost unchanged throughout the first year. The alveolar walls remain proportionately thick, the connective tissue stroma remains loose, and relatively great in amount, the size of the alveolar air spaces remains small in proportion to that of the

FIG. 8



Section of fetal lung at 5 months, showing development of bronchi; no alveoli



Section of infant's lung at 10 months, showing increased proportionate amount of parenchyma in comparison with the fetal condition; distended alveoli

bronchioles, and their number remains relatively small. After the first year, the development of the lung begins gradually to approach the adult type. It has been generally stated that the development is complete by about the end of the fourth or fifth year. I believe this statement to be only partly true. At this age the size of the alveoli has attained the adult proportion to the bronchioles, the walls of the alveoli have become thin, and the stroma has become firm and binding. But, in my experience, based on a number of observations of children's lungs, the proportion in number of alveoli to bronchioles still remains smaller than the adult proportion, and does not show complete development until toward the end of childhood.

THE STOMACH.—Both the position and the shape of the stomach change rapidly during the first year of life. The axis of the stomach gradually becomes less oblique, and by the end of infancy reaches the transverse direction seen in older children and in adults. The fundus develops rapidly during the first year, and although it does not reach its full development until late in childhood the tubular shape of the organ soon disappears.

The development of the capacity of the stomach is important in connection with the subject of infant feeding. There are two ways of estimating the gastric capacity. One is based on actual anatomic measurements made post-mortem. The other is based on measuring the amount of fluid which a normal infant takes at a single feeding. There is often considerable discrepancy between the anatomic and physiologic measures of gastric capacity. The following table shows some anatomic measurements at different ages made by Rotch and Holt respectively, and some physiologic measurements made by Rotch and Mosenthal respectively:

TABLE 9

Gastric Capacity in Ounces

	ANATOMICAL		PHYSIOLOGICAL	
	ROTCH	HOLT	ROTCH	MOSENTHAL
Birth	.80	1.20	.98	....
1 week	1.33	....	....	....
2 weeks	1.33	1.50	....	....
4 weeks	2.50	2.00	2.35	3.50
8 weeks	3.50	3.37	3.22	4.00
12 weeks	4.00	4.50	3.96	4.80
16 weeks	4.50	5.00	4.57	4.80
5 months	5.50	5.75	5.28	5.60
6 months	....	....	5.71	....
7 months	....	6.88	6.18	....
8 months	....	....	6.95	....
9 months	....	....	7.54	....
10 months	....	8.00	7.89	....
11 months	....	....	8.07	....
1 year	....	8.90	....	....

It will be noted that the figures for anatomic gastric capacity given by Rotch and by Holt are fairly close to each other, those of Holt being in general slightly larger than those of Rotch. The difference in the figures can easily be explained by some minor difference in the technique of taking the measurements. The figures given by Mosenthal for physiologic capacity are much larger than those of Rotch. This can probably be explained on the ground that the babies on which Mosenthal's figures were based were given all the milk they would take, and could take care of, whereas Rotch's figures are based on babies who were thriving on measured amounts without showing signs of hunger. The amounts given to these babies, also, were more or less based on Rotch's anatomic measurements. I believe Mosenthal's figures, which show the physiologic capacity to be larger then the anatomic, are better. The reason for the physiologic capacity being greater than the anatomic is explained by recent work on the digestion of milk. It has been shown that coagulation begins during the feeding, and the passage of the fluid portion into the duodenum begins before the feeding is completed. The exact bearing which measurements of gastric capacity have on the amounts of milk to be given at a feeding, will be discussed in the division on feeding.

The functional development of the stomach is so closely connected with the problems of digestion and nutrition that its consideration will be postponed, to be discussed in the division on gastroenteric diseases.

THE INTESTINE.—The growth of the small intestine is so extremely irregular that the widest variations are found at every age of childhood. Its most rapid and constant increase in length occurs during the first two months of life. The variations in the rate and amount of its growth appear to bear no relation to the general development of the child.

The position of the cecum is very variable in infants and young children, ranging normally from the lumbar region to the lowest part of the iliac fossa. In the majority of instances its position is higher than in adults, and for this reason the ascending colon is very short. In childhood, especially in infants and young children, the ascending colon very frequently has a mesentery, being so completely invested with peritoneum as to be absolutely free. Both the caecum and the ascending colon are always much more freely movable in the young child than in the adult.

The length and direction of the vermiform appendix are so very variable, that they have no significance.

The descending colon usually has no mesentery, but one may be present, especially in early infancy.

During the first four months of life there is little or no change in the length of the large intestine as a whole, but the upper portions grow at the expense of the sigmoid flexure, which at birth has a length of nearly one-half that of the entire large intestine. In the majority of cases, the sigmoid flexure assumes the adult proportion at four months, but sometimes remains relatively very long. After the age of four months, the large intestine increases in length throughout childhood, until the adult length is reached.

THE KIDNEYS.—The fetal lobulation of the kidneys observed at birth persists for a variable, but considerable, time. It is usually found at autopsy in children under one year. Evidences of the uric acid infarction usually disappear in about one week, but I have observed them at autopsy in a child three weeks old.

BONE MARROW.—Nothing definite is known as to the exact time when the red bone marrow of early life changes to the yellow marrow of a later period.

IV. HYGIENE AND CARE OF AND CHILDREN

CARE OF THE NEWBORN.—At the time ting the umbilical cord, the physician should newborn child cries lustily. The eyes should be water, but the mouth should not be washed. or if the mother has had a vaginal discharge, a per cent. solution of silver nitrate or ten per cen instilled into the eyes, before cleansing them wit may then be wrapped in warm flannels, and nurse has finished the immediate care of the mo

The bath should then be given. The body be gently rubbed with olive oil, to soften the then bathed in warm water at a temperature of of the cord and the surrounding parts should be dusted with sterile talcum, or other dusting should be wrapped in a pad of sterile gauze, and be enveloped rather snugly in a flannel band. then be examined for malformations or trau birth, after which it may be dressed, and pla should be covered with blankets, and if the e or if the lips and fingers are bluish, hot water flannel should be placed in the crib, care being not come in contact with the body. The baby in its mother's bed, but should occupy its own c ened room.

The stump of the cord must be kept dry, an turbed any more than is necessary to inspect rates from the fourth to the seventh day, lea which rapidly becomes covered with epitheli should be kept dusted with sterile talcum, and with a small pad of sterile gauze, about one-quar

The full bath should not be given until afte has healed. Until this occurs, the infant may be bath at a temperature of 100° F. Care must b baths at this early period of infancy be given in

The rectal temperature of the newborn infa twice daily until the umbilicus has healed. Al charges should be saved for the physician, until tirely disappeared. The baby need not be wei of the first week.

HYGIENE AND CARE

ewborn baby is considered in the division

ıl babies should be weighed once a week
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ding. Even if no basin bath is given at this
ce should be washed.

of the bath at the different ages is shown in

TABLE 10

mperature of the Bath for Different Ages

	CENTIGRADE	FAHRENHEIT
or four weeks	37.8°	100°
	35°	95°
months	34°	93.2°
four months	32.2°	90°
duce in summer to	30°	86°
arth year, if possible, reduce to	26.6°	80°
	23.8°	75°

of the room in which the bath is given should
80° F.

ld first wash the face in clear water, keeping the
wrapped up in a warm blanket. The face is then
t towel. She should gently cleanse the nose, the
es, and the external ears. The nose is especially
ıe infant's vitality is easily affected by occluded
e should then soap, wash off, and dry the scalp.

IV. HYGIENE AND CARE OF INFANTS AND CHILDREN

CARE OF THE NEWBORN.—At the time of ligating and cutting the umbilical cord, the physician should make sure that the newborn child cries lustily. The eyes should be cleansed with sterile water, but the mouth should not be washed. In hospital practice, or if the mother has had a vaginal discharge, a few drops of a two per cent. solution of silver nitrate or ten per cent. argyrol should be instilled into the eyes, before cleansing them with water. The child may then be wrapped in warm flannels, and laid aside, until the nurse has finished the immediate care of the mother.

The bath should then be given. The body of the infant should be gently rubbed with olive oil, to soften the vernix caseosa, and then bathed in warm water at a temperature of 100° F. The stump of the cord and the surrounding parts should be carefully dried, and dusted with sterile talcum, or other dusting powder. The cord should be wrapped in a pad of sterile gauze, and the abdomen should be enveloped rather snugly in a flannel band. The infant should then be examined for malformations or trauma received during birth, after which it may be dressed, and placed in its crib. It should be covered with blankets, and if the extremities are cold, or if the lips and fingers are bluish, hot water bottles covered with flannel should be placed in the crib, care being taken that they do not come in contact with the body. The baby should not be kept in its mother's bed, but should occupy its own crib, in a quiet, darkened room.

The stump of the cord must be kept dry, and should not be disturbed any more than is necessary to inspect it. It usually separates from the fourth to the seventh day, leaving a red surface, which rapidly becomes covered with epithelium. The umbilicus should be kept dusted with sterile talcum, and should be covered with a small pad of sterile gauze, about one-quarter of an inch thick.

The full bath should not be given until after the umbilical scar has healed. Until this occurs, the infant may be given a daily sponge bath at a temperature of 100° F. Care must be taken that all the baths at this early period of infancy be given in a warm room.

The rectal temperature of the newborn infant should be taken twice daily until the umbilicus has healed. All the intestinal discharges should be saved for the physician, until meconium has entirely disappeared. The baby need not be weighed until the end of the first week.

The feeding of the newborn baby is considered in the division on infant feeding.

WEIGHING.—Normal babies should be weighed once a week during the first year of life. The weighing is best done at the time the infant receives its daily bath. Infants who are very delicate, or sick, or in whom some change in the feeding has been instituted, should be weighed daily, unless their vitality is so low that they constantly have a subnormal temperature. During the second year, it is also better to weigh most babies as often as once a week, although such frequent weighing is not so necessary as in the first year. Older children should be weighed once a month.

BATHING.—Infants and children must be bathed daily. Infants should begin to have tub baths as soon as the cicatrix of the cord has healed. Theoretically the best time for the tub bath is in the morning, but giving it at this time, in most households, does not fit in so well with the child's general routine. The baby usually has to have its first feeding upon waking in the morning, and often goes out shortly after the second feeding. Also, in many households the rooms are not warm enough to bathe the baby so early in the morning. In most households it is found more convenient to give the tub bath at night, at bed-time, just before the feeding which is given about six o'clock. The baby may have a basin bath in the morning, before its second feeding. Even if no basin bath is given at this time, the infant's face should be washed.

The temperature of the bath at the different ages is shown in the table.

TABLE 10

Temperature of the Bath for Different Ages

AGE	CENTIGRADE	FAHRENHEIT
At birth	37.8°	100°
During first three or four weeks	35°	95°
One to six months	34°	93.2°
From six to twelve months	32.2°	90°
Twelve to twenty-four months	30°	86°
Then gradually reduce in summer to	26.6°	80°
In the third or fourth year, if possible, reduce to	23.8°	75°

The temperature of the room in which the bath is given should be from 76° F. to 80° F.

The nurse should first wash the face in clear water, keeping the body and limbs wrapped up in a warm blanket. The face is then wiped with a soft towel. She should gently cleanse the nose, the corners of the eyes, and the external ears. The nose is especially important, for the infant's vitality is easily affected by occluded nares. The nurse should then soap, wash off, and dry the scalp.

Especial care should be paid to the folds of the neck, the axillae, groins, genitals, and anus. The body and limbs having been thoroughly and quickly soaped, the nurse should gently lower the infant with its face up into the clear water in the bath, being careful not to frighten it or to drop it. After allowing the infant to kick and splash for a few seconds, it is taken back into the nurse's lap and carefully dried with a warm, soft towel. When the skin is perfectly soft, clear, and in a normal condition, no powder is needed. Where there is any slight irritation, which, at times, is liable to occur when the skin has not been kept sufficiently dry, especially if there is a decided redness in the folds of the skin, as of the neck, axillae, or groins, powder may be applied.

During the second year, the tub baths at night are continued, but in addition sponge baths in the morning should become part of the general routine. At first, the temperature of the water may be little if any lower than that of the tub baths, but after the end of the second year, an effort should be made to make the morning sponge baths colder. The baths must be very brief. The child should stand in a tub partly filled with warm water, and should be sponged all over very rapidly with the cooler water, for not more than half a minute. It should then be dried by vigorous rubbing. The temperature of the water used in giving the morning douche, at first 70° F., is gradually lowered, until the temperature of ordinary tap water is reached. At all times care must be taken that children react thoroughly from these baths. If at any time, after them, children shiver, look pale, or become slightly blue about the lips, the water used must be warmer, or cold sponging must not be used.

In the latter part of childhood, the warm bath at night should be omitted, except occasionally. At this time every morning, the child, standing on a bath mat, should be thoroughly washed with warm water, and may then plunge into a tub of cooler water. The temperature of the cold plunge should be not over 70° F., and should be as much colder as the reaction of the child will allow.

CARE OF THE MOUTH AND TEETH.—The teeth of the young infant should be cleansed each morning, when the morning toilet is made. Too much vigor must not be used in the mouth. Children should be taught to use the toothbrush at the earliest possible moment.

A protest should be made against the way in which the nurse, and also others who come near the infant, put their fingers into its mouth on all occasions. It would seem as though the infant's mouth was considered by those who ought to know better as something especially made to be felt. The fingers should always be thoroughly washed before entering an infant's mouth.

A nurse should be instructed that she is never to kiss the infant on the mouth, or allow any one else to do so. The micro-organisms of disease can well be transmitted in this way. Nor should the baby's hands or fingers be kissed, as they are constantly going into its mouth.

CARE OF THE SKIN.—The skin of the young infant is very delicate, and exceedingly liable to irritation. Napkins should be removed as soon as soiled and wet. In very fat infants, or whenever there is the slightest sign of irritation of the skin, some infant powder, containing stearate of zinc, starch, or talcum, should be used, especially in the folds and about the genitals. Intertrigo and eczema are usually due to faulty care of the skin. Children who perspire freely at night, should have the position of the head on the pillow changed occasionally, as facial eczema may be produced by the child's face resting all night on a sweat-soaked pillow.

CARE OF THE GENITALS.—In girls, cleanliness is the only requirement. In boys, the prepuce must be attended to, and the best time is the early weeks of infancy. In most cases the prepuce is adherent to the glans, and sometimes it is very long, with only a pin-hole opening. This condition is a marked source of discomfort and reflex irritation, from accumulation of secretions. When the prepuce is very long, and very tightly adherent, circumcision should be performed. I am not, however, an advocate of circumcision in all cases. In many cases, where the prepuce is comparatively short, the adhesion should be broken, and the prepuce retracted daily, with cleansing, and the application of a little vaseline.

CARE OF THE HAIR.—Parents do not usually seek the advice of the physician as to whether the child's hair should be cut long or short. In cases where the parent's aesthetic feelings lead them to keep the children's hair long, the physician should point out the sanitary reasons for keeping it short. Long hair makes the children perspire about the head and neck while active, and increases their chance of taking cold while quiet. It makes them hot, uncomfortable, and restless at night. Short hair causes a better growth of hair in later life.

CLOTHING.—In clothing the young infant, there are two important requisites. The clothing must be sufficiently warm to protect the infant from exposure, and it must be so arranged as to allow the most perfect freedom of motion for the legs and arms, and for the abdominal and respiratory muscles. Young infants have a greater body surface in proportion to their weight than have older children and adults, and this greater surface means greater opportunity for loss of heat by radiation. Also, the heat regulating appa-

ratus of the infant is not sufficiently developed to maintain so early a constant temperature under varying conditions of heat and cold. Infants, then, in general, require warmer clothes than older children. The community in general is so thoroughly impressed with the necessity of clothing infants warmly, that the tendency is to go to an extreme, and clothe them too warmly. The physician very rarely has to order warmer clothes for the infant, but frequently finds them dressed too warmly, especially during the summer months, and when indoors in winter.

The ordinary clothing which the infant wears all the time should be designed to meet the conditions prevailing during the greater part of the time in the particular season of the year. It should of course be lighter in the warmer than in the colder months. It represents a minimum rather than a maximum of clothing, designed so as not to overheat the child, but to keep it sufficiently warm under the conditions usually prevailing. Sudden changes to a colder degree of heat surrounding the child, such as when the child goes out in winter, or when the temperature falls in the mornings and evenings of summer, should be met by extra wraps. On the hottest days of summer, even this minimum of clothing should be reduced, and the baby should be allowed to play and kick in nothing but its band and diaper.

The clothes must be loose, so that they do not interfere with the natural activity of the circulation, do not press or bind anywhere, and do not interfere with full muscular activity. All the clothes must be supported from the shoulder, and not from the waist. They must be designed to go on and come off easily. For this purpose it is preferable that they fasten up and down the front rather than the back.

The best designed clothes which I have seen for infants are called the "Vanta Vesta." In them all buttons and all safetypins are entirely eliminated. They are fitted with tapes, which are so placed as to fasten them in exactly the best manner, and the tapes can be easily changed to suit the size of the particular infant. Even the diapers are fastened with these tapes.

The clothing worn by the infant until the age when it begins to creep, consists of the following articles: The flannel abdominal band is often worn throughout the first year. It is not a necessity, except for the first few months; after this period it should be changed for the knitted band, which fastens to the diaper below. The best material for this band is silk and wool. The diaper is best made of birdseye linen. The shirt is a garment with sleeves and high neck, and is almost as long as the "gertrude" and dress. It should fasten in front rather than behind. It is woven, the best material

FIG 9—CLOTHING FOR AN INFANT

First step in the dressing of a baby, showing band, diaper, and stockings

FIG. 10—CLOTHING FOR AN INFANT

Second step in the dressing of a baby, showing the shirt

FIG. 11—CLOTHING FOR AN INFANT

Third step in the dressing of a baby, showing the "Gertrude" or long flannel petticoat

FIG. 12—CLOTHING FOR AN INFANT

Final step in the dressing of a baby, showing the dress

being silk and wool. The petticoat, or, as it is usually called, the "gertrude," goes on over the shirt. It has a low neck and no sleeves, is somewhat longer than the shirt, but not so long as the old-fashioned "long clothes" in which the infant's feet were tightly swathed, and preferably fastens in the front. It may be made either of fine flannel, or of woven silk and wool; flannel is less expensive, and fully as satisfactory. The outer garment is the dress, which is made of some soft, white material, and has a high neck, and sleeves. It is of the same length as the gertrude. Many mothers put on a soft lawn gertrude between the dress and the flannel gertrude. The dress is the only garment which the nurse should be allowed to put on over the infant's head. The band, shirt, and gertrude should always be put on over the feet, and if they open in front, the baby may be dressed and undressed very rapidly and easily. The young infant does not need to wear stockings, except sometimes when he goes out in cold weather. There is no objection to short, knitted socks, if the mother wants to use them. The nightdress is a garment made of flannel, or of woven silk and wool, which goes on over the shirt. It should open all up and down the front, and the back should be longer than the front, to permit of its being folded over the feet.

When the infant begins to creep, short clothes take the place of the long clothes. The undershirt now takes the place of both the shirt and band. This should have sleeves, and should be made of silk and wool, or a fine all-wool material. The flannel petticoat is made of the same material as in earlier infancy, except that its waist is cotton; it has still a low neck and no sleeves, but is short, goes on over the head, and has no fastenings either in front or behind. Over it goes the white petticoat, similar in design, but made of some soft, white material with a cotton waist. Over these garments go the short dress, with high neck, sleeves, and buttons behind. Stockings must be worn at this period. These are preferably made of wool, and fasten to the diaper. When the child begins to stand and walk, soft kid shoes should be used. Care must be taken that the shoes are of proper shape, adapted to the natural curves of the child's foot. At night the child should wear a regular nightdress, made of soft flannel, with high neck and long sleeves, and buttoned behind.

When the child is two or three years old, and often before, the diaper is discarded, and the child begins to wear drawers. At this time the petticoats no longer have the cotton waists permitting them to go over the shoulders, and a new garment is worn, called the waist. The waist has no sleeves, but is supported by shoulder straps, and should fit loosely, especially about the child's waist. Its

principal function is to support the petticoats, drawers, and stockings. It is furnished with sufficient buttons at the waist, to permit the petticoats, drawers, and garters to be fastened to it, so that by means of this waist, everything hangs from the shoulders, without any constriction anywhere.

SLEEP.—The amount of sleep required by children at different ages varies very much with the individual. The newborn infant sleeps almost continually, waking only for its feedings, and during the first three months, it sleeps twenty to twenty-two hours out of the twenty-four. It sleeps somewhat less, sixteen to eighteen hours, during the second three months. In the second half of the first year, the child should sleep at night from about six p. m. to six a. m., and should have two two-hour naps during the day, one between nine and twelve in the morning, and one between twelve and three in the afternoon. The periods devoted to exercise, amusement, and play should be between six and nine in the morning, and between three and six in the afternoon.

The child should always be allowed to sleep as much as it will. The twelve-hour sleep at night should be continued until he is five or six years old. As they grow older, some children develop the habit of waking up earlier in the morning. If this occurs, the hour of their bedtime should not be made later, as this will disturb their routine, and will usually not correct the habit. The habit may often be corrected by making sure that the nursery is kept darkened in the morning.

The two day naps should be continued as long as possible, but are usually shortened by the child itself. At one year, the morning nap is usually shortened to one hour, and is given up in the last half of the second year. The afternoon nap should be continued, although children often will not sleep after they are five or six years old. They should, however, still be made to lie down and rest, until they are seven or eight.

It is very important that the child be trained from birth in proper habits of sleep. It should never be rocked to sleep, and preferably should not fall asleep in its mother's or nurse's arms, but should be accustomed to be put in its crib alone, in a quiet, darkened room, and to go to sleep of its own accord. All other artificial devices, such as allowing it to go to sleep on the breast, or with a nipple in its mouth should be avoided. Failure to go to sleep properly means noise, light, hunger, indigestion, or wet napkins.

FRESH AIR AND GOING OUTDOORS.—Nothing is of greater importance in insuring the normal development of the growing child, and in the prevention of disease, than abundance of pure, fresh air. This prime necessity in the hygiene of early life is more neglected

than any other, usually through fear of making the children ill by exposure. When children are delicate there is a special tendency to deprive them of the fresh air which they especially need.

During the first week or two of life, a baby is very easily disturbed by changes in the temperature, and by exposure to cold air. Its nutrition and heat regulating apparatus are not established, and it requires coddling and warmth. This does not mean that the young infant is not to be kept in well ventilated rooms, but only that it is not to go out doors, nor be exposed to any indoor airing which lowers the temperature of his surroundings below about 70° F.

After the first one, two, or three weeks of life, when the regular routine of early life has become established, we are confronted by the question of providing the infant with additional fresh air, by means of going outdoors, and by means of open windows. The transition from the protected routine of the early weeks, to that of later infancy must never be abrupt, but must be brought about gradually. The proper management of the fresh air problem depends upon the time of year.

With infants who are born in the summer months, there is no reason why they should not begin to go outdoors after the first week or two. The infant should then spend as much time as possible in the open air, sleeping outdoors in his carriage in the day time. When indoors, the windows of his room should always be open. In summer, all that is necessary is to guard against the very sudden drops in the temperature which are sometimes seen at our summer resorts. At such times, the infant may continue to go out, unless the temperature falls below 60° F., but extra wraps must be provided. Also, when indoors, he may still sleep with open windows, but if it is so cold that the temperature in his room would fall below 60° F., the windows must not be opened so wide. Only at those hours when the infant is bathed, or is partially undressed and allowed to kick and play, should the windows be closed.

Infants who are born in the spring or autumn should not begin to go outdoors until they are at least one month old, and then should not go out when the outdoor temperature is below 60° F. They should at first not be kept out more than ten or fifteen minutes, and the duration of the outing should be lengthened gradually. With infants who begin to go out in the autumn, it is possible to continue to take them out even as the season advances, if care is taken that they be kept in on the colder days, and that they be exposed very gradually to a lower outdoor temperature, so that often they may continue to go out on favorable days all winter.

In spring and autumn infants must be gradually accustomed to open windows in their sleeping rooms after they are a month old,

precautions being taken against sudden falls in the temperature of their sleeping rooms.

When infants are born in the late autumn or winter, it is more difficult to provide plenty of fresh air without too sudden a transition. When they are a month old, they may begin to have indoor airings. They are dressed for an outing, and then the windows of the room are opened wide for a few minutes. The minimum temperature of the room must be lowered very gradually, while the duration of the airing is very gradually increased. This substitute for an outing may be continued through the winter months, and the child will be ready for an actual outdoor airing with the first fine days of spring. After these indoor airings are instituted, the child may be gradually accustomed to sleeping in a room with open windows, the window at first being opened only the merest crack, and gradually opened more on the warmer nights.

When an infant is four or five months old, he should go out doors even in winter. In our middle and northern states, the weather conditions are such that it is not advisable for infants to be taken out every day. When it is very windy and dusty, when there is a snow storm, when the snow is melting, and when the temperature is below 20° F., it is better for the infant to receive his airing indoors. On fine, calm, sunny days, even if fairly cold, he should go out for two hours in the morning, and for two hours in the afternoon, but in winter, should not be out later than three o'clock. The best times for these outings are from ten to twelve in the morning, and from one to three in the afternoon. There is absolutely no objection to an infant sleeping outdoors in his carriage in winter. The nurse should protect his eyes from the direct rays of the sun, and his face from dust and strong wind.

Older children should be outdoors as much as possible, being kept in only when the outdoor weather conditions are very bad.

As long as an infant spends most of his time in bed while indoors, it is easy to keep the windows of his nursery open, and still protect him from exposure to draughts. When he has grown older, and has reached the age when he begins to creep, stand, walk, and play, this is more difficult in the colder months. The child will play on the floor, and if the window is open, no matter how carefully, and even if a window board is used, there are sure to be cold draughts on the floor. In most cases it is impossible to keep the window of the nursery open, even with a window board, while the child is playing. I am a great believer in an open fire, which is a great aid to ventilation, and from the draught of which the child may be protected by not allowing him to play in a direct line between the window and the fireplace. In any case, the nursery should always be thoroughly

aired whenever the child is out of it, and before he goes to bed at night. In the day time, if possible, he should be changed from one well aired room to another.

The practice has frequently been adopted in cases where infants were born in summer, and have become accustomed to sleeping outdoors even at night, to continue having them sleep in an outdoor sleeping-porch even in winter. I have seen this practice adopted without harm, and apparently with benefit. Great care must be taken with the coverings, and constant vigilance exercised against sudden changes in the weather. It is so difficult in our northern climate to care for children sleeping outdoors at night in winter, that I do not recommend it until children are at least three years old. The nursery with open window provides enough fresh air for the average baby. After the age of three, I am a great believer in the outdoor sleeping-porch, whenever it can be provided.

EXERCISE.—In young infants exercise is obtained by kicking its legs, and waving its arms, and does not require any very special attention. It is, however, a bad plan to allow infants to lie in their cribs all the time. This fault is more often seen in hospitals than in private practice, and sometimes leads to marked impairment of the health of the baby. The babies must be picked up and carried about occasionally and their position should be frequently changed. In private practice it is a good plan, once or twice a day, to take the baby into a warm room, remove its more cumbersome outer clothing, and let it kick about for a while on a large bed.

When the babies grow older, and reach the age when they begin to creep, they normally never fail to take sufficient exercise, and sometimes take too much unless they are restrained. By placing them on the floor on a large mattress, or on a large bed, in a warm room, opportunity may be given then to kick and roll about as much as they desire.

After babies have learned to walk, care must be taken to prevent them over-exerting to the point of becoming tired. Exercise in the open air is best, but they must not be allowed to walk too far. It is better to wheel them to some desirable spot, and then let them walk or run about for a time which, short at first, is gradually increased as they grow older, until finally, between three and five years of age, they walk during the entire outing. For indoor exercise in winter, an exercise pen, which keeps them off the floor, is often useful.

In older children, every form of outdoor exercise should be encouraged. In normal children, none of the usual games and sports are harmful, and opportunity should be given for the children to learn and practice as many as possible.

HYGIENE OF THE NERVOUS SYSTEM.—It must be remembered that the nervous system is relatively undeveloped throughout the whole of infancy and childhood. The first two years of life constitute the period in which the brain grows more rapidly than during all the rest of the child's life. This rapidly growing brain requires above all things rest and peace, and is especially sensitive to abuse. At all times the nervous system of early life is unstable, and extremely sensitive to stimulation and prone to excessive reaction. It is sensitive both to reflex stimulation, and to mental stimulation.

To insure normal development of the nervous system, care must be taken at every age that there is present no source of reflex irritation. The physician should carefully examine all children who come under his care for evidences of such conditions as phimosis, adherent prepuce or clitoris, adenoid disease, eye-strain, chronic indigestion, and others. The removal of any such possible cause of reflex injury to the nervous system is very important.

Mental over-stimulation is still more common as a source of injury to the growing child. It may easily be begun and carried to an injurious extent in the earlier months of infancy, without the mother being aware that any harm is being done. For the young baby, the chief form of over-stimulation which its nervous system receives, is too much or too active entertainment. In most families the first baby is the greatest sufferer, from parents at least, but often subsequent babies are allowed to become the plaything of the older children. Babies should not be played with much. If possible they should not be allowed to pass their time in rooms where the activities of family life are a constant source of noise, and where they are constantly attracting superfluous attention. A proud mother likes to show off her baby to visitors and friends, who like to hold it, and hand it about, and poke it to make it smile, and talk to it. As it begins to react more and more to its surroundings, by smiling, wriggling, and kicking, these actions are considered "cunning," and more and more efforts are made to bring them forth. All this is very bad for the baby. It is possible to produce in a very young baby an actual condition of nervous exhaustion, which first manifests itself by failure to gain weight, then by loss of appetite, than by restlessness and poor sleep, the baby jumping at the slightest sound.

Even with older infants and children, the nervous system can very easily be over-stimulated to the extent of producing symptoms of harm. Children are sometimes amused by exciting and unusual sights and sounds. Violent romping games, especially at night, and suggesting new and exciting forms of play, are also bad. It is better to allow children to invent their own play, than to play with them too

much. Moreover, especially when the children are bright and learn easily, their fond parents often delight in teaching them little sayings, or rhymes, at too early an age. This is also too stimulating to the nervous system of the young child, and the brighter the child, the greater the danger. The telling of exciting stories, or of stories about unfamiliar things which vividly stimulate the imagination, is also bad. Too much adult companionship is often not good for children, especially if new things are taught and suggested. The young child learns best from the companionship of other children of the same age. The type of the individual child should be considered. There is more danger from over-stimulation from teaching, novelty, and excitement in the bright child, than in the dull and phlegmatic child.

When they get older, the question of kindergarten and school arises. When a child has reached the age of six years, I believe that kindergarten is a good thing, provided that it is properly managed, with a maximum of play and exercise for the hands and body, and a minimum of teaching and exercise for the mind and eyes. The chief value of kindergarten, however, is the companionship of other children of the same age.

School is the next step after kindergarten. I believe that all children who have attained the proper mental and physical development, should go to school with other children, and should not have private lessons at home. Private teaching by a governess does not satisfy the very real need for companionship. At just what age a child should begin to go to school, and for how long, are questions which vary with the individual. At no time during childhood should school interfere with the large amount of outdoor exercise which is so essential to proper development in childhood. The hours of the public schools in many parts of this country, with two daily sessions, I believe to be too long.

The physical development of the child is very important in connection with the question of school. It has been shown that a child's capacity for mental work without injury to health, bears a direct relation to his physical development. The difficulty lies in measuring the physical development; height and weight alone cannot form the criteria on which the estimate is based. Everything, all the evidence obtained by careful physical examination, should be considered. Rotch suggested, as the best measure of development, the successive times of ossification of the carpal bones, and has designed a scheme which he called the "anatomic index." This has proved a very satisfactory basis in places where large numbers of children have to be grouped and classified according to their stage of development.

Some of the dangers and disadvantages of many of our school

systems are the following:—In the first place, there is the danger of too long hours. A second unfavorable feature often found in school life, is the fostering of a spirit of competition, by means of marks and prizes. There is another danger which pretty and precocious children in particular encounter, that of becoming "teachers' pets." These children are encouraged to seek promotion, and are shown off, and speak their little pieces whenever visitors are in the school. Many a nervous system has been injured in childhood by the over-ambitious teacher.

Precocity in children is always a sign that mental development should be guarded and restrained, rather than fostered. The life of the young child should always be more like the life of the young animal than like that of the adult. Children are little animals, not little men and women.

TRAINING AND DISCIPLINE, HABITS.—Children may frequently be trained to control the rectum and bladder at a remarkably early age. Such training should always be begun before the end of the first year of life. The mother should observe at what hour the baby usually has a movement of the bowels, which in most cases follows very shortly after a feeding, and the mother, immediately after this particular feeding, should put the baby on a small chamber. Many children learn very quickly to indicate when they want to have a movement. The training of the bladder, while rather less rapid, can be carried out in the same way. Children vary very much as to how quickly they learn to give signs when they want the chamber, but many intelligent children learn so quickly, that in the second year napkins can be dispensed with during the day.

While children are very young, no other habits should be taught by training, except the control of the rectum and bladder. During this early period of life, efforts at training should be directed at preventing the development of bad habits.

The earliest of these habits to appear, and one of the commonest, is crying. The crying habit develops in infants who are picked up and held and petted, or talked to and amused, whenever they cry. The cause of the first crying may have been something really wrong, but if crying is treated by picking up and amusing the baby, the habit will persist after the cause is no longer present. It must be remembered that it is a good thing for the young infant to cry a little every day, crying having a beneficial effect in providing exercise, favoring peristalsis, and causing deep inspiration and good expansion of the lungs. Too much crying in a well and unspoiled baby, always indicates that something is wrong, or at least, that some cause exists which can be removed. An infant cries from hunger, from indigestion, from fright, from soiled napkins, from

inflamed buttocks, from tight clothes. In all cases the crying should not be treated by picking the baby up and diverting him, but a painstaking search for the cause should be instituted, and we should not be satisfied till it has been found and removed. The baby who cries violently, but stops abruptly as soon as picked up, has become an habitual cryer, and has advanced the first step on the road which leads to the "spoiled child." Such cases must be treated by a short period of rigid discipline. If for a few times he is left to "cry it out," he will be cured. Mothers should be assured that this treatment will not injure the baby's nervous system as much as allowing the habit to continue, and that it will not cause either convulsions or rupture.

The crying habit is almost identical with another bad habit, that of being held constantly in the arms. A baby should be handled as little as possible, just enough to give it exercise. It should never be allowed to fall asleep in its mother's or nurse's arms. The habit of being held soon develops, and leads to the crying habit Its treatment is the same, when developed, but it may always be prevented.

Another habit, more inexcusable on the part of the mother or nurse, is that of sucking on a rubber nipple or "pacifier." Besides deforming the lips and jaws, the pacifier is always unsanitary. Thumb and finger sucking are bad habits of a similar character, and the habit of pulling the ears is sometimes seen. These can be cured by bandaging the hands, and in the case of thumb and finger sucking, putting a solution of quinine on the bandage. All sorts of curious rare habits are sometimes seen in children otherwise normal. One child I have seen recently has a habit of deliberately banging one part of his cranium against the bars of his crib.

The training of children in habits of good conduct and behavior, is as important as it is difficult. While they are very young, and unable to understand at all the meaning of right and wrong, it is absolutely useless to use either admonition or scolding. Fortunately, at this age, the properly cared for child will not be able to do very many things which are injurious. If he does begin to do things which are a real menace, such as pulling down table cloths, or putting beans and shoe-buttons into his nose, he must be corrected physically. At this age the baby is precisely like a young animal. Young animals learn the art of selfpreservation through the pain which attends their dangerous acts, and kittens or puppies do not walk into the fire. For young children who are beginning dangerous habits, there is nothing like a good spanking. The mother often fears that the child will dread her instead of loving her, but if the spanking always immediately follows the wrong-doing, and if the punishment is ***never remitted***, but always carried out, this will never occur.

For older children, much depends on a proper method of discipline. When children are old enough to understand commands, they must be trained to be obedient, and discipline must be carried out in the way which will cause the least stimulation to the sensitive nervous system of the young child. All punishment, at this age, should if possible be made to take the form of deprivation of a pleasure. The daily life of the young child has by this time become so varied, that some parts of the daily routine are looked forward to and enjoyed more than others. These times of enjoyment should be taken away when the child is naughty. But the great secret of carrying out this discipline properly, is to make each deprivation appear from the start as the natural and inevitable consequence of the naughty act. Each wrong-doing should be surely followed by its particular deprivation. It is essential that these punishments be *never remitted* under any circumstances. The mother in her affection, and desire to make her child happy, is strongly tempted to let the child off, if he will "be good next time." This is very bad. The penalty should appear to the child as coming inevitably, through the laws of nature, the mother being unable to prevent it. Once the penalty is remitted, it becomes for the child the arbitrary act of the mother, dependent upon her will, and the child will exercise every form of ingenuity to be as naughty as possible, and yet escape punishment.

Parents should of course never show anger, irritation, or annoyance in punishing children. They should never under any circumstances bribe children to be good with rewards or special pleasures; this is of all means the surest to produce the spoiled child. They should never appeal to the child's emotions, by showing grief, or by using the "this hurts me more than you" argument. Many parents believe they can discipline their children by appealing to their reason, and by affection alone. This can not be done; the child's intellect is not sufficiently developed to grasp the reasoning, but he assents, and promises, and learns to bribe his mother to overlook naughtiness by showing her excessive affection at times.

The neglect of the measures of training and discipline outlined above, leads to the development of the "spoiled child," which is a very distinct type. He is usually bright, precocious, sensitive, with a highly developed mentality; he is very affectionate and demonstrative toward his parents. On the other hand he is very naughty, given to tantrums, and violent fits of anger. He refuses to eat what is given him or to do what he is told, and when corrected, tries to frighten his mother by the display of all sorts of terrifying symptoms. His parents usually believe that his temperament and nervous system are so exceptional, that he cannot be properly disciplined without damage to his sensitive organization. As a matter of fact

he has almost unconsciously become an adept in managing his parents, and getting his own way.

Moderate cases of this description can be corrected, if the parents will adopt a proper system of discipline with sufficient firmness. The physician must aid them by assuring them that the symptoms which the child shows under discipline will not ruin his nervous system. Severe cases can only be cured by taking the child temporarily entirely away from his parents, and putting him in charge of a trained nurse.

To teach children good manners, nothing more is required than that the parents should use toward the children, and in the family, all the conventional politeness which they would use if strangers were present.

THE NURSERY.—This room, in which the child passes so much of its time, should always be adapted to fulfil its hygienic needs. It should be a large room, situated high up, on the second, or third floor. It should have a sunny exposure and large windows high enough from the floor to prevent the younger children continually pressing their faces against the glass to look out, and thus catching cold from the little currents of air which penetrate most window casings. Painted walls are better than papered, and a hard wood floor not highly polished, with a large rug is better than a carpet. The child should have his own bed, made of iron, and painted, with

FIG. 13

Infant's bed, Infants' Hospital

sides high enough to prevent him from falling out easily. The pillow and mattress should be of felt, folded so as to be soft and comfortable, and pillow and mattress cases should be used. The mattress should be protected by a rubber sheet. The nurse's bed should not be too close. The child should have its own closet, and its own bureau-drawers; the nurse's belongings should be kept in a separate room. The furniture should be simple, not complicated or cumbersome; stuffed furniture should be avoided. The windows should have both light and dark shades, and only simple muslin curtains. Toys made of woollen material or feathers, or which have colors which can be soaked off by saliva, should not be allowed.

Steam heat is not desirable in the nursery. The best means of heat is an open wood fire, and the next best a coal grate. The open fireplace is a great help in securing good ventilation. The window board is useful in securing fresh air, but if the room is always thoroughly aired when the child is out, the window need not be open when the child is playing, if an open fire is burning. The temperature of the nursery should not be above 70° F. when the child is playing, and should be lower when the child is sleeping.

If possible, a bathroom should immediately adjoin the nursery. This can also be used as a room for changing the diapers, and can be kept warm enough at night for this purpose, when the nursery is cold. The diapers should, if possible, never be changed, washed, or dried, in the nursery.

NURSERY-MAIDS.—The first essential of the attendant who cares for the young child is health. She should be free from any suspicion of tuberculosis or syphilis, or from any catarrhal affections of the mucous membranes. The idea that the child should be taken care of by an old, experienced nurse is a vicious one. The experience of nurses, as a rule, is that of ignorance rather than of intelligence. Every mother, as she is presumably more intelligent than the nurse whom she employs, and is surely more interested in the welfare of her child, should personally supervise and unhesitatingly investigate all that the nurse does to the child. The nurse's ideas as to what is needed for the child's hygienic surroundings, food, and clothing can well be dispensed with. The mother, learning from the physician what is best for her child, should give her directions to the nurse and see that these directions are strictly carried out. A nurse between the ages of twenty and thirty-five is preferable to one who is younger or older. She should be neat, healthy, strong, cheerful, gentle, and patient. She should be willing to refer small details of the nursery routine to the mother, as well as those which appear of greater importance. The chief attributes of a good child's nurse, in my opinion, are a desire to obey implicitly the orders which she

receives from her mistress, and a temperament in harmony with the sensitive nervous organization of her charge. In certain cities, such as New York, Philadelphia, Buffalo, and Boston, schools have been established for nursery-maids, where the nurses are trained to be servants as well as nurses.

PREVENTION OF EXPOSURE TO CONTAGION.—Infants and children are usually guarded from exposure to such well-known contagious diseases as scarlet fever and diphtheria, but no precautions are taken against exposure to such conditions as the common cold, influenza and bronchitis, all of which are contagious. Members of a household who have any of these respiratory affections should be quarantined as far as the children are concerned. Some parents believe that children should not be guarded from such contagious diseases as measles and whooping cough, on the ground that they must have them at some time. This is not necessarily true, and children should always be guarded against exposure to these diseases.

Whenever children assemble in crowds, especially indoors, the chances of contagion are much increased. The moving picture shows have become very fruitful sources of contagion, and young children should not be taken to them.

SUMMER RESORTS.—The physician is frequently consulted as to where the child is to be taken for the summer. In my experience, chiefly gained among children who pass the winter near the sea, our inland mountain summer resorts are far preferable to the sea-shore. Children may advantageously pass part of the summer in the mountains or inland, and part by the sea. A whole summer at the sea-shore should be avoided. It is probable that with children who live inland in winter, the sea-shore resorts in summer would not be open to objection.

THE DAILY ROUTINE OF THE NORMAL CHILD.—I have devoted considerable space to the hygiene and care of the normal child. It has come to be recognized that Preventive Medicine is one of the most important, if not the most important, branch of the art of medicine. It is especially important in early life, where a very large proportion of the pathological conditions which develop may be traced to some violation of, or lack of attention to, the small details of hygiene and care required by the undeveloped condition of the young human being. The modern mother understands the importance of all the little details of care in regulating the environment of the child, and it is on these points mostly that she consults the specialist in pediatrics. She no longer looks to the physician as a writer of prescriptions when the children are ill, but as the expert adviser to whom she can go when she wants to know how to bring up the child in such a way that illness is prevented. The

physician must be familiar with every detail of the child's daily life, and much of his work is the careful regulation of every detail of the child's environment.

This arranging of the daily routine is sometimes very difficult. It seems hard to make everything fit in right in the course of the day; meals conflict with outdoor airings, and naps with exercise. Many things have to be taken into consideration, such as the nurse's dinner, and other ways of the household difficult to change. As a guide in arranging the daily routine to meet the needs at various ages for food, sleep, and exercise, the following time table is submitted. It is intended to serve, not as an arbitrary rule, but simply as a model, to be modified according to circumstances.

TABLE 11

Time Table for the Daily Routine in Infancy and Early Childhood

FIRST 6 WEEKS	
6:00 A.M.	Feeding
7:30 A.M.	Morning Toilet
8:00 A.M.	Feeding
10:00 A.M.	Feeding
10:30 A.M. to 12:00 M.	Outdoors
12:00 M.	Feeding
2:00 P.M.	Feeding
2:30 to 4:00 P.M.	Outdoors
4:00 P.M.	Feeding
5:30 P.M.	Bath
6:00 P.M.	Feeding
10:00 P.M.	Feeding
2:00 A.M.	Feeding

6 WEEKS TO 5 MONTHS	
6:00 A.M.	Feeding
7:30 A.M.	Morning Toilet
8:30 A.M.	Feeding
10:00 to 11:00 A.M.	Outdoors
11:00 A.M.	Feeding
11:30 A.M. to 12:30 P.M.	Outdoors
1:30 P.M.	Feeding
2:00 to 4:00 P.M.	Outdoors
4:00 P.M.	Feeding
4:00 to 5:30 P.M.	Play
6:00 P.M.	Bath
6:30 P.M.	Feeding
9:00 P.M.	Feeding
2:00 A.M.	Feeding

5 TO 12 MONTHS	
6:00 A.M.	Feeding
7:30 A.M.	Morning Toilet
9:00 A.M.	Feeding
10:00 A.M. to 12:00 M.	Outdoors—Nap
12:00 M.	Feeding
1:00 to 3:00 P.M.	Outdoors—Nap
3:00 P.M.	Feeding
3:30 to 5:30 P.M.	Play
5:30 P.M.	Bath
6:00 P.M.	Feeding and Bed

12 TO 18 MONTHS	
7:00 A.M.	Feeding
8:00 A.M.	Sponge Bath
9:00 A.M.	Orange Juice
10:00 to 11:00 A.M.	Outdoors
11:00 A.M.	Feeding
11:30 A.M. to 12:30 P.M.	Outdoors
12:30 to 1:30 P.M.	Nap
1:30 to 3:00 P.M.	Outdoors
3:00 P.M.	Feeding
3:30 to 5:30 P.M.	Nap
5:30 P.M.	Bath
6:00 P.M.	Feeding and Bed

18 TO 24 MONTHS	
7:00 A.M.	Feeding
8:00 A.M.	Sponge Bath
9:00 A.M.	Orange Juice
10:00 to 11:00 A.M.	Outdoors
11:00 A.M.	Feeding
11:30 A.M. to 12:30 P.M.	Outdoors
12:30 to 2:30 P.M.	Nap
2:30 P.M.	Feeding
3:00 to 4:00 P.M.	Outdoors
5:30 P.M.	Bath
6:00 P.M.	Feeding and Bed

2 TO 6 YEARS	
7:00 A.M.	Sponge Bath
7:30 A.M.	Breakfast
10:00 to 12:00 A.M.	Outdoors
12:00 M.	Dinner
12:30 to 2:30 P.M.	Nap
2:30 to 4:00 P.M.	Outdoors
5:30 P.M.	Bath
6:00 P.M.	Supper and Bed

DIVISION II

DISEASE IN EARLY LIFE

I. ETIOLOGY AND CLASSIFICATION

The classification of diseased conditions has always presented almost insuperable difficulties for the medical writer. In earliest times diseases were classified upon the basis of their symptoms, or of unscientific, or superstitious theories as to their cause and nature. Later the spread of knowledge concerning pathological anatomy suggested a purely anatomical basis for the classification of disease. Such a basis, however, has proved unsatisfactory for many reasons, chief among which being the fact that many conditions recognizable as diseases present no distinctive anatomical features, and the fact that the lesions of disease bear no definite relation to the cause, symptoms or method of cure. If there were such a relation, classification would be a simple matter. In its absence, attention has been turned more and more toward etiologic factors as the most important conditions differentiating diseased processes from one another, especially since progress in bacteriology has set apart a large class of diseases as having a demonstrable specific cause. Treatment, also, is becoming more and more directed at the causes, rather than at the lesions of disease, and this suggests how advantageous would be an etiologic basis of classification, if it were possible.

Unfortunately, it is as yet impossible to classify diseased conditions solely upon the basis of their etiology, for two principal reasons. In the first place, there is no subject in the scientific aspect of medicine more obscure than that of etiology. The etiology of many recognizable diseased conditions remains wholly unknown. In the second place, a great many diseased conditions which can be recognized as fairly distinct disturbances, are produced by the coincident action of a number of different etiological factors. Moreover, the same etiologic factor may, at different times, produce wholly different lesions, or disturbances of bodily function, the probable explanation of this phenomenon being that there are other coexisting causes which cannot be recognized.

Nevertheless, etiology must not be discarded in any attempt to classify disease, but should be used as far as is possible, the deficiency in our knowledge being eked out by what we know of the essential manner in which various diseased processes are produced. We

w, for instance, that in some cases the result of the action of ause or group of causes is a definite anatomic lesion; in other :s the result is a disturbance of the function of a particular organ; ;till other cases a disturbance of the entire nutrition and meta- sm, with or without the formation of toxic substances in the)d.

y taking account both of causes, and the various kinds of dis-)ances produced in the body, a basis for the classification of ase may be constructed which will be sufficiently useful to meet requirements of the present stage of our knowledge.

THE CAUSES OF DISEASE

he following are the chief etiological factors in disease:

1. Inheritance.
2. The normal lack of development of early life.
3. Unknown internal causes.
4. Mechanical injury from without.
5. External factors in hygiene and environment.
6. Infection.

f these causes, the first three represent conditions existing within body of the child. The last three represent conditions of en- nment, acting upon the child's body from without.

NHERITANCE is an important, though rather obscure factor. nilis is the only disease often directly transmitted from parent nild, though there are rare instances of the transmission of other :tions. Inheritance plays its chief part in the transmission of : are commonly called constitutional weaknesses or tendencies. s is probable, there is an anatomical basis for these tendencies, not recognizable. Lack of resistance to other causes of disease we can see, the children being liable to certain infections, or rtain disturbances of function.

HE NORMAL LACK OF DEVELOPMENT OF EARLY LIFE)een thoroughly described in the first division of this book. In alone, it can not properly be called a cause of disease. It is, ver, a very important, if not the most important, etiological r in connection with other causes acting from without. It ins why children are less resistant to certain conditions of their onment.

NKNOWN INTERNAL CAUSES.—There are certain disturb- which occur only at times, and which cannot be traced to itance, or to any external cause. The cause may be external, f so, it has not been discovered. Unknown internal causes are led only as a possible explanation of these conditions.

MECHANICAL INJURY FROM WITHOUT needs no explanation. The effects of blows, of falls, of birth injuries, of foreign bodies in internal structures, of heat, of cold, of irritants, are well known.

THE EXTERNAL CONDITIONS IN THE HYGIENE AND ENVIRONMENT OF THE CHILD which can produce disease are, of all etiologic factors, the most numerous and important. Among them may be numbered improper feeding, unhygienic surroundings, neglect, and indeed any violation of the rules for the proper care of the child.

INFECTION means the invasion of the body from without by a living pathogenic parasite. The mode of action of these microorganisms and the mechanism of defense against them, will be described in other portions of this book.

HOW THE VARIOUS CAUSES ACT IN PRODUCING DISEASE

There are three principal ways in which the various known and unknown causes of disease act in the body.

1. By producing faulty anatomical development in intrauterine life.
2. By producing disturbance of function without primary anatomic lesions.
3. By producing primary anatomic lesions.

CONGENITAL FAULTS OF DEVELOPMENT.—In the first group the causes are entirely unknown. They may be inherited tendencies, or internal unknown causes, or external conditions acting on the foetus through the mother. This subject is still shrouded in mystery. Our knowledge of embryology tells us only just what phase of development goes wrong in embryonic life.

DISTURBANCE OF FUNCTION WITHOUT EVIDENT PRIMARY ANATOMIC LESION.—This is very common. Such disturbance is usually due to the coincident operation of several causes. Normal lack of development plus inherited weakness, for example, unite with faulty hygiene in producing a disturbance of function. All sorts of combinations and successions of events may exist. Thus the faulty hygienic surroundings of the mother may cause congenital lack of resistance on the part of the child, which permits disturbance of function under conditions which are normal and not injurious to normal babies. In most cases of disturbed function, multiple causes, involving both internal and external fac-

tors must be sought. Lack of balance between the child's functional development and the conditions of his environment is the usual finding.

The functions disturbed are various. A single organ, or several organs may be involved. There may be a disturbance of the entire function of nutrition and metabolism. The results of the disturbance are very manifold, showing a great variety of manifestations. For example, disturbance of nutrition and metabolism may give rise simply to retardation of growth and development, or to distinct diseases such as rachitis and scorbutus, or to toxemias.

TOXEMIAS.—One manifestation of disturbed function is so very striking, and has attracted so much attention recently, that it deserves special mention, if not a special place in the general classification of disease. I refer to toxemia. There exist a number of clinical manifestations, some of which can be proved to be due to the formation of toxic substances and their circulation in the blood, and others which can be best explained on this basis even without proof. These toxemias would hold a place in the classification of disease, were it not for the fact that we believe them to be manifestations of disturbed function, either of an organ, or more commonly, of the whole metabolism. These toxic substances may in turn cause secondary disturbances of function, which brings them into the class of etiological factors. They may even produce lesions, such as are seen in the kidneys in some forms of nephritis, or in the bones in rhachitis and scorbutus. Their mode of action is often not very different from conditions seen in infections, where there is also a toxemia, the only difference being that the toxins are of bacterial and not of metabolic origin.

In this group of functional disturbances are included all conditions in which disturbance of function, and not a lesion, is the *primary* result of the various etiologic factors. Lesions, however, may often be seen in these conditions. For example, improper feeding may cause disturbance of the function of digestion, which in turn causes irritation of the intestinal mucosa, which in its turn eventually may give rise to an intestinal lesion.

PRIMARY ANATOMIC LESIONS.—This is a very important manner in which the etiological factors produce disease. It requires little discussion, as this origin of disease is thoroughly understood. The two chief etiologic factors concerned here are mechanical injury from without, and infection. The former produces the various forms of lesion grouped under the general term of *traumatic*. The second produces a great variety of lesions peculiar to the particular form of infection involved. There is a class of lesions of unknown cause, represented by the new growths.

THE CLASSIFICATION OF DISEASE

Using as a basis both what we know of etiology, and what we know of the mode of action of various causes, the following classification of diseases is suggested, and will be followed in this book:

1. Malformations.
2. Mechanical injuries—
   a. From external causes—trauma.
   b. From unknown internal causes.
3. New growths.
4. Functional disturbances from multiple causes—
   a. Non-toxic.
   b. Toxemias.
5. Infections.

THE MALFORMATIONS represent the faulty intrauterine development described above, of which the cause is unknown. It includes all the congenital malformations.

THE MECHANICAL INJURIES from trauma need no explanation. Those from unknown internal causes are represented by such conditions as hernia and intussusception.

THE NEW GROWTHS represent very distinct and peculiar lesions, of which the cause is still unknown. Whether the origin of these lesions is developmental, or whether it is to be found in some as yet undiscovered agency acting from without, is for the future to determine. For the present these lesions can only occupy a class by themselves.

THE FUNCTIONAL DISTURBANCES trace their position as a separate class of diseases to the essential nature of the disease process, rather than to the nature of the causes, which are multiple. The essential feature is disturbance of function without primary anatomic lesions. Those disturbances in which the manifestations are chiefly chemical, caused by the formation of toxic substances and their circulation in the blood, are grouped under the subdivision of toxemias. Examples of non-toxic functional disturbances are indigestion from overfeeding, and enuresis; of toxic functional disturbances are spasmophilia, and some forms of nephritis.

THE INFECTIONS form the most distinct group etiologically.

II. PATHOLOGICAL ANATOMY

The peculiarities of the pathology of early life lie in the relative frequency of the different processes rather than in any marked modification of their character. The tissue changes which form the lesions of disease are the same at all ages. In childhood, however, the relative frequency of different kinds of lesions is different from that seen in adult life, and there are more or less characteristic modifications in the severity and distribution of the lesions. The variations characteristic of the pathology of early life are most notable in infancy, and become progressively less through childhood, until, at about the age of puberty, adult conditions are reached.

Congenital lesions form a comparatively large group in the pathological anatomy of early life, particularly in infancy. They may be subdivided upon an etiological basis into two classes, one being due to embryonic conditions—abnormalities of fetal development, the other being due to intrauterine disease. Closely connected with these congenital lesions is another group due to trauma sustained during birth.

Functional disturbances, without recognizable primary lesions, are comparatively common in early life. The multiple etiology of these conditions has been discussed in the section on etiology. In early life, disturbance of function is most common in those organs or systems of the body which in childhood show a relative backwardness of functional development. Consequently, functional disturbances in early life are most commonly connected with the *digestive system* and *nutrition*, and with the *nervous system*. The largest divisions of any work on pediatrics must be those dealing with nutrition, metabolism, and the gastro-enteric tract. The circulatory system in early life is comparatively well-developed, while it contains the tissues and organs which, having a mechanical function, show most markedly the effects of the strain of advancing years, and of active abuses. Consequently, functional disturbances of the heart and circulation are comparatively uncommon in childhood.

New growths are very much less common in childhood than in adult life. Carcinoma, the most common tumor of later life, is almost unknown in infancy and childhood. Full development and maturity appears to be an essential condition for the occurrence of many varieties of new growth. Thus the fibro-myomata of the uterus, and the cysts of the ovary, so common in adult life, are not seen in childhood. Sarcoma is the most common new growth encountered

in childhood, the commonest form being osteo-sarcoma, and sarcoma of the kidney originating in adrenal tissue. Gliomata also are often seen.

The inflammatory lesions characteristic of infection are comparatively common in early life, and form by far the largest group in the pathological anatomy of infancy and childhood. The great liability of children to the group of infectious diseases transmitted by contact, has led to the name of "children's diseases" being applied to this group. The frequency of the contagious diseases in childhood is, however, probably not chiefly due to an increased susceptibility at that period of life. Indeed, infants as compared with older children show a distinct immunity to certain contagious diseases, such as dipthheria and scarlet fever, which may be possibly explained by a partial transmission of immunity from the mother. The relative frequency of the contagious infections in childhood is to be explained chiefly by the fact that these diseases confer an immunity upon the patient, and are not seen in adult life because of the immunity acquired in childhood. The problem of susceptibility and immunity is, however, very complex, and undoubtedly there exists in the undeveloped tissues of the growing child an increased susceptibility toward certain infections.

The mucous membrane of the respiratory tract is particularly liable to infection in early life, and the inflammatory lesions caused by infection are the commonest found at autopsies on children. Pneumonia is very common, and the lungs are rarely found normal at autopsy after an acute infectious disease of long duration. The great frequency of acute inflammation of the middle ear is also a very notable feature in the pathology of infancy and early childhood. The nose, throat, and larynx show about the same susceptibility to catarrhal inflammation as in the adult. The mouth, however, is peculiarly liable to lesions in childhood, both of infectious and of traumatic origin.

There is a marked difference between childhood and adult life in the character of the lesions found in the heart. In infants, practically the only lesions found are of congenital origin. In older children, the heart, both endocardium and pericardium, are particularly liable to the inflammatory lesions characteristic of acute infection. The lesions due to chronic endocarditis and its mechanical effects, are less common than in adult life. At autopsy, acute endocarditis or pericarditis is the usual finding in cardiac cases.

Arteriosclerosis, and the whole group of lesions secondary to vascular changes, which are found in all the organs of the body, and which play so important a part in the pathology of adult life, are almost unknown in childhood. Such conditions are angina pectoris, chronic

myocarditis, "cardio-renal cases," chronic interstitial nephritis, hemorrhage into the internal capsule of the brain, and so forth, play little or no part in the pathology of childhood.

The nervous system in childhood presents a widely different pathology from that of adult life. These differences may be summarized as follows: 1. The greater frequency of congenital lesions. 2. The greater frequency of some forms of functional disturbance (chorea, epilepsy, pavor nocturnus) and the lesser frequency of others (hysteria). 3. The lesser frequency of processes showing the pathological anatomy of chronic degeneration. 4. The greater frequency of acute infections. In connection with the last two points, it is notable that such nervous diseases as tabes dorsalis, syringo-myelia, paralysis agitans, and similar conditions are very uncommon in childhood. On the other hand, both meningitis in all its forms, and poliomyelo-encephalitis are much more common in children than in adults.

Lesions of the lymph nodes play an important part in the pathological anatomy of early life. They are affected secondarily to the various inflammations of the mucous membranes, and the result of acute infection is the suppurative inflammation so common in infants, while prolonged chronic infection causes a condition of chronic lymph node hyperplasia. The same condition of chronic hyperplasia is also a very common secondary lesion in the various chronic nutritional disturbances. The thymus plays a part in the pathology of infancy only.

The serous membranes are less often the seat of lesions in childhood than in adult life. Empyema is rather commoner, as is tuberculous peritonitis, but the other forms of pleurisy and peritonitis are less often seen.

The skin in early life is very delicate. Consequently it is very often the seat of lesions caused by infection, or by mechanical irritation. The group of lesions seen in adults which represent degenerative conditions, the results of wear and tear, are not seen in childhood.

Certain diseases, chiefly infectious, which occur both in childhood and in adult life, present peculiarities in pathological anatomy characteristic of childhood. Notable among these diseases are tuberculosis, syphilis, typhoid fever, and rheumatic fever. The lesions characteristic of childhood, and the variations in the pathological anatomy from that of adults, will be described in detail in connection with the conditions in which they occur.

III. SYMPTOMATOLOGY AND DIAGNOSIS

THE HISTORY

The first step in the diagnosis and treatment of a diseased condition is the obtaining of the history. Here at once are encountered conditions fundamentally different from those met with in dealing with disease in adults. A child cannot give a description of its symptoms, and consequently the history must be obtained from a parent, nurse, or other attendant. This is particularly the case with very young children, who are not able to describe any symptoms nor to answer any questions, and with children in the first years of life, the entire history of the case must be obtained from the parent. Older children are able to tell their stories, and describe their symptoms, but their powers of description are often very deficient, and their statements very misleading. Even in older children, the main source of information is the story of the parent, corroborated and assisted by what the children are able to tell. For this reason, subjective symptoms play a comparatively small part in the symptomatology of infancy and early childhood, and such as can be obtained have to be accepted at second hand. As far as the history is concerned, particularly in young children, the physician is compelled to rely on the objective symptoms as observed and reported by another person, usually the mother, whose powers of observation and description are not trained, and are often very inadequate. This tends to diminish the importance of this history in early life. It must not be assumed, however, that the history is not important. The physician must understand the conditions under which it is obtained, and the comparative degree of importance to be attached to the various symptoms reported.

It is often supposed by the laity, and even by physicians who are not trained in the diagnosis of disease in early life, that this difficulty in obtaining a history constitutes one of the chief difficulties in pediatric practice. It is often said that practice among babies must be especially difficult " because they cannot tell how they feel." It is not true that this constitutes an especial difficulty, but it is rather that it constitutes a need for special training. The pediatrist must become especially practiced in the observation and interpretation of objective symptoms. Moreover, the inability of the patient to tell "how he feels" presents a distinct advantage from certain points of view. The long accounts of *feelings* presented by sick adults, often exaggerated by the results of prolonged

auto-suggestion which may even amount to hypochondriasis, are not of much value in diagnosis, and are often actively misleading. In children, the clinical picture is often all the more clear-cut, from the absence of subjective symptoms.

In obtaining the history it is well to let the mother tell the story first without cross-examination. When the story is complete, the physician should begin his questioning, not with the family history or previous history, but with the present illness. It is very disconcerting to a mother, whose mind is earnestly bent on describing clearly the illness of the child, and who has just completed her story, to be immediately asked what her other children died of, and whether there is tuberculosis in the family. Such questions give the impression that the physician is merely proceeding by rote, that he is not interested in her story, nor in the real trouble with the child, and they tend to trouble and confuse her. The physician should begin by asking questions on the various points mentioned in the mother's story, with a view to bringing out and defining the various symptoms. He should then ask the questions necessary for the completion of the history of the present illness. The family and previous history can just as well be obtained after the story of the present illness is complete.

SIGNIFICANCE OF SYMPTOMS IN EARLY LIFE. In children, as compared with adults, subjective symptoms are less marked, while objective symptoms are more marked. In infants, the symptoms reported by the mother are all objective. It must be remembered that there is often an unconscious tendency on the part of the mother to exaggerate the severity of the objective signs which she has observed. There is also a tendency to interpret them wrongly, and to call things by misleading names. Such symptoms as loss of appetite, loss of weight, vomiting, diarrhea, cough, convulsions, restlessness, insomnia, and so forth, are apt to be somewhat exaggerated. On the other hand, such symptoms as dyspnoea, apathy, clouding of the mentality, paralysis, and others, are apt to be insufficiently noticed.

In older children, subjective symptoms occur. There is often an unconscious tendency on the part of both the mother, and the physician. to underestimate the severity and importance of subjective symptoms. Such symptoms are not so varied, nor so common as in the adult. Pain is a much less common symptom in childhood, and in many diseased conditions, it is apparently less severe. When, however, subjective symptoms are present, they are very important in children.

THE FAMILY HISTORY.—In the majority of cases, this should begin with the parents. The general health and constitutional vigor of both parents should be ascertained. The diseased conditions having

most bearing on the child are tuberculosis, rheumatism, syphilis and alcoholism. The first two should be inquired about specifically. In the case of tuberculosis, the transmission from the parent usually depends rather upon exposure to contagion than upon transmission of the disease directly, or of a hereditary tendency toward the disease. It is consequently advisable not to limit the inquiry to tuberculosis in the parents, but to extend it in such a way as to ascertain if possible, whether the child has ever come into contact with a tuberculous individual. Neurotic tendencies in the parents should also be ascertained when present. It is usually unnecessary to ask direct questions as to syphilis or alcoholism, unless something in the history of the present illness, or in the physical examination, should suggest the advisability as to inquiries along these lines. The number of other children living, and their general health, should be ascertained. The number of dead children, and the cause of death, should also form part of the record. Also, in connection with the family history, it is advisable to gain as thorough as possible an insight into all the surroundings of the patient. It is particularly important to find out whether there has been any recent exposure to contagious disease.

PREVIOUS HISTORY.—There are four points of importance to be elicited in the previous history of the patient. These are, (1) the circumstances attending birth, (2) the feeding history, (3) the history of development, and (4) the history of previous illness.

THE BIRTH.—The physician should first inquire whether the child was premature, or born at full term. The character of the labor should be inquired into, whether natural, instrumental, unduly prolonged, or attended by complications. Inquiry should be made as to whether the child appeared normal at birth, whether it seemed vigorous, whether it breathed promptly, and whether it took the breast well. The nutrition of the child during the early days of life, and the existence of any symptoms, such as hemorrhage or convulsions, should be ascertained.

THE FEEDING.—The physician should next ask whether the child was breast-fed or bottle-fed, and if breast-fed, how long breast feeding was continued, when partial bottle feeding was begun, and when exclusive bottle feeding was instituted. If the story told by the mother suggests that the illness of the child is in any way connected with nutrition, or if the problem presented is one of feeding, all the data concerning the diet history should be obtained down to the minutest detail. Each method of artificial feeding used should be ascertained, with a full account of just how each food was prepared for the baby, and the various mixtures used in home modification should be translated by the physician into terms of the percentages of the various food elements. The diet of the child after the first

year should be taken up in similar detail. The symptoms exhibited by the child with each change of food should be ascertained. In particular, inquiry should be made as to gain or loss in weight, vomiting or regurgitation, the daily number and character of the intestinal discharges, colic and flatulence, and whether the child was satisfied or hungry. These inquiries should be brought up to include the food which the child is now taking.

DEVELOPMENT.—The best idea of development is to be obtained from the weight record, if there is one. In addition, inquiry should be made as to the appearance of the most prominent landmarks of normal development. For physical development, these are the time and order of appearance of the teeth, and the age at which the child could first hold up its head unsupported, sit unsupported, creep, stand, and walk. Mental development is estimated by the age at which the child could first recognize the mother or nurse, recognize the bottle, seize and handle objects, understand the meaning of words, and speak words, short sentences, and connected speech.

ILLNESSES.—All previous acute illnesses should be noted, particularly the acute infections, with the dates. Details as to the duration, severity, and complications of each attack should be obtained. It is also advisable to learn whether the child is particularly liable to certain varieties of disorder, particularly those affecting the upper respiratory tract, the digestive system, and the nervous system. Under the first are included frequent colds, tonsillitis, adenoid operations, earache or otitis media, croup, and bronchitis. Under the second are included attacks of vomiting, diarrhea, stomachache, and "bilious" attacks. Under the third are included restlessness, insomnia, night terrors, convulsions, and chorea. The date of the last successful vaccination should also be ascertained.

THE PRESENT ILLNESS.—The details of the present illness are obtained from the story of the mother or nurse, and from the cross-examination of the physician, which defines the various symptoms. The most important point in the history of the present illness is that of *duration*. The date of appearance and duration of each symptom should be exactly defined, and these details are often not volunteered by the mother, but have to be obtained on cross-examination. The *character of the onset* of disease in children is one of the most important points in diagnosis, and whether the onset was gradual or abrupt must be carefully ascertained. This can often be done only by asking when the patient was last quite well, and what was *the first symptom* of illness noted by the mother. The sequence of subsequent symptoms can then usually be clearly obtained. Individual symptoms must be analyzed as to their time and mode of onset, frequency, character, severity, and duration.

After the story of the mother and the cross-examination analyzing the symptoms are finished, it is advisable to conclude the history of the present illness with a few general questions as to the functions of the various physiological systems of the body, which have not been included in the story of the mother or nurse, nor in the physician's cross-examination. The digestive system may be investigated by questions as to appetite, the bowels, vomiting, colic, flatulence and abdominal pain; the respiratory system by questions as to cough, dyspnoea, respiratory obstruction, and pain in the chest; the circulatory system by questions as to shortness of breath, precordial pain, palpitation, and edema; the nervous system by questions as to headache, night terrors, restlessness, insomnia, convulsions, and paralysis; the urinary system by questions as to the frequency and amount of micturition, pain on micturition, and the amount and appearance of the urine.

Finally, the mother or nurse should be asked what in her opinion is *the chief complaint*, the symptom which has mainly caused her to seek medical assistance, and which most requires relief.

THE PHYSICAL EXAMINATION

If the history of the symptoms in the young child is of less diagnostic significance than in the adult, the physical examination is of proportionately greater importance.

METHOD OF EXAMINING A CHILD.—The method of examining a child is quite different from that pursued with adults. In the first place, the order of examination is different. The child will be either crying or not crying. If it be not crying, it is best to begin with that part of the physical examination with which crying most interferes. Crying interferes most with the palpation of the abdomen, and next with the auscultation of the heart. If the physician is accustomed to the examination of children, crying will not interfere with the auscultation of the lungs, but it is sometimes useful to auscult the lungs with the child quiet as well as crying. Consequently, if the child be quiet, it is best to palpate the abdomen, auscult the heart, and then auscult the lungs, at the very beginning of the examination. If the child be crying, these examinations should be postponed till the end, in the hope that the child will stop crying. The examination of the mouth, throat, and ears should always be postponed to the end, for if the child be not crying and resisting, this examination will almost certainly cause it to do so.

The time which is consumed in the physical examination of a child is important. Prolonged handling of a child, or a prolonged struggle with a resisting child, is very harrowing to the mother. In the adult, the element of time is not so important, but the pediatrist

must accustom himself to make his examination quickly. Thoroughness must not be sacrificed, but a rapid deftness in physical examination is a great asset in dealing with children. This can be gained by practice, and by a thorough knowledge of just how each part should be examined, and just how the child should be held. It is not a good plan with children to make the physical examination before taking the history. The history often brings out information which will greatly modify the relative time spent in different parts of the physical examination. It is not necessary, for example, to spend a long time on the examination of the heart and lungs in a case which is obviously one of nutrition, nor in the testing of all the functions of the nervous system in a case which is obviously one of infection of the respiratory tract. Sufficient attention, however, should be paid to all parts of the examination, to insure that nothing be overlooked.

The manner in which the examination is approached by the physician makes a great difference in the handling of a sick child. He should not approach it strenuously, nor tower over it, nor make any parade of preparation. It is best not to notice the child at first, but to talk with the mother. Then the physician may notice and handle the child's playthings. It is often a good plan to give the stethoscope to the child to hold at the beginning of the examination, for if this is done it will not be so terrifying when the time comes to use it. It is also often helpful to perform any act of handling like percussion, or the application of the stethoscope, first on a doll, teddy bear, or even on the mother, before performing it on the child. By smiling and making a sort of game of the examination, a good understanding with the child can often be maintained for a considerable time.

If, however, friendly relations cannot be maintained, and the child begins to cry, and resist, all blandishments should be stopped, and the physician should proceed to complete the examination as rapidly as possible. It is often difficult to get mothers to cooperate under these conditions, as they want to avoid using force, and to continue their coaxing methods. At this stage, coaxing is usually of no avail, and the physician should tell the mother exactly what he wants her to do, and just how she must hold the child, and should *make her do just what he tells her*. This method, though it involves the use of forcible restraint, is much better in the end, as the examination is shortened instead of being prolonged in a confusion of crying, struggling, and coaxing.

In many cases, the best position for the child undergoing physical examination is recumbent on a firm surface. The recumbent position on the back is, however, only essential when the abdomen is being examined, and in an infant, this can be obtained with the

child lying across the mother's knees. Placing the child on its back on a table or bed is often more apt to frighten it than if it be examined on its mother's lap, and in many cases it is better to conduct the examination with the child in the latter position. This is especially the case in examinations conducted in the home, where good examining tables are not always available.

EXAMINATION OF A YOUNG INFANT ON THE MOTHER'S LAP.—The naked child is first placed on its back across the mother's knees and the front of the body is examined. If the child be quiet, the physician begins with the palpation of the abdomen, and follows with the auscultation of the heart and lungs. Then he proceeds to the general examination of the body, and to the examination of the

FIG. 14

Proper position for the child when examined in a sitting position

cranium, facies, eyes, and neck. Next he completes the examination of the front of the chest and the abdomen, and examines the extremities and reflexes. The child is then turned face down and the back of the chest, the spine, the buttocks, and the anus are examined. The mouth, throat, and ears are examined last.

EXAMINATION OF AN OLDER INFANT OR YOUNG CHILD ON THE MOTHER'S LAP.—The child should sit upon the lap of the mother or nurse, leaning back upon the middle of her body, not held in the

ɔw of one arm. In this position, the physician first notes the ts included in the general examination of the body. He next nines the head as far as external inspection and palpation are erned, which includes the cranium, facies, eyes, and neck, leav- the mouth, throat and ears for the end. He then examines the t of the chest, the extremities, and the reflexes. If the child ggles, or attempts to interfere with the examination by grabbing ıe stethoscope or at the hands of the physician, the mother should aught to grasp each of the child's arms firmly just above the ws, and to hold them close to the sides of the body. When the nination of the front of the body is complete, the child should

FIG. 15

Method of holding an infant for examination of the back of the chest

ırned with its back to the physician, and held upright if an t, kneeling if larger, with its head on the mother's shoulder, ıer arm around its hips. In this position the back of the chest ımined. The diaper is then removed, and the baby is placed ; back across the mother's knees, for examination of the abdo- then turned face down for examination of the spine, buttocks .nus. If the child be too large to lie across the mother's knees, last part of the examination must be performed on a table or bed.

EXAMINATION UPON A TABLE.—This is performed in the same manner as with the young child across its mother's knees. If the child struggles or resists, the mother or nurse should stand on the opposite side of the table, and place one hand on the child's two hands, holding them stretched above its head, and pinning them firmly to the table, while the other hand grasps both ankles. If the child is too big to be held in this way, the mother or nurse should stand at the head of the table, and grasping each of the child's hands in one of hers, should hold them firmly to the table close to its sides. This will prevent the child from getting up or turning over, but the physician must be prepared to dodge the kicking heels. It is sometimes advisable, in examining the back of the chest, to have the child sitting on the table with its back bent forward, rather than lying upon its face.

FIG. 16

Position for examination of the throat

EXAMINATION OF THE MOUTH AND THROAT.—The child should sit upright on the lap of the mother or nurse, facing the light if a head-mirror is not used, with the light behind if a head-mirror is used. The strongest possible light is desirable. The essential feature of the position for the examination of the throat is that the child shall be held with its back reclining against the *middle* of the mother's or nurse's body, not in the crook of her arm. The mother then

grasps both the child's hands *in one of hers*, and places her other hand on the child's forehead, holding its head firmly against her body. The child's legs are held between the mother's knees. This position leaves both the physician's hands free, which is important when cultures are to be taken. If no culture is to be taken, the physician can use his free hand in helping to steady the head, or in bringing it to the proper angle by pulling slightly forward on the neck. The teeth and mucous membrane of the mouth are first examined, and then the tongue is depressed for the examination of the throat.

A young infant can be held for the throat examination with the mother or nurse standing. This permits her to approach close to a window or light. The child is swathed in a blanket, and is held with its back against the mother's shoulder, in the position shown in the illustration.

FIG. 17

Method of holding an infant for examination of the mouth and throat
This allows the child to be held in a good light close to a window

The various points of importance to be especially noted by a physician in the examination of a child, present certain differences, which should be familiar to every one practicing among children.

GENERAL INSPECTION OF THE BODY.—This is accomplished by inspection, almost at a glance, and by a rapid palpation of the neck, axillae, inguinal regions, and muscles. It includes the

nutrition, size, weight, skin, position of the body, mental condition, lymph nodes, bones, joints, and muscles.

THE NUTRITION, SIZE AND WEIGHT, are estimated by inspection. This first estimate will be confirmed later by weighing and measurement. The amount of subcutaneous fat can be judged at this time by inspection and palpation. It is essential that the physician should be familiar with the growth of the normal child in length and weight, as described in the section on Normal Development.

THE SKIN.—The skin should be thoroughly examined for visible lesions. The most significant in early life are eruptions, ecchymoses, desquamation, cicatrices, edema, sclerema, and angioneurotic edema. The color of the skin should be noted for pallor, cyanosis, and icterus. It must be remembered that the color of the skin is not so reliable an indication of anemia and cyanosis as is that of the visible mucous membrane. Edema is recognized by the fact that the swelling is most marked about the eyes, on the dorsum of the feet, on the legs, and on dependent portions, that the color of the skin is normal or pale, that the swelling is not hard nor accompanied by rigidity, and that *it pits on pressure.* Sclerema is seen only in the newborn, or in very feeble infants, the color of the skin is normal or slightly bluish, the swelling is hard and accompanied by rigidity, and it does not pit on pressure. Angioneurotic edema is usually circumscribed, and somewhat pink in color, is often accompanied by itching, and does not pit on pressure. It should also be noted whether the skin is hot or cold, moist or dry, smooth or rough, thin or thick. Rarer lesions sometimes seen in children are emphysema, rheumatic nodules, tumors, moles, and parasites. The bluish-black "mongolian spots," sometimes seen in the sacral and glutial regions, have no diagnostic significance.

POSITION OF THE BODY.—This is often of diagnostic significance, and should always be noted. A child may have a tendency to lie on one side or the other, to lie with the legs drawn up or the limbs in an abnormal position, or the spine may be abnormally curved either forward or backward.

MENTAL CONDITION.—This is one of the most important points in the physical examination of the sick child. A fairly accurate estimate of the mental condition can usually be made by inspection in the general examination of the body, although at times abnormalities can only be recognized by the special testing of the condition of the nervous system which is carried out later. The physician should note carefully the child's reaction to its surroundings, and in particular, how it reacts to the examination. Thorough familiarity with the behavior of normal children at every age is essential. The physician should note whether the child follows his movements

intelligently, and whether it resists intelligently or mechanically. By watching it closely, he can usually estimate its mental development, and can recognize the presence of such abnormalities as *apathy*, *stupor*, or *delirium*.

THE LYMPH NODES.—The cervical, axillary, epitrochlear, and inguinal lymph nodes, may be rapidly examined by palpation, and this can well be done during the general inspection of the body, as it does not usually frighten the child, and is a good preparation for the palpation and percussion which is soon to follow. The lymph nodes are examined chiefly for enlargement, tenderness, and suppuration. Enlargement of the lymph nodes may be general or local. It must be remembered that general enlargement of the peripheral lymph nodes in infancy is not of the same significance as in adult life. It occurs in practically all disturbances of nutrition, may include the epitrochlear and occipital nodes, and at this age does not point to syphilis, tuberculosis, or primary disease of the blood. In later childhood, general enlargement of the peripheral lymph nodes has the same significance as in adults.

Local enlargement of the peripheral lymph nodes is particularly common in early life. It has the same significance both in infancy and childhood as in adult life. The most common causes are, (1) acute lymphadenitis due to the absorption of toxic products from an infection in the area draining into the nodes involved; (2) acute infection of the lymph nodes themselves, usually leading to suppuration; and (3) tuberculosis. The lymph nodes of the neck are by far the most frequently involved in local enlargement, and are almost the only peripheral lymph nodes involved in tuberculosis.

THE BONES AND JOINTS.—The size, contour, and general appearance of the bones and joints can be estimated by inspection during the general examination of the body. If there is nothing in the history to suggest any abnormality of the bones and joints, their routine examination can well be completed at this time. The long bones and joints should be felt, and pressure should be applied. The chief signs to be looked for are deformities, swelling, tenderness, and redness. The significance of these various signs will be discussed under the examination of the extremities.

THE MUSCLES.—The condition of the muscles can be estimated by inspection and palpation at the same time as the nutrition and amount of subcutaneous fat are noted. The principal abnormal conditions in the muscles are atrophy, softness, and tenderness. The first is seen in many nutritional disturbances, particularly in those of gastro-intestinal origin. A soft, flabby, relaxed condition of the muscles is seen also in nutritional disturbances, particularly in rickets. Muscular tenderness is most often a sign of over-use. In the back

or extremities it is most often seen after over-exercise. Tenderness of the muscles of the abdominal wall is met with after excessive coughing or vomiting.

TEMPERATURE.—The period of the examination at which the temperature is taken depends on circumstances. If the child is quiet, it is often best to postpone it till the end of the general examination, just before the examination of the throat. It should always be taken either by mouth or by rectum; temperature taken in the axilla or groin cannot be relied upon. Whenever a child is too young to hold the thermometer properly in the mouth, the temperature should be taken by rectum.

FIG. 18

Obtaining pulse rate in infants

PULSE.—This is usually best examined during the general examination of the body, but if the attempt to take the pulse appears to annoy the child, it can well be postponed until later, and taken in connection with the examination of the heart, the limbs, or the taking of the temperature. The points to be noted are the rate, rhythm, volume, compressibility, and character of the wave. In cases showing signs of circulatory disturbance, the radial pulse should be compared with the ausculted heart sounds, and the capillary pulse should be sought.

The normal pulse rate at the different ages is given in the section on Normal Development. It must be remembered that an entirely

different standard from that of adults must be used in estimating abnormalities of the pulse in early life. Increase in the rate of the pulse is of much less significance in infancy and early childhood. The rate tends to increase very markedly from comparatively slight causes, such as exertion, excitement, or any slight disorder. Given any such cause, the absolute rate of the pulse is not very significant. If, however, in any severe disease the rate of the pulse is continually increasing, it points toward an increasing severity of the underlying pathological condition. The ryhthm of the pulse is also very easily disturbed; the pulse is normally irregular in the newborn, and easily becomes irregular from slight causes in infancy and early childhood. The volume and compressibility are more significant as evidence of the condition of the circulation.

THE RESPIRATION.—The respiration is best observed as a part of the general inspection, and should be examined as to rate, rhythm, and character. The rate varies normally with the age, with the individual, and according to whether the child is active or quiet, asleep or awake. It is therefore difficult to give a standard which is correct for the different ages; an approximate standard is given in the section on Normal Development. Irregularity of the respiration, which is so prominent in the newborn, continues normally during the first two years, and is not significant in infancy. In later childhood, irregularity is more important, and Cheyne-Stokes' types of diagnostic significance can often be recognized. Even in infants a *regular irregularity* is suggestive.

The points to be noted in connection with the character of the respiration, are whether it is painful, shallow, costal, or diaphragmatic. Painful respiration is betrayed by a contraction of the child's face, and by an expiratory groan or grunt. Shallow respiration is not of much significance, as this is a normal form in young children. Also up to the seventh year the respiration is normally predominantly diaphragmatic in character. The relation of inspiration to expiration should also be noted.

DYSPNOEA in early life is of two types, obstructive and non-obstructive. Each of these types may be further subdivided into inspiratory and expiratory. In the non-obstructive type, the respiration is increased in rate, and is often labored in character, but is not accompanied by any stridor, or other evidence of mechanical obstruction. It is seen in infections of the lungs and pleura, in disease of the heart, and in certain conditions of toxemia. Although often accompanied by an *expiratory* grunt, the type is mainly *inspiratory* in these conditions, the principal muscular effort being that of inspiration. In some toxic conditions, such as acidosis and diabetic coma, the inspiratory character is so marked that it is

called "air-hunger." Expiratory dyspnoea is seen chiefly in asthma, in which the expiration is labored, and though wheezing is heard, there is no evidence of any localized obstruction.

In the obstructive type, there is a stridor, or noise, suggesting the presence of a localized narrowing of the air passages. The location and character of this sound is evidence of the location and character of the obstruction. When the sound is a coarse stridor without musical tone, and appears to come from the throat or nose, the obstruction is usually in the nose or naso-pharynx. The dyspnoea is usually inspiratory, when the obstruction is due to such conditions as rhinitis, adenoids, or enlarged tonsils, but in retropharyngeal abscess the most marked sound is usually heard during expiration. When the sound is crowing, with a more or less musical tone, the obstruction is in the larynx, and the dyspnoea is of a marked inspiratory type. Severe inspiratory dyspnoea is further to be recognized by inspiratory retraction of the supra-clavicular, intercostal, and epigastric spaces. It must be remembered, however, that inspiratory recession of the epigastrium is normal in the early months of life. When the stridor comes from the chest, it is usually less marked. The commonest cause is pressure upon the trachea or primary bronchi by an enlarged thymus, or enlarged mediastinal lymph nodes. The dyspnoea is usually of the expiratory type.

THE HEAD.—After the general examination of the body has been completed, the physician proceeds to the examination of the various anatomical parts. This should be carried out systematically in a definite order. It is convenient to begin with the head.

THE CRANIUM.—The cranium should be examined with reference to size, shape, the fontanelles, the sutures and ossification, the hair, and the superficial veins of the scalp.

The absolute size of the head varies with the individual, and the important point is the relative size in comparison with the thorax. The normal relations are given in the section on Normal Development. The most common cause of enlargement of the cranium is hydrocephalus, and of an abnormally small cranium is microcephalus.

The most common abnormalities in the shape of the cranium are those caused by rickets and hydrocephalus. They are recognized by inspection, and will be described in connection with those diseases. Certain deformities resulting from compression during labor are often present at birth, but disappear during the early weeks. There is also a marked congenital asymmetry of the head seen at times, which persists for a number of years, but usually disappears before the child is seven years old. Flattening of the back or side of the head may occur as a result of the child lying too much in one position. This is easily recognized and corrected.

The fontanelles are examined by palpation with reference to size, delay in closure, depression, and bulging. An open posterior fontanelle after the sixth week, and an open anterior fontanelle after the nineteenth month, are abnormal. Delay in closure, or abnormally large size of the fontanelles is due most commonly to rickets, and next, to hydrocephalus, but is sometimes seen as a peculiarity in normal children. It is well to measure the diameters of the anterior fontanelle; they should not be over 2.5 by 3 cm. Depression of the fontanelle is caused by a decrease in the quantity of intracranial fluid, and is seen chiefly in acute diarrhea, and chronic conditions of malnutrition. Bulging of the fontanelle is caused by increased intracranial pressure, and is seen chiefly in meningitis and hydrocephalus, sometimes in brain tumor, brain abscess, and brain hemorrhage.

The sutures are examined for failure to close, and for overlapping. Except that the upper part of the frontal suture may normally remain open for a few weeks, any separation of the cranial bones is abnormal. It is seen most often in rickets and hydrocephalus. The cranium should also be examined for *cranio-tabes*—a softening of the bones of the skull. It is recognized by pressure upon various parts of the cranium with the tips of the fingers. Soft spots are found which give the sensation of tightly stretched parchment. They are found most often about the fontanelles and sutures, and over the occipital and parietal bones. Craniotabes is usually a sign of rickets, but is sometimes seen in syphilis. If it be present *at birth*, it represents a congenital developmental anomaly rather than rickets or syphilis.

The hair is examined as to amount and texture. The fine hair present at birth is not permanent, but is replaced. There is great variation in the period when this occurs, so that while some babies soon have a thick growth of hair, others remain bald for a considerable time. Loss of hair on the back of the head is a sign of restlessness, from whatever cause. Coarse hair should suggest the possibility of cretinism.

The superficial veins of the scalp are examined for enlargement. This is a constant sign in chronic internal hydrocephalus, and is often seen in syphilis. It is sometimes seen in rickets and other nutritional disturbances.

THE FACIES.—Inspection of the face often reveals abnormal signs. The most important point to be noted is the facial expression, which often gives useful evidence as to the mental condition. The expression may be placid, stupid, anxious, or pinched. There are various types of facies which are difficult to describe, but which are easily recognized from experience. The most familiar is the adenoid facies, characterized by open mouth, and stupid, vacant look, and sometimes

by a flattened bridge of the nose, narrowing of the face toward the chin, prominent upper lip, and obliteration of the naso-labial folds. There is an "abdominal facies," often seen in severe abdominal disorders such as peritonitis or intussusception, which is characterized by a peculiar sunken-eyed appearance difficult to describe, but easy to recognize. There is also a cerebral facies, seen in patients with severe acute intracranial lesions such as meningitis, characterized by a peculiar staring expression.

Facial paralysis is recognized by inspection of the face, but is often overlooked unless the child laughs or cries. In repose, the chief sign is obliteration of the naso-labial fold on one side; on crying, the lessened movement on one side is plainly apparent.

THE EYES are inspected for inflammation of the conjunctiva, icteric staining of the conjunctiva, conjunctival discharge, strabismus, nystagmus, and inequalities of the pupil.

Inflammation and conjunctival discharge suggest infection, and a purulent discharge should always be examined bacteriologically. Nystagmus and irregularities of the pupils suggest either disease of the central nervous system, or local disease of the eye. I have often seen nystagmus in albinos. In connection with strabismus, it should be remembered that coordination is not well established in young infants, and a normal baby does not fix its eyes until it begins to recognize objects at about the age of six weeks, or even somewhat later. In these early weeks, temporary and varying strabismus is of no diagnostic significance. A constant strabismus is suggestive of a congenital abnormality of the muscles of the eyeball. In older infants and children, if strabismus be found, it is necessary to ascertain if the condition has been present since birth, in which case it is due to the same local cause. If the strabismus has not been present from birth, but has developed as a symptom of the present illness, it is important in diagnosis, pointing toward a disturbance of the central nervous system.

The pupillary reaction can best be tested at this period of the examination. The test is best performed either by bringing a light from above downward in front of the eyes, or by suddenly snapping on a pocket flash-light held in front of the eyes. The normal reaction to light is present immediately after birth. Failure to react suggests disease of the central nervous system. It is useless to attempt to test the reaction to accommodation in infants and young children.

THE NOSE, in routine examination, is investigated by simple external inspection. The most important signs in diagnosis are widening of the bridge of the nose, and discharge from the nostrils. In infancy and early childhood the bridge of the nose is normally

slightly developed and relatively wide, but a pronounced widening is seen in cretinism and Mongolian idiocy. A nasal discharge in infants as well as children, is usually due to a simple catarrhal rhinitis, and should not suggest syphilis unless other signs be present. A thin, irritating, blood-tinged discharge, especially if coming from one nostril, suggests nasal diphtheria, and a culture should always be taken. A unilateral discharge also suggests a foreign body in the nostril.

THE MOUTH is best examined at the end in connection with the throat, but the signs to be noted are most conveniently described in connection with the systematic examination of the head. The physician should first note whether the mouth is kept open or closed. An open mouth suggests obstruction in the nose or naso-pharynx, most commonly from adenoids, but it should be remembered that young children when deeply interested often keep their mouths open. The physician should next inspect the lips as to their color, and as to the presence of fissures, ulcerations, herpes, and deformities. In infants fissures and ulcerations are suggestive of syphilis; while in older children they are usually due to chapping. Herpes is seen in acute infectious diseases. The most common deformity is hare-lip.

The throat stick or handle of a spoon is now inserted between the lips, which are retracted for the inspection of the teeth and gums. The number and arrangement of the teeth are noted, and compared with the normal at that age. The appearance of the teeth, whether carious or deformed, is also noted. Delay or irregularity in the appearance of the first set of teeth is a sign of rickets, but may be only an inherited or individual peculiarity, and should not be attributed to rickets unless other signs of the disease be present. Other disturbances of nutrition do not usually cause delayed dentition, but may cause the formation of imperfect teeth with a tendency toward early decay. The permanent teeth show similar changes if nutrition is disturbed in early childhood. The Hutchinson teeth, characteristic of syphilis, are seen only in the second dentition.

In the inspection of the gums, the physician should note their color, and whether they are swollen, spongy, hemorrhagic, ulcerated, or separated from the teeth. Hemorrhagic lesions of the gums are characteristic of scurvy, while the other changes are seen in the various varieties of stomatitis.

The tongue depressor is now inserted between the teeth, and the cheek on each side is retracted for inspection of the buccal mucous membrane. Disease signs to be particularly looked for are the *redness of catarrhal stomatitis, the mucous patches of syphilis, the Koplik's spots of measles, the eruptions of the exanthemata, and the characteristic lesions of thrush, stomatitis herpetica, and ulcerative stoma-*

titis. There are two appearances in young infants which must not be mistaken for any of the above lesions, namely, *Epstein's pearls*, and *Bednar's aphthae*. The former are one or more small white or yellowish-white nodules in the median line of the hard palate near its junction with the soft palate, and are accumulations of epithelial cells. The latter are symmetrical erosions or ulcerations of mechanical origin on each side of the hard palate where the mucous membrane over the tips of the sphenoidal hamular processes is very thin.

The shape of the roof of the mouth should be noted. It is flatter in infancy than in later childhood. While normal variations in the arch of the hard palate are very great, excessive arching is suggestive of obstruction to nasal respiration, the most common cause being adenoids.

The tongue is next inspected. The physician should note whether it is pale, red, cyanotic, dry, moist, smooth, rough, or coated. The mouth is normally relatively dry in early infancy. Later the salivary secretion increases rapidly in amount, and until the infant learns to swallow it, drooling is a normal condition. The tongue is normally lightly coated during early infancy. The size of the tongue should be noted. Enlargement and protrusion may rarely represent a congenital malformation, but is more often a sign of cretinism. The tongue may show any of the lesions characteristic of the various forms of stomatitis, and of syphilis. Ulceration of the under surface of the tongue about the frenum, as a result of the mechanical irritation of the lower incisor teeth, is not uncommon in infancy. The frenum should also be inspected for tongue-tie. The mucous membrane of the tongue in children may show the peculiar appearance of "geographical tongue." Enlargement of the papillae of the tongue is an important sign of scarlet fever.

THE THROAT.—The proper position of the child in the examination of the throat has already been described. If the child refuses to open its mouth, the tongue depressor can usually be worked in gradually from the side, being pushed in little by little when the child cries. It is rarely necessary to pinch the nose. As soon as the depressor is over the tongue, downward pressure will cause the child to open the mouth. It is necessary, in examining the throat, to make the child gag. This cannot be accomplished by pressure upon the front or middle of the tongue, but the depressor is placed on the back of the tongue and pressed downward and forward. The physician should practice his powers of observation so that he sees all there is to see in a brief space of time.

In the examination of the throat, all visible portions of the mucous membrane should be inspected for *redness*, *eruptions*, *exudate*, *ulceration*, and *membrane*. The localization of any of these signs, whether

upon the soft palate, uvula, pillars, tonsils, or posterior pharyngeal wall, should be noted. Whenever exudate or membrane is observed, a culture should be taken. Redness alone suggests simple catarrhal pharyngitis, or scarlet fever. Eruptions suggest the exanthemata. Exudate suggests follicular tonsilitis, but is sometimes seen in scarlet fever or diphtheria. Ulceration suggests Vincent's angina, syphilis, or tuberculosis. Membrane suggests diphtheria, scarlet fever, or membranous angina.

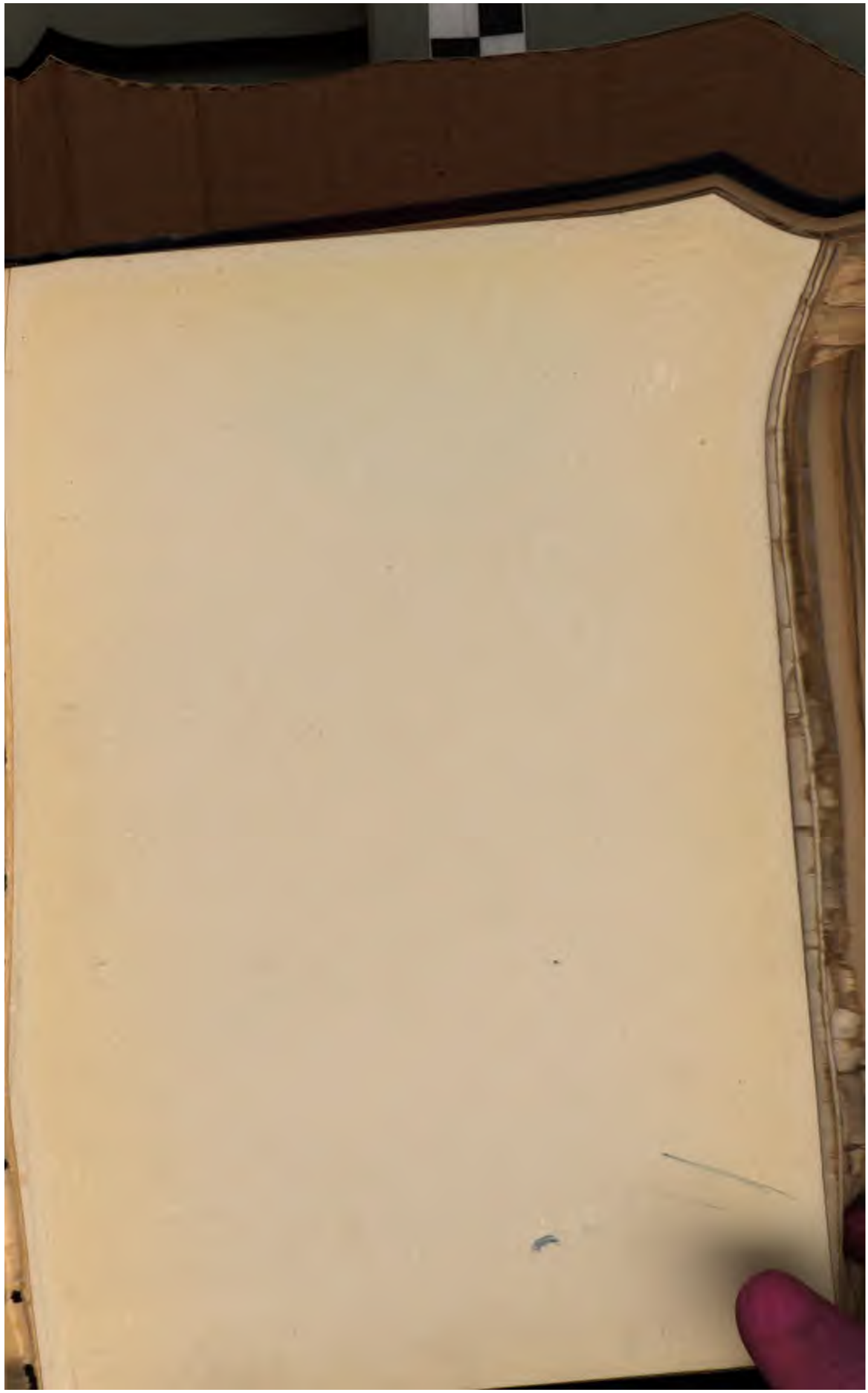
The physician should note whether the soft palate rises properly when the child gags; failure to do so is suggestive of diphtheritic or bulbar paralysis. He should also note whether the uvula is elongated or edematous.

The size and appearance of the tonsils should be carefully noted. It should be remembered that throughout childhood the tonsils normally are relatively larger than in the adult. An enlargement of one tonsil, or a pushing of one tonsil toward the median line, is an important sign, suggesting tonsillar or peritonsillar abscess. Enlargement of both tonsils suggests tonsillar hypertrophy.

The posterior wall of the pharynx is inspected for the swelling characteristic of retropharyngeal abscess, for the enlargement of the lymph follicles seen in chronic catarrhal inflammation, and for the excessive muco-purulent or purulent secretion seen in rhino-pharyngitis. It must be remembered, however, that retropharyngeal abscess is often invisible to simple inspection. Therefore, *in the examition of the throat, whenever there are any signs of obstruction either to respiration or to deglutition, a digital examination should be made.* A gag should not be used, as in retropharyngeal abscess the wide separating of the jaws sometimes results in sudden death.

THE NASO-PHARYNX.—The naso-pharynx cannot be examined by ordinary inspection. Evidence of disease in this region is obtained from the presence of pathological secretion coming from the nose or seen in the throat, and from evidences of obstruction to the respiration. In young children, the naso-pharynx cannot be examined with the mirror. This can be done in some cases in children old enough to cooperate, but in many cases it is impossible throughout childhood. Consequently the physician must depend on digital examination. This should never be undertaken as part of a routine examination, but should be carried out only when it is desired to confirm the presence of adenoids, or when there are signs of respiratory obstruction.

For digital exploration of the naso-pharynx, the child is held sitting on the nurse's lap, with its left side against her body, and its arms held to its sides. The physician stands behind the child, and with his left hand holds the mouth open, either with the fore-



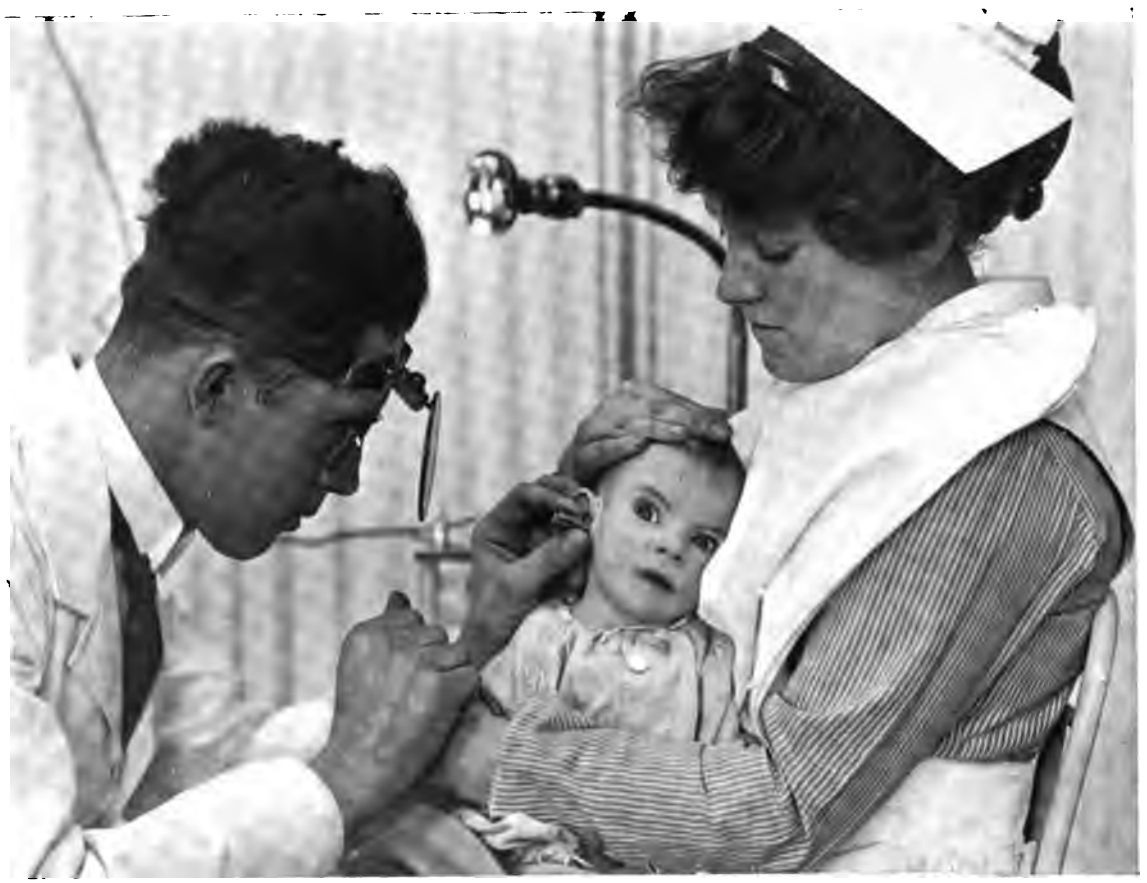


FIG. 19—Position for examination of the ears

finger, or with a gag if the child be too large. The right forefinger is introduced into the mouth with its dorsum downward. In reaching the naso-pharynx, care must be taken to hook the finger under the soft palate, for if the soft palate be pushed ahead of the finger, a very false impression will be gained from palpation.

THE LARYNX.—Inspection of the larynx is so difficult in children, that laryngoscopy is only undertaken in special cases. Most of the common diseased conditions in the larynx can be recognized by the sound of the respiration, the sound of the voice, and the character of the cough.

THE EAR.—Examination of the ears in children is somewhat disturbing, and need not be a part of the regular routine in every variety of case. When a satisfactory explanation of the symptoms is found elsewhere, as in a case of gastro-intestinal or nutritional disorder without fever, there is no necessity of examination of the ears. The ears, however, should always be examined as a routine under the following circumstances: (1) When there is fever, or when there are any symptoms which could be caused by otitis, which are not adequately explained; (2) in all acute infectious diseases, and the examination should be frequently repeated in the course of the disease; (3) whenever there are any symptoms suggesting otitis media. It must be remembered that disease of the middle ear in childhood exists more often without symptoms than with them, and cannot be ruled out by the absence of such symptoms as discharge, pain, putting the hand to the head, restlessness, mastoid tenderness, or even fever.

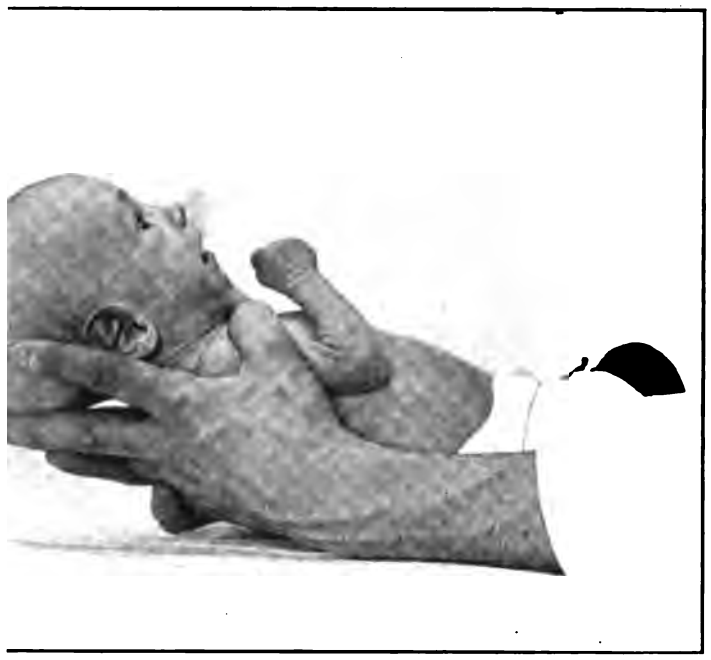
The ear is examined with a head mirror and a speculum. A specially small speculum is needed for the examination of an infant's ears. It must be remembered that the direction of the external auditory canal is different in infants, and that to straighten the canal the ear must be pulled downward and a little forward, instead of upward and backward as in older children and adults.

The points to be looked for are reddening of the drum, bulging of the drum, and disappearance of the landmarks. The drum is more horizontal in infancy than later.

THE NECK.—Among the most important physical signs in early life are rigidity of the neck, tenderness of the back of the neck, and retraction of the head. Examination with reference to these signs should be made with every sick child. The proper method for the physician is to place both hands—one from each side—behind the occipital region of the head. The head is tilted forward and back for rigidity, and then the physician presses on the back of the neck for tenderness. Retraction of the head is recognized by inspection. These signs are by no means diagnostic of meningitis, but suggest meningeal irritation, whether from infection or from toxemia. The

ld also be examined with reference to torticollis, spinal venous fulness, abnormal pulsation, abnormal swellings, ement of the salivary glands, thyroid, or lymph nodes.

FIG. 20



Examination for rigidity of the neck

HEST. INSPECTION.—The chest is examined with refer- :e, shape, symmetry, mobility, and deformities. The nor- arities of the size and shape of the chest in infants and dren has been described in the section on Normal Develop- ıe most common deformities of the chest in the first two e are due to rickets, and are enumerated under the descrip- .t disease. The physician should look in particular for the ɔsary, consisting of bead-like enlargements at the junc- : ribs with their cartilages. Flattening of the sides of the ng of the lower ribs, Harrison's groove, "pigeon breast," ıest," should all be looked for. The last is more often a malformation than due to rickets. Marked deformity of in infants is sometimes produced by certain less common

I have seen it as a very conspicuous feature of con- monary atelectasis, and of myatonia congenita. Deformity st in older children is caused most commonly by tuber- the spine, disease of the pleural cavities, and paralysis

or weakness of the muscles. Prominence of the region of the sternum is seen in forms of cardiac disease associated with marked enlargement of the heart. The amount and character of this deformity is valuable as evidence as to the period in life when the cardiac disease was acquired. In congenital cardiac enlargement, the prominence is most marked in the middle of the sternum, the curve receding both above and below. In cardiac disease acquired in early life, the prominence is most marked over the lower part of the sternum. In cardiac enlargement acquired late in childhood, there is usually no precordial prominence.

PALPATION.—The rachitic rosary is not always evident to inspection, and should always be sought by palpation. It must be remembered that in thin babies, the line of junction of the ribs with their cartilages is palpable. The rosary is recognized by the fact that the prominence can be felt at the sides of the junction as well as just at the point of junction.

THE THYMUS.—The normal thymic dulness in infancy is very difficult to detect. Very light percussion will show slight dulness under the manubrium, which in my experience is continuous with the cardiac dulness, although some writers state that there is a zone of vesicular resonance between. The thymic dulness is said to diminish gradually and to disappear at about six years. Practically, any marked degree of dulness under the manubrium should suggest enlargement of the thymus, but this diagnosis cannot be made with any certainty on routine physical examination. The thymus may normally extend a little above the sternal notch but is not palpable; if it is felt in this region, it is certainly enlarged.

THE HEART. INSPECTION.—The precordia should first be inspected for pulsation, and the position of the apex beat should be noted. It must be remembered that in early infancy the cardiac impulse is normally rarely visible and is often not palpable. In childhood, on the other hand, the impulse is relatively more distinct than in the adult. The physician should also note the character of the impulse, whether localized or diffuse, faint or forcible.

PALPATION.—The evidence obtained by inspection as to the cardiac impulse should be confirmed by palpation. In placing the position of the apex beat (which means the point farthest out and down in which the impulse is visible or palpable), the physician should remember the anatomical conditions characteristic of infancy and early childhood. In infancy, the apex beat is normally higher up and farther out than in later life. At this age it is in the fourth interspace, about 1 cm. (3/8 in.) outside the nipple line. From this point it gradually moves inward and downward, reaching the

fifth space in the nipple line at about seven years, and com'ng inside the nipple line before the thirteenth year For purposes of record, it is best to record the position of the apex in relation to the nipple line, rather than to the median line, because the distance from the median line varies with the size of the child.

The physician should also confirm by palpation his observations as to the force and character of the cardiac impulse, and should note whether any thrill be present.

PERCUSSION.—The outlines of the cardiac dulness are now determined by percussion. The normal outlines at the various ages of infancy and childhood have been given in the section on Normal Development. Both the superficial or absolute dulness, and the deep or relative dulness, should be noted. Many authorities state that the absolute dulness is more difficult to determine than is the relative dulness. In my experience, it is just the other way; the absolute dulness is easier to determine, but is less reliable as evidence of the actual size of the heart than is the relative dulness. It is for this reason that it is better to record both outlines.

Percussion in infants and young children should be light and delicate. In determining the left border, the physician should place his finger first on the area between the left nipple and the left border of the sternum, and should percuss toward the left until the tone becomes resonant. In determining the boundary of absolute dulness on the right, he should begin at the same point and percuss toward the right until there is a change of tone. In determining the boundary of relative dulness on the right, the physician should begin percussion entirely to the right of the cardiac area, and should percuss toward the left until a modification of the resonance is noted. In determining the upper border of dulness, he should percuss from above downward.

AUSCULTATION.—In children, auscultation should be performed as in the adult, the most significant areas being those of the apex beat, the middle of the precordia near the left border of the sternum, the base on each side of the sternum, and the lower part of the sternum itself. The character of the heart sound should be noticed with reference to rate, rhythm, tone, loudness, and relative accentuation or diminution in the different areas. A peculiar character of the first sound which lacks the booming quality of later life, and resembles the second sound, is normal in early infancy and should be remembered. It should also be remembered that in infancy and early childhood, the first heart sound is fainter at the base than in later life.

The pulmonic second sound is normally louder than the aortic throughout the whole of childhood, and for this reason accentuation of the second pulmonic sound is difficult to recognize, and not of

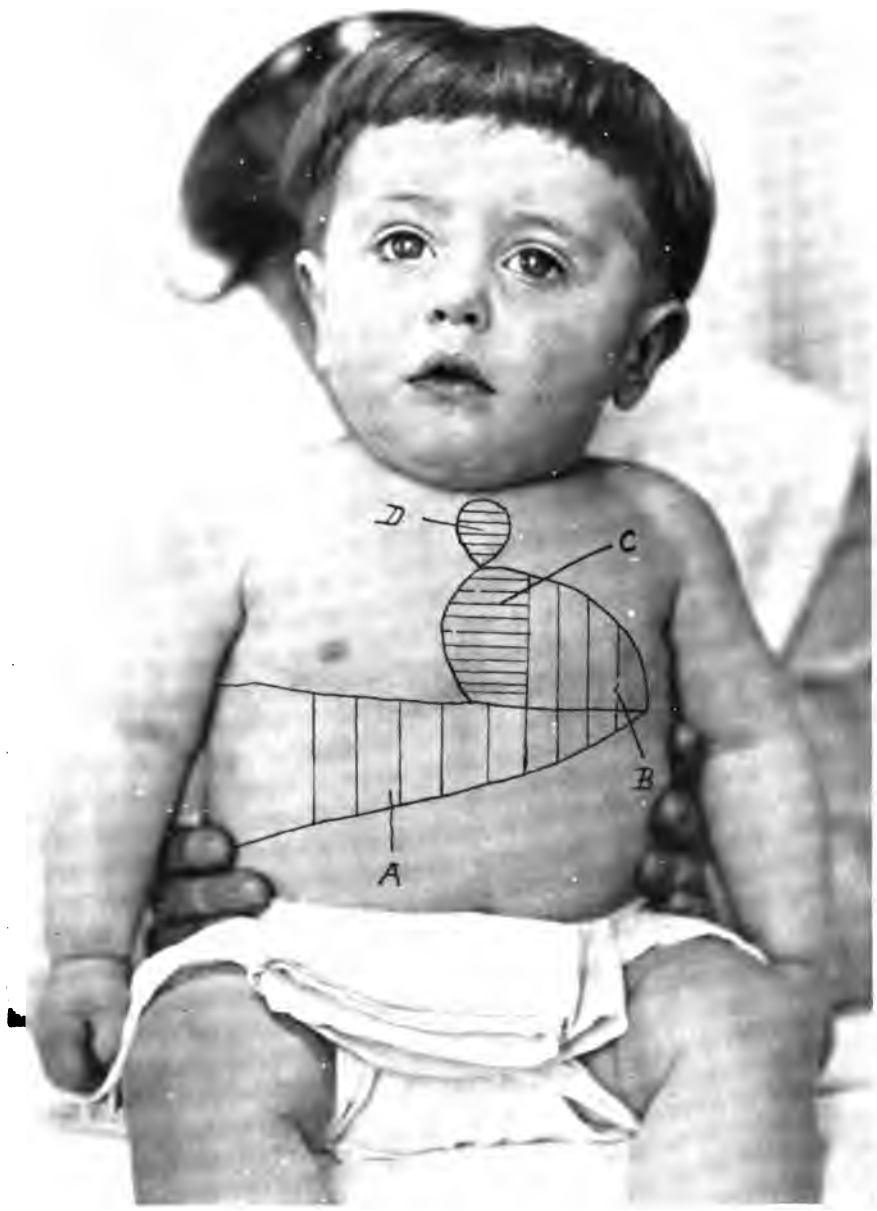


FIG. 21—Normal areas of dulness to percussion of the front of the chest

A. Hepatic dulness
B. Superficial or absolute cardiac dulness
C. Deep or relative cardiac dulness
Thymic dulness in an infant

much diagnostic significance. Reduplication of the second sound is also often heard in normal children, and should not be considered pathological unless there are other evidences of cardiac disease.

The presence of such adventitious sounds as murmurs and friction-rubs should be carefully noted. With reference to murmurs the following points should be recorded: (1) The region in which they are heard and their point of greatest intensity; (2) their time with reference to the cardiac cycle—systolic, presystolic, or diastolic; (3) their loudness; (4) their character—soft, harsh, or musical; (5) their transmission—whether they are heard in the axilla, back, or over the vessels of the neck; (6) whether or not they replace the heart sounds. It is to be remembered that in the first two or three years of life, murmurs due to acquired cardiac disease are very uncommon. At this age, murmurs are either functional, or represent a congenital cardiac lesion. The physician in considering the relative weight of evidence pointing toward the functional or organic origin of a murmur, should remember that in an infant, if the evidence points toward organic disease, the lesion is usually congenital. The significance of murmurs will be considered in detail in the division on Diseases of the Heart.

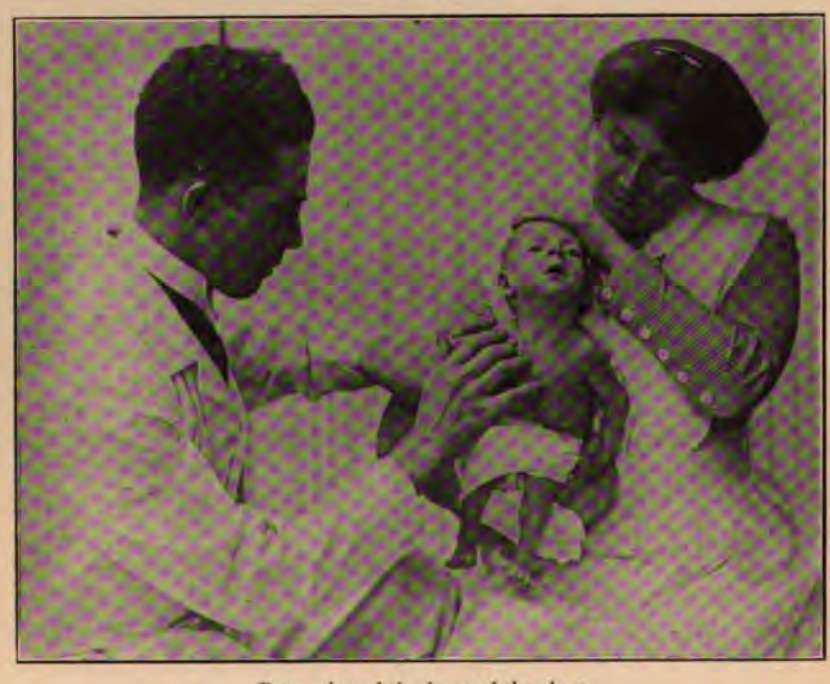
LUNGS. INSPECTION.—The observations as to the rate and character of the respiration, and as to the presence or absence of cough, have been made during the general preliminary examination of the body. The physician should therefore note whether the movement of the chest on both sides is equal, whether there is inspiratory retraction of the intercostal spaces, and whether there is increased circumference of one side with bulging of the intercostal spaces.

PALPATION.—Palpation over the lungs is used to determine whether there is increase or diminution in the tactile fremitus caused by the voice sounds. This is much less useful in children than in adults, because in many cases it can only be obtained when the child is crying, and the crying voice sounds are so loud, and the chest wall so thin, that the finer differences cannot be detected. It is also less valuable in diagnosis, especially in young children, as good tactile fremitus is often felt over an effusion. In older children, who are able to speak as ordered by the physician, the results of palpation are more valuable. The tactile fremitus should always be tested, but the physician should not rely too much on its results in young children, as compared with the results of other methods of examination. Rales are often palpable in young children.

PERCUSSION.—It is more difficult in infancy and early childhood to recognize changes in the percussion note, than in later childhood. When a young child is crying hard, the resonance of all parts of the lungs is impaired. When, however, a clear difference in the resonance of the two sides is made out, the evidence is just as valu-

able as it is in later childhood, unless the only abnormality found is slight dulness over the right base. This may be due to the relatively large size of the liver in infants. Percussion in children should be particularly light and delicate, on account of the thinness of the chest wall, and the small size of the parts. Strong percussion will produce confusing sounds coming from a distance, and cannot elicit a slight relative dulness. The finger, not a percussion hammer, should always be used. The results of percussion are entirely unreliable if an infant is lying on its side, because the chest is so compressible that the resonance of the lower lung is perceptibly impaired. The infant may, however, be lying on its back, or on its face, may be sitting upright, or may be held upright facing the shoulder of

FIG. 22



Percussion of the front of the chest

the mother. Even in the latter case, there is some possibility of compression, and the infant should be transferred to the other shoulder if there is any suspicion of dulness on one side; also, if possible, the arms should be placed in approximately the same position.

It is difficult in young children to determine accurately the borders of the lungs, and usually impossible to determine by percussion the mobility of the pulmonary borders. The borders in infancy are about one space higher than in later life. The relation of the various lobes of the lung to the chest wall is practically the same in infancy

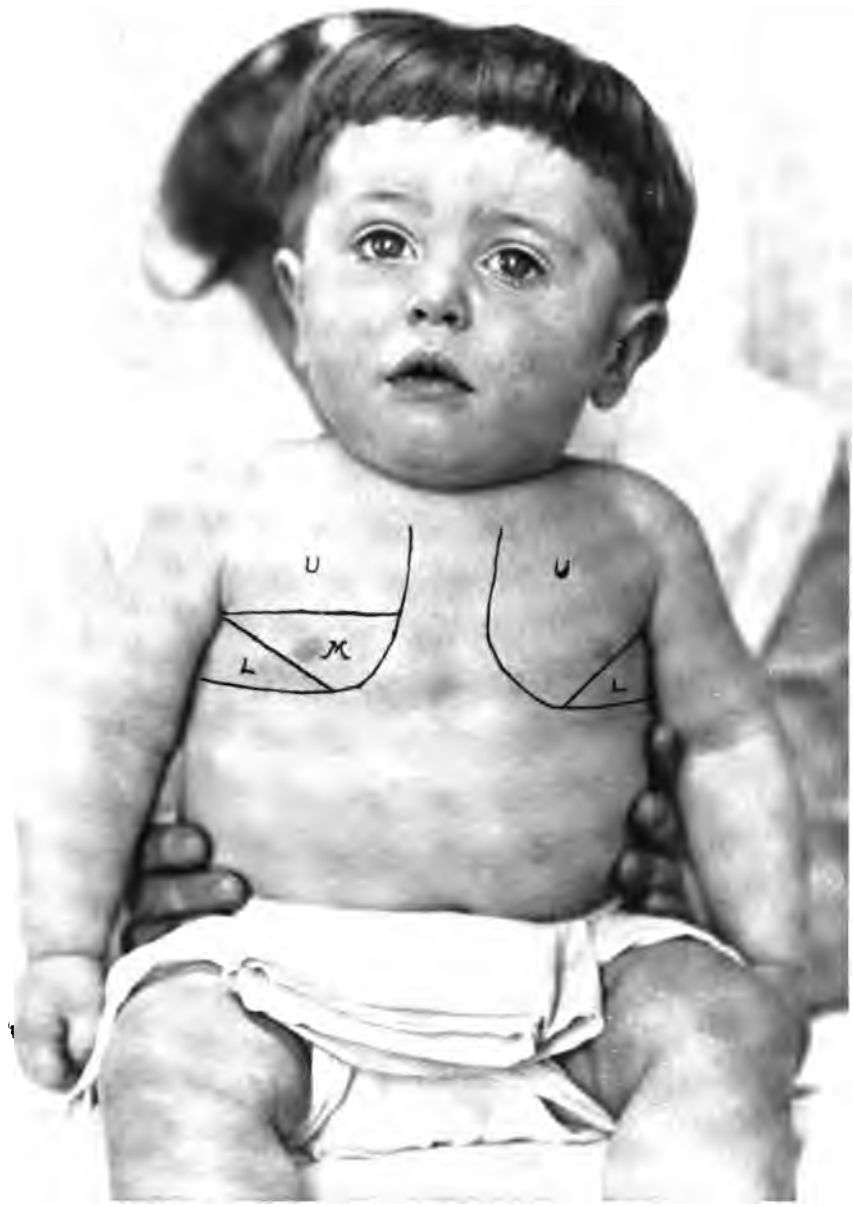
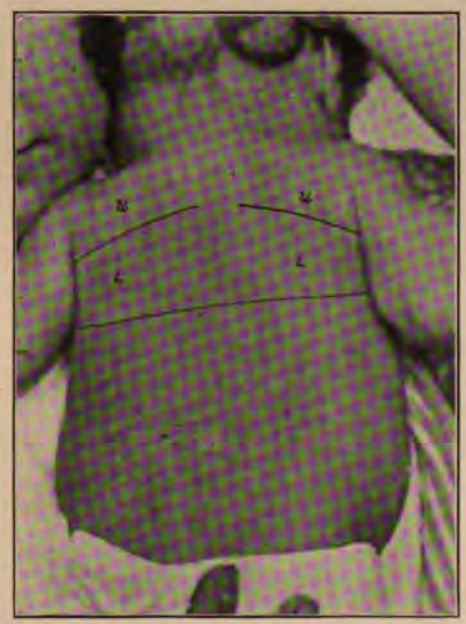


FIG. 23—Boundaries of the lobes of the lungs from in front

and childhood as in adult life. The lines between the upper and lower lobes start from the vertebrae at the level of the spines of the scapulae, pass through the mid-axillary lines at the level of the fourth rib, and reach the borders of the lung at the sixth rib, in the nipple line on the left, in the parasternal line on the right. The boundary between the upper and middle lobes starts on the right at the fourth costal cartilage, runs into the axilla, where it is close above the lines separating the upper and lower lobes, and reaches this line at the outer border of the scapula.

FIG. 24



Boundaries of the lobes of the lungs from behind

The principal points to be noted are relative dulness, flatness, hyperresonance, and tympany. It is useful to remember the following points characteristic of infancy and childhood: 1. The percussion note is normally more resonant than in adults, except in a crying infant 2. The note at the left base is more tympanitic because of the proximity of the stomach, and this tympany may be very marked when the stomach is distended with gas. 3 The percussion note is not higher pitched over the right apex, as in adult life. 4. Up to about ten years of age, there is slight dulness under the inner third of the left clavicle; this is very difficult to elicit in infancy

It is very important in early life to distinguish between dulness and flatness on percussion, and this is one of the most important differentiating signs between pulmonary consolidation, and pleuritic

effusion. Light percussion must be used, because the layer of fluid in children is very thin, and heavy percussion will obtain some pulmonary resonance, even though fluid be present.

More important often than the *sound* obtained by percussion, is the *sense of resistance*. This can also be elicited by tapping directly on the chest wall with the tips of the fingers, and this method of examination should not be neglected. It is particularly valuable in infancy, at which age an increased sense of resistance is an important diagnostic sign of fluid in the pleural cavity.

FIG. 25

Percussion of the back of the chest

AUSCULTATION.—The features to be noted on auscultation in children as in adults are, the character of the respiratory murmur, the vocal resonance, and the presence or absence of adventitious sounds The standard by which abnormality is recognized is, however, so different in infancy and childhood from that of adult life, that there is no part of the physical examination which so greatly requires the special knowledge and training of the pediatrist, as the auscultation of the lungs. The peculiarities are most marked in infancy, and grow progressively less marked throughout childhood. In children approaching the age of puberty, the adult standard has been reached. The normal peculiarities encountered in the auscultation of infants and children, are due to peculiarities of anatomical

development. The principal causes are, the shallowness of respiration in early life, and the greater proportion of bronchial air space to alveolar air space in the lungs.

The respiratory murmur in infants and young children is higher pitched and harsher than in the adult. If pitch be taken as one of the criteria by which bronchial respiration is recognized, the normal respiratory sound of the young child approaches the bronchial. But the criteria used in recognizing bronchial respiration in the adult,

FIG. 26

Percussion of the apex of the lungs

should not be used in childhood, and should be entirely banished from the mind of the physician when he is examining a sick child. Unless he does this, the harsh, high pitched respiratory murmur of early life, which is called *puerile*, will often be mistaken for bronchial respiration. The recognition of bronchial respiration in children depends in no way upon loudness, harshness, or pitch. It differs from vesicular respiration only in the character of the sound, and in the greater length of expiration. Unfortunately, the latter feature cannot always be recognized. The expiration is frequently replaced by the cry; sometimes in young infants it cannot be heard because the child is breathing very quietly, and when an attempt is made to cause it to draw a longer breath, it begins to cry, and only inspiration is audible. Prolonged expiration is a valuable sign of bronchial

breathing when found, but it is so often not found, that the physician must accustom himself to recognizing bronchial breathing mainly by the character of the sound which is heard on inspiration as well as on expiration. The character of the sound in the bronchial breathing of childhood must be learned mainly by experience. It is difficult to describe. It is high pitched, but so is puerile breathing. It is decidedly not harsh as compared with normal puerile breathing. It can best be described as having a peculiar nasal quality, and as sounding close to the ear as if it came from some point intermediate between the ears of the physician and the bell of the stethoscope. It should be remembered that when the character of the respiration is the same on both sides of the chest, front and back, it cannot be bronchial. In case of doubt, the physician should listen to the respiratory murmur over the second dorsal spine of the vertebrae. In this situation, the breathing is normally purely bronchial, and gives a good standard for comparison as to the quality of the sounds heard in other parts of the chest.

The term "increased" is sometimes applied to the respiratory murmur as heard in adults, the meaning being increase in the loudness, or intensity of the sound. This term should never be applied to the description of the respiratory murmur heard in childhood. There is no limit to the loudness or intensity of the sound normally heard. When there is a difference in the loudness of the respiratory murmur in the corresponding areas of the two sides of the chest, in the vast majority of cases in childhood, the louder side represents a normal condition, while the fainter side is evidence of a pathological condition. Occasionally, especially in older children, the bronchial breathing heard over the consolidation is louder than the normal breathing heard on the other side. In such a case the breathing might be called relatively increased, but its bronchial character is always plainly apparent. A relative increase in loudness alone, should never be considered abnormal. It is not uncommon for physicians or students unaccustomed to the examination of children, to diagnose lobar pneumonia on the wrong side. The breathing over a consolidation in children is frequently so diminished in loudness that its bronchial character is not recognized, especially if the child is breathing quietly; and the loud, harsh, puerile breathing on the normal side is mistaken for bronchial breathing. Whenever the respiratory sound in infancy is so feeble that its character cannot be determined, the baby must be made to cry and thus take a long breath. The baby should never be examined when lying on its side, because in this position the compression of the chest will lead to a diminution of the respiratory murmur on the lower side.

It should be remembered that bronchial respiration is normally heard over a greater area at the root of the lungs in infancy and early child-

hood than in later childhood and adult life. The slightly prolonged expiration heard at the right apex in adults is not found in children.

THE VOCAL RESONANCE, like the tactile fremitus, is not so valuable as diagnostic evidence in childhood as it is in adult life. Children cannot usually be made to say "one, two, three," or "ninety-nine," as can adults, and vocal resonance can only be obtained when they are crying. The child's cry is so loud, and the chest walls are so thin, that the sound heard on auscultation is often too overpowering for an estimate of its character and intensity. A pronounced degree of bronchophany can usually be heard, even when the child is crying. Diminution in the vocal resonance is not only more difficult to detect in young children, but is less constant as a sign of pleuritic effusion, and is often present over consolidation when there is no fluid. In older children the value of the vocal resonance approaches that of adults. In some cases, however, even in infants, a change in the voice sounds on one side can be recognized before any change in the character of the respiration.

RALES have the same character, varieties, and significance in children as in adults, and require no detailed description. They are often louder in infancy than in adults. One error frequently made in the examination of the lungs of infants and young children, is the mistaking of rales originating in the nose or naso-pharynx for true bronchial rales. The former are frequently transmitted to the chest, but should easily be distinguished from rales coming from the lungs. Bronchial rales are never exactly alike over both lungs, and are not heard over the trachea or cheeks, whereas rales made in the upper air passages have the same sound over both lungs, the trachea, and the cheeks.

PLEURAL FRICTION SOUNDS are very rarely heard in infancy, although pleurisy is not uncommon at this age. The reason for this is unknown. In childhood they have the same significance as in adults, but in my experience, are somewhat less commonly heard.

THE ABDOMEN. INSPECTION.—The abdomen should be inspected with reference to size, prominence, retraction, shape, irregularities of outline, tension of the wall, the condition of the umbilicus, herniae, superficial veins, respiratory movements, epigastric pulsation, and visible peristalsis. The normal appearance of the abdomen at the various ages has been described in the section on Normal Development.

Enlargement of the abdomen is very common in infancy. The most common cause is various disturbances of digestion and nutrition, which act in two ways in producing abdominal enlargement. Disturbed digestion may produce an increased formation of gas in the intestine. Enlargement of the abdomen from this cause is seen

also in older children in chronic indigestion from an excess of carbohydrate, the abdomen being enlarged chiefly in the upper portion. In disturbances of nutrition the walls of the intestines suffer with the rest of the tissues and become relaxed, so that even without increased formation of gas there may be increased accumulation of gas. The enlargement of the abdomen seen in rickets and in most chronic digestive disturbances, is produced in this latter way.

In older children enlargement of the abdomen is suggestive of some more serious condition, and the possibility of the same conditions in infancy must not be forgotten. Among these causes are ascites, tuberculous peritonitis, enlargement of the liver or spleen, sarcoma of the kidney, and congenital dilatation of the colon. Irregularities in outline are suggestive of enlargement of one of the solid viscera, or sarcoma of the kidney. A prominence in the lower portion in an infant may be due to a distended bladder.

Retraction of the abdomen is usually due to lack of intestinal contents, both gaseous and fluid. It is often seen in acute diarrheas, in meningitis, and in conditions characterized by severe vomiting, such as acidosis. It is never due in children to hysteria or lead poisoning.

Distention of the abdomen is due to the same causes as enlargement, representing only a greater degree.

Inspection of the umbilicus is particularly important in the newborn. Umbilical herniae are easily recognized. Epigastric herniae are not very rare in infants and young children.

Visible peristalsis is a very important diagnostic sign in infancy. When over the epigastrium it is suggestive of pyloric stenosis. Over other parts of the abdomen, it is suggestive of tuberculosis or intestina obstruction.

PALPATION.—In the general palpation of the abdomen, the points to be noted are the tension and resistance of the walls, and the presence or absence of spasm of the muscles, tenderness, fluctuation, and abnormal solid bodies. Spasm and resistance must be localized, and it must be determined whether they are voluntary or involuntary. If tenderness be present, its seat must be noted, and whether it is superficial or deep. Abdominal tenderness is a less common sign in childhood than in adult life, and is not often seen in disturbances of the stomach or intestine. It should always suggest the possibility of some serious condition, such as appendicitis, peritonitis, or intussusception. A sense of fluctuation is often obtained in infants and young children due to the liquid character of the intestinal contents, which may be mistaken for ascites. The fluid wave of ascites is obtained as in the adult, and has the same diagnostic significance. It is often difficult to tell whether masses felt in the abdomen are fecal accumulations, or the masses of tuberculous peritonitis, and it

is sometimes necessary to repeat the examination after the bowels have been emptied, in order to clear up this point.

PERCUSSION of the abdomen is employed mainly to determine the presence of fluid, to determine the character of masses felt, and to map out the outlines of the abdominal organs. The sign of free fluid is dulness in the flanks, shifting with change of position. Care must be taken in interpreting this sign, as in infants and young children the liquid feces tend to gravitate to the flanks, and will give a dulness which shifts with change of position. Very marked shifting dulness, or a fluid wave is necessary for a conclusive diagnosis of ascites.

THE STOMACH.—Percussion of the stomach is difficult in infancy and childhood, and the results obtained are unreliable. A dilated stomach, when distended with gas, can sometimes be recognized both by inspection and percussion. Visible peristalsis is an important sign of pyloric stenosis.

THE LIVER is investigated by palpation and percussion. The upper border must be determined by percussion. The lower border in infants and young children is best determined by palpation, as percussion is unreliable on account of the thinness of the edge. The upper border of the liver flatness is at the fifth rib in the right nipple line in infancy. From this point it gradually descends, the adult position being reached at about six years. The lower border in infancy may normally extend 3 cm. (1-1/8 in.) below the costal border in the mammillary line, and 6 cm. (2-1/4 in.) below the tip of the ensiform. It is palpable in the mammillary line up to three years, and may sometimes be felt after this time in children with relaxed abdominal walls if the fingers are pushed up under the costal border. The liver in early childhood is easy to feel on account of the thinness of the abdominal wall. It is, however, very often missed, because the physician, not realizing how soft and thin is the edge, and how superficially it lies, palpates too deeply and forcibly.

The liver is examined for enlargement, tenderness, and irregularity of outline. Examination of the gall-bladder is unsatisfactory in early life, but is hardly necessary, as this organ is very rarely diseased in childhood.

THE SPLEEN.—Percussion of the spleen is so difficult in early life, on account of the small size of the organ, that it is hardly worth while to undertake it. The outline may be determined in older children, but palpation of the spleen is so easy in infancy and childhood that it is better to trust to this method of examination.

The spleen should be palpated with the physician on the *right* side of the patient. Many text-books recommend palpation of the

spleen from the left side, with the tips of the curved fingers. In my experience palpation from the right is infinitely better. The right hand should be placed almost flat upon the abdomen, with the fingers almost straight. In the child the abdomen wall is so thin, and the spleen is so superficial, that care must be taken not to palpate too deeply, for the spleen may thus be pushed down with the abdominal wall without being detected. The extreme tips of the fingers should be placed against the costal border, and then are lightly and quickly depressed. By repeating this operation, drawing the hand forward and back, the spleen will not be missed if palpable. The proper position for the hand is shown in the illustration.

The spleen is normally not palpable at any age, except that in infants with lax abdominal walls, the normal spleen can sometimes be felt if the fingers are pushed up under the costal border. Whenever the spleen is palpable below the costal border, it is safe to conclude that it is enlarged. When there is marked splenic enlargement, forming a large mass on the left side of the abdomen, it is easier in the child than in the adult to recognize the mass as spleen, on account of its very superficial position, and flatness on percussion; moreover, the notch can usually be clearly felt.

THE KIDNEYS.—The outline of the normal kidneys cannot be percussed either in infancy or in childhood. The normal kidney can only rarely be palpated even when the abdominal walls are very thin and lax. If the kidney is palpable, it can be concluded that it is enlarged or displaced. Floating kidneys are very rare in infancy and childhood, and if present, usually represent a congenital abnormality Palpable masses in the region of the kidney usually represent tumors, the most common being sarcoma. Congenital cystic kidney, and pyelo-nephrosis are seen at times. Tumors of the kidney are recognized by the fact that they do not move with respiration, and usually show some tympany on percussion, as the colon lies in front of them.

Tenderness on deep palpation in the region of the kidney is sometimes seen in pyelitis. It should suggest either pyelitis or perinephritic inflammation.

THE BLADDER.—In infancy the small oblique pelvis is almost wholly filled by the rectum, and consequently almost all of the bladder lies in the abdominal cavity above the crest of the pubes. Distention of the bladder forms a rounded tumor in the lower part of the abdomen, which lies close to the abdominal wall, and may even reach to the umbilicus. Most of the anterior surface is uncovered by peritoneum. After the child gets upon its feet, the weight of the urine and the anatomical changes in the shape of the

pelvis gradually cause the bladder to assume the adult position, which is reached in middle childhood.

This high position of the bladder in infancy and early childhood has caused many bad mistakes in diagnosis. A distended bladder has often been mistaken for ascites, or a new growth. Whenever there is any question of these conditions, the bladder should always be emptied by catheterization.

In connection with the examination of the bladder, the groins should be carefully examined. Abnormalities to be looked for are hernia, hydrocele of the cord, undescended testicles or misplaced ovary, and enlargement of the lymph nodes.

THE EXTERNAL GENITALS.—Inspection of the external genitals should always form a part of the physical examination, particularly in females, and in the newborn. The principal abnormalities to be looked for are malformations and discharge. The commonest malformation in females is adhesions of the nymphae. The prepuce is normally adherent to the clitoris throughout infancy and early childhood. In males the physician should look for phimosis, undescended testicles, and hydrocele. In infants the glans penis is normally covered by the prepuce, which is adherent. In phimosis the prepuce is so narrowed that it cannot be retracted. The testicles may be wholly or partially undescended. Tumors of the testes, varicocele, balanitis, ulcerations, and urethral discharge (in the male), are rare in children. In the female, vulvo-vaginitis is fairly common. It is often of gonorrhoeal origin, but may be due to uncleanliness or some other source of irritation. Whenever there is the least sign of discharge a bacteriological examination should be made.

THE ANUS.—The buttocks should always be retracted and the anus inspected, particularly in infants. The condition of the skin about the anus should be noted. Fissures of the anus are not uncommon in infancy. Hemorrhoids are uncommon in childhood as compared with adult life. Prolapse of the rectum, on the other hand, is common. Ulcers, condylomata, and mucous patches are often found about the anus in syphilitic infants. Fistula, ischiorectal abscess, and pilonidal sinus are seen at times.

THE R[illegible] l examination is not necessary unless there [illegible]ry suggesting its advisability. Whenever [illegible] intestinal obstruction, intussusception, or [illegible] the abdominal cavity, a rectal examina-

[illegible]amining the extremities the physi- [illegible]ts in mind: *Relative size, position,* [illegible]*es, the joints, spasm, and paralysis.*

RELATIVE SIZE.—The limbs are normally alike in circumference on the two sides. A difference is more apt to be due to atrophy on one side than to hypertrophy on the other. If any apparent difference be present, it should be confirmed by measurement. Care must be taken that measurements are made in exactly the same place. Differences of less than 1 cm. are within the limits of error in measurement, and should be disregarded.

It is also important to determine whether the legs are of equal length. This can only be determined with the child lying on its back. A difference may be an actual difference in length, or due to congenital dislocation of the hip. The latter is recognized by the high position of the trochanter.

POSITION.—Abnormal positions of the extremities are usually due either to temporary muscular spasm or to permanent muscular contractures. The details of the examination for spasm will be described under the examination of the nervous system.

A limb is sometimes held in an abnormal position without spasm, the cause being some painful lesion which is more comfortable when the limb is held in this way. The most common cause is arthritis.

DEFORMITIES.—Deformities of the extremities are either congenital or acquired. The former variety includes such conditions as club-foot, club-fingers, web-fingers, congenital dislocation of the hip, and a variety of rare gross malformations.

The most common acquired deformities are due to rickets, and are enumerated in detail under the description of that disease. There is a sabre-like deformity of the tibiae which is especially characteristic of late syphilis in childhood. It is often mistaken for a rachitic deformity, but occurs later in childhood. The limbs are also deformed in cretinism and chondrodystrophy fetalis.

Contractures from disease of the nervous system may cause a condition which is more than an abnormal position of the limbs, and amounts to actual deformity.

THE LONG BONES are examined for tenderness, swelling, and fractures. The most common cause in infancy of tenderness and swelling over the shafts of the long bones is scurvy; periostitis, and osteomyelitis stand next. In older children, scurvy is not seen, and the most common cause is periostitis. Tumors, chiefly osteosarcoma, are seen at times. The fingers should be examined for tuberculous dactylitis.

THE JOINTS.—The physician should note the contour of the joints for swelling, should palpate them for tenderness and swelling, and should manipulate them for pain on motion and limitation of motion. It is useful to remember that in infancy acute inflammation of the joints usually represents a septic arthritis, while in older children,

FIG. 28—Examination of the flexibility of the spine

it usually means rheumatic fever. Chronic inflammation of the joints at any period of early life is most commonly due to tuberculosis. A traumatic chronic arthritis is not uncommon in children. The chronic "rheumatoid" conditions, osteo-arthritis, and so forth, so often seen in adults, are rare in childhood.

THE SPINE.—The spine is examined best with the patient lying on its face on a flat surface. The points to be noted in connection with the spine are *curvatures*, *mobility*, *tender points*, and *spina bifida*. The peculiarities of spinal curvature normally characteristic of early life have been described in the section on Normal Development. Curvatures due to disease will be apparent with the patient lying on its face A kyphosis is most often due to tuberculosis, while a backward curve in children is always suggestive of meningeal irritation. The lateral curvatures are common in childhood.

The flexibility of the spine is tested by grasping the heels of the infant with one hand, while the other hand holds down the shoulders. The heels are lifted up, and swung in various directions, as shown in the illustration.

THE NERVOUS SYSTEM.—Some of the examinations made in obtaining evidence as to disturbance of the nervous system have already been described. It is, however, useful to consider the physical signs of disease of the nervous system together, although the various steps in the investigation are more conveniently made in connection with other parts of the physical examination.

THE MENTAL CONDITION is estimated during the general examination of the body which precedes the detailed examination of the various organs and systems. It is often advisable, when there is any abnormality in the apparent mental attitude of the child, to make a more detailed investigation of the mental condition. This cannot always be accomplished at a single examination, and requires more or less prolonged observation. Apathy, stupor, or delirium will usually be plainly apparent before the examination is finished. Also, pronounced cases of mental impairment, the severer grades of idiocy and imbecility, will not escape notice. It is the milder types of imbecility and backwardness that are not always apparent on a single examination. Much depends upon the physician's powers of observation, and upon a very thorough familiarity with the behavior of a normal child at every stage of development.

PARALYSIS AND SPASM.—These signs are most conveniently investigated during the examination of the extremities.

Paralysis is often difficult to detect in young children. Older children, who can cooperate with the physician, will attempt to make various movements as directed, and in them the recognition of paralysis is easy enough. Infants and young children will not do this. The

motor function of the arms can be tested by offering them things to play with, or showing them something which they particularly like, such as the bottle or a favorite toy. The power of the legs is tested by tickling the feet, or pricking them with a pin. Sometimes it works well to place a limb in an abnormal strained position, and note if the child moves it back. When a child is unconscious, the only way in which paralysis can be detected is by lifting a limb and then letting it fall, noting the manner in which it drops. A completely paralyzed limb will drop like a dead weight, while if there be no paralysis, the limb will drop neither so promptly nor so lifelessly. Further evidence may be obtained by manipulating the limb and noting the amount of resistance to passive motion. Flaccid paralysis usually shows a notable difference.

It is very important in infancy to distinguish true paralysis from the pseudo-paralysis which comes from failure to use the extremities on account of pain. Evidence of pain can usually be found in the latter condition, and the two conditions can usually be distinguished without causing too much suffering.

Spasm is also often difficult to detect in infants. In young infants, and in all infants who are notably emaciated, there is a hypertonicity of the muscles which must not be mistaken for contractures of nervous origin. This hypertonicity is most marked in the flexor group of muscles, and often prevents complete extension of the limbs. When true contractures are present, there is usually a difference in the amount of resistance to passive motion on the two sides.

The test for spasm is made by passive motion of the limbs at the various joints. It is often difficult to distinguish voluntary resistance from temporary spasm. The former can usually be overcome by rapid repetitions of the same movement, while spasm cannot thus be overcome. When temporary spasm is present, it is important to note the position assumed by the extremities. In early life, spasm is most often due either to disease of the central nervous system, or to hyperirritability of the peripheral nerves (Spasmophilia).

THE REFLEXES.—The reflexes to be tested in children as a routine are the pupillary reaction, the knee jerks, and the plantar reflex, and certain special signs. The abdominal reflex is inconstant in childhood, and the cremasteric reflex is not of much diagnostic significance. The method of obtaining the pupillary reaction has been described under the examination of the eye.

The knee-jerk is often hard to elicit in an infant. The leg must be relaxed, and great patience is often necessary before a satisfactory test can be made. Too much stress must not be laid upon an absent knee-jerk in an infant, and it is wisest to disregard it as evidence unless there are some other signs of disease of the nervous system.

An exaggerated knee-jerk has its full diagnostic significance. The best method of obtaining the knee-jerk in an infant is to place the hand under the lower part of the thigh and lift the knee from the bed or table. The ligamentum patellae is tapped, the foot still resting on the bed, the angle of the leg being varied by moving the knee up and down. Then, if no response is obtained, the knee is lifted so that the foot hangs clear of the bed, and the tapping is repeated. In older children the knee-jerk is best tested with the child in a sitting posture, and the leg hanging free.

FIG. 29

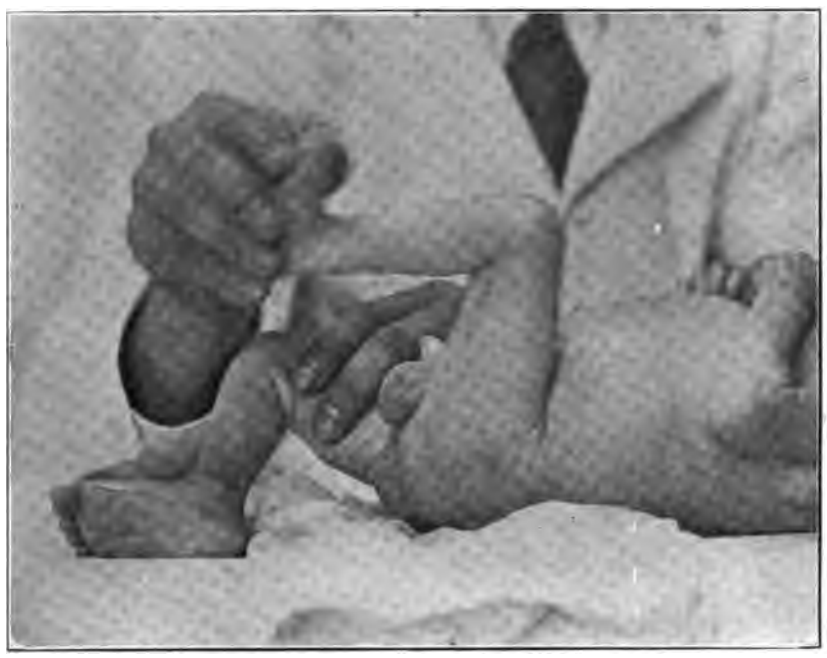
Testing the knee jerk

The plantar reflex is tested in the ordinary manner. In infancy its presence is more often shown by extension than by flexion of the toes. *Babinski's sign* is a simultaneous extension of the big toe and flexion of the other toes, and in adults and older children it points toward disease of the higher motor tracts. It has no diagnostic significance in infancy.

SPECIAL SIGNS —There are certain special signs of great importance in the investigation of the nervous system in early life. These are Kernig's sign, Brudzinski's sign, ankle clonus, the contralateral reflex, Chvostek's sign, Trousseau's sign, and the peroneal reflex.

KERNIG'S SIGN consists in a limitation of the extension of the leg upon the thigh when the thigh is at a right angle with the body. Under normal circumstances, when the thigh is at a right angle with the body, the leg can be extended to an angle of 135° at the knee, at least, and in infants often to a greater angle. Kernig's sign is obtained when the leg cannot be extended to an angle of 135°. The sign can be tested by placing the thigh at a right angle to the body and attempting to extend the leg, or by holding the leg straight at

FIG. 30



Examination for Kernig's sign

the knee and bringing the thigh to a right angle with the body, noting how much flexion this causes at the knee. The former method is perhaps a little the better. In very young, or much emaciated infants, the muscular hypertonicity must be taken into account.

A positive Kernig's sign is almost constant in all forms of meningitis except the tuberculous, in which it may or may not be present. It is often positive in conditions other than meningitis, but usually means meningeal irritation of some kind.

BRUDZINSKI'S NECK SIGN.—This consists in a movement of the legs when the neck is flexed forward. Under normal conditions forward flexion of the neck causes no movement of the legs. To test the neck sign, the child must be lying on its back. The physician

holds the chest stationary with one hand, and brings the head sharply forward with the other. When the sign is positive, this will cause a flexion of the legs both at the hips and at the knees, or at the hips alone. The movement is sometimes present on one side only. The sign when positive has been regarded by some writers as diagnostic of meningitis. It is certainly seen mainly in meningitis, but is not present in all cases of that disease, and I have observed its presence repeatedly in conditions of meningeal irritation which were proved not to be meningitis.

FIG. 31

Examination for Brudzinski's neck sign

ANKLE CLONUS.—This sign is elicited by lifting the leg a few inches from the table, holding it straight at the knee and making sharp dorsal flexion of the foot upon the leg, the foot being held by the toes. Ankle clonus is present if this causes a rhythmical jerking of the foot back and forth at the ankle. The sign is seen in a great variety of lesions of the central nervous system, affecting the upper motor segment. It usually accompanies spastic paralysis. It is one of the few signs which may be present in meningitis, but which I have never observed in meningismus.

THE CONTRALATERAL REFLEX is present when passive flexion of one leg causes a reflex movement of the other leg. The reflex move-

llow the light. Another way is to approach the hand
yes, noting whether the child winks. Care must be
w a wrong conclusion from winking caused by the
duced by the movement of the hand. Hearing in
tested by making a sudden noise near the ears, and
child jumps. The test is not always satisfactory
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METHODS OF EXAMINATION

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in children for purposes of diagnosis, but which
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familiar with the technique of these various

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ubes should be boiled also. The hands
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n. Tincture of iodine is useful to dis-
nbar spines where the puncture is to be
the proper holding of the patient. An
e of the patient places one hand under
ces the other arm about the patient's
flexed by drawing up the knees with
oulders forward. Pressure should be
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hetic is unnecessary in the majority
when muscular spasm prevents proper
cases in older children the site of the
nsitive with cocaine or ethyl chloride.

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In either case, the general direction is horizontally
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e than 5 c. c. should be withdrawn. If there
pressure, as shown by spurting of the fluid,
e continued until the fluid drops at a normal

properly performed, there should be no dif-
in the great majority of cases. Care should
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l canal, as in such a case the fluid will prob-
nd if the needle enters the tissues in the
ll flow. On the other hand, care must be
ushed far enough to enter the canal. If
fore entering the canal, it is a sign either
edle is not right, or that the spine is not
the needle to pass between the bones.
le must be entered again with corrected
smaller needle must be used, or increased
btained.
tain fluid may be due to several causes.
needle has become plugged in passing
Physician should clear the needle by
be also that through some fault of
tomical condition, the needle has not
such a case the puncture should be
ue. If fluid still fails to appear, the
below the one first chosen, should
hen the operator is sure that the
dry tap occasionally occurs; this is
Or pathological cause, which prevents
hing the lumbar portion of the canal,

or to the fact that the fluid contains fibrin or thick pus, and is thus unable to pass through the needle.

After the withdrawal of the needle, the puncture is covered with a little piece of sterile absorbent cotton held in place by collodion.

EXPLORATORY THORACENTESIS.—This is a most important procedure in the diagnosis of pleural effusion. The point chosen for the puncture should be that which shows the greatest flatness on percussion. When the flatness is extensive, involving the whole lower portion of the pulmonary area, the point of selection should be in the posterior axillary line, at the sixth interspace on the left, and at the fifth interspace on the right. Fluid should always be obtained at these points when the pleural cavity contains free fluid; but in cases of encapsulated fluid, the point for the puncture must be determined by physical examination. The needle used in making the puncture should be about one millimeter in diameter. The needle and syringe should be boiled.

The child is held firmly in a sitting position, the hand on the side to be punctured being brought up over the opposite shoulder, and the skin is rendered surgically clean. The needle, attached to the syringe is introduced between the ribs, nearer to the upper than the lower border of the rib, in order to avoid injuring the intercostal artery which runs along the lower border. The distance which the needle enters is from 1 to 2 cm. The entrance into the pleural cavity can usually be detected by a sudden diminution in the resistance. The piston of the syringe is then gently drawn, and if fluid be reached it will enter the syringe. If fluid is not obtained, it should not be sought by moving about the point of the needle, but the needle should be withdrawn, and after a second careful physical examination, it should be introduced in another place. After withdrawing the needle upon failure to obtain fluid at any point, it is well to make sure that the lumen of the needle is clear. The wound left by the needle may be covered by a bit of sterile cotton and collodion. In seeking for encapsulated fluid, almost any part of the chest may be safely entered, but it is well to avoid the region near the heart. Performed in this way, exploratory puncture is practically without danger, even if the needle enters the lung.

EXPLORATORY PUNCTURE OF THE PERITONEAL CAVITY.—This procedure is very rarely employed. The diagnosis of ascites should depend upon the results of physical examination, and exploratory puncture is not a safe procedure to determine the presence or absence of fluid. It is only made in cases in which the diagnosis of ascites is clear, for the purpose of obtaining fluid for bacteriological and cyto-diagnostic examination. The syringe, needle, and preparations are the same as for thoracentesis. It is essential that

rst emptied by catheterization — The child should
ture, and the needle is introduced half-way between
umbilicus.

Y PUNCTURE OF THE PERICARDIAL CAV-
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be made to obtain fluid for examination. The
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therapeutic measure. The technique of this
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OF GASTRIC CONTENTS AND RETEN-
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18 French scale. This is connected by a
a rubber tube attached to the nozzle of a
te nozzle of the glass bulb, another piece
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as given. After two or three hours, the
In withdrawing the gastric contents, the
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ODENAL CATHETER.—The tech-
ribed under the heading of Spasm of

ICAL REACTION.——This requires
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ose. The essentials are, an appli-
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be an appliance for measuring the
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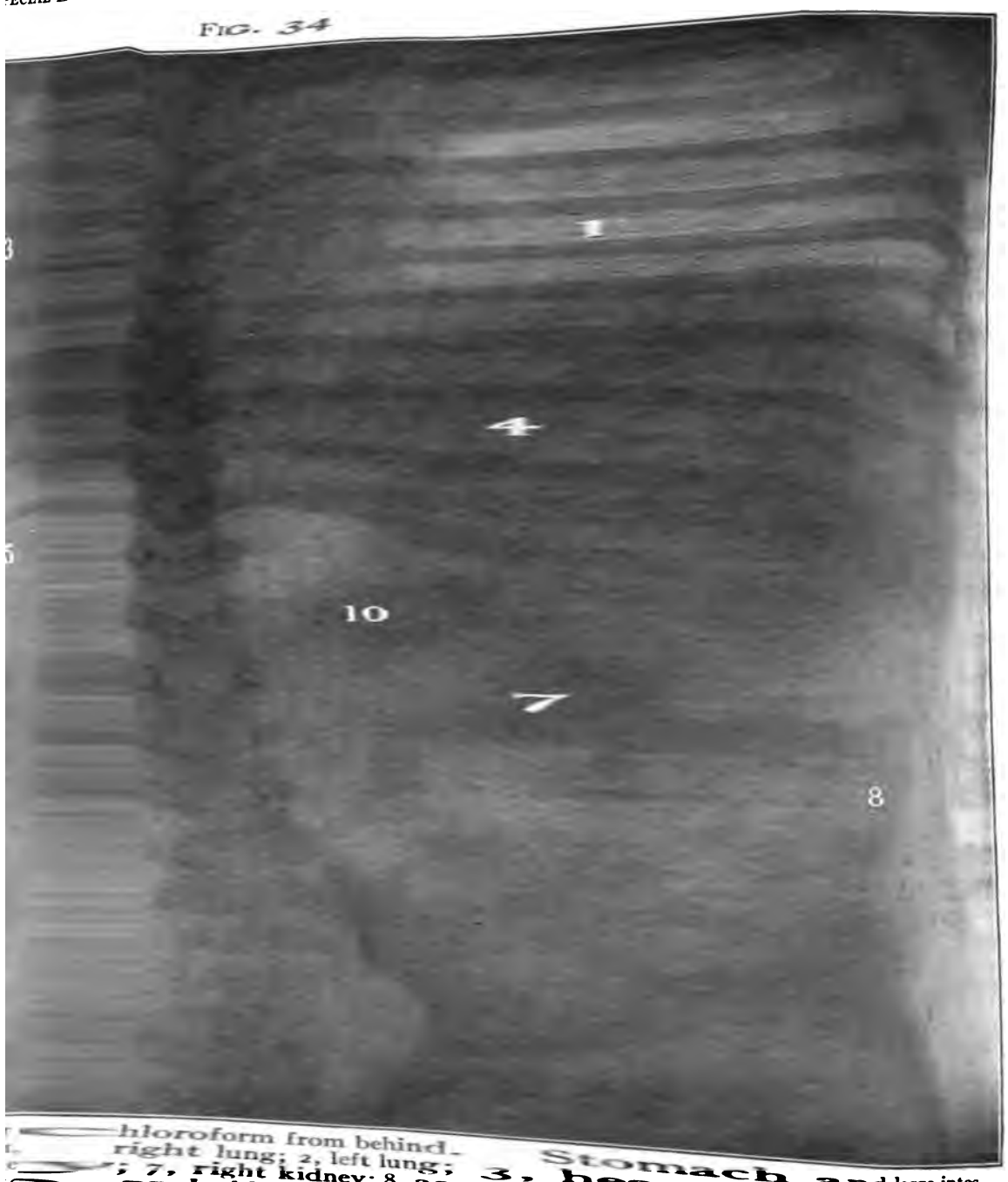
front of the chest or any portion of
g electrode is applied to the nerve

FIG. 34



Chloroform from behind. Stomach and large intes-
right lung; 2, left lung; 3, heart; 4, liver;
, 7, right kidney; 8, ascending colon;
probably the head of the pancreas

ment may be identical (flexion), or reciprocal (extension). It is one of the signs of disease of the central nervous system.

CHVOSTEK'S SIGN is tested by tapping upon the facia nerve or its branches where they cross the jaw. Under normal conditions, no contraction of the facial muscles is caused by this procedure, but when the sign is positive there is a contraction which involves either the muscles supplied by the whole nerve, or those supplied by one of its branches. The contraction is observed either about the mouth, about the eye, or about both. It is a sharp, quick contraction which cannot be mistaken for voluntary movement. The sign cannot be obtained when the child is crying. It is sometimes called the facial phenomenon, and the contraction about the eye, caused by tapping the upper branch of the facial nerve is sometimes called Weiss' sign. Chvostek's sign when positive is diagnostic of spasmophilia.

TROUSSEAU'S SIGN.—This is obtained by placing a constriction band about the upper arm near the fork of the biceps. The continued pressure on the nerve trunks normally causes no reaction, but in spasmophilia this often causes the hand to assume the typical spasm of tetany. The sign when positive is diagnostic of spasmophilia, but its testing is painful, and the diagnosis can usually be made from other signs. It should never be tried in a case of spasmophilia characterized by laryngospasmus.

THE PERONEAL REFLEX is obtained by tapping upon the peroneal nerve near the head of the fibula. In eliciting this reflex, a percussion hammer is better than the finger. The sign is obtained when the tapping causes a sharp reflex movement of the foot. A positive peroneal reflex is one of the diagnostic signs of spasmophilia.

SENSATION.—In older children who are able to answer questions intelligently, sensation may be tested for touch, pain, and temperature, as in adults. Testing the temperature sense is however usually unnecessary in children. In young children who are unable to cooperate with the physician, only the sensation of pain can be satisfactorily tested. This is performed by touching the skin with a sharp point and noting whether the child's expression shows pain, or whether it moves the limb out of the way.

SPECIAL SENSES.—For purposes of diagnosis the special senses to be tested are those of sight and hearing. In older children who are able to answer questions intelligently, the tests are made as in adults. In young children, sight is tested by showing the child some object in which it is specially interested, such as the bottle, or a favorite toy. This object is moved from side to side, the physician noting where the patient's eyes follow. In very young babies not old enough to recognize objects, the sense of sight is tested by moving a bright light back and forth in front of the eyes and noticing whether the movements

of the eyeball follow the light. Another way is to approach the hand rapidly to the eyes, noting whether the child winks. Care must be taken not to draw a wrong conclusion from winking caused by the current of air produced by the movement of the hand. Hearing in young children is tested by making a sudden noise near the ears, and noting whether the child jumps. The test is not always satisfactory in very young babies.

SPECIAL METHODS OF EXAMINATION

There are certain special methods of examination which frequently have to be performed in children for purposes of diagnosis, but which do not form a part of ordinary routine examination. It is essential that the physician be familiar with the technique of these various operations.

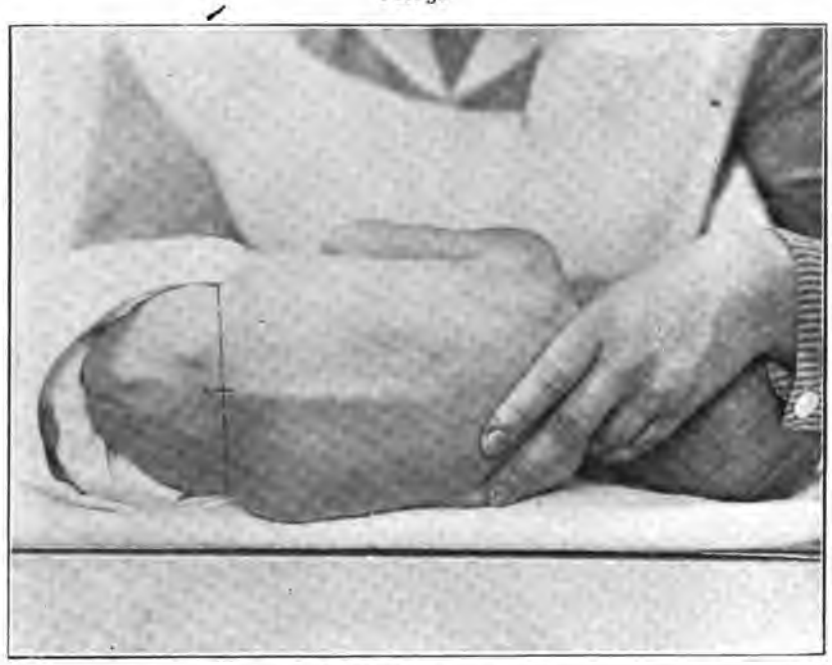
LUMBAR PUNCTURE.—This is one of the most important diagnostic procedures used in infancy and childhood. It is much more frequently employed in children than in adults, because not only meningitis but many conditions simulating meningitis are particularly common in early life. There are also other conditions more common in early life in which lumbar puncture is used, such as hydrocephalus and poliomyeloencephalitis.

The technique of lumbar puncture is comparatively simple, but success depends upon attention to details. Needles are better than trocars for lumbar puncture. The size of the needle should be adapted to that of the child. For very young babies, an ordinary antitoxin needle should be used. For bigger babies and older children, the needle should be larger. Several needles and two test tubes should be boiled, and if the fluid has to be transported for examination, corks to fit the test tubes should be boiled also. The hands of the operator and the lumbar and sacral regions of the patient extending around as far as the highest point of the crest of the ilium, are rendered surgically clean. Tincture of iodine is useful to disinfect the point over the lumbar spines where the puncture is to be made. Much depends upon the proper holding of the patient. An attendant on the opposite side of the patient places one hand under the bend of the knees, and places the other arm about the patient's shoulders. The spine is then flexed by drawing up the knees with one hand and pressing the shoulders forward. Pressure should be made on the region of the shoulders rather than on the neck. It is important that the spine be flexed as much as possible without the use of undue force. An anesthetic is unnecessary in the majority of cases, but should be used when muscular spasm prevents proper flexion of the spine. In some cases in older children the site of the puncture may be rendered insensitive with cocaine or ethyl chloride.

In many children, however, this procedure appears to cause more discomfort and terror than the puncture itself.

The operator takes as a landmark a line drawn between the highest points of the crests of the ilium on each side. This is the upper limit of safety, and the spinal canal can be entered anywhere below this level without danger of piercing the cord. It is unnecessary to count and locate the lumbar spines by number. The operator should take as his first point of selection the second space below the line described above, which gives him a margin of one space above and below the point first chosen, in case fluid is not obtained at the first attempt. The spaces are located with the forefinger of the left hand. Some

FIG. 32



Position for lumbar puncture
The dotted line shows the upper limit of safety, below which the puncture must be made

operators insert the needle directly in the median line, to pass between the spines, others a little to one side of the median line, to pass between the laminae; in the latter case, the needle must be inclined slightly toward the median line, so that after insertion the point of the needle will be in the median line.

The advantage of the first point of insertion is that the proper direction is much easier to follow, and of the latter point that there is more room for the needle to pass between the laminae than between

the spines. I am accustomed to use the latter method, but believe that both are equally good, the choice depending upon the individual operator. In either case, the general direction is horizontally forward, and the needle is pressed in until the resistance opposing its progress is felt suddenly to diminish, when the fluid should immediately begin to drop or spurt from the outer end of the needle. The fluid is caught in one of the sterile test tubes.

The advantage of boiling two test tubes lies in the fact that part of the fluid will sometimes be blood-stained and part clear, and blood-stained fluid interferes somewhat with the diagnostic inferences to be drawn. If at any time the character of the dripping fluid changes from clear to blood-stained, or from blood-stained to clear, the second test tube should be substituted.

The amount of fluid to be withdrawn depends upon the purpose for which lumbar puncture is performed. If lumbar puncture is undertaken for diagnosis only, and there are no evidences of intradural pressure, not more than 5 c. c. should be withdrawn. If there is evidence of intradural pressure, as shown by spurting of the fluid, the withdrawal should be continued until the fluid drops at a normal rate.

If lumbar puncture is properly performed, there should be no difficulty in obtaining fluid in the great majority of cases. Care should be taken that the needle is not pushed in far enough to touch the anterior wall of the spinal canal, as in such a case the fluid will probably be blood-stained, and if the needle enters the tissues in the anterior wall, no fluid will flow. On the other hand, care must be taken that the needle is pushed far enough to enter the canal. If the needle strikes bone before entering the canal, it is a sign either that the direction of the needle is not right, or that the spine is not sufficiently flexed to permit the needle to pass between the bones. In the former case, the needle must be entered again with corrected direction; in the latter case, a smaller needle must be used, or increased flexion of the spine must be obtained.

A "dry tap," or failure to obtain fluid may be due to several causes. The most common is that the needle has become plugged in passing through the tissues, and the physician should clear the needle by passing in the stylet. It may be also that through some fault of technique, or some peculiar anatomical condition, the needle has not entered the spinal canal. In such a case the puncture should be repeated with corrected technique. If fluid still fails to appear, the intervertebral spaces above and below the one first chosen, should be successively tried. Even when the operator is sure that the needle has entered the canal, a dry tap occasionally occurs; this is due to some peculiar anatomical or pathological cause, which prevents the cerebrospinal fluid from reaching the lumbar portion of the canal,

or to the fact that the fluid contains fibrin or thick pus, and is thus unable to pass through the needle.

After the withdrawal of the needle, the puncture is covered with a little piece of sterile absorbent cotton held in place by collodion.

EXPLORATORY THORACENTESIS.—This is a most important procedure in the diagnosis of pleural effusion. The point chosen for the puncture should be that which shows the greatest flatness on percussion. When the flatness is extensive, involving the whole lower portion of the pulmonary area, the point of selection should be in the posterior axillary line, at the sixth interspace on the left, and at the fifth interspace on the right. Fluid should always be obtained at these points when the pleural cavity contains free fluid; but in cases of encapsulated fluid, the point for the puncture must be determined by physical examination. The needle used in making the puncture should be about one millimeter in diameter. The needle and syringe should be boiled.

The child is held firmly in a sitting position, the hand on the side to be punctured being brought up over the opposite shoulder, and the skin is rendered surgically clean. The needle, attached to the syringe is introduced between the ribs, nearer to the upper than the lower border of the rib, in order to avoid injuring the intercostal artery which runs along the lower border. The distance which the needle enters is from 1 to 2 cm. The entrance into the pleural cavity can usually be detected by a sudden diminution in the resistance. The piston of the syringe is then gently drawn, and if fluid be reached it will enter the syringe. If fluid is not obtained, it should not be sought by moving about the point of the needle, but the needle should be withdrawn, and after a second careful physical examination, it should be introduced in another place. After withdrawing the needle upon failure to obtain fluid at any point, it is well to make sure that the lumen of the needle is clear. The wound left by the needle may be covered by a bit of sterile cotton and collodion. In seeking for encapsulated fluid, almost any part of the chest may be safely entered, but it is well to avoid the region near the heart. Performed in this way, exploratory puncture is practically without danger, even if the needle enters the lung.

EXPLORATORY PUNCTURE OF THE PERITONEAL CAVITY.—This procedure is very rarely employed. The diagnosis of ascites should depend upon the results of physical examination, and exploratory puncture is not a safe procedure to determine the presence or absence of fluid. It is only made in cases in which the diagnosis of ascites is clear, for the purpose of obtaining fluid for bacteriological and cyto-diagnostic examination. The syringe, needle, and preparations are the same as for thoracentesis. It is essential that

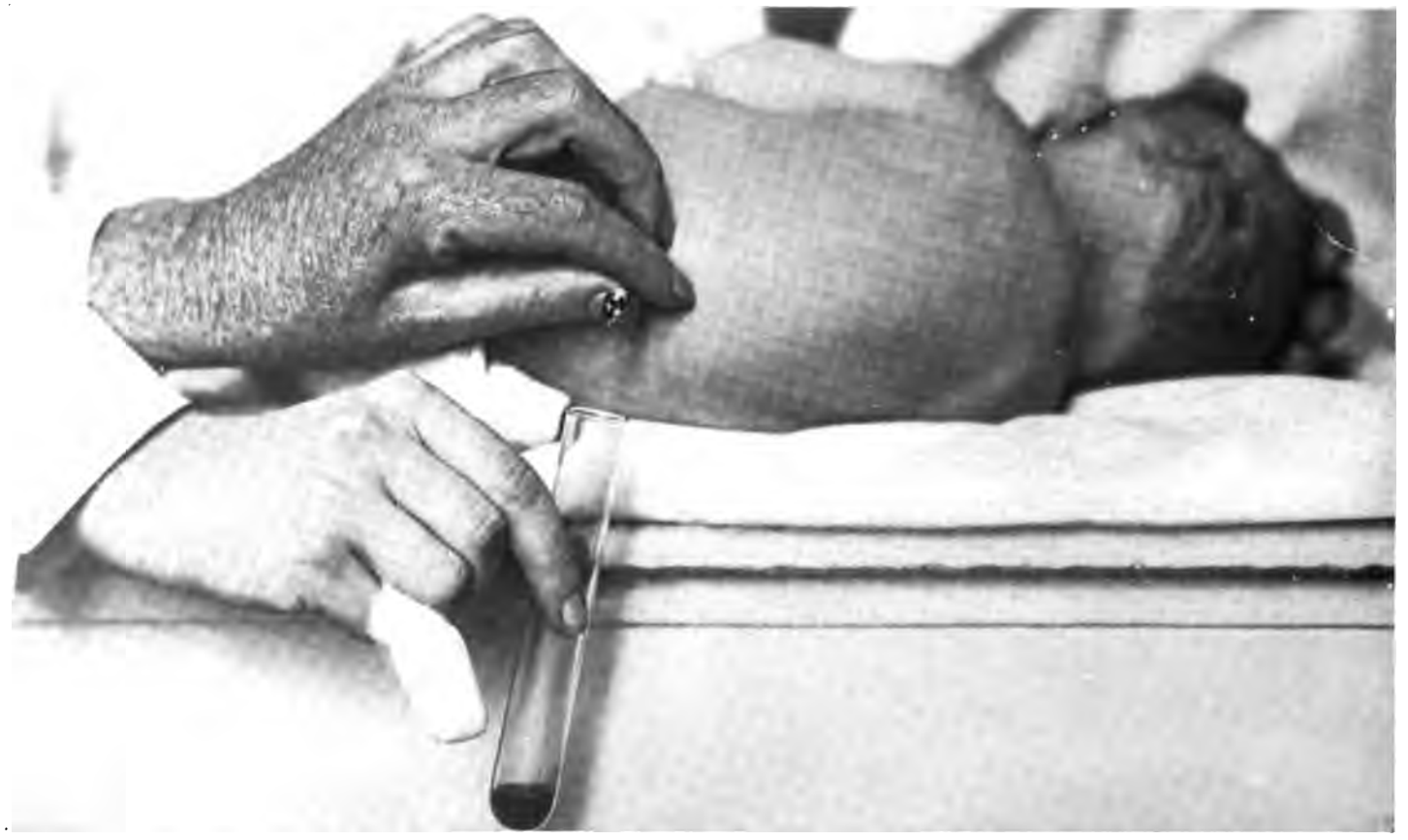


FIG. 33—Lumbar puncture

the bladder be first emptied by catheterization. The child should be in a sitting posture, and the needle is introduced half-way between the pubes and the umbilicus.

EXPLORATORY PUNCTURE OF THE PERICARDIAL CAVITY.—Puncture of the pericardial cavity is also rarely employed for diagnostic purposes. The diagnosis of pericardial effusion should rest on the results of physical examination. In rare cases exploratory puncture may be made to obtain fluid for examination. The usual purpose, however, of entering the pericardial cavity is the withdrawal of fluid as a therapeutic measure. The technique of this operation will be described in the article on Pericarditis.

EXAMINATION OF GASTRIC CONTENTS AND RETENTION TIME.—This procedure is of importance in the diagnosis of pyloric stenosis and pyloric spasm. It also often throws light upon gastric digestion, and sometimes reveals the presence of dilatation of the stomach. The apparatus used consists of a soft rubber catheter of a size from 14 to 18 French scale. This is connected by a piece of glass tubing with a rubber tube attached to the nozzle of a glass bulb; to the opposite nozzle of the glass bulb, another piece of rubber tubing is attached. A measured quantity is given at a feeding, and the contents are withdrawn at whatever period after the feeding time it is desired to obtain them.

In estimating gastric retention time, it is desirable as a routine to withdraw the contents after one hour, and again after three hours. After one hour, the stomach is usually not empty, but much less should be obtained than was given. After two or three hours, the stomach should be empty. In withdrawing the gastric contents, the catheter is passed into the stomach, and the physician applying his mouth to the tube draws the fluid into the bulb. The amount of fluid withdrawn should always be measured.

EXAMINATION BY DUODENAL CATHETER.—The technique of this operation is described under the heading of Spasm of the Pylorus.

TESTING THE ELECTRICAL REACTION.—This requires special apparatus. There are a number of good batteries on the market available for this purpose. The essentials are, an appliance for reversing the direction of the current, so that the anodal and cathodal reactions may be tested without changing the position of the electrode. There must also be an appliance for measuring the strength of the current used, graduated in milliamperes. The indifferent electrode is applied to the front of the chest or any portion of the body, while the differentiating electrode is applied to the nerve or muscle to be tested.

LARYNGOSCOPY.—This procedure is mainly used in older children. The technique is that for adults.

OPHTHALMOSCOPY.—This is very difficult in infants on account of the movements of the eyeball. The technique in childhood is the same as in adults.

RECTAL EXAMINATION.—This procedure is frequently indicated in children, in obscure conditions in the abdominal cavity. The child should be lying on its back. The physician inserts the oiled finger into the rectum, while with the other hand he makes palpation through the abdominal wall. There is no danger in introducing the forefinger into the rectum, even a small baby's, provided that the introduction be made slowly enough to dilate the sphincter. A much greater area may be reached in infants than in older children and adults, and for this reason rectal examination often throws very valuable light on the diagnosis in infants.

ROENTGEN-RAY EXAMINATION.—This is by far the most important and valuable of all the special diagnostic procedures used in early life. Owing to the fact that the soft parts in children are less thick, the results of Roentgen-ray examination are more valuable in children than in adults, and in babies than in older children. Whenever possible, Roentgen-ray examination of the chest should be carried out in all cases of suspected tuberculosis, or suspected disease of the lungs, pleura, heart, pericardium, or thymus. It should be carried out in all suspected cases of disease of the bones or joints. It is sometimes valuable in intracranial disease. One of the most important uses of Roentgen-ray examination is in the investigation of the gastro-intestinal system. This is carried out by means of the bismuth meal, or the bismuth enema.

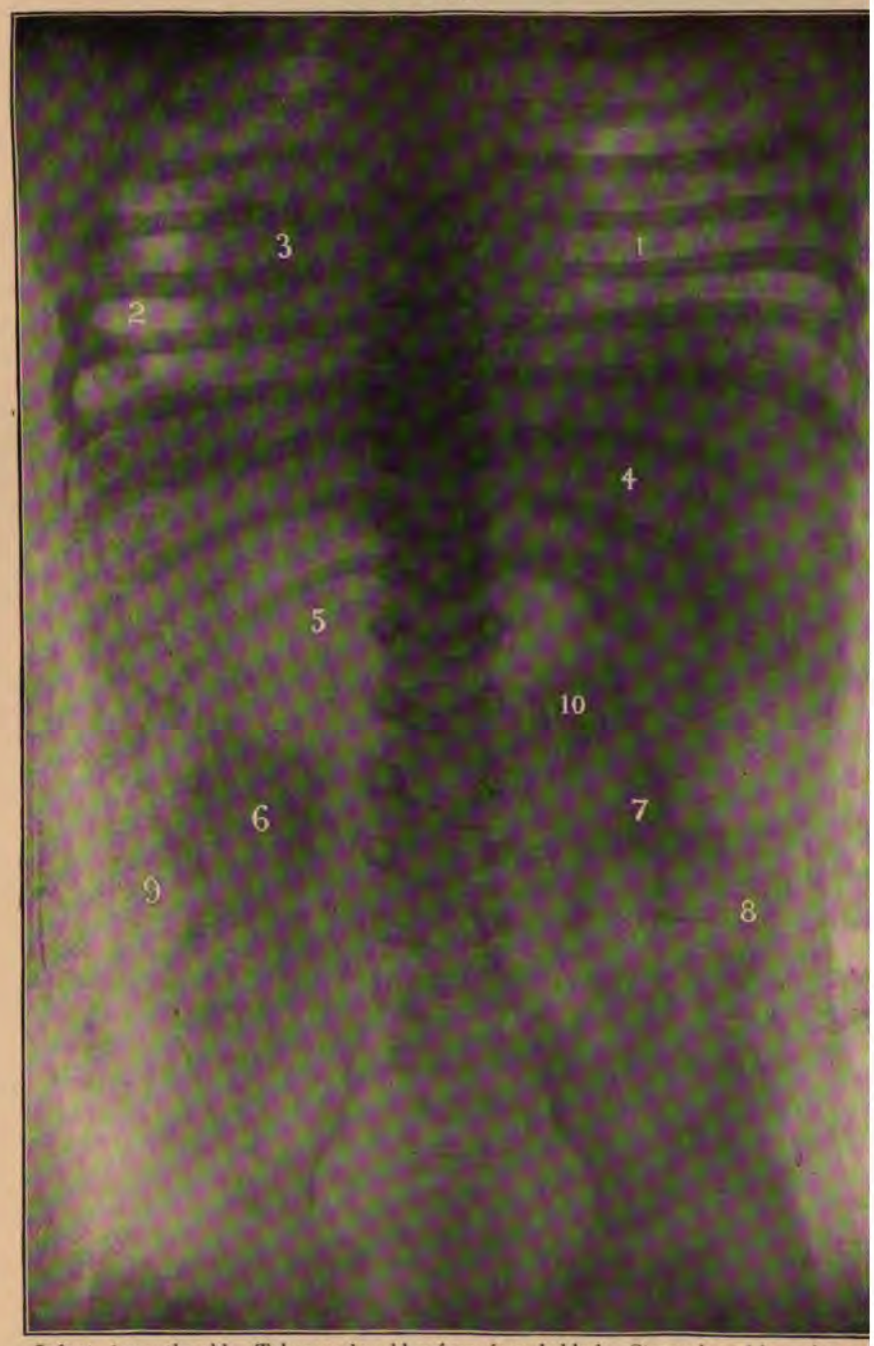
For the technique of these investigations and the interpretation of their results, the reader is referred to standard textbooks on roentgenology.

LABORATORY METHODS OF DIAGNOSIS

The importance of laboratory methods of diagnosis in the diseases of early life cannot be overestimated. The laboratory has come to occupy a more and more important place in modern diagnosis. There is, however, a tendency to neglect laboratory methods in the diseases of infancy and childhood. This is a very great mistake. Indeed, there are some conditions almost peculiar to early life, such as for example, pyelitis, in which the diagnosis depends entirely upon the results of laboratory investigation.

The laboratory methods of diagnosis which are used in infancy and childhood, do not differ either in character or in technique

FIG. 34



Infant, 6 months old. Taken under chloroform from behind. Stomach and large intestine distended with gas. 1, right lung; 2, left lung; 3, heart; 4, liver; 5, stomach; 6, left kidney; 7, right kidney; 8, ascending colon; 9, descending colon; 10, probably the head of the pancreas

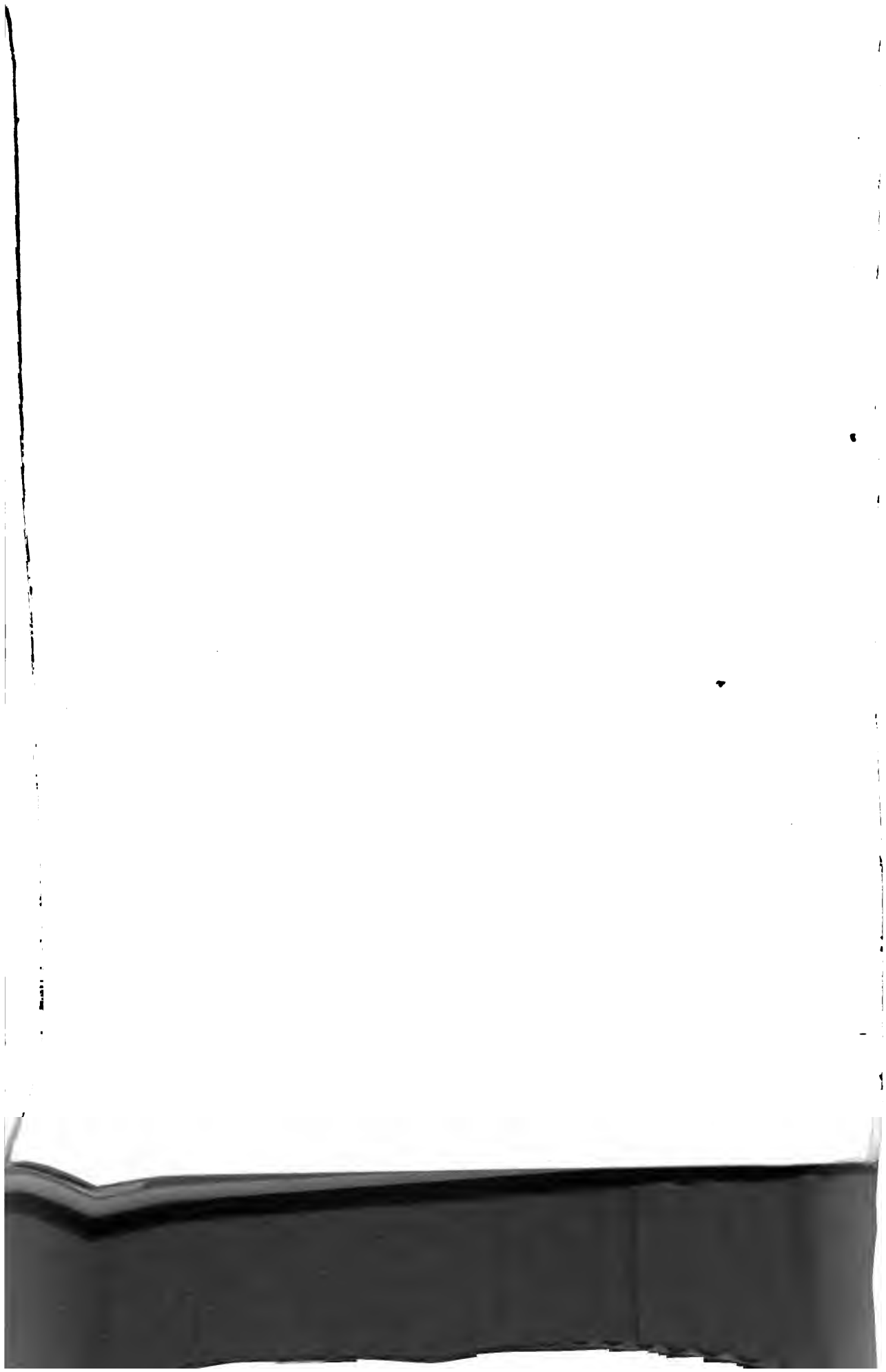
from those used in investigating disease in adults. The significance and relative importance of the various tests are however in many cases widely different in early life and in adult life. It is for this reason that the tests are here enumerated and described. The technique of the various tests is described for purposes of convenience of reference and of completeness.

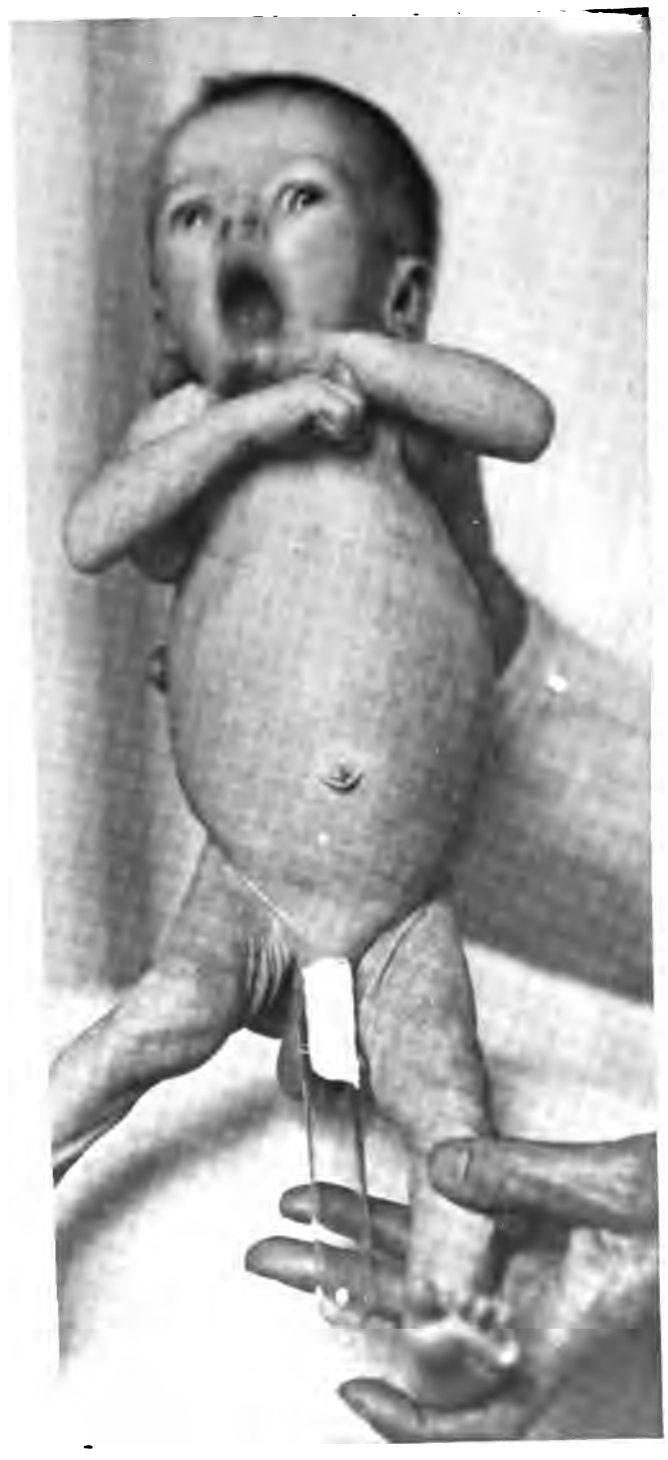
THE URINE.—The examination of the urine in childhood is often neglected. This is due partly to the impression that the results of the clinical examination of the urine in children are not of such great diagnostic significance as in adults, but mainly to the difficulty of obtaining urine from infants. The examination of the urine should be a routine measure in the investigation of every case of disease in infancy and childhood.

TECHNIQUE FOR OBTAINING THE URINE OF INFANTS.—In male infants, the best method of obtaining urine for examination is by the application of a clean test tube to the baby's penis. This is held in place by a strip of adhesive plaster, as shown in the illustration. If the test tube be properly applied and left in place, the urine passed will be collected in the test tube. It is possible by this method to obtain all the urine excreted in twenty-four hours.

In female infants, the obtaining of a specimen of urine for examination is more difficult. I am accustomed to use an apparatus like that shown in the illustration. It is simply placed in the proper position inside the diaper, which holds it in place. In many cases it is impossible to obtain immediately a specimen of urine. In such cases catheterization, which is a very simple procedure in female infants, should be employed.

CLINICAL EXAMINATION OF THE URINE.—The chemical and microscopic tests used in the examination of the urine of children do not differ from the tests in adults. For this reason it is, perhaps, unnecessary to describe them. Nevertheless, as there are differences in the relative importance of the various tests in childhood and in adult life, and also some differences in the diagnostic significance of the tests, they will be briefly described here for purposes of pointing out these differences, and of completeness. It is important to note the frequency of micturition in all cases. It should be remembered that in infants the normal frequency is very great when the baby is awake, the urine often being passed two or three times an hour; in sleep it is retained longer, from two to six hours. After the first two years, the normal time in which the urine is retained during sleep is very variable, and as the child grows older, the frequency by day gradually diminishes. The age at which control of the sphincter of the bladder is attained, is also very variable. Under normal





. 35—Obtaining urine from male in'ant

FIG. 36—Apparatus for obtaining urine from a female infant

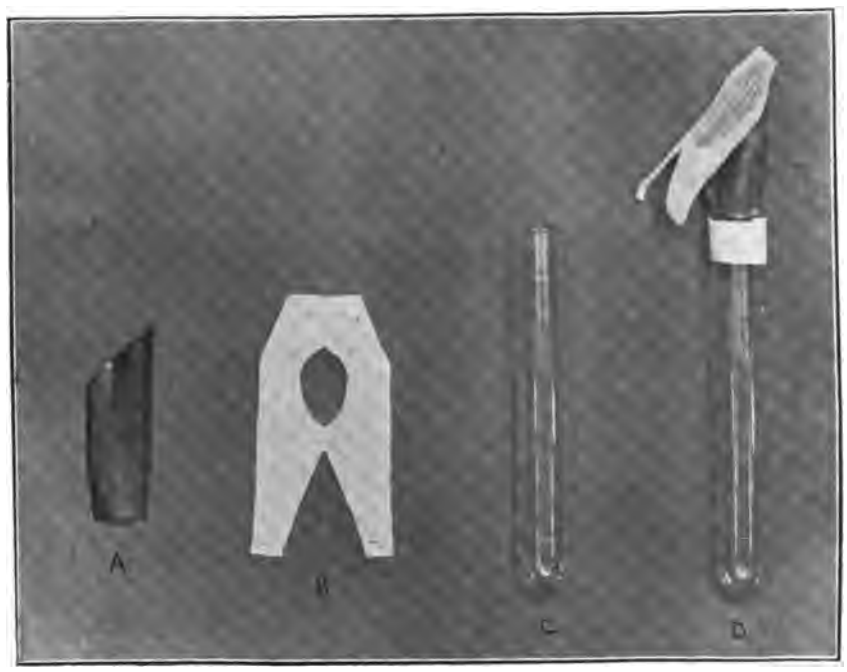


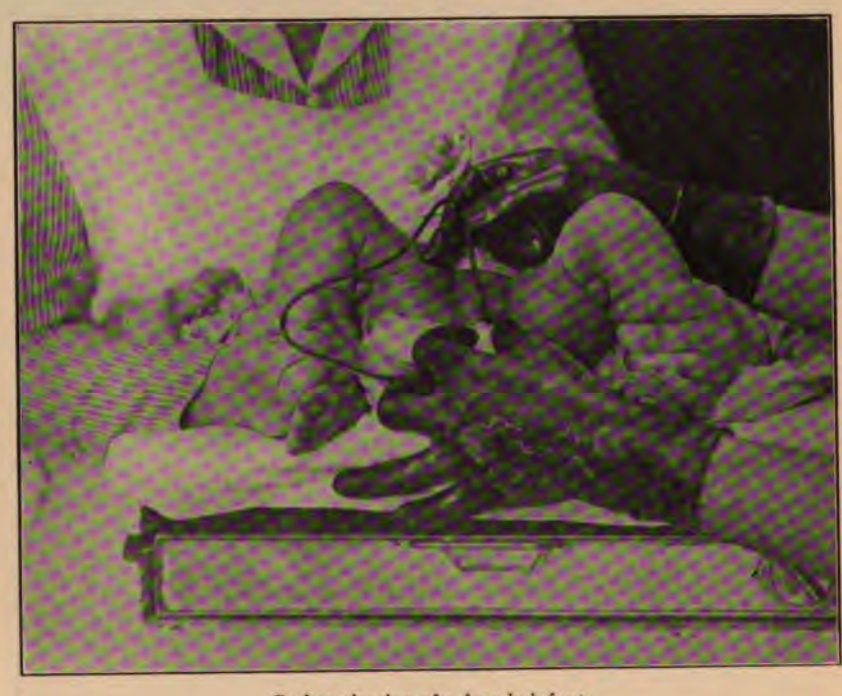
FIG. 37—Method of collecting urine from a female infant
A. Cut rubber finger cot. B. Adhesive plaster for attachment. C. Test tube.
D. Apparatus complete

circumstances, the diaper may sometimes be dispensed with as early as one year, and in other cases, not until two years, or even later.

The tests may be divided into those which should form part of every routine examination, and those which need only be employed occasionally for a definite purpose. The tests used in the *routine examination* of the urine will be described first.

PHYSICAL CHARACTER AND REACTION.—The color, odor, specific gravity, turbidity, and amount and character of sediment should

FIG. 38



Catheterization of a female infant

first be noted. The urine of the newly born infant is usually somewhat highly colored. During the rest of infancy the normal color is pale in comparison with that of older children and of adults. The normal odor has no peculiarity in infancy and childhood. A strongly ammoniacal odor in infants is seen in some cases of malnutrition and gastro-intestinal disease. Except in the first few days of life, the reaction of the urine in infancy is faintly acid or neutral. In older children it is acid. In the first days of life it is strongly acid, and often shows a deposit of urates or uric acid, which appears as a pinkish or reddish-yellow stain upon the napkin. The normal specific gravity at the various ages is as follows:

TABLE 12

The first two or three days	1.010 to 1.012
Fourth to fourteenth day	1.003 to 1.006
Fourteenth day to sixth month	1.004 to 1.010
Six months to two years	1.006 to 1.012
Two to eight years	1.008 to 1.016
Eight to fourteen years	1.012 to 1.020

Slight turbidity of the urine is not uncommon in infancy. Under normal conditions it may be due to the presence of mucus, in which case it cannot be entirely removed by filtration, or to the presence of urates. Cloudiness due to urates will disappear on heating. Pathological causes of turbidity are phosphates, bacteria, or pathological sediment, which in early life is usually pus. The cause of turbidity should always be determined. Cloudiness due to bacteria is recognized by the fact that it is not diminished by ordinary filtration. Phosphates are recognized by the fact that the turbidity disappears when a few drops of acetic acid are added. Pus is suspected from exclusion of the other causes, the suspicion being confirmed by the microscopic examination.

ALBUMEN.—For routine use, the best test for albumen is the heat test, which is performed as follows: Filter about 10 c.c. of urine into a test tube, and boil the upper half of the fluid. Add one or two drops of acetic acid (36 per cent.), and boil again. A precipitate which appears on boiling, and persists after the addition of the acid, or which appears on the second boiling, is albumen. Care must be taken not to use an excess of acid.

An approximate idea of the quantity of the albumen can be obtained from the heat test by allowing the albuminous precipitate to settle. 1/100 per cent. or less causes turbidity but no precipitate. With 1/20 per cent. the bottom of the test tube is covered; 1/10 per cent. occupies 1/10 the volume of the column of urine; while 1/2 per cent. occupies 1/3 of the volume; and one per cent. half the column. Albumen amounting to two or three per cent., becomes converted into a compact coagulum.

When in the performance of the heat test the addition of acetic acid gives a heavy precipitate which partially clears on boiling, it suggests the presence of the Bence-Jones body (often incorrectly spoken of as albumose). The presence of this substance is confirmed after filtering the contents of the tube while hot by the appearance of a cloud in the filtrate on cooling.

Albumen may be normally present in small amount in the urine during the first days of life. Later its presence has the same significance as in adults.

SUGAR.—Fehling's test is best for routine qualitative examination. Mix in a test tube 5 c.c. of each of Fehling's two solutions. Boil, and then add slowly 5 c.c. of urine. Separate in two portions

in two test tubes. Boil one of these portions. If no precipitate appears, sugar is absent; a precipitate may or may not be sugar. Set the other portion aside without further heating. If a precipitate appears, the presence of sugar is confirmed. When the sugar is very small in amount, the precipitate may take from fifteen minutes to several hours in appearing. For the methods by which the presence of sugar is confirmed by other tests, the reader is referred to standard works on physiological chemistry.

It is usually stated that sugar may be present in the urine of normal infants during the first two months. This is true, but I have also found it occasionally in the urine of normal infants, or of infants suffering from mild gastro-intestinal disturbances, up to the age of two years. It is very much more frequently absent than present. Creatinin is normally present in the urine of infants, and occasionally is present in sufficient amounts to reduce Fehling's solution. Many writers have mistaken this reduction for sugar. Creatinin can be excluded by shaking the urine with picric acid, filtering, and repeating Fehling's test, which will be negative if the reduction was caused by creatinin. In connection with the appearance of sugar in the urine of infancy, the statement of Grósz is often quoted, that sugar appears if the quantity of sugar given in the food is increased to a certain amount in proportion to the body weight of the infant. In some experimental work performed by Dr. Langley Porter and the writer, the results failed to confirm this statement. We were unable to cause the appearance of sugar, or an increase in the amount of sugar, by giving large amounts of lactose or maltose in the food.

For the most accurate quantitative test for dextrose in the urine, such as that with Benedict's solution, the reader is referred to standard works on physiological chemistry.

The simplest quantitative estimation for routine use can be made by means of the fermentation test. To 100 c.c. of urine of known specific gravity, add one-third of a yeast cake broken into very small pieces. Set in a warm place for twenty-four hours. If at the end of this time there is no reduction with Fehling's solution, take the specific gravity; otherwise continue the fermentation till no reduction is obtained. Multiply the difference in specific gravity before and after fermentation by 0.23 to get the per cent. of dextrose present.

ACETONE.—The test for acetone should form part of the routine examination of the urine in infancy and childhood. The presence of the acetone bodies is of great diagnostic importance in the diseases of early life. Their significance will be considered under Acidosis.

To one-sixth of a test tube of urine add a crystal of sodium nitroprusside. Shake thoroughly until the crystal is dissolved, and then

add a few drops of glacial acetic acid, shaking again. Pour carefully down the side of the test tube 5 c.c. of ammonium hydrate. A purple ring where the ammonia meets the urine demonstrates the presence of acetone.

SEDIMENT.—Whenever there is any visible sediment, it should be examined microscopically as a routine. The things to be looked for are pus, casts, blood, epithelial cells, crystals, fat, and bacteria.

The following tests need not be made as a routine, but are of occasional value and should be made whenever their need is suggested by the clinical features of the case.

BILE.—The reagent consists of tincture of iodine one part, alcohol fifteen parts. Pour 1 c.c. of this reagent on the top of the urine in the test tube. A green ring at the junction of the two fluids shows the presence of bile. This test should be performed whenever there is any suspicion of jaundice.

BLOOD is recognized from the color of the urine, and from the examination of the sediment. Blood pigment may be present in solution, as in haemoglobinuria. Its presence is demonstrated by the guaiac test, which will be described under the examination of the feces.

INDICAN.—To 15 c.c. of urine add 3 c.c. of a 20 per cent. solution of lead acetate. Filter. To the filtrate add an equal volume of a reagent made up of 0.4 gram ferric chloride in 100 c.c. of concentrated hydrochloric acid. Shake for two minutes. Add 3 c.c. of chloroform and again shake. If indican be present in considerable quantity, the chloroform will assume a deep blue color. This is known as Obermeyer's test. An excess of indican in the urine of infants and children is usually significant of acute or chronic intestinal disease. It is more or less a measure of the amount of protein putrefaction going on in the intestine. Indicanuria is also seen in extensive suppurative processes without drainage in various parts of the body, such as empyema. Indicanuria is not of very great importance either as a diagnostic measure, or as a measure of the results of treatment in intestinal diseases. It is said to be an important finding in pellagra.

DIACETIC ACID.—Add a strong aqueous solution of ferric chloride to one-third of a test tube of urine. A Burgundy-red color shows the presence of diacetic acid. If the reaction takes place after the urine has been previously boiled, it is not due to diacetic acid. Diacetic acid is one of the acetone bodies which are found in acidosis. The test is performed whenever acidosis is suspected, or when the routine test for acetone is positive. Whenever diacetic acid is found in the urine, it is probable that B-oxybutyric acid is also present.

TUBERCLE BACILLI.—To find tubercle bacilli in the sediment, the following technique should be employed: 1. Centrifugalize.

Decant supernatant fluid, dilute with water, recentrifugalize, and make a cover glass preparation from the sediment. This should be spread thinly, and held in the fingers over a flame of a Bunsen burner until dry. Place the cover glass in the forceps and fix by passing three times through the flame. 2. Cover the preparation thoroughly with carbol-fuchsin solution and steam over the flame for half a minute. Do not allow the staining solution to dry on the surface of the cover glass, but add more stain if necessary. 3. Wash in water. 4. Decolorize for twenty seconds in 20 per cent. sulphuric acid. 5. Wash in water. 6. Wash in 95 per cent. alcohol for thirty seconds, or until no more color will come out. 7. Wash in water. 8. Cover the preparation with Löffler's methylene-blue solution for thirty seconds. 9. Wash in water, dry and mount. Tubercle bacilli are bright red; nuclei and other bacteria are blue.

GONOCOCCUS.—The technique of staining the sediment of the urine for gonococci is the same as that used in the examination of the vaginal discharge, and will be described under that heading.

EXAMINATION OF THE BLOOD.—For ordinary clinical examination, blood is obtained from infants and children as in adults. The lobe of the ear is cleansed with water, thoroughly dried, and quickly pierced with a clean surgical needle.

HEMOGLOBIN.—For the ordinary estimation of the per cent. of hemoglobin, Tallqvist's scale is sufficiently reliable. If it be desired to make an accurate estimation, some such apparatus as the Fleischl-Miescher or Sahli must be used. The presence or absence of anemia can be determined by Tallqvist's method. The standard by which anemia is estimated in early life is, however, entirely different from that used in adult life. The percentage of hemoglobin in the blood is highest in the newly born, and rapidly falls in the first few days of life. It is considerably lower than in adult life throughout childhood, being lowest during infancy, and gradually increasing after the second year up to the age of puberty, when the adult standard is reached. As measured by the adult standard, the lowest normal limit would be represented by 65 per cent on Tallqvist's scale. The usual range in infancy and childhood is from 65 to 85 per cent.

LEUCOCYTOSIS.—The presence or absence of leucocytosis is determined by means of the white count. Draw the blood into the Toma-Zeiss white blood counter up to the 0.5 mark, and then draw in 0.5 per cent. acetic acid up to the 11 mark. Mix thoroughly by shaking and revolving the counter. Count all the corpuscles on the ruled field. Clean the slide and make a second count. Add the two counts together, multiply by 100, and the product is the number of white corpuscles per cubic millimeter.

The normal standard differs notably from that of adult life. Normal variations in infancy are from 8,000 to 15,000, and in later childhood from 6,000 to 13,000.

THE RED CORPUSCLES.—The count of the red corpuscles is made as follows: Draw the blood into the Toma-Zeiss red counter up to the 0.5 mark, and dilute with Gower's solution up to the 101 mark. Mix thoroughly by shaking and revolving the counter. Place a drop of blood on the slide of the counter, cover with the glass, and count the corpuscles in twenty-five small squares at each of the four corners of the ruled field, and multiply the total by 8,000. This gives the number of red cells in a cubic millimeter of blood. For greater accuracy it is well to repeat this procedure with a second slide, and take the average.

The number of red corpuscles at birth is high, although there is some diversity of opinion as to the actual number. It is between 5,350,000 and 6,000,000. There is an increase in the first two or three days of life, and the count may be from 6,000,000 to 7,500,000. This increase is probably only relative, being due to loss of fluid, and the delay in the establishment of nutrition. After the first few days, the count falls rapidly, reaching the normal figures for infancy at about two weeks. The normal standard for infancy ranges from 5,000,000 to 6,000,000. It gradually falls during early childhood, reaching the adult standard of 4,500,000 to 5,000,000 at about six years.

EXAMINATION OF THE STAINED SPECIMEN.—Cover glasses should be thoroughly washed with soap and water, and wiped dry. The center of a cover glass held by its edges is touched against the summit of the drop of blood, touching the skin being avoided. Drop the cover glass upon another clean cover glass, allowing the blood to spread evenly between them without pressure. Hold the cover glasses by their edges, and, keeping their faces parallel, draw them quickly apart. When dry, they are ready for staining. No heat is required. Cover the specimen with five drops of Wright's modification of Leishman's blood stain, for one minute. Add with a medicine dropper ten drops of water, and let the diluted stain remain on the slide for two minutes. Wash in water (not running water) until the film has a pinkish color. Dry and mount.

The features to be noted in the microscopic examination of the stained specimen, are the following:

1. The relative number of the different varieties of white corpuscles.
2. The appearance of the red corpuscles. The points to be noted are variations in size (makrocytes and mikrocytes), variations in shape (poikilocytosis), loss of color (achromia), stippling, abnormality of staining (polychromatophilia), tendency toward predominance of

large or small forms, and the presence and relative number of nucleated forms (normoblasts and megaloblasts).

3. The number of blood platelets.

4. The presence or absence of parasites, particularly those of malaria.

Five varieties of white corpuscles are found normally in human blood, and these are classified as follows by Ehrlich:

1. Lymphocytes (Fig. 39). These are about the size of a red corpuscle, and contain one large, round, deeply staining nucleus which entirely fills the cell. The protoplasm is not granular and stains faintly or not at all.

2. Large Mononuclear (Fig. 39). These cells are much larger than the lymphocytes. They have one large oval nucleus with a broad margin of non-granular, almost colorless protoplasm about it.

3. Transitional (Fig. 39). These cells are derived from the last form and are similar in size and color. The nucleus is indented on one side as the result of the beginning of nuclear division.

These three varieties are sometimes called basophiles.

4. Polynuclear Neutrophiles (Fig. 39). These are round cells, smaller than the large mononuclear, having a peculiar polymorphous deeply staining nucleus. The nucleus is long, irregular or twisted, and when stained often appears segmented. The protoplasm contains fine granules which are stained by both the acid and basic stains.

5. Polynuclear Eosinophiles (Fig. 39). These are usually about the size of neutrophiles and have a deeply stained polymorphous nucleus. The protoplasm contains granules which are much coarser than those of the neutrophilic cells, and which stain readily with acid stains.

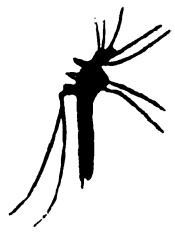
These cells are sometimes called oxyphiles.

6. Myelocytes (Fig. 39). These are round or ovoid cells with one, rarely two, large round or slightly bent nuclei which stain light blue. There are two varieties, neutrophilic and eosinophilic. The first has a protoplasm crowded with fine neutrophilic granules. The second contains coarse eosinophilic granules. Myelocytes rarely appear in normal blood, but are much increased in some of the pathological states.

7. Mast-Cells.—These are coarse, granular basophiles, usually with a trilobar nucleus. The protoplasm with Wright's stain is nearly unstained, with a number of round dark blue spots against a faintly stained background, representing the basophilic granulations.

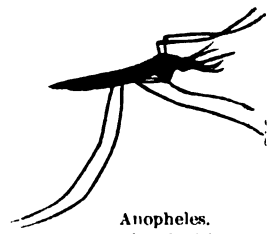
The relative number of these different varieties of white corpuscles is estimated by means of the *differential count*. The physician should write down the list of the different cells, and then count the corpuscles in the stained specimen, making a mark after each variety of cell.

PLATE XV.



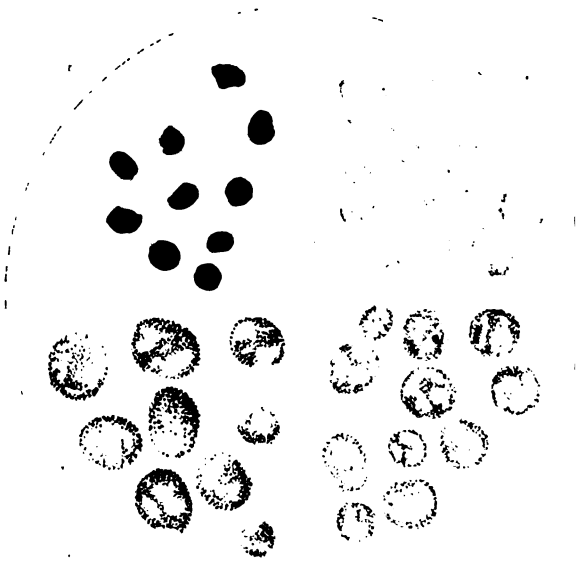
Culex.
(Resting Position.)

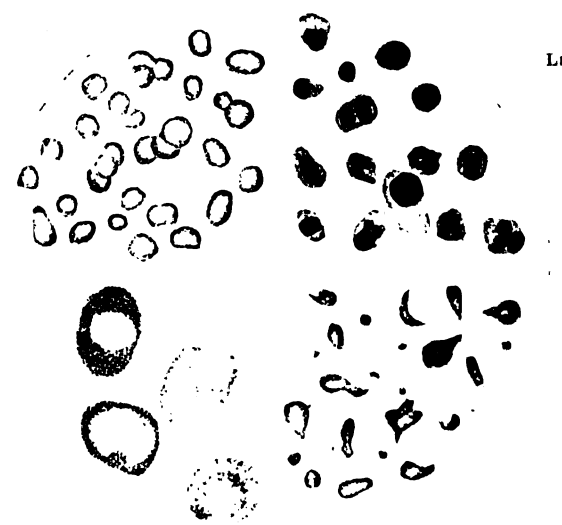
Anopheles.



Anopheles.
(Resting Position.)

Plasmodium Malariæ
Oil Immers. REICHERT $\frac{1}{12}$.
Ocular No. 3.





LEITZ Oil Immers. $\frac{1}{12}$, Ocular No. 3.

1. Normal Red Corpuscles.
2. Hæmatoblasts or Nucleated R Corpuscles:
   a. Normoblasts.
   b. Megaloblasts.
3. Microcytes.
4. Poikilocytes.
5. Lymphocytes or Small Mo nuclear.
6. Large Mononuclear:
   a. Transitional.
7. Polynuclear Neutrophiles.
8. Polynuclear Eosinophiles:
   a. Dwarf Eosinophile.
9. Myelocytes:
   a. Neutrophilic.
   b. Eosinophilic.

At least 250 white corpuscles should be counted, and the number of each variety should be reduced to a percentage of the total number counted. The number of any normoblasts or megaloblasts seen during the count should be noted and recorded, ***but should not be included in the total of the count, nor expressed in figures of percentages.*** A specimen of the method of making and of recording a differential blood count is shown in the following tables:

TABLE 13

Method of Making Differential Blood Count

Polynuclear neutrophiles..	𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸	130
Basophiles.....	𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸 𝍸	110
Eosinophiles.....	||	2
Myelocytes.....	𝍸 |||	8
		250
Normoblasts.....	|||	3
Megaloblasts.....	𝍸 |	6

$\frac{130}{250} \times 100 = 52\%$ $\frac{110}{250} \times 100 = 44\%$ $\frac{2}{250} \times 100 = .8\%$ $\frac{8}{250} \times 100 = 3.2\%$

TABLE 14

Method of Recording Results of a Specimen

Examination of the Stained Specimen

DIFFERENTIAL COUNT OF 250 WHITE CORPUSCLES

Polynuclear neutrophiles	52.0%
Basophiles	44.0%
Eosinophiles	0.8%
Myelocytes	3.2%
	100.0%

The red corpuscles showed slight poikilocytosis, and moderate variation in size, makrocytes predominating. There was marked achromia, some stippling, and some polychromatophilia. During the count were seen—

Normoblasts 3
Megaloblasts 6

The blood platelets appeared normal, and no parasites were seen.

PECULIARITIES OF THE DIFFERENTIAL COUNT IN EARLY LIFE.—The variations in the percentages of the different kinds of leucocytes in the blood of infants and young children, according to the age of the child, is most clearly demonstrated by means of chart 3.

It will be seen from the chart and from the figures giving the total leucocytosis at birth and in the subsequent periods of childhood, that in the first day of life polynuclear leucocytosis exists which declines rapidly during the next nine days, and then very gradually until the sixth month, when it begins to mount, and at the end of the sixth year has almost reached the adult level. The lymphocytes, or small mononuclear leucocytes, are, on the other hand, at their minimum at birth, and in the subsequent ten days increase with about the same rapidity as the polynuclears decline in numbers. They raech their maximum about the sixth month and then gradually decline to the sixth year, while the polynuclears are proportionately increasing. The chart also shows clearly that the lymphocytosis so characteristic of the early months of life is almost wholly dependent upon an increase in the lymphocytes or small mononuclear cells, the large mononuclear and transitional leucocytes being but little changed. The eosinophiles show a count somewhat higher than in adults, ranging from two to six or even eight per cent. Their presence in such proportions in early childhood is, therefore, of less significance than in later life.

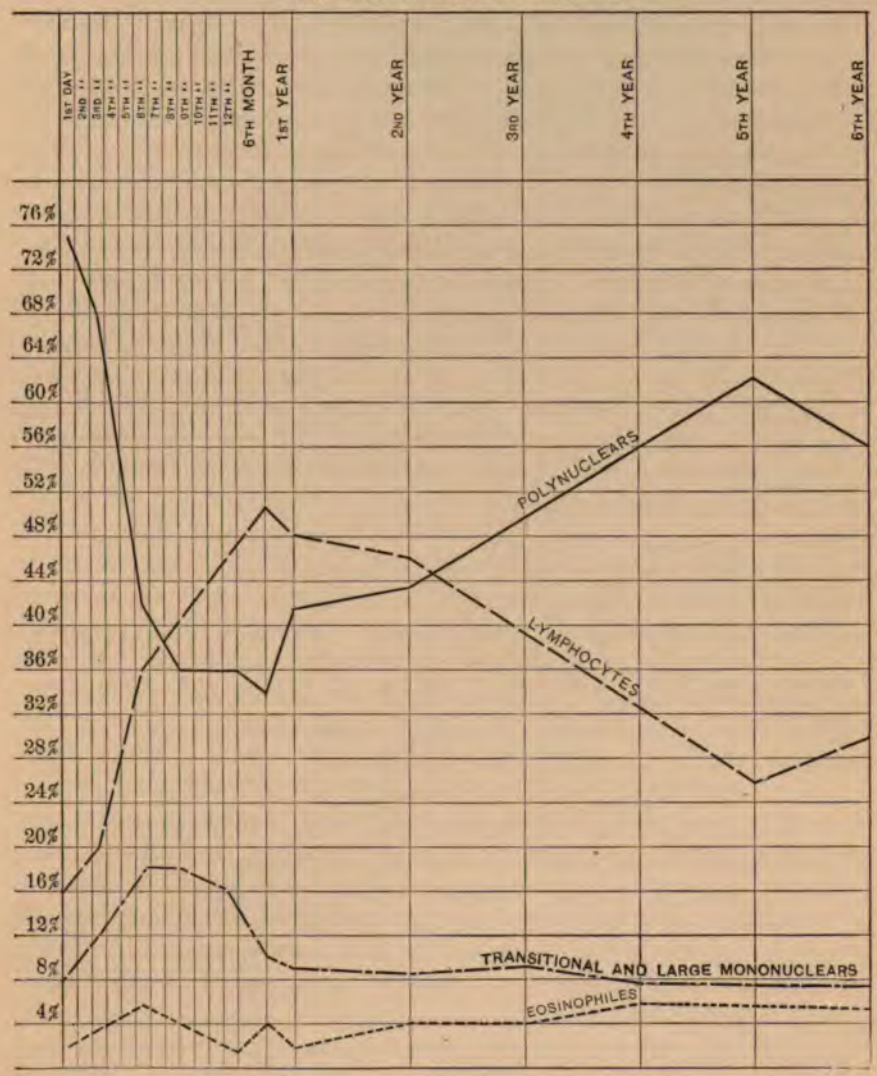
PECULIARITIES OF THE BLOOD PICTURE IN DISEASE IN EARLY LIFE.—It is recognized that all the signs by which sickness is shown in the blood of adults are exaggerated in that of children. Their blood is much more sensitive to the action of adverse influences, such as poor air, improper hygienic surroundings, improper food, and numerous other causes, and anemia is much more easily induced. This is especially noticeable in the impoverishment of the blood which follows gastro-enteric disease. Not only is a condition of anemia acquired by these influences, but the development of the blood may be retarded. In this class of cases the blood of a child three or four years old may show no higher development than that of a healthy infant in the second year.

Again, the erythrocytes under certain morbid conditions may revert to an earlier type. The function of the bone marrow in infancy is chiefly occupied in the production of the erythrocytes. As the marrow still has at birth many of its fetal characteristics, any strain put upon it by disease makes it much more likely to revert to its fetal function, and, therefore, to manufacture and pour out into the circulation the cells which are characteristic of that period. The presence of nucleated red cells, myelocytes and eosinophiles in infancy and early childhood are, therefore, of less significance than in later life. Opinions differ as to the time at which the haematoblasts disappear in the normal blood of infants. They may be present in small numbers during the first week, but after that their presence is abnormal. In diseased conditions in infancy they appear with much less provocation than in older children and adults.

CHART 3

SHOWING APPROXIMATELY THE RELATIVE PROPORTIONS OF THE LEUCOCYTES FROM BIRTH TO SIX YEARS.

(ADAPTED FROM CHARTS BY CARSTANJÉN.)



In the secondary anemia due to a large number of diseased conditions, a diminution in the number of red cells as well as of hemoglobin is more common in infancy and early childhood than later. The chloro-anemia (chlorosis) type, with lower hemoglobin in proportion to the red count, is not so common in infancy. It should also be remembered that in all diseased conditions, variations in the size and shape of the red cells are much more easily produced in infancy and early childhood, than later.

The white corpuscles are affected in a temporary or permanent manner by many physiological and pathological conditions in the body at large as well as in the blood-forming organs. The most common change from the normal is that of leucocytosis. Leucocytosis was originally described by Ehrlich as the presence in the blood of a greater number of white cells than normal for the individual, or a relatively increased number of any variety of white cells, with or without an increase in the total number of leucocytes. Cabot has restricted the term leucocytosis to an increase of leucocytes in which the polynuclear cells predominate, and uses the terms lymphocytosis and eosinophilia to describe a relative increase of the lymphocytes and eosinophiles. Physiologically, we find a leucocytosis after meals, often called digestive leucocytosis. It is at its height about two hours after a meal, when the number of leucocytes may be increased by 33 1/3 per cent. of the normal number. Pathologically, a leucocytosis follows a considerable number of diseases, and seems in a general way to depend upon two factors, the severity of the infection and the amount of resistance in the individual. We find a pronounced leucocytosis in most fevers and in most septic processes. In these cases the increase is almost wholly composed of the polynuclear neutrophiles, which may make up from 90 to 98 per cent. of the entire leucocyte count. The leucocytosis does not depend on the degree of fever, does not always occur with it, and conversely inflammatory leucocytosis may appear before the fever.

The diseases which generally show a leucocytosis are pneumonia, pericarditis, endocarditis, pleuritis, erysipelas, acute rheumatism, purulent meningitis, pharyngitis, diphtheria, septicemia, osteomyelitis, scarlet fever, variola, tonsillitis, bronchitis, peritonitis, acute nephritis, certain gastro-enteric infections, some profound anemias whether primary or secondary, leukemia, hemorrhage, malignant new growths, abscess of any kind, including appendicitis, and many skin diseases.

The diseases in which the leucocytes are approximately normal are malaria, measles (after the appearance of the efflorescence), typhoid fever, pulmonary tuberculosis (unless there is a secondary infection of other bacteria), rubella, and most cases of epidemic influenza. In tubercular meningitis and peritonitis, leucocytosis may or may not be present. Comparing these two lists it will be seen that there are instances in which the leucocyte count may be of great importance to the physician in making a differential diagnosis.

The diagnostic value of leucocytosis in early life is usually stated to be approximately the same as in adult life. In my experience, the value of the white count in diagnosis is not so great in infancy and childhood. Exceptions to the ordinary rules appear to be more numerous.

In diseased conditions in infancy and early childhood affecting the

blood, myelocytes appear more often than in later life. They are usually to be found in all the severe secondary anemias of infancy.

There are certain diseases in which there is an alteration in the differential count of the white cells. The lymphocytes are increased in pertussis, syphilis, and in leukemia. An increase in the eosinophiles is seen regularly in leukemia, asthma, with certain intestinal parasites, and in some forms of chronic skin disease. In infancy and early childhood an increase in eosinophiles is not uncommon in secondary anemia, and the count may show ten per cent. without any very obvious cause.

THE FECES.—The examination of the feces is in infancy the most important of all the laboratory methods of diagnosis. In older children it is important, but is not necessarily a matter of routine.

The stools are examined macroscopically and microscopically.

THE MACROSCOPIC EXAMINATION should include first an observation of the *frequency* and *size* of the dejecta, and of their *consistency*, and *form*. The *color*, *odor*, and *reaction* should be noted. Abnormal constituents to be looked for are *curds*, *mucus*, *blood*, *pus*, *membrane*, *masses of undigested food*, and *intestinal parasites*.

FREQUENCY AND SIZE.—The normal number of stools in the breast-fed infant is three or four daily. In the bottle-fed, the daily number is less, varying from one to three or four. A certain amount of constipation is a normal condition in bottle-fed infants who are otherwise doing well. The size of the stools varies with the frequency. Abnormally small stools suggest relative starvation. Increase in the frequency of the stools suggests irritation of the intestinal mucosa, such as occurs in various forms of indigestion and infection.

CONSISTENCY AND FORM.—The normal stool of the breast-fed infant is of a soft mushy consistency. In infants fed on cow's milk, the stools are of firmer consistency, and may even be formed. Increase in consistency is seen in constipation. Loose stools suggest irritation of the intestinal mucosa, with increased peristalsis.

COLOR.—In observing the color of the stools, it must be remembered that the surface of the fecal mass is easily discolored by contact with the air, and the center should be used as a basis for description.

The color should be recorded as yellow, white, brown, green, or black. The significance of the various colors in infancy is as follows:

Yellow stools are seen in breast-fed and bottle-fed infants who are doing well. In the breast-fed the normal color is a golden yellow. In infants who are thriving on cow's milk mixtures, the color is very often lighter yellow, probably because some of the bilirubin is converted into hydrobilirubin, or because there is a slight excess of unabsorbed protein.

Brown stools are not abnormal, or a sign that an infant is not thriving. Various shades of brown are seen when the amount of protein in the food is high in proportion to the fat. This often occurs when infants are being fed on skimmed milk or whey, or on lactic acid milk or albumen milk. Brown stools are also normal when maltose is substituted for lactose as the extra sugar added to cow's milk modifications. When the infant begins to take a more varied diet, containing starch, beef juice, or broth, the stools assume a brownish color.

Gray stools are abnormal. A gray color may be due to the presence in the stool of a large amount of fat. While not normal, a gray color is not in itself an indication that the amount of fat should be reduced. A gray color is also seen when bile is absent or insufficient.

Green is the most common abnormal color. The shade may vary from a light grass green, to a dark green. The darker the green, the greater the pathological significance. A stool which changes from yellow to green after exposure to the air is not abnormal. The abnormal green color is due to the oxygenation of bilirubin into biliverdin, and this apparently may take place when the stool is either excessively alkaline, or excessively acid, or as a result of various forms of bacterial activity. Green stools are seen in all forms of indigestion, and are not characteristic of any one type. They are particularly common in acute stages of indigestion from fat or carbohydrate, and in indigestion with fermentation.

White stools are due to the presence of an excessive quantity of undigested fat in the form of soaps. They may be soft or hard. White stools are always a sign that the digestion and absorption of fat is not normal, but are not necessarily an indication for cutting down the fat in the food.

Black stools are usually due to the giving of bismuth, or iron, or to the presence of blood.

ODOR.—The odor of the stools depends on the composition of the food, on the completeness of digestion and absorption, and on the degree and variety of bacterial activity. The normal stool has a sour or aromatic odor. In fat indigestion the odor is that of butyric or lactic acid. In carbohydrate indigestion the odor of lactic or acetic acid predominates. Excessive protein, even when not causing indigestion, produces a cheesy or fecal odor. A peculiar musty odor is also suggestive of a relative excess of protein in the food. A very foul or putrefactive odor is seen in some cases of acute indigestion with fermentation, and of infection of the intestine.

REACTION.—The reaction of the normal stool is slightly acid. When the relative quantities of the fat and protein are well balanced, it is probable that the normal slight acidity of the stools is maintained

by the fermentation of the carbohydrate residue which is not absorbed, and that Nature designs that there shall be an excess of carbohydrate to maintain a normal condition of the intestinal flora, and the normal chemical reaction. When the unabsorbed carbohydrate residue is in excess, it is never excreted as such, but undergoes fermentation, and consequently the acidity of the stools may be increased. When unabsorbed fat is in excess, there is increased formation of the fatty acids, and the acidity of the stools may also be increased. A relative excess of protein will cause increased activity of the proteolytic bacteria whose products are alkaline, and may cause an alkaline stool, which is not necessarily pathological. An excessively acid stool, therefore, is seen in carbohydrate indigestion, and in the acute exacerbations of fat indigestion. An alkaline stool means only a relative excess of protein in the food in the majority of cases, and has no pathological significance. It is, however, occasionally seen in cases of indigestion with fermentation due to an excessive activity of bacteria of the putrefactive group.

CURDS.—There are two kinds of curds found in the stools of an infant. One is primarily composed of casein, the other of fat combined with sodium and potassium in the form of soaps. The casein curds are larger, varying in size from that of a bean to that of a peanut, and are white or yellowish-white in color. They are tough, cannot be broken up by pressure, are insoluble in ether, and when placed in formalin become very hard. The fat curds are smaller varying in size from that of a pinhead to that of a pea, and are white, yellow, or green in color according to the color of the stool. They are soft, can be broken up by pressure, are soluble in ether when acidified, and are unaffected by formalin.

Casein curds suggest indigestion from a relative excess of protein. Fatty curds always suggest indigestion from a relative excess of fat. Not only that, but fatty curds suggest a degree or stage of fat indigestion which is more serious than that characterized simply by an excess of fat in the stools in the form of soaps, and is usually an indication for dietetic treatment.

MUCUS.—Mucus is present in most normal stools, but in amounts which can only be detected by microscopic examination. In the majority of abnormal stools, an excess of mucus is present, and an amount visible macroscopically is always pathological. Undigested starch may be mistaken for mucus, but is distinguished by the fact that it is stained blue with iodine, and shows a definite structure (starch granules) under the microscope, whereas mucus is not stained, and shows no evidence of structure.

An excess of mucus in the stools is caused by any condition which irritates the intestinal mucous membrane. It may be seen in all

forms of indigestion. A very great excess of mucus, or a stool composed mainly of mucus, especially if there is also blood, suggests infectious diarrhea or intussusception.

BLOOD in the stools is always pathological. It can usually be easily recognized on macroscopic examination, but sometimes it is necessary to prove that the appearance seen in the stools is certainly blood.

The *guaiac test* is the best for blood. The reagent consists of ten drops of freshly prepared tincture of guaiac and thirty drops of hydrogen peroxide. To 10 c.c. of stool or other suspected material in a test tube add 2 c.c. of glacial acetic acid and 15 c.c. of ether. Insert a cork, and invert several times. When the ether has separated, decant it, and add the ethereal solution to the reagent. A blue color indicates the presence of blood.

When blood appears as a spot or streak of fresh blood on the outside of a hard stool, it indicates some slight local lesion about the anus, usually a fissure, rather than a hemorrhoid. When the blood is mixed with mucus, but not homogeneously, occurring usually in spots or streaks, it means some form of ileo-colitis, the most common being that seen in infectious diarrhea. When blood is homogeneously mixed with mucus, it suggests intussusception. When blood occurs without excess of mucus, usually as a tarry mass, it suggests gastro-intestinal hemorrhage from some other cause.

PUS, visible macroscopically, means a severe inflammatory condition of the intestinal tract.

MEMBRANE is rarely seen, because the patients in whom membranous colitis is found post-mortem, usually die before the membrane appears in the stools. It means a very severe inflammation.

UNDIGESTED MASSES of food can sometimes be recognized macroscopically. Their significance is obvious.

INTESTINAL PARASITES.—The commonest in children in northern latitudes are round worms (ascaris) and pin-worms (oxyuris). Segments of the several varieties of tape worms (taenia) are seen at times, usually the beef worm (taenia saginata). Hook worms (uncinaria) are found in some localities. Other parasites, including the amoebae, are rare.

BILE PIGMENT is a normal constituent of the stools. Its absence is sometimes of great diagnostic significance. The test is as follows: Mix about one-quarter of the stool thoroughly with 100 c.c. of a concentrated solution of corrosive sublimate, and set aside in a covered glass dish for twenty-four hours. All particles containing hydrobilirubin will have a red color, while those containing bilirubin will be green.

MICROSCOPIC EXAMINATION OF THE STOOLS.—This is of the first importance in all the nutritional disturbances of infancy. In older children it is often of diagnostic value, but need not be made a routine measure. The examination is divided into three parts as follows: (1) examination of the fresh specimen; (2) microchemical examination for fat; (3) microchemical examination for starch. The feces should be thoroughly mixed in preparation for these examinations, and if hard, should be rubbed together with a little water.

EXAMINATION OF THE FRESH SPECIMEN.—A portion of the stool is placed on a slide, covered with a cover glass and put under the microscope. This reveals undigested meat fibres, and such pathological elements as pus, blood, eggs of parasites, and so forth.

MICROCHEMICAL EXAMINATION FOR FAT.—A portion of the stool is spread on a glass slide, and mixed with a saturated alcoholic solution of Sudan III. It is then covered with another slide (a slide is better than a cover glass) and put under the microscope. Neutral fat drops, and fatty acid crystals, stain red, while soaps do not stain. Next a drop or two of glacial acetic acid is thoroughly mixed with the contents of the slide. It is better to add the acid by removing the top slide, rather than by letting it run under the edge. The top slide is reapplied, and the slide is held over the Bunsen flame until it begins to bubble. This converts the soaps into neutral fat and fatty acids, which appear under the microscope as large stained drops. The amount of fat in the form of soap is estimated from the number of these drops.

If the stain with Sudan III before the addition of acetic acid shows fat, it can be determined whether it is neutral fat or fatty acid, by staining another specimen with a dilute solution of carbolfuchsin (1 part carbolfuchsin to 4 parts of water). With this solution neutral fat does not stain, fatty acids stain a deep red, and soaps stain a dull rose-red.

In estimating the quantity of fat in the form of soap, it must be remembered that under normal conditions all the fat ingested is not absorbed, and that a certain quantity of soaps in the stools is normal. The difficulty in the microchemical examination of the stools for fat lies in determining what quantity shall be termed an excess. With the technique described above the only standard for estimating the amount of fat is the number and size of the fat-droplets in the microscopic field, after boiling with acetic acid. The standard is much influenced by variations in the technique, depending on the quantity of feces taken, the amount of its dilution, the length of the boiling, and on variations in the rapidity and completeness with which soaps made with different alkaline bases are broken up. Every physi-

cian must establish his own standard, by adopting a uniform technique, noting the results obtained with numerous babies fed on different quantities of fat, and comparing these results with the macroscopic appearance of the stools. The following may prove useful as a suggestion:

1. Fat droplets crowding field and running together.... = large excess
2. Fat droplets numerous in every field..... = moderate excess
3. Fat droplets few in every field..... = slight excess
4. Fat droplets not in every field..... = no excess

The presence of neutral fat indicates an imperfect digestion of the fat, and usually, but not always, means that the fat in the food is in sufficient relative excess to cause nutritional disturbance. The presence of an excess of fat in the form of soaps indicates that the fat is normally split by the digestive ferments, but that it is not normally absorbed, the quantity in the food being in excess in proportion to the power of assimilation. Such an excess may or may not cause nutritional disturbance.

MICROCHEMICAL EXAMINATION FOR STARCH.—This of course, is carried out only with the stools of infants and young children who are taking foods containing starch. The staining reagent is Lugol's solution (iodine 2; potassium iodide 4; distilled water 100). The stools are placed on a slide, mixed with this solution, and examined under the microscope. The starch granules stain blue or violet. There are certain microörganisms which also stain blue with this solution, and the presence of these so-called iodophilic bacteria suggests a disturbance of carbohydrate digestion.

The presence of starch in the stools indicates that the quantity of starch in the food is too great for the digestive power, and that any symptoms of indigestion present are probably caused in part at least by this excess.

BACTERIOLOGIC EXAMINATION.—In the present stage of our knowledge, this is very unsatisfactory. Our knowledge of intestinal bacteriology is very limited, and in comparison with what remains unknown, hardly a scratch has been made upon the surface of this very complicated subject. We know that bacteria, and bacterial activity, play a very important part in gastro-intestinal disturbances, but this subject is still so complicated that it has not afforded any very valuable methods of laboratory diagnosis. There are certain special tests, such as those for the gas bacillus and the dysentery bacillus, which will be described under infectious diarrhoea. For further methods, clinicians must wait for the research workers, who are making very slow and discouraging progress at the present writing.

THE CEREBROSPINAL FLUID.—In the routine macroscopic examination of the cerebrospinal fluid, the points to be recorded are

the *quantity obtained*, the *pressure*, the *turbidity*, and the *presence or absence of fibrin formation*. The *pressure* is estimated by the rate and force of the flow through the lumbar puncture needle. Normally the fluid falls from the needle in discrete drops, with an appreciable pause between each drop. If the fluid drops more rapidly, but still in discrete drops, the pressure may be recorded as slightly increased. If the fluid runs in a continuous stream, the pressure is increased, and if it spurts from the needle, it is markedly increased. The pressure may be accurately measured by special apparatus, but this is not necessary in ordinary clinical work.

With regard to *turbidity*, the fluid is recorded as clear, faintly cloudy, opalescent, turbid, or purulent. In a purulent fluid, a distinct zone of pus settles at the bottom of the test tube.

Fibrin Formation is shown by the appearance in the fluid of a gossamer-like web on standing.

In the routine microscopic examination of the cerebrospinal fluid, the points to be noted are the cell count, the character of the cells, and the presence or absence of bacteria.

CELL COUNT.—The estimation of the cell count should be made first, when the fluid is as fresh as possible and before the fibrin clot has had time to form. The fluid in the test tube is thoroughly shaken for one or two minutes. The Toma-Zeiss white blood counter is used, not as in blood examination to obtain a dilution, but simply for purposes of mixing. Glacial acetic acid is drawn into the blood counter to about the 0.5 mark, accuracy not being essential, as the only purpose of the acid is to dissolve any red corpuscles which may be present, and to make the nuclei of the white cells stand out sharply. The counter is then inserted into the cerebrospinal fluid, which is drawn up until the counter is filled. It is then thoroughly shaken for several minutes, and a drop of its contents is placed on the Toma-Zeiss slide in the same way as for a blood examination. All the cells included within the ruled field are counted. Ten counts are made in this way, and the sum of these ten counts gives the cell count or number of cells in one cubic millimeter of cerebrospinal fluid.

The error caused by the slight dilution with acetic acid is so small that it may be neglected. If, however, the fluid is contaminated with blood, a variable error is introduced depending upom the proportion of blood present. If the fluid is only slightly blood-stained, the error is negligible, but if there is evidence that considerable blood has entered the fluid, the results of the cell count are useless for diagnostic purposes. The normal cell count is not over ten cells per cubic millimeter, which is only an average of one cell to each ruled field.

CYTO-DIAGNOSIS.—During the performance of the cell count, the physician can usually sufficiently differentiate the mononuclear from

the polynuclear cells to form an estimate of the predominating type. For greater accuracy the differential count may be made upon the stained specimen. A cover glass preparation can be stained with Wright's modification of the Leischman stain, or the differential count can be made on a cover glass which has been stained for bacteria. The results are recorded in percentages of polynuclears and mononuclears.

BACTERIOLOGIC EXAMINATION.—While it is advisable to take cultures from all suspected specimens of cerebrospinal fluid, the results of the examination of cover glass preparations are more valuable for diagnostic purposes than are the results of culture, because all of the bacteria commonly encountered in the cerebrospinal fluid can be found in cover glass preparations, whereas the diplococcus intracellularis is of uncertain growth in cultures. The best method of making a cover glass preparation is to wait until the fibrin clot has formed. This is removed with a platinum loop and is thoroughly rubbed and kneaded upon the surface of a cover glass. If no fibrin clot be formed, the preparation should be made from the centrifugalized sediment. For routine examination the best stain is Löffler's alkaline methylene-blue. With this the presence and morphology of all the organisms which cause meningitis can be determined, except the tubercle bacillus, which requires its special stain. The diagnosis can usually be arrived at with this stain, but for purposes of completeness, or in case of doubt Gram's stain should be used on another slide.

The *diplococcus intracellularis* is recognized by its occurrence in biscuit-shaped pairs, the cocci composing a pair lying side by side with reference to their long axis. It is decolorized by Gram's method. The *pneumococcus* occurs for the most part in pairs, but sometimes in short chains; in cerebrospinal fluid the chains are rarely more than four cells in length. The cells composing the pairs lie end to end with reference to their long axis. This organism retains the Gram's stain. The *streptococcus* in the cerebrospinal fluid almost always forms long easily recognizable chains, and shows no tendency to occur in pairs. The *staphylococcus* is recognized by its typical occurrence in clumps. Both staphylococci and streptococci retain the Gram stain. The *bacillus of influenza* is the most difficult organism to recognize. Although a bacillus, the bipolar staining is usually so prominent that the appearance of the organism suggests a diplococcus. For this reason the influenza bacillus is often mistaken both for the diplococcus intracellularis and for the pneumococcus. The bipolar dots of the influenza bacillus are smaller than either of these two varieties of cocci. Also in most specimens, while many forms resemble a diplococcus, involution forms are usually present. In these involution

forms the morphology is distinctly that of a bacillus. They do not look like the typical bipolar forms, and are often quite large. As a result of these peculiarities, the impression given by the slide is that of a mixed infection of diplococci and bacilli, which is puzzling unless the tendency to the appearance of involution forms is remembered, when the appearances become an aid in diagnosis. The bacillus of influenza is decolorized by Gram's method. In case of doubt cultures should be made upon blood agar and plain agar, as the influenza bacillus only grows in the presence of hemoglobin.

The following points in the bacteriological diagnosis of the cerebrospinal fluid are the results of a very wide experience with cerebrospinal fluids, and while not strictly scientific, may prove of value: Whenever diplococci are very few and difficult to find, the evidence points toward the intracellularis. Whenever there are many organisms included within the leucocytes, the evidence points toward the intracellularis. Whenever diplococci are very numerous, although few are included within the leucocytes, the evidence points toward the pneumococcus.

The stain for the tubercle bacillus is precisely the same as that used in the sputum, urine, and other suspected materials. Cover the preparation with carbolfuchsin solution and steam over a flame for thirty seconds, not allowing the staining solution to dry on the glass, but adding more stain if necessary. Wash in water, and then decolorize for twenty seconds with 20 per cent. sulphuric acid. Wash in water, and then wash in 95 per cent. alcohol until no more color will come out. Wash in water. Cover the preparation with Löffler's methylene-blue solution for thirty seconds. Wash in water and mount. Under the microscope the tubercle bacilli are bright red, while the nuclei of the cells are blue.

The finding of the tubercle bacillus in the cerebrospinal fluid is often very difficult. If the organism is not found in a preparation made from the fibrin clot, it is advisable to digest the clot in artificial gastric juice, and then centrifugalize thoroughly. The fluid from the bottom of the centrifugal tube is put upon the slide and allowed to dry in air.

The following table shows the characteristics of cerebrospinal fluid in some of the most common conditions, in which they are of diagnostic significance. The term meningismus as used in the table refers to a condition of meningeal irritation from a variety of causes without infection.

SPECIAL TESTS.—There are a number of special tests which can be applied to the cerebrospinal fluid, the final value of which is still under investigation. These tests depend upon the fact that in the normal cerebrospinal fluid the amount of protein present is too small to be

recognized by ordinary clinical methods, whereas in all inflammations of the meninges there is an increase of protein substances, among which the globulin is most used in tests. These tests have as their chief advantage over the simple routine method of examination described above that they are more delicate. Among the principal ones are to be mentioned Nonne's test, Noguchi's butyric acid test, Lange's colloidal gold test, and Boveri's permanganate of potash test. The technique of these tests is too complicated for ordinary clinical use. At present their chief place is in the research laboratory. They will not be described here, but the reader is referred to recent literature for a description of their technique and diagnostic value.

EXUDATES AND TRANSUDATES.—The laboratory examination of effusions into the pleural, peritoneal, and pericardial cavities is often an important diagnostic procedure.

APPEARANCE.—First note the appearance of the fluid, and in particular whether it is clear, cloudy, or purulent. The color of serous fluids should be noted, whether pale, straw color, orange, red, or bile stained. The presence or absence of fibrin formation should also be noted.

SPECIFIC GRAVITY.—Take the specific gravity of all serous fluids. This is important in distinguishing between exudates and transudates.

ALBUMEN.—The fluid should be tested for albumen, and the per cent. of albumen present should be estimated by some quantitative method. It can be estimated roughly from the amount of sediment in the heat test as described for the urine. A more accurate estimate may be made with Esbach's graduated tubes.

THE SEDIMENT.—The sediment should be examined microscopically, and the character of the cells and other bodies should be noted.

CYTO-DIAGNOSIS.—Place the fluid in clean centrifuge tubes and centrifugalize thoroughly. Pour off the supernatant fluid by inverting the tube, and stir up the sediment in the remaining drops of fluid with the platinum loop. Spread a drop of this mixture on a cover glass and allow it to dry spontaneously without heat. Stain with a fluid made up of Wright's modification of Leischman stain three parts, and pure methyl alcohol one part. The slide is stained by the method described for the examination of the stained specimen of blood. The same method as for blood is used in making a differential count of the cells.

BACTERIOLOGICAL EXAMINATION.—Bacteria are stained by the solution used for cyto-diagnosis. In case of doubt as to their morphology, another slide may be stained with Löffler's methylene-blue solution. Further evidence as to the type of microörganism present

TABLE 15

Diagnostic Characteristics of the Cerebrospinal Fluid

FLUID	QUANTITY	PRESSURE	APPEARANCE	FIBRIN	CELL COUNT	PREDOMINANT CELL	BACTERIA
Normal.....	Normal	Normal	Clear	Absent	Normal 1-10	Mononuclear	Absent
Meningismus.....	Normal or Increased	Normal or Increased	Clear	Absent	Normal 1-10 or Increased 10-60	Mononuclear	Absent
Tuberculous meningitis.....	Increased	Increased	Clear, (occ. slightly cloudy)	Present (occ. absent)	Increased 30-1000 usually over 100	Mononuclear	Tubercle Bacillus
Other forms of meningitis..	Increased	Increased	Cloudy	Present	Increased usually over 300	Polynuclear	Present
Poliomyelo-encephalitis....	Normal	Normal	Clear	Absent	Increased in early stage only	Mononuclear	Absent
Hydrocephalus.....	Increased	Increased	Clear	Absent	Normal	Mononuclear	Absent
Cerebral hemorrhage.....	Normal	Normal or Increased	Red or Yellow	Absent	Normal	Mononuclear	Absent

may be gained by staining with Gram's method as described for the sputum. The method of staining for tubercle bacillus is the same as that used for all suspected tuberculous material. Cultures should be taken from all exudates.

DIFFERENTIAL DIAGNOSIS.—The following points in the examination of serous fluid serve to distinguish exudates from transudates.

TABLE 16

Distinction Between Exudates and Transudates

	EXUDATES	TRANSUDATES
Specific gravity	1016–1026	1008–1018
Albumen	2–6%	.2–2%
Fibrin	Present	Absent
Sediment	Leucocytes	Epithelial cells

The sediment of all exudates except the tuberculous is composed chiefly of polynuclear leucocytes. Tuberculous exudates show a much higher proportion of mononuclear cells, which may predominate. Bacilli are often difficult to demonstrate in tuberculous effusions.

EXAMINATION OF THE VAGINAL DISCHARGE.—This is very important, particularly in infancy. Vaginal discharge is not uncommon in early life, and in a large proportion of cases it is due to the gonococcus. So common is gonorrheal infection in female infants, and so contagious is it that an examination of the vaginal discharge should be made as a routine measure with every infant admitted to a hospital, whether or not macroscopic discharge be visible. The material is obtained by gentle swabbing of the vulva, and is spread on a cover glass, dried, fixed in the flame, and stained. The following method of staining should be employed: Cover for one minute with Stirling's gentian violet, or with anilin gentian violet. Wash in water. Apply Gram's iodine and iodide of potash solution for thirty seconds. Wash in water. Wash in 95 per cent. alcohol until the blue color ceases to come out. Wash in water. Counterstain with a solution of carbolfuchsin diluted 1 to 8, without heat, or with a saturated aqueous solution of Bismark brown. Wash in water and mount. All diplococci within the leucocytes which have not retained the blue stain, but have taken the red or brown counterstain are to be considered gonococci.

EXAMINATION OF THE SPUTUM.—The examination of the sputum does not hold so important a place among diagnostic procedures in infancy and early childhood as it does in adult life. Infants and young children habitually swallow their sputum, and it is consequently much more difficult to obtain a specimen of sputum for examination. Not only that, but the evidence obtained from the sputum examination in children is not so valuable as in adults. The forms of tuberculosis which affect young children are not the phthisis of

adults, and do not show tubercle bacilli in the sputum with anything like the same frequency as does phthisis. The bacterial picture shown by the sputum of even lobar pneumonia in children is not so clear cut and definite as it is in adults. For these reasons the examination of the sputum cannot be called a necessary routine measure. In the pulmonary affections of early life it is often however of diagnostic value, and should be employed in all cases of doubtful diagnosis. The specimen of sputum for examination can best be obtained from the pharynx. The examination of the swallowed sputum expelled in the vomitus or obtained from the stomach, is not satisfactory. In obtaining sputum, cough should be excited by irritation of the pharynx. This causes sputum to be brought into view and it can be caught upon a cotton swab. It is easy to obtain sputum in this way, but the value of the examination is lessened by the fact that the physician can never be sure that the sputum comes from the lungs and is not sputum which has trickled down from the naso-pharynx.

Macroscopic examination is of little diagnostic value in children. The sputum should be stained separately for the tubercle bacillus and for other organisms.

STAIN FOR THE TUBERCLE BACILLUS.—In preparing a cover glass for the tubercle bacillus stain, select if possible purulent or cheesy particles. These can be removed by means of a stiff platinum wire, and should be smeared as thoroughly as possible upon a cover glass. The preparation held in the forceps is dried over the flame of a Bunsen burner and fixed by being passed three times through the flame. Cover the preparation with carbolfuchsin solution and steam over the flame for half a minute, adding more stain if necessary, to prevent the staining solution from drying on the surface of the glass. Wash in water. Decolorize for twenty seconds in 20 per cent. sulphuric acid. Wash in water. Wash in 95 per cent. alcohol until no more color will come out. Wash in water. Cover the preparation with Löffler's methylene-blue solution for thirty seconds. Wash in water and mount. Tubercle bacilli are stained bright red, nuclei and other bacteria being blue.

ROUTINE STAIN FOR OTHER ORGANISMS.—The cover glass should be smeared with sputum as thinly as possible. It is dried and fixed in the flame by the usual method. Cover with Stirling's gentian violet or anilin gentian violet, and heat over the flame until the stain just steams. Wash in water. Cover with Gram's iodine and iodide of potash solution for thirty seconds. Wash in 95 per cent. alcohol until the blue color ceases to come out. Stain with Löffler's methylene-blue solution, heating to the steaming point. Wash in water, dry, and mount.

By this method of staining the pneumococcus will be stained blue-

black, and its characteristic morphology can usually be recognized. The capsule will not stain, but the various special methods for staining capsules do not give constant results and are not necessary in routine examination. The influenza bacillus will be stained blue, and the fact that it does not take the Gram stain is usually apparent. The influenza bacillus is the smallest organism found in sputum. Owing to the bipolar staining it usually gives the impression of a very small diplococcus. In case of doubt as to the results of the Gram stain, a second preparation may be stained by Gram's method, and counterstained with Bismark brown instead of Löffler's blue. The bacillus mucosus capsulatus can generally be recognized by its morphology. If doubt exists, if a specimen is stained with carbolfuchsin, the capsules will show. There are various methods of staining the sputum which give prettier preparations, but they are rather complicated for routine use.

EXAMINATION OF THE GASTRIC CONTENTS.—This procedure is less valuable as a means of diagnosis in the diseases of early life than in those of adult life. Organic disease of the stomach is so rare in childhood that it hardly enters into consideration. In the various functional disturbances so common in infancy and childhood, the examination of the gastric contents throws little light upon the diagnosis. The whole etiology and classification of gastro-intestinal disease is entirely different in infancy and childhood from that of adult life, and calls for entirely different methods of diagnosis. There are almost no conditions met with in children in which there is an absence of free hydrochloric acid.

BLOOD.—The most common test called for in examining the vomitus or gastric contents of infants and children is that for blood. The guaiac test should be used as described under the examination of the faeces.

FREE HYDROCHLORIC ACID.—This may be tested for by the same tests as are used in adults, such as Gunzburg's, Töpfer's and ∞ tropaeolin. These tests are so rarely required in children that they will not be described here, and the reader is referred to the various adult text-books.

WIDAL REACTION FOR TYPHOID.—This procedure has the same diagnostic significance in children as in adults.

Collect three or four drops of blood in a small test tube, and let it stand until the serum is exuded. When dried blood is employed, it must be mixed with a few drops of water, this mixture taking the place of serum. In making the test, use a bouillon culture of typhoid bacillus from twelve to twenty-four hours old. Examine a drop of this culture under the microscope in order to make sure that the bacilli are unclumped and in active motion. Mix nine drops of the typhoid

FIG. 40—Scarifying for von Pirquet tuberculin test

culture with one drop of blood serum upon one end of the slide. Upon the other end of the slide make a 1 to 50 dilution by mixing one drop of the 1 to 10 dilution with four drops of water. Drops of equal size are obtained by using a platinum loop. Cover both the 1 to 10 and 1 to 50 dilutions with cover glasses. Make controls in the same way except that the blood serum is omitted. The serum reaction is regarded as positive when before the end of one hour there is complete clumping of the bacilli, and an absolute cessation of motility in both dilutions, the controls remaining unclumped. The reaction is regarded as suggestive but not positive when only the 1 to 10 dilution is agglutinated. A positive reaction is diagnostic evidence of typhoid infection or antityphoid vaccination.

THE VON PIRQUET REACTION FOR TUBERCULOSIS.—The tuberculin reaction and its theory, the various methods of applying it, and its diagnostic value, are discussed in detail under tuberculosis. For routine use, von Pirquet's cutaneous test is the one to be employed. Wash the skin of the forearm by rubbing with alcohol and ether, and apply two small drops of Koch's old tuberculin undiluted, about 10 cm. apart. A scarifier resembling a tiny chisel which has been sterilized by heating, is used for the inoculation. Apply this first to the skin between the tuberculin drops and rotate with a boring motion. Repeat this procedure on the skin through each of the tuberculin drops. The boring must be sufficient to remove only the superficial layers of the skin. The blood-vessels of the deeper layers should show through, but blood should not be drawn. As a precaution against rubbing, cover the site of the inoculation with a piece of sterile gauze. A positive reaction is shown by the appearance of reddened areolae about the two points of the inoculation, while no reddening is seen about the control. The reaction usually begins within twenty-four hours, reaches its height during the second twenty-four hours, and slowly fades in one or two days. If the areola is less than 5 millimeters in diameter, the reaction should be considered as doubtful rather than positive. In my experience, however, with infants, based on the results of autopsies, a doubtful reaction usually means tuberculosis.

In infants and children a negative reaction has no diagnostic significance. A positive reaction means tuberculosis in the great majority of cases.

CUTANEOUS TEST FOR FOOD IDIOSYNCRASIES.—The technique of this test is exactly the same as that of the cutaneous tuberculous test, except that a small bit of the suspected food substance is used in place of the tuberculin. Food idiosyncrasies are not rare in infancy. The idiosyncrasy most commonly shown is against some protein food substance, such as lactalbumen, egg albumen, or

casein. Whenever there is a history of symptoms following the ingestion of some particular food substance, a cutaneous test should be employed. A positive reaction indicates that the symptoms are caused by a food idiosyncrasy toward the special substance.

WASSERMANN REACTION FOR SYPHILIS.—The complement fixation test of Wassermann is of the greatest value in the diagnosis of syphilis in early life. The technique of the test is altogether too complicated for routine use, and the test can only be satisfactorily made by one trained in its performance. There are

FIG. 41

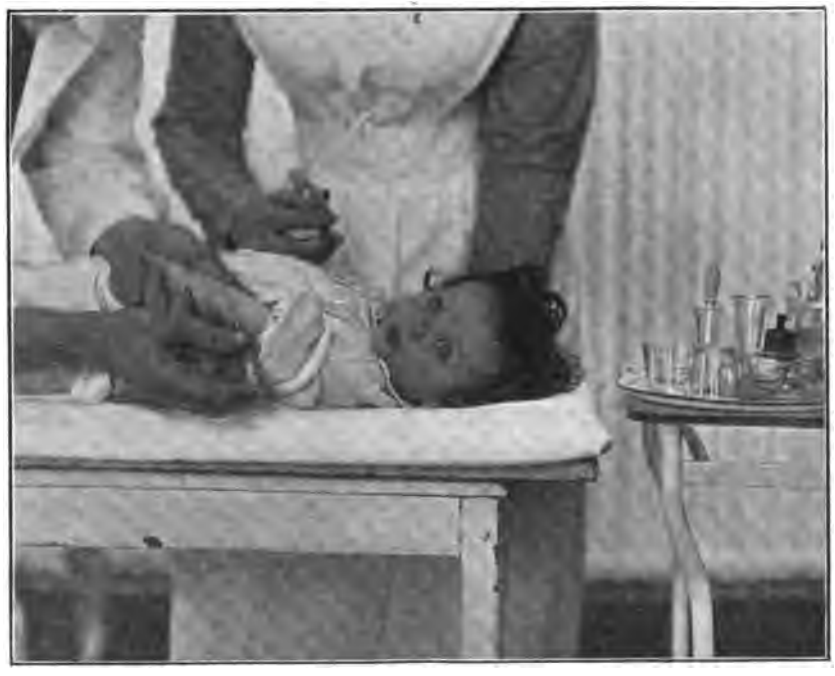
Obtaining blood for the Wassermann reaction from the longitudinal sinus of an infant

however a certain number of laboratories where this test is being habitually made, and in every case of suspected syphilis, a specimen of blood should be sent to a Wassermann laboratory for examination. About 10 c.c. of blood should be required for the test, and it is difficult to obtain so much blood from infants by the ordinary methods.

The best method of obtaining blood from infants for examination is to take advantage of the open fontanelle. The longitudinal sinus which lies immediately under the anterior fontanelle is a very large vein which can be very easily reached. A small Luer syringe and hypodermic needle are sterilized, and the skin over the anterior

fontanelle is cleansed. A small area may be shaved if there is much hair. The needle attached to the syringe is entered at the posterior angle of the fontanelle exactly in the median line. The needle passes through the skin and dura with a perceptible sense of suddenly lessened resistance. The piston of the syringe is then drawn, and the syringe immediately fills with blood. This method of obtaining the blood is greatly preferable to the gashing of the ear or heel, which was necessary for obtaining enough blood for the Wassermann reaction. My assistant at the Infants' Hospital recently obtained in this way specimens of blood from ten babies in eleven minutes. The specimen in a sterile test tube should be tightly corked before being sent to the Wassermann laboratory.

FIG 42



Injecting diphtheria toxin for the Schick reaction

THE SCHICK REACTION TEST FOR IMMUNITY AGAINST DIPHTHERIA.—This test is mainly useful in hospital practice, but it is sometimes useful in private practice in determining whether a prophylactic dose of diphtheria antitoxin is indicated in children with whom there has been exposure or suspicion of exposure to diphtheria. Its value as a reliable test of immunity has been confirmed by the majority of recent investigations, although it is perhaps not finally proved.

Diphtheria toxin is used for the test. The minimum lethal dose of the toxin for a guinea pig weighing 300 grams must be known. This is so diluted that 0.1 c.c. of fluid contains 1/50 of this minimum lethal dose. This is injected with a very small needle, not under the skin but into the skin. If the injection is properly made, it should cause a small raised white looking spot. If the reaction is negative, there is no further discoloration at the site of the injection. If the reaction is positive, the area within twenty-four hours becomes reddened and slightly edematous. This passes off in forty-eight hours, leaving a brownish pigmentation. A positive reaction indicates that the individual has less than 1/30 of a unit of natural antitoxin in 1 c.c. of blood, and such individuals are to be considered susceptible to diphtheria. A negative reaction indicates that the individual has more than 1/30 of a unit of natural antitoxin in 1 c.c. of blood, and is immune to diphtheria. The reaction has also a diagnostic value, because children sick with diphtheria show a positive reaction before antitoxin is given.

It has been shown that most newly born infants are protected by natural antitoxin, and that about 57 per cent. of children in the first year of life, and 37 per cent of children between two and five years, are immune.

THROAT CULTURES.—The chief purpose for which cultures are taken from the throat is in the diagnosis of diphtheria. A certain amount of evidence can also be gained as to other infecting organisms, not only by the examination of cultures from the throat, but also from examination of cover glass preparations made from the material obtained by swabbing.

Cultures should be taken from all cases in which the examination of the throat shows the presence of exudate or membrane. The swab should be applied to the edge of the membrane and should be drawn rather forcibly across the suspected area. The swab is then rubbed upon the surface of a blood serum culture medium. The culture should be incubated for twelve hours, and then examined by means of cover glass preparations. The best routine stain is Löffler's methylene-blue solution. The diphtheria bacillus is recognized by its peculiar morphology.

BLOOD CULTURES.—In a great number of infectious conditions in infancy and childhood, the bacteria enter the blood stream. Cultures from the blood are often very valuable in diagnosis, but this method is not employed as much as it should be on account of the supposed difficulty of obtaining blood from infants and young children under proper sterile conditions. The introduction of the method of obtaining blood from infants from the longitudinal sinus has very much simplified the technique of blood cultures. Exactly the same

procedure is employed as in obtaining blood for the Wassermann reaction. In older children with closed fontanelles, the blood can be obtained from one of the veins of the arm, which can be made to stand out by a tourniquette. The blood obtained should be inoculated upon the various culture media, according to the resources of the bacteriological laboratory. For routine use, inoculations should be made in bouillon and upon blood agar. The special method of examining blood cultures for typhoid will be described under that disease.

PHENOLSULPHONEPHTHALEIN TEST FOR RENAL FUNCTION.—Within the last few years various methods of testing renal function have been introduced. These tests have appeared to be of notable diagnostic value in many diseased conditions of the kidney. The most useful single test among them is that with phenolsulphonephthalein.

Under aseptic precautions, the patient is given an intramuscular injection of 1 c.c. of a sterilized solution containing 6 mgm. of phenolsulphonephthalein. Any urine passed during the next two hours is saved, and at the end of that time the patient is made to empty his bladder, or is catheterized. All the urine collected in this way is made definitely aklaline, and is diluted to make 1 liter. A standard solution of alkaline reaction, containing 6 mgm. of phenolsulphonephthalein in 1 liter, is made up. Some of the urine dilution is put in a test tube, and a series of test tubes of equal diameter are made up containing various dilutions of the standard solution. In this way, by colorimetric readings, the amount of the drug excreted in the urine can be estimated. More accurate results can of course be obtained by the use of Antenreieth-Königsberger or other type of colorimeter.

With normal renal function, 60 to 80 per cent. of the drug should be excreted in two hours. If the amount is less than this, a disturbance of renal function is indicated, and the degree of the disturbance is measured by the amount of the decrease.

DIFFERENTIAL DIAGNOSIS

When all the evidence which can be obtained from the history, physical examination, and laboratory investigation is at hand, the physician is confronted with the problem of diagnosis. In certain cases, there is something in the evidence which is so positively diagnostic, that a decision can be arrived at at once. Care must be taken, however, not to base a positive diagnosis upon symptoms or signs whose presence or absence is not absolutely diagnostic. Care must be taken also not to overlook the possibility of more than one pathological condition.

In other cases, the evidence obtained suggests only a number of possibilities, without anything positively diagnostic of any one of

them. In these cases a differential diagnosis must be made through a careful weighing of the evidence. In making a differential diagnosis, it is unnecessary to consider every possibility that could go with every symptom. The best method of arriving at a conclusion is for the physician to select the most prominent symptom or group of associated symptoms, which demand explanation. He should then make a written or mental list of all the conditions which could produce this group of symptoms. He should then consider how many of these possibilities can be positively excluded by the different kinds of diagnostic evidence at hand or obtainable. If more than one possibility be left, he should arrange these alternatives in the order of their probability as suggested by the evidence.

Care must be taken in excluding possibilities in diagnosis. A distinction must be drawn between clinical evidence which positively *excludes* a certain condition, and clinical evidence which is only *against* a certain condition. The latter class of evidence can only be used in arranging possibilities in the order of their probability.

When an opinion is formed as to the diagnosis, the evidence should again be reviewed with reference to whether the diagnosis is adequate to account for all the symptoms and signs of disease which are present. If it is not adequate, the possibility of two things must be considered; one is that some false step in the reasoning has led to a mistaken diagnosis; the other is that more than one diseased condition may be present. The clinical evidence must be reconsidered with a view to settling these alternatives.

The ability to make an accurate diagnosis in early life depends upon the weighing of the clinical evidence. If the standards used in diagnosing disease in adults are used, many mistaken diagnoses will be made with children. The physician must be thoroughly familiar with the anatomy, physiology, and development of the normal child, and with all the peculiarities of the pathology, symptomatology, and diagnostic methods of childhood.

IV. PROGNOSIS IN EARLY LIFE

The most important factor in the prognosis of disease in early life is the age of the patient. The general rule is that the younger the patient, the worse is the prognosis in all the diseases of childhood. It is for this reason that the mortality of the first year of life is so high, and that it shows a progressive diminution throughout the remaining years of childhood. I shall not take time here to review the much discussed question of infant mortality, nor to show in detail the relative frequency of the different causes of death at the different ages.

The causes of the high mortality of infancy may be summarized as follows: In the first place, there is the feeble resistance of the infantile organism in general to all the external causes of disease. This feebleness of resistance is shown not only against those external causes which produce disturbance of function, but also against infections. In the second place, the feeble resistance of the undeveloped organism may be increased both by inherited conditions and by congenital faults of development. A third cause is the prevailing ignorance as to the hygienic demands of the young organism and the conditions which are essential to its proper health and growth.

In the first year of life, gastro-intestinal conditions rank first as a cause of death, with acute respiratory tract infection second, congenital malnutrition third, and other acute infections fourth. In the second year, diseases of the gastro-intestinal tract still hold first place. It is difficult to draw a distinction between acute infections of the respiratory tract and the acute contagious diseases, because death so often occurs when the former is a complication of the latter. Measles, diphtheria, and pertussis, with their pulmonary complications appear to hold second place, but in my opinion, the statistical figures of infant mortality show altogether too low a proportion for acute tuberculosis and tuberculous meningitis. After the second year, the acute infections rank ahead of all other causes.

In considering questions of prognosis, it must be remembered that in general, infants and young children succumb easily to acute conditions. They also show much more markedly the effects of chronic functional disturbance. On the other hand, numerous diseases accompanied by organic lesions in early life, show a remarkable possibility of complete recovery as compared with adult life. Many organic conditions which in adults would be permanent can in children be "outgrown," provided that the patient can be placed under proper hygienic surroundings. This power of recovery from organic

disease is due to the peculiarly rapid metabolism of childhood. There are also a certain number of infectious diseases which are less severe in infants than in adults, owing to certain peculiarities of the anatomy of early life.

In children, it is lack of development and peculiarities of functional resistance which form the unknown factor in prognosis, just as in adults it is the effects of all abuses, such as alcohol, and various forms of wear and tear which constitute the unknown factor. The prognosis in any diseased condition can only be estimated from a knowledge of the peculiarities of the particular disease in early life, and from an estimate of the constitutional peculiarities of the individual patient. In children of apparently robust constitution and normal development, the prognosis in those acute diseases which do not tend inevitably to a fatal ending, is fairly good. In children of poor development and poor constitutional resistance, the prognosis is proportionately bad. In chronic functional disturbances, the prognosis depends largely upon the length of time which the disease has existed, and the degree of malnutrition and lowered resistance which has been brought about.

In giving a prognosis in early life, the physician must remember two things: One is the liability in infancy to sudden and unexpected death from various unsuspected causes. The other is the liability of infants and children to develop unexpected complications. The prognosis of the various diseased conditions as compared with the same conditions in adults, will be discussed in detail in the consideration of the various diseases.

V. TREATMENT

GENERAL PRINCIPLES

The one great principle in the modern treatment of disease is removal of the cause. This is particularly applicable to the diseases of infancy and childhood. A very large group of the disturbances to which children are particularly liable belong in the class of functional disturbances from multiple external causes, and tend toward spontaneous recovery as soon as the causes are found and removed. Not only are these diseases curable through timely removal of the causes, but they are still more easily preventable. A second large group is represented by the infections, in which the cause, while not removable after infection has taken place, is nevertheless also often preventable.

Prophylaxis is therefore the most important department of the treatment of disease in early life. There is no more promising field in the whole of medicine than the prevention of disease in childhood. The fundamental principles in preventive medicine are two in number: (1) The removal of the external factors in hygiene and environment which interfere with normal development, and contribute to disease; (2) the prevention of infection. The first principle can be applied only by the education not only of the medical profession, but also through them of the general public, in the proper hygiene of early life. The second can be realized through continued study of the mechanism of infection and the principles of immunity on the one hand, and of such problems as disinfection, sanitation, and quarantine on the other. In these last problems the public health authorities have their great field.

When disease has actually occurred, the true diagnostic problem which confronts the physician is not the mere finding of a name for the condition, but the finding of the cause. No name is of any value which does not connote all that is known of etiology, and nothing is worse than for a physician to hide ignorance of etiology and pathology under vague names like grip, marasmus, rheumatism, biliousness, and so forth, which are not based on distinct etiological conceptions. Taking refuge in names which have a definite if incorrect signification to the general public may satisfy the parents of our patients, but should not satisfy us, unless we go further, so that our diagnosis suggests all that is known of etiology. It is for this reason that so much stress is laid upon etiological factors throughout this book.

Modern treatment is primarily aimed, not at the mere relief of symptoms, but at the cause. This does not mean that symptomatic treatment does not have its place. It is obviously our duty to relieve symptoms as far as possible. But symptomatic treatment is longer established, better known, and easier of application than is treatment aimed at causes, and there is danger that we may lose sight of the latter in self-satisfaction at our ability to apply the former.

SPECIAL INDICATIONS IN EARLY LIFE

The peculiarities of disease in childhood which modify the treatment are chiefly due to the incompleteness of anatomical and functional development at that period of life. Three groups of conditions besides the acute infections are particularly common in childhood. These are (1) functional disturbance of the digestive system, (2) functional disturbance of the nervous system, and (3) functional disturbance of the general nutrition and metabolism. Even in other diseases, such as the infections, these three varieties of functional disturbance are particularly common as manifestations. The treatment required by these three kinds of disturbance is mainly hygienic and dietetic, and consequently these forms of treatment are relatively of the first importance in childhood.

On the other hand, the great variety of subjective symptoms which demand treatment in the adult are much less common in childhood. Consequently symptomatic treatment directed at such manifestations plays a relatively unimportant part. Moreover, certain important systems of the body suffer less from disease in children than in adults. The chief example of this difference is the heart and circulatory system, which is most damaged by the wear and tear of the adult life, and consequently often calls for symptomatic treatment, but which is relatively competent to combat disease in childhood. Drug treatment plays its most important part in combatting subjective symptoms and circulatory disturbance. The rôle of drugs is consequently relatively smaller in early life.

Treatment may be divided into *specific*, *hygienic*, *dietetic*, and *symptomatic*.

SPECIFIC TREATMENT

In the infectious diseases, the first aim in therapeutics has always been to find some remedial agent which will act directly on the cause of the disease. This effort has led to much study of the mechanism of transmission and infection, of the mechanism of defence, and of the complicated problems of immunity. It has been finally demonstrated that the human organism has various means of defence against the occurrence of infection, and various means of combatting infection when once it has occurred. These natural methods of

combatting infection are the cause of the self-limited character of many acute diseases, and of the fact that in most infections there is a natural tendency toward eventual recovery.

The first object of treatment is to hasten recovery—to overcome the infection as quickly as possible. In the search for therapeutic measures which are *specific*, that is which act directly on the particular microörganism causing the disease, attention was first turned to *drugs*—chemical agents which when introduced into the body, would kill or injure the microörganisms or prevent their development. The idea of the possible value of drugs in combatting infection was based on the fact that many drugs have a demonstrable pharmacological action, modifying and influencing various bodily functions in various ways, and as the symptoms of infection are due to a disturbance of bodily function, a drug which caused the opposite effect, which lessened the disturbance of the function, was considered of value against the microörganism causing the trouble. It has been found, however, that *in-so-far as the bacterial infections are concerned, there is no drug which can be safely introduced into the body which will act directly on the microörganisms causing the disease, and that consequently, there is no specific drug therapy for the diseases caused by bacteria.*

It has been supposed that the influence of drugs upon bodily function might be utilized in bringing about recovery, through the finding of drugs which aid the natural methods of resistance, defence, and disinfection. A drug which has such a power would be considered specific. It has been found, however, that the natural methods of recovery from bacterial infections depend upon much more complicated biological processes than the simple physiological functions which can be influenced by drugs. The most that drugs can do in infections is to strengthen and support functions whose disturbance threatens a fatal ending before the natural methods of defence have time to act, or to aid in the promotion of the general resistance of the body against adverse influences. The action of drugs in bacterial infections is therefore in no way specific.

The same is true of the various non-medicinal therapeutic measures. Their rôle is the relief of subjective symptoms, the support of threatened function, and the strengthening of general resistance.

For a true specific therapy in the bacterial infections, we must look further. The study of the natural methods of defence, and of the complicated subject of immunity, has given us two methods of aiding or hastening the natural methods of recovery. These are *serum therapy*, and *vaccine therapy*.

SERUM THERAPY.—This consists in the introduction into the patient's circulation of the serum of an animal which has been immunized by inoculations with the bacterium causing the disease.

Immunological research has demonstrated that one of the methods of defence through which recovery from infection takes place, is the formation in the blood serum of the patient of *immune bodies*. These immune bodies are of various kinds; some neutralize the toxin formed by the bacteria (antitoxins), others kill the bacteria (bactericidal substances), others prevent the multiplication of bacteria, and others injure the bacteria in such a way that they fall an easy prey to phagocytic tissue cells (opsonins, or bacteriotropins). If an animal be inoculated with bacteria, or preparations made from bacteria, under proper conditions, its tissues will be stimulated to the formation of immune bodies, which will be contained in its blood serum. The therapeutic use of such immune animal sera is serum therapy.

The most conspicuous example of success in serum therapy is *diphtheria*. This success led to the hope that a similar specific therapy would be applicable to all the infectious diseases. This hope has not been realized. The immune body in diphtheria is an antitoxin, and it has been found that only in infections with an organism which secretes a soluble toxin as does the diphtheria bacillus, can an effective antitoxic serum be obtained through animal inoculation. The only other effective antitoxic serum is that produced by animal inoculation with the bacillus of *tetanus*. In the other infections, the organisms do not secrete a soluble toxin, but liberate endotoxins, and the mechanism of immunity is much more complicated. Hence serum therapy has made slow progress.

The most successful example of serum therapy besides diphtheria, is in *epidemic cerebrospinal meningitis*. Immune sera have been obtained and widely employed against infection with the *streptococcus*, the *pneumococcus*, the *bacillus of dysentry*, the *bacillus of influenza*, and the *tubercle bacillus*. Most of these sera have given strong laboratory evidence of value. Some of them have given some clinical evidence of value, but on the whole, in these infections, serum therapy cannot be said to have proved markedly successful.

The reasons for the failure of complete success of serum therapy against the infectious diseases, may be summarized as follows:

1. In some diseases certainly infectious, the specific organism has not been demonstrated. Examples are scarlet fever, measles, rubella, varicella, mumps.

2. Some diseases cannot be sufficiently reproduced in animals to permit the production of an immune serum. Examples, typhoid fever, gonococcus infection.

3. In some diseases immunity depends on tissue reactions of a very complicated nature, in which the formation of immune bodies in the blood serum probably plays a very small part. Example, tuberculosis.

4. In some diseases the specific organism has been so recently discovered, that there has not been sufficient time to investigate the problem of immunity, and to build a satisfactory foundation for serum therapy. Examples, pertussis, poliomyeloencephalitis.

5. In some infections the specific organism does not represent a single type, but includes a variety of strains, each with its particular immune reaction of varying complexity. Much time will be required for the study and differentiation of these strains and their immune reactions, before an effective serum therapy will become practical. Examples are the pneumococcus, the streptococcus, rheumatic fever.

It will be seen from the causes of failure up to the present time, that the outlook for further progress in serum therapy is by no means hopeless or discouraging. The majority of the difficulties are of a character to suggest that they are capable of solution. It is for this reason that among the various fields of medical research, the greatest amount of activity is found in the study of infection and immunity. We must look mainly to the results of this study and research for further advances in specific therapy.

VACCINE THERAPY.—This consists in the inoculation of the patient with a bacterial preparation with a view to stimulating the patient's tissues to the production of immune bodies, or to an immune reaction.

Immunological studies have demonstrated that when bacteria are killed by heat, the substances contained in the bacterial cells which excite the formation of immune bodies in the patient, are not destroyed. In some instances the injurious endotoxin is not the same as the antigens, or substances which excite antibody formation. It is mainly the multiplication of bacteria in the body which causes harm, and this is prevented by the method of preparation. The majority of vaccines consist of suspensions of dead bacteria in normal salt solution, the dose being measured by the number of dead bacteria given. In using a vaccine, the number of dead bacteria in a certain quantity of the solution must be known, in order to regulate the dose. These vaccines are injected subcutaneously.

The tuberculosis vaccine known as tuberculin is an extract of the tubercle bacillus, and the dose is measured in milligrams. The vaccine used against small-pox is the living unknown virus of cow-pox, and is inoculated into an abrasion of the skin.

The chief value of vaccine therapy is prophylactic rather than curative. The efficacy of the inoculation of bacterial products in producing an active immunity against certain diseases, has been established beyond the possibility of a doubt. Except in the case of vaccination against small-pox, prophylactic vaccination has at present a very small place in pediatrics. It is only under exceptional

circumstances of epidemic and exposure that prophylactic inoculation is indicated in childhood.

The chief use of vaccine therapy in early life is as a curative measure. Vaccine therapy has been tried in almost every known form of bacterial infection. In general, the results have been disappointing. In those general infections, in which there is a profound disturbance of the physiology of the entire body, such as pneumonia, typhoid fever, streptococcus and staphylococcus septicaemia, ulcerative endocarditis, and the various forms of meningitis, the clinical evidence of good results is not sufficient to justify the routine use of vaccine therapy. There is evidence of a good effect in some individual cases, but it must be remembered that in these diseases the body is already inoculated and overtaxed with toxic substances, and it is difficult to see how good may be expected from adding to the burden by artificial inoculation.

In certain strictly localized infections, on the other hand, there is abundant clinical evidence that the infectious process can often be favorably influenced by bacterial inoculations. The most conspicuous examples are the various localized *staphylococcus* infections, of which furunculosis is typical. But furunculosis represents really a series of infections, rather than a single one, and the effect of vaccine therapy is probably chiefly as a prophylactic against recurrent infection. The more nearly a localized infection approaches a type resembling furunculosis, the more promising is the outlook for vaccine therapy. In other infections, not so strictly localized, but intermediate in character between the widespread and limited types, such as pyelitis, gonococcus infection, certain forms of arthritis, and so forth, vaccine therapy must be considered as a possibility, of which the value is not yet either established or disproved.

In the selection of the vaccine, the physician must choose between the employment of an *autogenous vaccine* or a *stock vaccine*. An autogenous vaccine is one prepared from a culture taken from the patient and contains the particular strain or type of infecting organism that is producing the disease. The researches of recent years have shown that one of the chief elements leading to chronicity and resistance in an infectious process, is a change in the infecting organism by which it becomes less susceptible to the attack of the immune bodies of the host. Artificial active immunization, if it is to be effective, must take cognizance of such elements as bacterial resistance and "organ specificity," and consequently a vaccine should be derived from an organism possessing the properties of those concerned in the infectious process—an autogenous vaccine. The technique of the preparation of autogenous vaccines varies somewhat with the type of organism concerned, and for the details the reader is referred to standard works on bacteriology.

Stock vaccines are prepared from stock cultures. The advantage attending their use is only that they are more easily obtainable. There is evidence that in some types of infection stock preparations are effective. We cannot, however, be sure of the type of infection without a culture, and if a culture is obtainable, the preparation of an autogenous vaccine should be possible. Even if we grant that a correct bacteriological diagnosis can be made from clinical observation, stock vaccines have the further disadvantage that they may not meet the final requirements of the particular strain of organism in the individual case. This disadvantage may be partly overcome by uniting bacteria from different strains of the same species in a polyvalent vaccine. The usual reason however for the employment of a stock vaccine is that the physician is not master of the technique of the preparation of an autogenous vaccine. While their use under certain circumstances may be justifiable, it encourages loose thinking and neglect of the fundamental principles on which vaccine therapy is based.

The use of "mixed vaccines," or of non-specific mixtures of the products of bacterial growth, cannot be too strongly condemned. Such "shot-gun" empiricism in matters so important as infection and its treatment is not only wholly unscientific, but stands an excellent chance of being actually extremely dangerous.

The indication for vaccine therapy and its method of employment will be considered under the various diseases in which it holds a place as a prophylactic or therapeutic measure.

SPECIFIC MEDICINAL TREATMENT.—Failure of drugs as a specific weapon against infectious processes applies to the bacterial diseases only. In infections produced by animal parasites such as protozoa and others, there is evidence that drugs may have a specific value against the pathogenic invader. In pediatrics there are two diseases of this class, malaria and syphilis. The use of quinine in malaria, and of mercury, salvarsan, or iodide of potassium in syphilis, may be considered as specific treatment.

HYGIENIC TREATMENT

In those diseased conditions which are directly due to faulty hygiene, hygienic treatment may be considered as specific. It holds also a very important place in all the diseased conditions of early life. The resistance of the organism against adverse influences of all kinds, whether they be infections, or whether they be due to constitutional peculiarities or external injuries, is largely dependent upon the normal development and general health of the child. Proper hygiene is essential to the proper development of a constitution which is able to resist disease. For this reason hygienic measures have their place in practically all diseases.

The details of hygienic treatment have been described at length in the first division of this book under the heading of "The Hygiene and Care of Normal Children." The physician should always devote the greatest pains to discovering whether there are any faults in the hygiene and environment of his youthful patient, and should correct such faults. It is always the first essential to place the patient in proper hygienic surroundings. The particular hygienic measures which are demanded by certain special diseased conditions will be described under the several divisions.

DIETETIC TREATMENT

A large group of the diseased conditions encountered in infancy and early childhood are due to the relatively low resistance of the functions of digestion and metabolism against adverse influences. In this group, treatment is mainly dietetic, and here, dietetic treatment may be considered specific. The susceptibility of the gastro-intestinal system in early life must be taken into consideration also in all the diseased conditions of childhood, and consequently the proper arrangement of the diet plays a very important part in the therapeutics of early life. The details will be considered at length in this book in the divisions on Feeding and on the Gastro-Intestinal Diseases.

SYMPTOMATIC TREATMENT

This is directed at the various functional disturbances which appear as manifestations of disease. It must be remembered, however, that in children the evidences of disturbed function are not always an indication for treatment. Symptomatic treatment is indicated under two conditions: (1) When the manifestations of disturbed function are uncomfortable or painful, and (2) when they are harmful or dangerous. The mere fact that a child has fever or a rapid pulse, does not mean that these symptoms are necessarily to be treated. The indications for symptomatic treatment vary with the disease and with the gravity of the symptoms.

The remedial measures employed in symptomatic treatment can be roughly classified according to the particular physiologic system whose function is disturbed. Measures directed at the reduction of bodily temperature are called antipyretic. Measures directed at remedying disturbed function of the circulatory system include stimulants and counterirritants. Measures which aid the renal function are called diuretic. The measures directed at the relief of pain are called analgesic, and include the opiates. Disturbance of the function of the nervous system is controlled by the sedatives. Measures designed to aid the general metabolism may be classified as tonics.

The measures employed in the symptomatic treatment of diseases may be classified in another way, according to whether they do or do not involve the use of drugs. They will be described under this classification.

THERAPEUTIC MEASURES OTHER THAN DRUGS

These play a very important part in the therapeutics of infancy and childhood.

HEAT AND COLD. The Ice Bag.—The application of cold is useful in many conditions. Applied to the head, it is useful both for the reduction of temperature and for the relief of pain. In other parts of the body it is often indicated as an analgesic measure, and when applied to the precordia may have some favorable influence on cardiac function. It is useful in many forms of local inflammation. In very young and delicate children it should be used with caution. Cold is usually applied by means of an ice bag. On the head, an ice cap made like a helmet, may be used. On the eyes, cold compresses are employed, which are frequently changed.

The Hot Water Bag.—The application of the hot water bag is useful in symptomatic treatment of pain, particularly when referred to the abdominal cavity.

Poultices.—These are useful for the treatment of pain, and in certain local infections in hastening the formation of definite suppuration. The flaxseed poultice, or a poultice made from hot antiseptic solutions may be used.

The Hot Pack.—The naked body of the child is covered with towels wrung from water at a temperature of 100° to 108° F., and is then rolled in a thick blanket. The hot application may be changed every half hour until the required amount of free perspiration is produced. The chief use of this procedure is in nephritis.

The Hot Air Bath.—After removal of the clothing, the patient is laid upon the bed with the bedclothes raised about a foot above the body by means of some sort of wicker support. The bedclothing is fastened tightly about the neck of the patient. Hot air from a lamp of some kind is conducted through a tin pipe beneath the bedclothes. The hot air bath may be continued from fifteen to thirty minutes at a time. Free perspiration will usually be produced in fifteen or twenty minutes. The chief use of this procedure is in nephritis.

HYDROTHERAPY. The Hot Bath.—The patient is put into water at a temperature of 100° F., and the temperature of the water is gradually raised by the addition of hot water to from 103° to 106° F. The body of the patient should be thoroughly rubbed while the patient

is in the bath. A thermometer in the bath must be carefully watched. The chief use of the hot bath is in promoting reaction in cases of shock or collapse.

THE MUSTARD BATH.—Four tablespoonfuls of powdered mustard are mixed with one gallon of tepid water, and enough water is added to this to make five gallons, at a temperature of 100° F. The mustard bath is useful in cases of shock, collapse, or sudden heart failure, especially in infancy. The duration of the bath should be not more than ten minutes, but it may be repeated in an hour if necessary.

THE TEPID BATH is given at a temperature of 95 to 100° F. The patient should be left in the bath about ten minutes. This procedure is mainly useful in conditions of irritability of the nervous system, and to promote sleep. It is often more effective than drugs.

THE COLD SPONGE.—For this purpose equal parts of alcohol and water are used at a temperature from 80° to 85° F. The naked child is laid upon a blanket, and the body should be sponged for from ten to twenty minutes, with occasional rubbing of the skin. After the sponging, the child is wrapped in a blanket without further dressing.

This procedure is occasionally indicated in reducing high temperature. Its chief value, however, is in relieving symptoms of irritation of the nervous system in certain diseases. It is less effective, but also less depressing than the cold pack.

THE COLD PACK.—The naked child is laid upon a blanket, and the entire trunk is enveloped in a sheet wrung from water at a temperature of 100° F. Ice is now rubbed upon the outer surface of the sheet, first in front, then behind, so that the entire trunk is included. The child is then left in the pack for a varying time, according to the circumstances of the case, during which the rubbing with ice is once repeated. The child is then wrapped in a blanket without removing the wet sheet, and left in this about an hour, when it may be dried and wrapped in another blanket.

This is the most effective means of reducing the temperature in infancy and childhood. It has the advantage of avoiding all shock and fright. It may be necessary to apply heat to the lower extremities while the child is in the pack..

THE COLD BATH.—This is sometimes employed as an antipyretic measure, but much less often in children than in adults. The cold pack is to be preferred in most cases. The child is placed in a bath at a temperature of 100° F. The temperature of the bath is then gradually lowered by the addition of cold water to from 80° to 85° F. The body of the child should be vigorously rubbed while it is in the bath, and water should be applied to the head. The bath should be continued for from five to ten minutes, after which the child is removed, dried quickly and rolled in a blanket.

COUNTERIRRITATION. Mustard Paste.—This is made as follows: Take one part powdered mustard and six parts flour, mix with warm water, and spread between two layers of muslin. This is applied to the region where counterirritation is desired, and held in place by a swathe or simple bandage. The effect on the skin should be carefully watched, and the paste should be removed as soon as a decided redness of the skin has been produced. In most cases, this will not take much longer than five minutes. The paste may be made somewhat stronger for older children than for infants. The application of a mustard paste may be repeated as often as every three hours if desired.

This is the most efficient means of producing a rapid counter-irritation over a large surface. It is sometimes useful in pulmonary diseases.

The Mustard Pack.—The naked child is laid upon a blanket and its body is wrapped in a large towel or small sheet, which has been saturated with mustard water of a strength of one tablespoonful of mustard to one gallon of warm water. The patient is then rolled in a blanket. The pack is continued until a notable redness of the entire body has been produced.

The mustard pack is useful in the same conditions as the mustard bath, namely, sudden collapse, great prostration, and internal congestion. It is less efficient than the mustard bath, but has the advantage of being less disturbing to the patient.

The Turpentine Stupe.—Wring a piece of flannel out of water as hot as can be borne by the hand, sprinkle upon it ten or twelve drops of spirits of turpentine, apply directly to the affected part, and cover with dry flannel. Care must be taken in using turpentine stupes in infants, as they easily produce blistering of the skin. The chief use of the turpentine stupe is in abdominal pain, or inflammation, or in abdominal distention.

Bleeding.—Venesection is almost never indicated in the diseases of infancy and childhood. Local bloodletting by means of leeches are sometimes useful in certain conditions such as mastoid disease, or thoracic disease with marked dyspnoea and cyanosis. Dry cupping may also be employed in some similar conditions. Wet cupping is never indicated in early life.

IRRIGATIONS AND SPRAYS. Irrigation of the Colon.—This is one of the most important of all the therapeutic procedures used in infancy and early childhood. Irrigation of the colon must not be confused with enemata. The object is to flush the entire large intestine with fluid, injected as high up as possible.

The apparatus required is a fountain syringe holding at least two quarts, four or five feet of rubber tubing, and a soft rubber catheter

ze about number 26 French scale. The catheter is connected he rubber tubing coming from the syringe by means of a bit s tubing. In place of the fountain syringe, a funnel and may be employed. Double current tubes, or two catheters, outflow, the other for inflow, are unnecessary. The syringe about two feet above the level of the child, and is filled with gating fluid, which in routine cases should be normal salt .

FIG. 43

Irrigation of the colon

is placed upon its back with the thighs flexed, and the ɔught to the edge of the bed or table. A Kelly pad or : should be arranged to form a trough leading into a tub n. The catheter is oiled, and the water is turned on and ɔw until the cold water in the tube has escaped. The end ter is then introduced through the anus with the water is gradually pushed upward as far as possible, usually ɛe of twelve or fourteen inches. The pushing of the ndered easy by the fact that the running water distends ahead of the tube. If on account of the peristalsis ıtheter turns on itself and comes back through the anus, introduced and pushed up again. There is no danger tion of the intestine, as active peristalsis is always ex-

cited and the water comes back through the anus around the catheter. Usually a pint or more will enter before the fluid begins to return. The irrigation should be continued until the water comes back entirely clear, but at least two quarts should always be given. During the irrigation gentle massage of the abdomen will facilitate the reaching of the upper part of the colon. After the irrigation, the rubber tube is disconnected and the remaining water is allowed to escape through the catheter. Some is usually retained and passed later.

The temperature of the water used in irrigation for ordinary purposes should be from 95° to 100° F. It may be varied according to special indications, as when there is high fever, or when there is pain and tenesmus; in these cases colder irrigating fluid is of advantage. In some cases, instead of normal saline solution, plain water or some medicated fluid may be used.

Irrigation of the colon should never be used more than twice in twenty-four hours. It is wise for the physician to make sure that the nurse thoroughly understands the technique, as otherwise only a rectal injection will be given. It is often a good plan for the physician to give the first irrigation himself. The essential points are the high introduction of the tube, and the use of a large quantity of water.

GASTRIC LAVAGE.—Stomach washing is frequently indicated in the diseases of infancy and early childhood, and is one of the most valuable therapeutic procedures which we have. It is not, as is often supposed, attended by shock or prostration in infants, and it is entirely free from danger.

The apparatus for stomach washing consists of a soft rubber catheter, a glass funnel attached to a rubber tube, and a glass "window" connection between the other end of the rubber tube and the catheter. The child is wrapped up with the arms confined and is held in a sitting position, with a large basin at the nurse's feet. The physician depresses the tongue with the forefinger of the left hand, and with the right hand passes the catheter rapidly backward into the pharynx and down into the esophagus. The passage of the catheter past the pharynx will cause gagging, and for this reason this part of the introduction should be made rapidly. The catheter is now pushed downward more slowly. A second gagging will usually betray the moment when the catheter passes the cardiac orifice and enters the stomach.

The catheter is now passed a little further downward. The entrance into the stomach is often shown by the escape of curdled milk from the funnel. The distance from the teeth to the stomach varies with the size of the child. It is approximately about ten inches, but the physician can usually recognize the entrance into the stomach by the distinct reflex at the cardiac orifice. When the

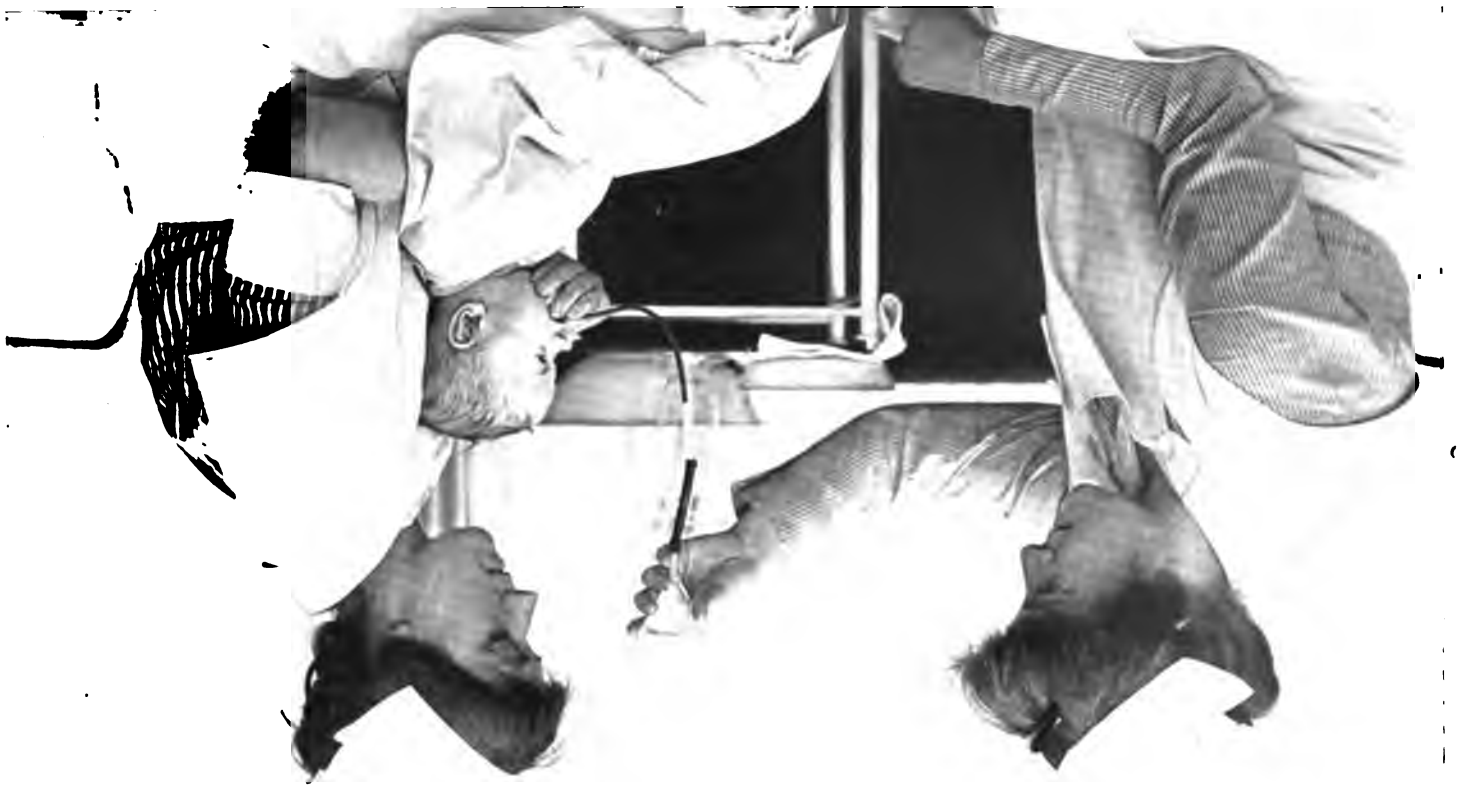
catheter has entered the stomach, the funnel should be raised as high as possible, to facilitate the escape of any gas which may be present, and should then be lowered in order to siphon out any fluid contents. The funnel is then raised and warm water at a temperature of about 100° F. is poured into it from a pitcher. The amount of water varies with the age of the child, and should be approximately equal to the quantity of milk given at a feeding at that age. The funnel is then lowered and the water is siphoned out. This procedure is repeated a number of times until the fluid comes back clear. A number of repeated washings are often required to break up the large curds which sometimes interfere with the flow of fluid.

Stomach washing is practically never employed in children over three years old, and is most commonly indicated in the first two years of life. Indications for this procedure will be given under the several diseases in which it is used.

FIG. 47

Irrigation of the ear

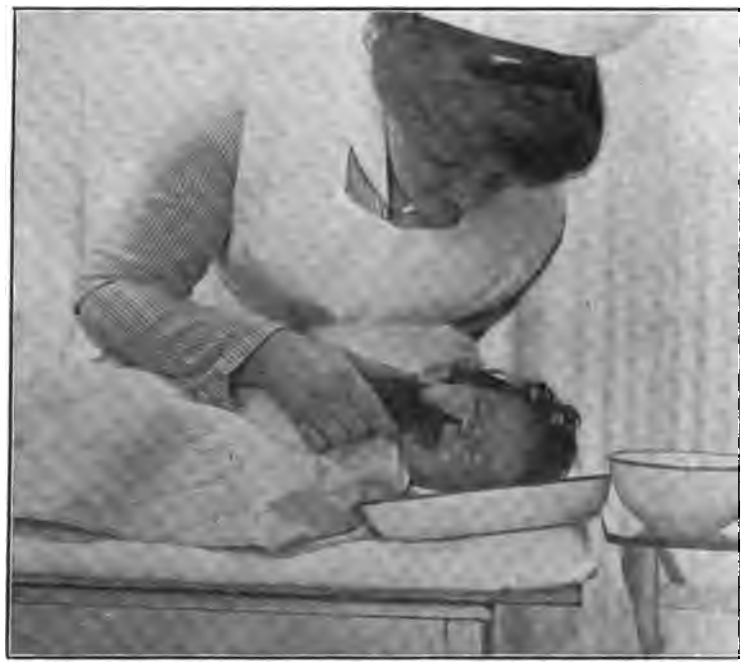
SYRINGING THE EARS.—This is best performed with a syringe made tirely of soft rubber. The child should be lying on its back in e position shown in the illustration. This is one of the most fre- ent procedures in the therapeutics of early life, owing to the very mmon occurrence of middle ear disease.



General Therapeutic Measures

Nasal Irrigation.—This may be given by means of a
syringe, or with any form of hand syringe which has a soft
tip and with which the pressure can be easily regulated. Th
tain syringe should not be higher than two feet above th
The patient lies on its side with its mouth open. The irrig
given through the upper nostril, and then the child is turnec
other side for irrigation through the other nostril. The fluic
return either through the lower nostril or through the mouth
must be taken in using the hand syringe not to employ tc
pressure, as the fluid may be forced into the eustachian tube

Fig. 48



Irrigation of the nose

Spraying the Nose and Throat.—This procedure is best pe
with some form of atomizer. Either aqueous or oily solutions a
the character of the solution being determined by that of the

Gargles.—Gargling the throat is impossible with infants
only used in older children. The character of the gargle var
that of the condition in which it is indicated.

Syringing the Mouth and Throat.—This is sometimes ir
in children too young to gargle. The child should either l
on its side, or held in a sitting posture with the head inclined f
Any form of syringe may be used in this procedure.

[ONS.—These are very useful in various affections of y tract. They are given in the form of vapor, either eing employed, or vapor from boiling water to which ıl agent has been added. In the mildest cases, a bowl er may be surrounded with a newspaper folded in such ncentrate the vapor, and the child is held over the bowl) breathe the steam. To be thoroughly effective, how- nt should be under a tent. Such a tent can be impro- rib in various ways by means of a sheet. A rubber better than an ordinary cotton one. To generate the ort of croup kettle or vaporizer may be employed, and and safe means must be devised to conduct the steam) kettle or vaporizer to the tent. Many of the croup market are dangerous, but there are some good ones. ious forms of apparatus for giving medicated inhala- Cresolene lamp is in common use, but steam inhala- and croup kettle are preferable.

.—Enemata are used for four purposes, (1) as a means r cleansing the bowel, (2) as a means of introducing as a means of introducing medication, (4) as a means fluid or of stimulation.

R CLEANSING ENEMATA.—For the purpose of simply bowel or of cleansing the rectum, the ordinary soap- best. In marked constipation when the fecal mass y, enemata of olive oil are used—about one ounce,— often be advantageously followed by a soapsuds enema. iate and certain emptying of the bowels is desired, a glycerin in one ounce of water should be injected for ger quantity being used for older children.

ENEMATA.—Peptonized milk was formerly recom- ım convinced that the most efficient nutrient enemata of a solution of dextrose (glucose). A six per cent. be employed, and the quantity given at each injec- y with the age of the child. Young infants will not n one or two ounces, older infants not more than two older children four to six ounces. The interval be- ions should usually be four hours.

ENEMATA.—The giving of drugs by rectum is often ancy and childhood, either on account of vomiting f the unpleasant taste. The medicine may be given gruel. The quantity of diluent used should be the trient enemata. With both nutrient and medicated seful in infancy to press the buttocks together for er the injection, to prevent expulsion.

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STIMULANT ENEMATA.—These are employed mainly for the purpose of introducing fluid into the circulation. Stimulating drugs are best given hypodermically or intravenously. The normal saline solution may often best be introduced by rectum. Enemata of normal saline solution may be given at intervals in the same quantities as are used for nutrient enemata, the object being to give as much fluid as possible without producing intolerance of the rectum.

Another procedure is to give the normal salt solution by means of the drop method. A fountain syringe connected by means of a rubber tube with a soft rubber catheter is used. The syringe is hung at an appropriate height above the bed, and is kept warm by being surrounded with some form of thick covering. The solution should be changed when it becomes cold. An attachment is placed upon the rubber tube, by means of which compression of the tube can be varied, and in this way the rate of flow of the solution can be regulated. It may be started at ten drops per minute, but if the patient begins to pass fluid from the rectum, the rate of flow should be decreased, the object being to give as much fluid as can be retained and absorbed. In this way fluid can be given continuously without producing intolerance.

GAVAGE AND NASAL FEEDING.—In a great number of conditions in early life patients are unable or unwilling to swallow sufficient nutriment. Gavage is a very important procedure in pediatrics. The same apparatus is employed as in stomach washing—soft rubber catheter, connecting tube, and funnel. The technique is the same as that described for stomach washing, except that it is preferable to have the child lying upon its back instead of in a sitting posture. The catheter is introduced into the stomach through the mouth, and after the funnel has been raised to allow the gas to escape, the food is poured into the funnel. Before withdrawing the tube, it should be tightly pinched in order to prevent any fluid from trickling into the pharynx, and the tube should be withdrawn quickly.

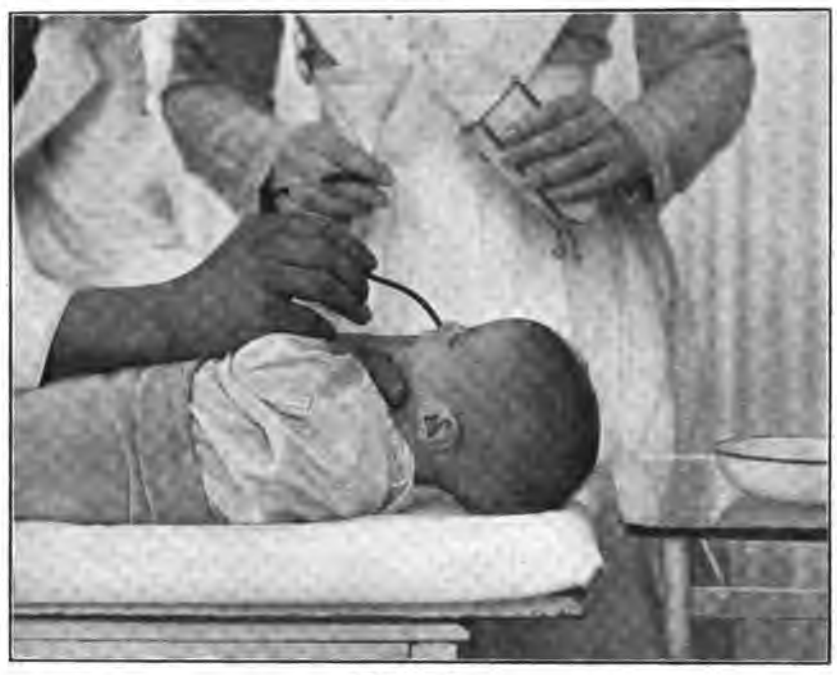
The introduction of the catheter through the nose is possible, and was formerly recommended for gavage. The introduction of the catheter through the mouth is preferable, but there are certain cases in which nasal feeding must be used. When gavage is necessary in children over two years old, the tube cannot be passed through the mouth without the use of a gag, and then only after much struggling. There are also certain cases in which, on account of operations about the throat or lesions in the throat, nasal feeding is better.

MASSAGE.—This is useful in older children in the same conditions as those for which it is employed in adults. It is best whenever possible to have a trained masseur. In infancy true massage is not

often indicated. Massage of the abdomen is occasionally used in constipation. Friction of the skin either with the bare hand or with cocoa butter is sometimes indicated in infants. Cod liver oil is widely used for this purpose, but is very disagreeable, and has no advantages over cocoa butter.

HYPODERMOCLYSIS.—The introduction of fluid under the skin is often a very valuable therapeutic measure in infancy and childhood. The object is to compensate for loss of fluid, to give addi-

No. 51



Nasal feeding

tional fluid, and to stimulate the general circulation. It is an alternative procedure to the giving of normal salt solution by rectum, and is a preferable method in certain conditions. In other conditions, the skin is used in addition to the rectum, or is substituted for the rectum when the latter becomes intolerant. Sterile normal salt solution at body temperature should be employed. It is injected by means of an appropriate syringe and an ordinary antitoxin needle, both needle and syringe being sterilized. Injections may be made between the scapulae, into the skin of the abdomen, or in the lateral thoracic region. The amount injected at one time in an infant should be from one to four ounces, and in an older child from four to six ounces.

INTRAVENOUS INJECTIONS.—This procedure has hit been very little if at all employed in infancy and childhood, p pally on account of the difficult technique caused by the sma of the veins. I have recently adopted the plan of employin longitudinal sinus for purposes of intravenous injection in infant open fantanelles. This greatly simplifies the technique, and I been gradually using intravenous injections more and more in in

Drugs—including the various stimulants from which quick is desired—salvarsan in syphilis, and sodium bicarbonate in aci may be given in this way. It is a most efficient means of

Fig. 52

Intravenous injections in infancy—tapping the cerebral ventricles
A. Point where the needle is introduced in obtaining blood for examination, intraveneous injections and transfusion
B. Point where the needle is introduced in tapping the cerebral ventricle

ducing normal salt solution rapidly into the general circul Nutriment also may be introduced directly into the blood in th of dextrose by means of this procedure. I have found intrav dextrose injections of great value in certain cases of extreme tion, atrophy, and malnutrition, and have been able to demon that most if not all of the dextrose thus introduced is actually u for the purpose of energy production, only a little if any being ex in the urine.

The technique is simple. If the injection is of a small bulk which ın be contained in a small syringe, no more apparatus is required ıan a sterile syringe and hypodermic needle. If the injection is of .rger bulk, as in the case of normal saline, sodium bicarbonate, or extrose injections, a somewhat larger needle must be used, and this connected with a rubber tube, which in turn is connected with a .rge syringe. A funnel may be employed instead of a syringe, but syringe has the advantage that with it the physician can make ıre that the needle is actually in the sinus.

FIG. 53

Intravenous injection in infancy

Before beginning the injection the entire apparatus is sterilized. Two assistants are required, one to hold the child's head firmly, the ther to manage the syringe. The syringe is filled with the fluid to e injected, connected with the tube and needle, and all air is expelled. The region of the fontanelle is sterilized and the physician enters he needle in the median line at the posterior angle of the fontanelle. The entrance of the needle into the sinus can usually be recognized y a sudden lessening of the resistance. The physician now holds he needle steady while the assistant in charge of the syringe slightly ithdraws the piston. If the needle is in the vein, blood will imme-

FIG. 49—Tube feeding, first step

diately appear at the glass window of the tube. The assistant then reverses the pressure and slowly injects the fluid into the sinus. Distention of the veins of the scalp is a sign that the fluid is being injected too rapidly. The injection should consume from ten to fifteen minutes, and the amount injected should be one sixtieth of the infant's body weight. In dextrose injections a 5 per cent. solution is used.

USEFUL DRUGS IN INFANCY AND CHILDHOOD

The number of drugs used in the treatment of disease in infancy and childhood is comparatively small. It is far better for the physician to understand thoroughly the use of a few drugs than to use many at random. It is essential also that the physician shall thoroughly understand the action of every drug used, and that he should never prescribe a drug unless the indications for its use are clear. In considering the indications for drug treatment, it is important to remember the idiosyncrasies of children in general toward drugs, and also to be on the watch for individual idiosyncrasy.

In giving a drug, the physician should proportion the dose to the age of the patient, and when a drug has been given, he should watch carefully its effect. If the desired effect is produced without toxic symptoms, it is a sign that the dose is sufficient. If toxic effects occur, the drug should be omitted for a time, and when it is resumed smaller doses should be given. If no effect is observed, increase the dose, carefully watching for either a good or a toxic effect. If no effect is then produced, the drug or particular preparation must be changed.

In the following list, those drugs which are most important and most frequently used in the treatment of infants and children are indicated. It is assumed that the physician is familiar with the general description of these drugs and with the facts about their action, elimination, toxic effects, and contraindications. The purpose of the list is mainly to point out the particular purposes for which the drugs are used in early life, to point out any peculiarities which attend their administration to infants and children, and to indicate the doses appropriate at the different ages. It is extremely difficult to classify the drugs which are most useful in Pediatrics. They cannot well be divided on a basis of their importance on account of the great diversity of the purposes for which they are used. The classification used in the list is not by any means complete. Many of the drugs listed under one heading have also a pharmacological action which would bring them under another heading. The classification is used mainly for convenience, and no effort is made to list them in the order of the frequency of their employment. The doses given are minimum, those with which it is safe to begin.

STIMULANTS AND DIURETICS.—This group includes drugs whose principal action is upon the circulatory system or upon renal function, and their principal use is in the symptomatic treatment of disturbance of these functions.

CAFFEIN.—This is the most generally useful circulatory stimulant in the diseases of early life. It is used particularly in threatening circulatory failure of toxic origin such as occurs in many of the acute infectious diseases. This circulatory failure is usually due as much to vasomotor paralysis as to cardiac weakness, and caffein acts both as a vasomotor and cardiac stimulant. More specific symptomatic indications for its use will be given under the various diseases in which it is employed. It is best given in the form of caffein-sodium-benzoate, or caffein-sodium-salicylate. It is most often used hypodermically, but may be given by mouth. The hypodermic dose is as follows:

One to six months,	gr. 1/8
Six months to two years,	gr. 1/4
Two to five years,	gr. 1/2
Over five years,	gr. 1

Given every four hours, or as indicated.

DIGITALIS.—This is used as a cardiac stimulant in circulatory disturbance caused by disease of the heart itself. It is almost useless in circulatory weakness resulting from causes outside the heart, and is of no use in the toxic circulatory disturbance of acute infections. Its chief indication is in cardiac insufficiency from disease of the endocardium, myocardium, or pericardium. In children it is more useful in the treatment of chronic than of acute lesions of the heart, and it is particularly valuable in cardiac disease associated with edema, on account of its diuretic action. The usual preparation is the tincture of digitalis (U. S. P.). Indications for its use are very rare in children under one year. The dose is as follows:

One to two years,	m. II
Two to five years,	m. IV
Over five years,	m. V to X

Given three times a day. It is given by mouth.

The following preparations and substitutes for tincture of digitalis may be used: *Digipuratum*, the patented preparation put up in tablets of which each is equal in strength to m. xv. of the tincture. It is expensive. *Tincture of strophanthus* (U. S. P.). The action is very similar to that of the tincture of digitalis and it is given in the same doses. It should be remembered that the absorption is slow and that twenty-four hours or more is required for the drug to produce a result. Excretion is slow, and action may be cumulative.

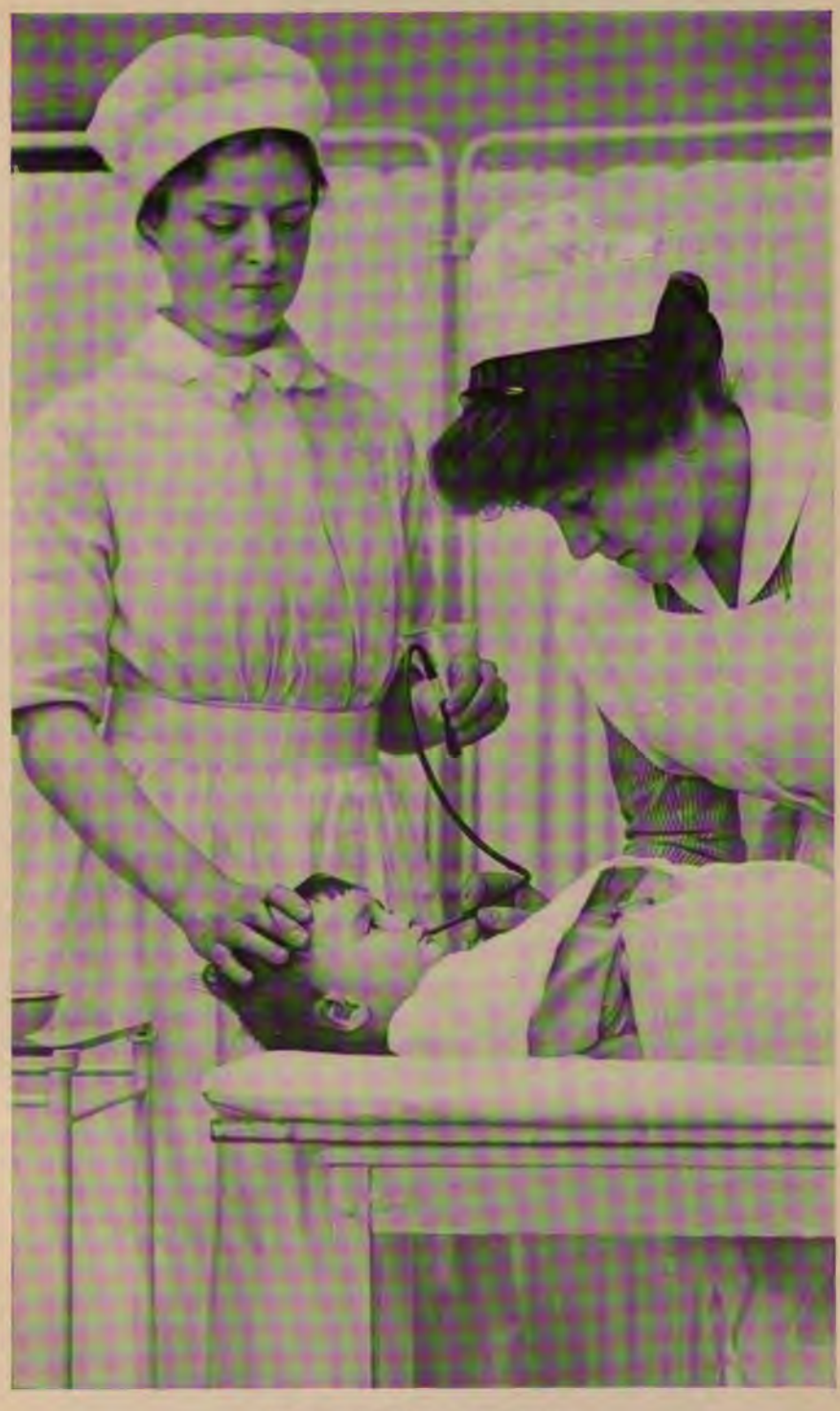


FIG. 50—Tube feeding, second step

CAMPHOR.—This is useful as a rapid stimulant in sudden circulatory collapse. It is given only subcutaneously or intravenously in the form of a 10 per cent. solution in oil. The doses of this solution are as follows:

One to six months,	m. II
Six months to a year,	m. III
One to two years,	m. V
Two to five years,	m. VII
Over five years,	m. X

ADRENALIN.—This is occasionally useful in sudden circulatory collapse, especially when of vasomotor origin. The preparation is a one to one thousand solution of adrenalin chloride. Parke, Davis & Company's is a good preparation. It is given only hypodermically or intravenously. The dose is as follows:

One to six months,	m. I
Six months to one year,	m. II
One to two years,	m. III
Two to five years,	m. IV
Over five years,	m. V

STRYCHNIN.—This is used only rarely as a stimulant in early life, in certain particular conditions. Its indication will be noted under the diseases in which it is occasionally useful. It is given hypodermically in the form of strychnin sulphate. The dose is as follows:

One to six months,	gr. 1/800
Six months to one year,	gr. 1/500
One to two years,	gr. 1/300
Two to five years,	gr. 1/200
Over five years,	gr. 1/100 to 1/60

THEOBROMIN-SODIUM SALICYLATE.—This is a double salt of theobromin-sodium and sodium salicylate. "*Diuretin*" is a trade-name of a similar preparation. It is described in "New and Non-Official Remedies, 1914." Theobromin-sodium salicylate is a diuretic, acting directly on the renal epithelium. It is slightly irritating to the kidneys and is contraindicated in acute nephritis. Its toxic effect is vomiting. The chief indication in children is in cardiac disease with edema.

To young children who do not mind the bad taste, the drug may be given in solution; to older children it may be given in capsules. The dose is as follows:

One to six months,	gr. I
Six months to one year,	gr. II
One to two years,	gr. III
Two to five years,	gr. V
Over five years,	gr. V to X

Given four times a day by mouth.

A substitute having a similar action is the patented preparation known as *theocin*. It is used in smaller doses, from one-fifth to one-half the quantity of diuretin being given at a dose.

SEDATIVES.—This group includes drugs whose principal action is upon the nervous system. In it are described drugs which are given for the purpose of quieting nervous excitability, and also drugs other than the opiates which are used for the relief of pain, or to produce sleep.

SODIUM BROMIDE.—The bromides are by far the most generally useful sedatives in infancy and childhood. The sodium salt is to be preferred to the potassium or ammonium salts for use in childhood. Owing to the fact that the nervous system in early life is particularly liable to disturbance of function, sodium bromide is indicated in the symptomatic treatment of a great variety of conditions. It is also indicated in the more pronounced forms of nervous irritability, such as convulsions. It is well borne by children and is proportionately less likely to produce vomiting than in adults. Toxic effects are vomiting, acne, coryza, and somnolence.

Sodium bromide is administered in watery solution well diluted. It is given by mouth except in cases where vomiting is present or where swallowing is impossible. Under the latter conditions it is given by rectum. The dose varies somewhat with the purpose for which the drug is prescribed. As a sedative in comparatively mild conditions, the dose is as follows:

	BY MOUTH	BY RECTUM
One to six months,	gr. 1/2	gr. I
Six months to one year,	gr. I	gr. II
One to two years,	gr. I-1/2	gr. III
Two to five years,	gr. II	gr. IV
Over five years,	gr. III to IV	gr. VI to X

given every four hours.

When sodium bromide is given for the purpose of checking serious symptoms such as convulsions or vomiting, the doses must be somewhat larger, as follows:

	BY MOUTH	BY RECTUM
One to six months,	gr. I-1/2	gr. III
Six months to one year,	gr. II-1/2	gr. V
One to two years,	gr. V	gr. X
Two to five years,	gr. VII	gr. XV
Over five years,	gr. X to XV	gr. XX to XXX

Given as a single dose and repeated as often and in whatever doses as are necessary to produce the desired effect.

CHLORAL.—The chief use of chloral in early life is to check severe convulsions in infancy. It is well borne even by quite young infants. It should always be given in solution by rectum. The dose is as follows:

One to three months,	gr. I
Three to six months,	gr. II
Six to twelve months,	gr. III
One to two years,	gr. V

TRIONAL.—This is occasionally indicated in older children for the same purpose for which it is used in adults, namely, to produce sleep. Insomnia, however, is a symptom which much less often requires treatment in children than in adults, and is usually produced by those forms of nervous over-excitability which are best treated by the administration of bromide. In infants trional is almost never indicated, sodium bromide being preferred.

Trional is insoluble and is given by being placed upon the tongue and washed down with water. The dose is as follows:

Two to five years,	gr. II
Five to thirteen years,	gr. III to V

SALICYLATE.—This represents the most useful drug for the relief of certain kinds of pain in childhood. In addition to being analgesic, salicylate is antipyretic and diaphoretic. It also increases nitrogen elimination in the urine, acts as a cholagogue, and has some diuretic action. The toxic effects are tinnitus, headache, vomiting, erythema, and in very large doses, drowsiness or delirium. It is very well borne by children. It is contraindicated in acute nephritis or when there is an idiosyncrasy.

The indications for salicylate are rheumatic fever, and various other conditions belonging in the ill-defined rheumatic class which are characterized by pain. It is used for pain in arthritis, pericarditis, endocarditis, neuritis, neuralgia, and similar conditions. It is an open question whether its action in rheumatic fever is only to relieve pain, or whether it has some actual curative action. There is evidence that it has curative value in some cases of chorea.

It is comparatively rarely used in infants, as they do not often suffer from rheumatic fever or from the various forms of pain which are influenced by salicylate.

The preparation most commonly used is the sodium salicylate (U.S.P.). Children do not mind the taste of sodium salicylate as do adults, and consequently the drug is best given in solution which may be so arranged that one teaspoonful contains the desired dose. It is usually unnecessary to add any vehicle for the purpose of disguising the taste. The dose is as follows in rheumatic fever:

Two to five years,	gr. III
Five to eight years,	gr. V
Eight to thirteen years,	gr. V to VII

Given every hour until relief of pain has appeared, and after this every four hours, unless some toxic effect is produced. For pain in other conditions than rheumatic fever smaller doses may be sufficient.

A common substitute for sodium bromide is acetylsalicylic acid which is a proprietary preparation sold under the trade name of

aspirin. In children this has no particular advantage over sodium salicylate. The doses are the same.

PHENACETIN.—This was formerly a proprietary preparation, but it is now official under the name of *acetphenetidinum*. It is a coal-tar derivative. Its action is analgesic, antipyretic, mild diaphoretic, and sedative. The toxic effect is circulatory depression, but it is comparatively well borne by children. It is used in a great variety of conditions, rarely as an antipyretic, more often as an analgesic or sedative.

Phenacetin has the disadvantage of being insoluble, and is given in tablet form or as a powder. The dose is as follows:

One to six months,	gr. 1/4
Six months to one year,	gr. 1/2
One to two years,	gr. I
Two to five years,	gr. I-1/2 to II
Over five years,	gr. II to IV

Repeated every two to four hours until the desired effect is produced.

OPIATES.—Strong objections have been urged against the use of opium in the diseases of early life. It is true that the opiates are less often required in infancy and childhood than in adult life, and this applies especially to infancy. Nevertheless, cases are frequently encountered in which an opiate is required for the relief of pain. Moreover, small doses of an opiate are the most effective means which we have of controlling excessive cough.

PAREGORIC.—This is the most generally useful preparation of opium for use in young children. Its chief uses are to check cough, check certain forms of diarrhea, and occasionally to control pain. The doses are as follows:

One to three months,	m. I
Three to six months,	m. II
Six months to one year,	m. III
One to two years,	m. V
Two to five years,	m. X to XX
Five to ten years,	m. XX to XXX

Given every four hours by mouth in a little water.

MORPHIN.—This is the best preparation of opium for the control of severe pain. It is not well borne by children and the doses must be proportionately smaller. Morphin is most commonly given hypodermically in the following doses:

One to three months,	gr. 1/1500
Three to six months,	gr. 1/1000
Six months to one year,	gr. 1/600
One to two years,	gr. 1/300
Two to fiveyears,	gr. 1/100 to 1/48
Five to ten years,	gr. 1/48 to 1/24
Over ten years,	gr. 1/24 to 1/12

Repeat when necessary, not sooner than in two hours. When given by mouth the doses must be slightly larger.

CODEIN.—This opium derivative is chiefly employed for cough, or for the treatment of pain. It is less effective than morphin but does not have the constipating effect. The dose is as follows:

One to three months,	gr. 1/300
Three to six months,	gr. 1/200
Six months to one year,	gr. 1/100
One to two years,	gr. 1/60
Two to five years,	gr. 1/60 to 1/10
Five to ten years,	gr. 1/10 to 1/8
Over ten years,	gr. 1/8 to 1/6

Given by mouth and repeated when necessary, or every four hours.

DOVER'S POWDER.—This is occasionally used instead of paregoric, and for the same indications. The dose is as follows:

One to three months,	gr. 1 20
Three to six months,	gr. 1/10
Six months to one year,	gr. 1/8
One to two years,	gr. 1/4
Two to five years,	gr. 1/2 to I
Five to ten years,	gr. II to III
Over ten years,	gr. III to IV

PURGATIVES AND LAXATIVES. CASTOR OIL (Oleum ricini U.S.P.).—This is the most useful general purgative for use in infancy and childhood. Nothing surpasses it in producing rapid and complete emptying of the bowels. Its chief indication is in various forms of indigestion, especially those characterized by diarrhea. Infants and very young children do not object to the taste, and even older children will often not mind the taste if it has not been suggested to them that the taste is bad. When the taste is objected to, castor oil may be given with orange juice, lemon juice, or brandy. The doses are as follows:

First year,	1 teaspoonful
Second year,	2 teaspoonfuls
Two to five years,	3 teaspoonfuls
Over five years,	1 tablespoonful

CALOMEL.—This is a purgative and diuretic, but in children is used almost wholly on account of its purgative action. The toxic effects are renal irritation and stomatitis, but the purgative doses used with children never produce these effects.

Calomel is useful as a substitute for castor oil when vomiting is present, and is also useful in the treatment of vomiting, as when given in repeated doses it tends to correct reversed peristalsis. It is dispensed in the form of tablets or powders. When tablets are given they should be crushed into powder with the back of a teaspoon, and the powder is given by being floated on the surface of a teaspoonful

of water. In almost all cases calomel is best given to children in divided doses as follows:

One to six months,	gr. 1/20, every half hour for ten doses
Six months to two years,	gr. 1/10, every half hour for ten doses
Two to five years,	gr. 1/8, every half hour for ten doses
Over five years,	gr. 1/4, every half hour for eight doses

In infants it is unnecessary to follow the calomel with a saline cathartic. In older children it is well to follow it with a dose of magnesia.

MAGNESIA.—This is the most useful mild laxative for use in infancy and childhood. In the first two years of life the best preparation is Phillips' milk of magnesia. After two years the best preparation is citrate of magnesia.

Milk of magnesia, which is a suspension in water, is used in infancy for two purposes. The first is as a mild measure for the emptying of the bowels. The dose is as follows:

One to six months,	1 teaspoonful
Six months to one year,	2 teaspoonfuls
One year to eighteen months,	3 teaspoonfuls
Eighteen months to two years,	1 tablespoonful

The other use for milk of magnesia is in the treatment of constipation. For this purpose it is better not to give the magnesia in single daily doses, but to give it with every feeding. The dose required varies with the severity of the case and will be discussed under constipation.

In children over two years of age citrate of magnesia is the best preparation. The dose is as follows:

Two to five years,	oz. II
Over five years,	oz. IV

Given usually at bedtime.

MAGNESIUM SULPHATE.—This is occasionally used in childhood as a hydrogogue purge. Its chief indication is in dropsy, whether of cardiac or of renal origin. It is contraindicated when there is marked weakness, emaciation, or vomiting. The dose in young children is two teaspoonfuls, in older children two to four teaspoonfuls, given well diluted in water on an empty stomach.

CASCARA.—(Fluidextractum Rhamni purshianae, U.S.P.). (Fluid Extract of Cascara Sagrada). This is a mild laxative useful in constipation and in a variety of other conditions as a means of keeping the bowels open. It is not so good in the treatment of constipation in infants as in older children. The dose in infants is from one to five drops, in older children from five to twenty drops. It is given in water, preferably at bedtime.

AGAR AGAR.—This substance swells enormously by the absorption of water, is not digested, does not ferment, and is consequently useful in stimulating peristalsis and sweeping out the intestine. It is used in chronic constipation usually in conjunction with other forms of treatment. The dose is from a teaspoonful to a tablespoonful once or twice daily. Powdered agar agar may be eaten on cereal. Granulated agar agar may be eaten mixed with milk or water. There are also agar agar wafers on the market.

RUSSIAN OIL.—This is liquid paraffin similar to the petrolatum liquidum of the U.S.P. The Russian oil is, however, a more refined product and does not have the yellowish color and unpleasant taste which characterizes American petroleum. It is useful as an intestinal lubricant which passes unabsorbed and undigested through the intestine. It is employed in some cases of chronic constipation. The doses are as follows:

Under two years,	1 teaspoonful
Two to five years,	2 teaspoonfuls
Five to ten years,	2 to 4 teaspoonfuls

Given three times daily after meals.

TONICS. ALCOHOL.—This is well tolerated by children, even by young infants. It has, however, been much abused through being used as a stimulant. The chief value of alcohol in early life is as a tonic, particularly in conditions of malnutrition. It is useful in many chronic conditions, and in acute febrile conditions late in the course of the disease. Alcohol is best administered to children in the form of brandy or whisky. The *twenty-four amounts* with which treatment with brandy is begun in children are as follows:

One to six months,	1/2 teaspoonful
Six to twelve months,	1 teaspoonful
One to two years,	2 teaspoonfuls
Two to five years,	3 to 4 teaspoonfuls
Over five years,	4 to 6 teaspoonfuls

This may be given in divided doses with the feedings or meals.

IRON.—This is as useful as a tonic in the anemias of infancy and childhood as it is in those of adults. The best preparation for use in the anemias of infancy is the *citrate of iron* given subcutaneously. An aqueous solution of the citrate is put up in ampules, sterilized, and each ampule contains a single dose. A platinum needle must be used. The dose during infancy is three-quarters of a grain every other day.

An alternative preparation for use in infancy is the saccharated carbonate in doses of 5 gr. three times a day. With older children nothing is better than Blaud's pills in doses of 5 gr. three times a day.

ARSENIC.—This is indicated as a tonic in a variety of conditions encountered in infancy and childhood. It is usually given in the

form of Fowler's solution (liquor potassii arsenitis, U.S.P.). Arsenic is well borne by children. The doses of Fowler's solution for ordinary tonic purposes are as follows:

One to six months,	1 drop once a day
Six to twelve months,	1 drop twice a day
One to two years,	1 drop three times a day
Two to five years,	2 drops three times a day
Over five years,	2 to 5 drops three times a day

Given in water after meals.

COD LIVER OIL. (Oleum Morrhuae, U.S.P.).—This is a very useful tonic in infancy and childhood, used in a variety of conditions. In the case of infants, the pure oil is to be preferred to an emulsion. The dose is 10 to 20 drops three times a day after meals. To older children any good emulsion of cod liver oil may be given in doses of 1 to 4 teaspoonfuls three times a day after meals.

MALT PREPARATIONS.—These are sometimes of value as tonics, particularly in conditions in which cod liver oil is not well borne. Malt extract may also often be advantageously combined with cod liver oil. The dose is 1 to 4 teaspoonfuls three times a day after meals.

TINCTURE OF NUX VOMICA.—This is the only bitter tonic which is much used in the diseases of early life. It is especially useful in conditions attended with loss of appetite. The dose is as follows:

One to six months,	m. 1/4
Six months to one year,	m. 1/2
One to two years,	m. I
Two to five years,	m. II
Over five years,	m. III to V

Given three times a day before meals.
It may be combined with water in such proportions that each teaspoonful contains the required dose.

COMPOUND SYRUP OF THE HYPOPHOSPHITES. (Syrupus Hypophosphitum Compositus, U.S.P.).—This is occasionally useful as a tonic in older children. The dose is 1 teaspoonful three times a day after meals.

DRUGS USED UNDER SPECIAL INDICATIONS. SALVARSAN.—This is a proprietary preparation. Its action is to kill certain pathogenic organisms in the living body. It may irritate the kidneys or liver, but apparently has no toxic effect for other organs. Its toxic effects are as follows: Signs of renal irritation or diminution of renal function; jaundice; erythema; hyperemia and swelling at the site of the syphilitic lesions; fever developing gradually.

The only indication for salvarsan in infancy and childhood is *syphilis*.

Neosalvarsan has an action like that of salvarsan but less powerful in equal dosage, and with less danger of toxic effects. The dose of salvarsan for an infant is 0.05 gm.; the dose of neosalvarsan is 0.1 gm. The method of administering salvarsan will be described under syphilis.

MERCURY.—This is used in early life in the treatment of congenital syphilis. Mercury is usually given to infants by inunction. Equal parts of unguentum hydrargyri and vaselin are ordered, and an amount the size of a pea is rubbed in daily. If for any reason the inunctions are objectionable, the gray powder (hydrargyri cum creta, U.S.P.) may be given by mouth. The dose is gr. 1/2 three times a day.

POTASSII IODIDUM.—Iodide of potash has two actions of importance in the therapeutics of infancy and childhood. These are: 1. It causes the disappearance of the tertiary lesions of syphilis. 2. It increases the fluidity of mucus in the respiratory tract. In over doses it causes acne, catarrh of the respiratory organs, gastric disturbance, and possibly delirium. Its chief indications are in tertiary syphilis, in bronchitis with sticky expectoration, and empirically in asthma.

Iodide of potash is well borne by children, and for tertiary syphilis may be given to older children in the same doses as to adults, namely, 10 to 20 gr. three times a day. In infancy the usual dose in syphilis is 5 gr. three times a day. As an expectorant the doses are half those given for syphilis. Usually, however, as an expectorant in infancy and childhood, it is better to use as a substitute *syrup of hydriodic acid*. The doses are as follows:

One to six months,	5 drops three times a day
Six months to one year,	10 drops three times a day
One to two years,	15 drops three times a day
Two to five years,	20 drops three times a day
Over five years,	1/2 teaspoonful three times a day

QUININ.—This is a specific for malaria. It is well borne by young children and relatively larger doses are required than for adults. The various preparations of quinine, the doses, and the methods of administration will be described in detail under malaria.

HEXAMETHYLENAMIN.—This drug is excreted in an acid urine in the form of ammonia and formaldehyde. The setting free of formaldehyde acts as a urinary antiseptic. When the urine is alkaline or neutral in reaction, hexamethylenamin is excreted unchanged, and is inefficient. In such a case the urine should be made acid by the administration of acid sodium phosphate, but this drug should not be administered *with* hexamethylenamin because they are incompatible.

Hexamethylenamin is especially indicated in pyelitis and cystitis;

also in typhoid fever, to prevent bacilluria and cystitis. The dose is as follows:

One to six months,	gr. II three times a day
Six months to one year,	gr. III three times a day
One to two years,	gr. V three times a day
Two to five years,	gr. VII three times a day
Over five years,	gr. X three times a day

The drug is given in solution which may be so put up that a teaspoonful contains the required dose. It should be given with plenty of water.

"Urotropin," "formin," and "aminoformin," are proprietary names applied to hexamethylenamin.

SODIUM BICARBONATE.—This is used in infancy and childhood in a great variety of conditions, among which are the following: (1) In the modification of cow's milk for infant feeding; (2) as an antacid acting locally in certain disturbances of digestion; (3) as a means of making the urine alkaline; (4) as a means of diminishing the acidity of the blood. The dosage and method of administering vary widely with the different purposes for which bicarbonate of soda is used, and will be given under the various indications.

BISMUTH SUBNITRATE.—This a mild astringent and antacid, and combines with the sulphuretted hydrogen in the intestine to form a black insoluble sulphate. In childhood it is chiefly used in the treatment of certain forms of diarrhea. A good preparation for infants is the milk of bismuth (Parke, Davis and Co.), which can be given in the feedings; each teaspoonful contains 5 gr. of bismuth subnitrate. The doses are as follows: Infants, gr. V. Older children, gr. X. Given every four hours, or repeated after each loose movement.

POTASSIUM CHLORATE.—This is chiefly used in the treatment of certain forms of stomatitis in infancy and early childhood. Its beneficial effect comes from the fact that it is almost wholly excreted with the saliva. When given in over-doses, it is toxic, the symptoms being drowsiness, suppression of urine, cardiac weakness, and sometimes cyanosis. The best method of administering it is to put the entire twenty-four hour amount into a glass of water, and to divide the doses in such a way that they will be given as frequently as possible. The following is the quantity which can be taken in twenty-four hours at the different ages:

Under one year,	gr. X
One to two years,	gr. XV
Two to six years,	gr. XX
Six to eight years,	gr. XXV
Eight to fourteen years,	gr. XXX

SWEET SPIRITS OF NITRE. (Spiritus aetheris nitrosi).—This is used in infants as a mild diuretic and antipyretic. The doses are as follows:

One to six months,	m. II
Six months to one year,	m. III
One to two years,	m. V
Two to five years,	m. X
Over five years,	m. XV to XX

Given every four hours in water.

IPECAC.—This drug is used in infancy and childhood chiefly as an expectorant, but occasionally as an emetic. Its chief value is in croup. The best preparation for children is the Wine of Ipecac. Expectorant doses are as follows:

One to three months,	m. 1/4
Three to six months,	m. 1/2
Six months to one year,	m. I
One to two years,	m. II
Two to five years,	m. III
Over five years,	m. V

Given every four hours in a little water.
As an emetic or in croup, larger doses are required.

SODIUM BENZOATE.—This is occasionally used in early life, particularly in the treatment of pyelitis. The purpose of its administration is to make the urine acid. The dose in infancy is 1 to 5 gr.; in older children, 5 to 30 gr. It is given in solution.

POTASSIUM ACETATE.—This is occasionally used in early life, chiefly for the purpose of making the urine alkaline. The doses are the same as those of sodium benzoate.

ATROPIN AND BELLADONNA.—The chief use of this drug in early life is in the treatment of enuresis. The dosage and method of administration will be described under that disease. Atropin and belladonna are occasionally used for their action in checking excessive secretion from the respiratory mucous membrane. Atropin is also used with morphin for its counteracting effect. The doses are as follows:

For Atropin—	
One to three months,	gr. 1/5000
Three to six months,	gr. 1/3000
Six months to one year,	gr. 1/2000
One to two years,	gr. 1/1000
Two to three years,	gr. 1/500
Three to five years,	gr. 1/300
Five to seven years,	gr. 1/200
Seven to ten years,	gr. 1/150
Ten to fourteen years,	gr. 1/100
For Tincture of Belladonna—	
One to six months,	m. 1/4
Six months to one year,	m. 1/2
One to two years,	m. I
Two to three years,	m. II
Three to five years,	m. III
Five to seven years,	m. V
Seven to ten years,	m. VII
Ten to fourteen years,	m. X

THYROID EXTRACT.—This is used in infancy in the treatment of cretinism. There are various preparations, among which I have found that of Burrough's and Welcome to be very satisfactory The doses should be at first gr. 1/4 three times a day, and this is gradually increased. Signs of over-dosage are wakefulness, sweating, tachycardia, or rise of temperature.

ROUTINE TREATMENT OF AN ACUTE SELF-LIMITED DISEASE

In order to avoid needless repetition in the description of the treatment of various diseases, the routine treatment of an acute self-limited disease will be described here. It will then only be necessary in the description of the particular diseases of this class to enumerate any special modifications called for by the special etiology and symptoms of the several diseases. The duty of the physician in an acute self-limited disease may be summarized as follows:

1. The hygienic care of the patient.
2. Directing the diet.
3. Attending to the bowels.
4. The treatment of symptoms.
5. Watching for complications.

To these may be added in certain diseases—

6. The establishment of quarantine and the carrying out of proper means of disinfection.

HYGIENIC CARE.—Children with an acute self-limited disease should invariably be kept in bed during the entire febrile period, and for a certain time after the fever has subsided. The length of time during which they should be kept in bed after the temperature has come down permanently to the normal varies with the several diseases. The sick-room should preferably be one with a sunny exposure. Plenty of fresh air should always be secured at all times. In winter an open fire in the sick-room is very desirable.

Every detail of the nursing should be carefully supervised, and good nursing is the prime essential in the treatment of the acute febrile diseases. It is usually best for the child not to walk or be carried to the bathroom, but to remain continuously in bed, using the bedpan. Sponge baths should be given in bed. Plenty of water should be given in all febrile diseases.

The management of convalescence is important. The time when the child is allowed to get out of bed depends mainly on the rapidity with which its strength returns, and varies with the character and severity of the disease. It is usually best to keep children in bed for some time after they are anxious to get up, even though recovery

in children is usually rapid. Patients should first be allowed to sit up in bed for an increasing length of time each day. They may then be allowed to sit up in a chair for a little while in their dressing gowns, and if this does not tire them, they may be given their clothes. On the first day after a child gets its clothes, it should not be allowed to walk farther than from the bed to a chair, and should only be dressed for a short time. As the strength of the child increases, it may be allowed to be dressed for a longer period each day, and to gradually walk about more. When the child shall be allowed to go out doors depends upon the rapidity of the return of strength and upon the prevailing weather conditions.

THE DIET.—The diet to be given in acute febrile diseases varies somewhat with the type of infection. In breast-fed babies no change should be made from the normal diet, except possibly slight dilution of the breast milk by giving boiled water, or water and lime water immediately before each nursing. In artificially-fed infants, it should be remembered that in acute febrile diseases the digestive power is lowered. Consequently the composition of the food must be adapted to the digestive power of the child by a reduction in the percentages of all the food elements, and fat is especially badly borne in acute illness. On the other hand, the child should be fed up to the limit of its digestive power. The feeding must be carried out on the principle described in the division on Feeding. It is unnecessary under ordinary circumstances to change the amounts given at a feeding and the intervals between feedings.

Older children with acute febrile diseases must be fed more frequently with smaller quantities than in health, the usual interval being every four hours during the day, and sometimes an additional night feeding is given.

Children should always be fed at regular intervals, should never be fed oftener than every three hours, and should never be given milk between feedings when they ask for a drink. The diet should consist mainly of farinaceous food. In children under five years of age, the milk must usually be somewhat diluted with water or lime water. When children do not take milk readily, they may be given Kumyss, beef juice, or broth, as substitutes. Broth and beef juice are also often a valuable part of the routine diet in addition to the milk and farinaceous foods, when they can be digested. Ice cream and jellies are likely to disturb the stomach and should not be given. Raw eggs beaten up with a little sherry, a little sugar, and cracked ice are usually taken well, and form a valuable addition to the diet. Orange juice or grape juice may be given with feedings at which no milk is taken. Among the farinaceous foods, oatmeal and barley gruels are the best.

The physician should note carefully the total amount of food taken each day. It is undesirable to force food upon children who have marked loss of appetite. In an acute febrile disease, anorexia is Nature's method of protecting the patient against disturbance of digestion. On the other hand, anorexia may be so extreme that an insufficient amount of food is taken. If this occurs only during the first one or two days of the illness, no attention need be paid to it, even if practically no food be taken. If, however, it continues longer than the first two days, forced feeding may be necessary. In such cases gavage must be resorted to. Gavage is most often indicated in infants, but is occasionally required in older children also. If, however, in older children insufficient food is taken on account of anorexia rather than on account of delirium or coma, an effort should be made to increase the amount of food taken by persuasion, before resorting to gavage.

THE BOWELS.—The nurse should be directed to give an enema every other day as a routine, if the bowels do not move. When the physician finds that these enemata are required, he should order a mild laxative to be given daily, provided that it does not upset the stomach. Magnesia, or some mild saline may be used for this purpose.

It is usually a good plan to empty the bowels at the onset of an acute febrile disease. For this purpose castor oil is best; if there is vomiting, calomel should be used.

THE TREATMENT OF SYMPTOMS.—As there is no specific drug treatment for the acute febrile diseases, no drug should be given as a routine. The majority of cases of acute febrile disease in children require no drug treatment whatever. It is, however, very commonly said that it is difficult in private practice to retain the confidence of the parents of the patient if no medicine is given. They do not realize that the hygienic care, nursing, and attention to the diet and bowels are the chief essentials of treatment, and feel that if nothing is prescribed, nothing is being done for the child. In my experience, the old superstition as to the necessity of medicinal treatment is gradually passing away; the modern mother of the educated classes, in this community at least, often not only knows that routine medicinal treatment is not necessary, but is even apt to judge any physician who uses it as old fashioned and behind the times. I realize, however, that among many classes of people, and in other communities, the parents of sick children demand drugs. In such cases the physician should take care to prescribe nothing which can possibly do any harm. Sweet spirits of nitre in small doses will sometimes satisfy the parents. On the other hand, the physician should watch most carefully for every symptom which *does* require treatment, or which can be benefited in any way by treatment. When such symptoms arise, the proper treat-

ment should be instituted at once. Even in these cases, however, the various therapeutic measures other than drugs are usually of the greatest value.

COMPLICATIONS.—The physician should watch most carefully for the appearance of complications, as they are not uncommon in the acute febrile diseases. An adequate physical examination should never be neglected at any visit. One of the chief dangers in the acute self-limited diseases is the liability to complication, and their early recognition and proper treatment is of the first importance.

QUARANTINE AND DISINFECTION.—The measures used in preventing the transmission of the infection to other persons will be described under the contagious diseases.

ROUTINE TREATMENT OF A CHRONIC DISEASE

In many chronic diseases it is not necessary for the child to be kept in bed. Whether or not bed is necessary depends entirely upon the patient's strength. Chronic cases should have as much open air treatment as possible, and whenever weather conditions permit, the child should be taken out doors or its bed should be placed out doors. The hygienic measures applicable to chronic diseases are the same as those indicated for the healthy child, with whatever modifications may be demanded by the character of the disease. The same is true of the diet. In my experience, many children with chronic disease are kept in bed too much.

DIVISION III

DISEASES OF THE NEWBORN

I. MALFORMATIONS

The commonest diseased conditions seen in newborn infants are the various gross developmental lesions known as malformations. The malformations are so very numerous, that their detailed description would occupy much space. Almost any part of the body may be involved. Usually the malformation is external, and its nature is recognizable at once. The only question which confronts the physician is whether the condition is one which offers hope of relief through surgical treatment. Occasionally, the malformation is internal, and gives rise to symptoms of functional disturbance of so vague a character that a diagnosis cannot be made at once. Such cases play an important part in diagnosis.

The most important malformations are described in the divisions devoted to the different organs or tracts of the child's body.

II. TRAUMATIC CONDITIONS

CAPUT SUCCEDANEUM

This is a lesion of the scalp, seen in newborn infants, and caused by the mechanical conditions attending labor, especially when pro-

FIG. 54

Caput succedaneum. Male, 2 hours old

longed. There is an extravasation of serum, or blood, into the subcutaneous tissues of the scalp. A swelling is seen, sometimes on one side, sometimes on both sides of the cranium. The tumor is soft

and flabby, and does not fluctuate. It is at its height at birth, and always disappears spontaneously in the course of two or three days.

CEPHALHEMATOMA

This condition also is due to an injury received during birth, which causes a rupture of the sub-periosteal blood-vessels. The result is a hemorrhage between the periosteum and the bones of the cranium, which manifests itself as a swelling of the scalp. This swelling is usually not present immediately after birth, but appears in the first three or four days of life. The tumor is soft and fluctuating, reaches a variable size, and is usually situated over one parietal bone, rarely over both. The tumor usually continues to increase in size for about a week, becoming more tense, and may become so large as to cover the whole parietal bone. After a stationary period, it begins

FIG. 55

Double cephalhematoma. Infant, 4 days old

to diminish very slowly, becoming softer, and often it does not completely disappear until as late as the fourth month. The skin over the tumor appears unchanged, and there is no tenderness, nor other signs of inflammatory reaction. After the tumor has been present a few days, an elevated ridge presenting a bony edge can be felt surrounding the tumor.

Cephalhematoma is distinguished from caput succedaneum, in that it is not at its height at birth, but continues to increase in size, is fluctuating, becomes more tense, and does not disappear so rapidly. It is distinguished from abscess of the scalp by the absence of signs of inflammation. In some cases, a cephalhematoma becomes con-

verted into an abscess through infection. In case of doubt, the diagnosis can be settled by aspirating the contents of the tumor with a hypodermic needle under the strictest aseptic precautions.

Cephalhematoma disappears spontaneously, and requires no treatment other than protection from external injury. When infected, the treatment is that of an abscess.

HEMATOMA OF THE STERNO-CLEIDO-MASTOID MUSCLE

This condition also is a traumatic birth injury. Either from the violence of the expulsive efforts of the uterus, or from too great violence on the part of the obstetrician, the sterno-cleido-mastoid muscle is torn in such a way that there occurs a hemorrhage into its sheath. Soon after birth it is noticed that the infant holds its head to one side, and when the cause of the torticollis is sought, one sterno-cleido-mastoid muscle is found contracted, and along the tense muscle a small tumor is felt. Sometimes, especially in infants with fat necks, the tumor is difficult or impossible to feel. It is soft at first, and tender to the touch; later it becomes smaller and harder. Efforts to straighten the neck cause crying.

The tumor disappears spontaneously, but the torticollis is apt to persist. It is probable that most cases of torticollis in young infants are due to this cause.

No treatment should be employed until after all pain and tenderness have disappeared, when massage, and gentle manipulations designed to stretch the shortened muscle should be begun. If these measures fail, the child should be referred to the orthopedic surgeon, for treatment by apparatus or operation.

INTRACRANIAL HEMORRHAGE

ETIOLOGY.—The commonest cause of the intracranial hemorrhage seen in newborn infants is trauma, the injury being due to the violence attending parturition. Hemorrhage is seen following both operative and normal labor. Everything which increases or prolongs pressure upon the head during labor may be a predisposing cause. Such conditions as difficult or prolonged labor, precipitate labor, difficulty in extracting the head in breech presentations, and difficult instrumental deliveries, may act as causes. Occasionally intracranial hemorrhage is seen as a manifestation of hemorrhagic disease of the newborn. Rare cases may be associated with syphilis, or infection of the newborn.

PATHOLOGICAL ANATOMY.—The hemorrhages may occur over the cortex of the brain, or at the base. They are found most frequently beneath the tentorium, usually over the cerebellum, or the occipital lobes of the cerebrum, but hemorrhages over the cortex

are not uncommon, and even hemorrhage into the ventricles has been reported. The extent of the hemorrhage and amount of extravasated blood are very variable. There may be a single large clot, or diffuse hemorrhage. The lesion may be confined to one hemisphere, or extend over both. The extravasation may be beneath the pia mater, or in the arachnoid space between the pia and the dura.

The source of the hemorrhage is also variable. In a large proportion of cases, lacerations of the tentorium have been found. The blood may come from a laceration of one of the sinuses of the dura from overlapping of the bones, but more frequently, when there is no laceration of the tentorium, the blood comes from one of the large veins empyting into the sinuses, or from the rupture of a small vessel in the pia.

SYMPTOMS.—In intracranial hemorrhage, one would expect the symptoms to be those either of increased intracranial pressure, or of cortical irritation. In certain cases such symptoms are present to such a well marked degree, that the picture of a cerebral lesion is very plain. Such cases are frequently asphyxiated at birth, and are only resuscitated after considerable effort. They may show cyanosis, difficult, shallow, or very irregular respiration, bulging fontanelle or slow pulse. They may show twitching of the extremities, or even convulsions, especially in cases which last several days. There may be spasm of the extremities, or paralysis of the face, or of one or more of the limbs, and there may even be a general rigidity or opisthotonus. In these cases the deep reflexes are increased, the pupils may be dilated, or more commonly, contracted, and there may be strabismus or nystagmus.

The localizing symptoms of cortical irritation are more frequently seen when the hemorrhage is situated over the cortex. When the hemorrhage is at the base, the symptoms are those of increased pressure. In these pronounced cases, showing symptoms of this kind, death may occur within twenty-four hours, and with very large hemorrhages, the children are usually still-born.

Nevertheless, pronounced cases giving these distinct symptoms of a cerebral lesion, are comparatively less common than those in which the symptoms are much vaguer, and less suggestive either of increased intracranial pressure, or of cortical irritation. In such types the diagnosis is equally important, but more difficult.

The symptoms are frequently not seen at birth, but develop at some time during the first four days of life, usually at about the second day. The commonest and most suggestive symptom is refusal to nurse. The babies are pale, and show either restlessness, or seem very dull and stupid, rousing with difficulty. There is often at this time a slight facial edema, which, when present, is a sign of

great significance. The appearance of refusal to nurse in a newborn baby should always suggest the possibility of intracranial hemorrhage, while pallor and facial edema greatly strengthen suspicion. Careful examination for cyanosis, disturbed breathing, tense fontanelle, slow pulse, or signs of cortical irritation will often confirm the suspicion, but it must be remembered that *localizing nervous symptoms are frequently absent.*

DIAGNOSIS.—When there are found the symptoms of refusal to nurse, pallor, and possibly facial edema in a newly born baby, intracranial hemorrhage should always be suspected. When in addition are found signs either of increased intracranial pressure, or of cortical irritation, the diagnosis is confirmed. In the absence of these confirmatory signs, the condition should always be suspected. In doubtful cases, lumbar puncture should be performed. If the fluid obtained is bloody, the diagnosis of intracranial hemorrhage is verified, provided that the operator has performed the puncture carefully, and is sure he has not wounded a local blood-vessel. If the fluid is clear, intracranial hemorrhage is not excluded, as only in cases where the hemorrhage is below the tentorium, will the fluid be blood stained. In a case which remains doubtful after a negative lumbar puncture, confirmation of the diagnosis may be obtained by aspiration of the subdural space, as advocated by Henschen. The technic is simple. The aspiration is made through a fontanelle or suture, care being taken not to wound the dural sinuses. The most favorable point is at the lateral angle of the anterior fontanelle. The needle is entered in a direction almost parallel with the surface of the skull, and if blood is not immediately obtained, may be carefully swept in various directions. The middle line must not be crossed on account of the falx. If blood is found, as much may be removed as will flow through the needle.

PROGNOSIS.—The prognosis is always serious. Hemorrhages below the tentorium are probably always fatal, as are large hemorrhages in any situation. Death may not occur for four or five days. When the hemorrhage is situated over the convexity of the brain, and is not too large, the child may survive. In such cases there is always serious damage to the brain, which usually manifests itself later as a cerebral paralysis. The full extent of the damage may not become apparent for a number of years.

It follows that in cases in which the signs of cortical irritation are not marked, but in which the signs are those of intracranial pressure, a fatal ending is most certain.

TREATMENT.—Prophylaxis is the most important measure available in connection with so serious a condition as intracranial hemorrhage. This is mainly a matter for the obstetrician. It

must be remembered also that slight and unavoidable degrees of trauma may be followed by hemorrhage. The indications are to shorten prolonged and tedious labor, to avoid compressing the head too violently with the hand through the perineum, and to avoid difficult high forceps operations whenever possible.

Aspiration of the clot through the fontanelle or suture will sometimes give relief of symptoms. If, when this procedure is undertaken for diagnosis, a considerable amount of blood can be withdrawn, it may be wise to wait before trying anything further. If little or no blood is obtained, the only treatment is by operation.

The operative treatment of intracranial hemorrhage of the new-born consists of craniotomy and washing out of the clot. The operative risk is necessarily great, but a certain number of cases have recovered. Out of seventeen cases thus treated prior to 1914, seven recovered.

OBSTETRICAL PARALYSIS

ETIOLOGY.—Paralysis in new-born babies is due to trauma received during parturition. It may be one of the symptoms of intracranial hemorrhage, as already described. The form known as obstetrical paralysis, or birth palsy, is caused by an injury to the peripheral nerves. Two types of paralysis are seen, one involving the muscles of the face, the other involving the muscles of the shoulder and upper arm. In the facial type, the injury is usually due to the use of forceps, which press upon the facial nerve, either near the point of exit from the stylo-mastoid foramen, or where it crosses the ramus of the jaw. While pressure from forceps is the usual cause, some cases have been seen in which the forceps were not used. In these cases, it is believed that the injury is received from pressure following long arrest of the head at some point in its passage through the pelvis.

In the upper arm type, the injury received during labor may be caused in a variety of ways. It is seen more commonly after artificial labor, but may occur when labor has terminated naturally. In vertex presentations, it may be caused by pressure from a forcep's blade which extends down to the neck, by traction upon the head while it is in a rotated position, or by traction with the obstetrician's finger in the axilla. The injury is much more frequent in breech presentations, in which it may be produced by traction upon the shoulder in delivering the head, or by pulling or twisting the arm, especially in bringing down the arms when they are above the head.

PATHOLOGICAL ANATOMY.—In facial birth palsy the injury is usually comparatively slight, and consists of exudation produced within the sheath of the nerve bundle by pressure. In the upper arm type, often known as Erb's Paralysis, the injury affects the

nerves which form the brachial plexus, the nerve trunks involved being usually the fifth and sixth, and sometimes the seventh cervical nerves. In the severer cases, the stretching may result in the rupture of the plexus, or in hemorrhage about the plexus. In milder cases there is hemorrhage within the connective tissue sheath of the cord of the plexus, with a resulting fusiform fibrous swelling. In the mildest cases the lesion is simply exudation within the sheath from stretching of or pressing upon the nerve bundle. The muscles most commonly affected are the deltoid, supra- and infra-spinatus, brachialis anticus, biceps, supinator longus, and probably also the supinator brevis.

FIG. 56

Showing characteristic position of the arm in obstetrical paralysis in a girl of 20 months

SYMPTOMS.—Facial paralysis and severe upper arm paralysis are usually noticed on the first or second day. Mild upper arm paralysis may not be noticed for a week. Facial paralysis is recognized by lack of symmetry in the two sides of the face. This is not very apparent when the child is in repose, but it can usually be noticed that the eye on the affected side remains open. When the baby

cries, the whole side of the face is seen to be affected, the paralyzed side appearing smooth and full, while the mouth is drawn toward the sound side. Nursing is not interfered with, as the lips are little used in sucking, and the tongue is not involved.

In Erb's Paralysis, the arm hangs limp and motionless, close to the side; the humerus is rotated inward, and the forearm is pronated so that the palm looks outward. It is notable that the movements of the wrist and hand are not affected, nor is the triceps, so that the forearm can be extended, but cannot be flexed. The reaction of degeneration is present in the severer cases. Atrophy usually begins after a few weeks, but the muscles are comparatively so small, and so covered with fat, that it is usually not perceptible until paralysis has persisted for one or two years.

DIAGNOSIS.—Facial birth paralysis has to be distinguished only from a facial paralysis of central origin. It is extremely rare that facial paralysis is the sole manifestation of intracranial hemorrhage, and it can usually be easily excluded by the absence of signs of intracranial pressure or of cortical irritation. Moreover, in facial paralysis of central origin the upper segment of the facial nerve is not affected, there is no paralysis of the orbicularis palpebrarum, and consequently the eye can be closed.

The upper arm type of obstetrical paralysis is also easily recognized. Here again, in a paralysis of central origin, it is extremely rare for the arm alone to be affected. The characteristic grouping of the muscles affected in obstetrical paralysis, together with the absence of the signs of intracranial pressure or irritation, usually make the diagnosis sufficiently clear. The reaction of degeneration, when present, is further proof of the peripheral origin of the paralysis.

Erb's Paralysis, however, has at times to be carefully distinguished from surgical injuries received during birth, such as fractures, dislocations, or separation of an epiphysis in the shoulder. Careful examination, especially for tenderness, will exclude these conditions.

PROGNOSIS.—The prognosis of the facial type of birth palsy is good. Only in cases very rare and very severe, may the paralysis be permanent, or even last for several months. In the majority of cases it disappears without treatment in about two weeks.

The prognosis of the upper arm type is very variable, depending on the extent and character of the injury to the nerve plexus, and upon the treatment. As time passes, partial recovery is always seen, even with untreated cases. In some cases spontaneous recovery is seen in the course of a few months. Even when recovery does not occur in untreated cases, the flexion of the forearm at the elbow and the abduction of the arm from the trunk are usually regained. Pronation of the forearm, and external rotation of the humerus are

the movements which remain most persistently poor. With treatment begun at the proper time, recovery usually occurs after a time varying from a few months to one or two years.

In the majority of untreated cases, and in a few of the severer cases even under proper treatment, the paralysis persists, to a varying degree. Atrophy of the muscles becomes noticeable, especially

FIG. 57

Obstetrical paralysis

in the deltoid, the shoulder droops, and there is a lack of development of the bones of the shoulder girdle as a result of disuse. The arm appears shrunken, and shorter than on the sound side. Contractions in the muscles of the forearm and hand may eventually develop. Finally, in the worst cases, fortunately rare, there is loss of response to either the galvanic or faradic current, and the arm remains comparatively useless.

It is a noticeable feature in this type of paralysis, that even in severe cases, atrophy takes place comparatively slowly. In comparison

with similar paralysis due to poliomyelitis, there is, as pointed out by Thomas, a notable lack of flaccidity, hypotonia, and atrophy in the affected muscles. This fact is of value in prognosis, as showing that considerable good may be expected in cases which have gone for a long time untreated, if proper therapeutic measures are instituted.

The presence or absence of the reaction of degeneration is of some value in prognosis. Its presence points toward a severer type of lesion.

TREATMENT.—The earlier treatment of these cases is by massage, electrical stimulation, and educational gymnastics. Treatment should be begun at the end of the first month, and should be continued regularly and systematically for many months.

Electrical stimulation has always been a conventional part of the treatment of these cases. I have never been able to convince myself that it is of very great value. It has been given up at the Children's Hospital, partly because it is much less effective than the other measures, and partly because the muscles which need exercise the most, namely, the spinati, cannot be reached by electrical stimulation.

The treatment by massage and educational gymnastics, as practised by Dr. Thomas of the Children's Hospital, seems to me to be most satisfactory, and I have seen a number of strikingly favorable results from its use. As described by Thomas, the treatment is carried out as follows, consisting of three parts:

1. Passive movements of the joints, shoulder, elbow, wrist and fingers, best done by some one who knows the anatomical possibilities and limitations of these joint movements. These movements need to be continued until the arm has entirely recovered.

2. Massage of the gentle kneading form, to stimulate growth, which may be discontinued when the arm keeps up in growth with the unaffected one.

3. Exercises, to induce the child to use the arm voluntarily, which vary with the age of the child.

III. NON-TRAUMATIC MECHANICAL DISTURBANCES

This group represents certain lesions of a mechanical nature, of which the anatomy and mechanism is understood, but of which the ultimate etiology is unknown. The most common conditions of this group are the various forms of hernia seen in newborn babies. Both inguinal and femoral hernia are sometimes seen at birth, but are not strictly to be classed as diseases of the newborn. There are other mechanical conditions which, while they may be seen at birth, occur more frequently at a later age; examples of such conditions are volvulus and intussusception.

UMBILICAL HERNIA

This is the only common mechanical condition strictly to be classified among the diseases of the newborn, and even this often does not

FIG. 58

Large umbilical hernia. Infant 5 months old

appear for a considerable time after birth, its development being favored by poor nutrition. When it appears shortly after birth, it is most frequently seen in premature, or congenitally weak infants.

There is a soft tumor at the umbilicus, caused by a protrusion of a small part of the abdominal contents through the umbilical ring, which is found dilated. The tumor varies much in size, the usual hernia being about half an inch in diameter, but occasionally it may be very large. It rarely becomes strangulated, and is usually not serious.

TREATMENT.—In the first two or three months, after the cord has separated, a firm pad should be placed over the navel, kept in place by a rather snug abdominal band. In cases coming under observation at a later period, the pad and band are not enough, and adhesive plaster should be used. A coin or button covered with kid may be placed over the umbilicus, and kept in place by strips of adhesive plaster. I prefer the procedure which we use at the Infants' Hospital, to the use of a coin or button. After the parts are well powdered, and the hernia is reduced, the skin of the abdomen on each side is picked up between the finger and thumb of each hand, in such a manner as to make two vertical folds. These folds are then brought over to the middle line, so that they touch one another over the center of the navel. They may then be held in place by the finger and

FIG. 59

Adhesive strap for umbilical hernia

thumb of one hand, while the other hand applies snugly a broad strip of adhesive plaster. This holds the two folds of skin together over the navel in such a way that they form the pad which retains the hernia. This plaster may be changed every few days, and must be worn for months.

Very large herniae, or persistent herniae can only be cured by surgical operation. Operation. however, is seldom necessary.

There is a rare condition of hernia into the umbilical cord, which is entirely different from the commoner umbilical hernia. This is a malformation, due to some fetal defect. It varies in size from a comparatively small tumor to complete eventration in which most of the abdominal organs are in the sack and outside the body. Such severe cases, with complete eventration, are fatal. Milder cases, even though the tumor is quite large and contains a number of coils of intestine, may be cured by surgical treatment.

PROLAPSE OF MECKEL'S DIVERTICULUM

This is a condition which produces a tumor at the umbilicus in newborn babies, and with it there is usually an umbilical fistula. The tumor is formed by a prolapse of the mucous membrane of Meckel's Diverticulum. Normally the omphalo-mesenteric duct closes completely during fetal life. The duct may however remain patent to a varying extent, the most common finding in such cases being a blind pouch a few inches in length given off from the lower part of the ileum. In rare cases it may be patent at birth throughout its entire extent, and a prolapse of its mucous membrane produces a tumor at the umbilicus with a fistula at its summit.

These tumors are usually small, not larger than a common marble, but may be much larger. They are smooth, of a pink color, and cannot be reduced. A mucous discharge oozes from their surface, and a slight fecal discharge comes from the fistula. Tumors are sometimes seen without a fecal fistula, but microscopic examination shows that their covering is the same as in cases with a fistula, the structure being that of the intestinal mucosa. In other cases, the fecal fistula may exist without a tumor. These fistulae may exist for months or years, may close spontaneously, or be closed by operation. The small tumors most often seen are easily cured by ligation. The rare large tumors are usually associated with serious malformation of the intestines.

IV. NEW GROWTHS

While various forms of new growths may be present at birth, their occurrence is so rare, that they constitute pathological curiosities.

UMBILICAL GRANULOMA

After the cord has fallen off, there is sometimes seen at the umbilicus a reddish, moist, secreting mass of granulations. This condition is usually due to improper care of the stump of the cord, in which case a mild infection may play an etiologic rôle. The condition is commoner in hospital out-patient than in private practice. The usual story is that the navel will not heal. The tumor varies in size from the head of a pin to a pea. The discharge is irritating, so that the skin around the umbilicus may be eczematous. A neglected overgrown granuloma becomes a polyp. These polyps are more vascular than the simple granuloma, and have a pedicle.

TREATMENT.—The simple granuloma is best treated by one or more applications of a 50 per cent. nitrate of silver solution, and by keeping the part thoroughly powdered with some absorbent powder. Polyps should not be treated by cutting the pedicle and applying nitrate of silver, as hemorrhage is sometimes severe. The pedicle should be ligated, and the polyp, kept freely powdered, will dry up and fall off.

NAEVUS

This is a congenital new-formation of tissue in the skin; the new-formed tissue may be pigmentary or vascular.

ETIOLOGY.—The etiology of naevus, as of new growths in general, is obscure. None of the theories as to the cause of naevus advanced at various times, have been supported by any proof. One theory is that birth marks are produced by intrauterine pressure; the only evidence in support of this theory is the frequent occurrence of birth marks at the back of the head or neck, and on the face. Virchow called attention to the frequency of naevi at embryonic fissures of the skin, where slight irritation might easily give rise to abnormal vascular activity.

PATHOLOGICAL ANATOMY.—Various forms of pigmented moles are seen at times, of various shades of brown or black. Some are smooth, some rough and warty, some hairy.

The commoner forms of naevi are vascular, and include the well-known so-called birth marks. They vary in histology, in size, and in appearance, from the small capillary angioma, to the large, disfiguring "port wine" stain. The appearances vary with the number, and degree of dilatation of the new-formed vessels. True vascular naevi become pale on pressure.

FIG. 60

Naevus of face and neck in an infant 4 months old

TREATMENT.—The smaller pigmented moles are best treated by excision. It must be remembered that metastasis and general sarcomatosis have occasionally followed mechanical interference with certain forms of moles. The same thing, however, occurs with equal if not greater frequency when there is no attempt at excision, and it is probable that children are not so liable to metastasis as adults. Smooth pigmented moles should be left alone, if not in a disfiguring situation. The warty variety, in my opinion, should be excised.

Large hairy moles, and vascular naevi, are treated by electrolysis, or multiple puncture. Some small capillary naevi will disappear under the pressure produced by frequent applications of collodion (Kerley).

The best results in the treatment of vascular naevi, have been obtained by freezing with liquid air or carbon dioxide snow.

V. FUNCTIONAL DISTURBANCES

CONGENITAL PULMONARY ATELECTASIS

This condition is one in which the whole or parts of the lung remain in a fetal condition, air not entering the alveoli.

ETIOLOGY.—It is usually stated in textbooks that atelectasis is closely associated with *Asphyxia Neonatorum*, this statement being based on the fact that infants born dead, or dying as the result of asphyxia show a complete or partial condition of atelectasis post-mortem, and that most cases in which infants die in the first few days of life, and in whom the lesions of atelectasis are found post-mortem, have shown symptoms of asphyxia at birth. I believe, however, that the connection between asphyxia and atelectasis is not so close as is generally supposed. It is a well-known fact that the expansion of the lung is not normally completed immediately after birth, but is a gradual process, occupying a number of days before complete expansion of the lung is attained. It follows that a certain amount of atelectasis would always be found post-mortem in infants dying in the first few days of life, whether they die from asphyxia, or from any other cause. Further, most of the conclusions upon the cause of atelectasis are based on post-mortem examinations of infants dying in the first few days of life, and in these cases asphyxia may well have been the cause of death, or it may have been a symptom of a feeble vitality which was the cause of death. In my experience, infants who survive the early days, and later show the lesions or signs of atelectasis, do not give a history of asphyxia at birth.

Atelectasis becomes a pathological condition when it persists beyond the early days of life. The cause is congenital weakness of any kind, such as is seen in premature infants, in congenitally weak or malnourished infants, and in infants suffering from congenital syphilis or cerebral hemorrhage. Anything which impairs the vitality of the newborn infant, causes weakened respiratory efforts, and asphyxia undoubtedly may be numbered among the causes of such weakness. In addition to weakened respiratory efforts, in feeble infants there is weakened heart action, leading to pulmonary congestion or edema, and the two together form a vicious circle of which atelectasis is the result.

Asphyxia is usually due to some intrauterine condition interfering with the placental circulation, such as prolonged labor, the use of ergot in the second stage, pressure on the cord, winding of the cord about the neck, or early separation of the placenta. It is a condition

:he pediatrist, its diagnosis and treatment falling more
obstetrician, and it is so adequately described in text-
trics, that it will not be described here.

;ICAL ANATOMY.—In infants who are born dead
, atelectasis is complete, the lung showing a fetal
ere are no alveoli, the tissue consisting of closely packed
; and blood-vessels, and is recognizable as lung tissue
ɔronchioles with their characteristic epithelium. In
:athe at birth, but who die from asphyxia or congenital
ıy time in the first few days of life, the amount of un-

FIG. 61

ɔngenital atelectasis of the left lung. Hypertrophy of the heart

is very variable, varying to a certain extent with the
the child survives. At the borders of completely col-
·e areas where some air-containing alveoli are seen, there
ıl transition to the areas where the lung is completely
n in the most collapsed areas are found some air spaces.
to the fetal condition, there is frequently edema
ɔanded alveoli of the atelectatic areas. The blood-
ıgested.

In children who survive the early days of life, there is little evidence available as to the pathological anatomy. It is probable that there is a persistence of the fetal condition in more or less extensive areas of the lung.

The portions of the lung most frequently involved in congenital atelectasis are the paravertebral and central portions, particularly the area just behind the hilus. The outer portions and borders expand better. The best expanded portions are usually the anterior borders of the upper lobes. The left lung is more frequently and more fully involved than the right.

SYMPTOMS.—In the beginning, at birth, the symptoms of atelectasis are the same as those of asphyxia. In other words, a certain amount of asphyxia is frequently present at birth in cases of atelectasis. During the first few days of life the symptoms are those of general feebleness and low vitality. The infants are somnolent, do not cry strongly, and do not take their nourishment well. Their faces are pale and expressionless. In the severer cases, the respiration is notably defective, being irregular and shallow, with occasional long pauses followed by a single deep gasping inspiration, after which shallow irregular breathing is resumed. During these deep inspirations, which may be brought about artificially by slapping the child with the hand, or by applying cool water to the chest, crepitant rales may frequently be heard in various parts of the lungs. Sometimes it may be noted that the respiratory movements are not symmetrical, one side of the chest moving more than the other.

In the majority of cases of atelectasis physical signs on auscultation and percussion cannot be found. In rare cases there is considerable dullness and diminished breathing over a varying area of the lung.

The further course of these cases is very striking. The children do not thrive, but seem feeble, gaining very slowly, or not at all in weight, with a tendency toward cyanosis, cold extremities, and subnormal temperature. Their cry remains notably weak. If they survive the early weeks, they remain small and delicate. A child with very slight atelectasis may eventually develop normally, but in these cases, the atelectasis is usually not to be recognized. Occasionally a child survives with an extensive atelectasis. Such children remain very much under-sized in both height and weight. I know of one such child, who has reached the age of twelve years. One lung is still almost wholly collapsed, showing dulness and diminished breathing over the greater part of the left chest. The chest wall has sunk in over the collapsed lung, causing a marked deformity of the chest. This patient was very delicate during infancy and early childhood, but is now strong and healthy, though very much undersized.

:S.—Any child who dies in the first days of life will :electasis post-mortem. In children who survive the : diagnosis is difficult. If asphyxia has been present if the children show impaired vitality with notable f the respiration, atelectasis may be suspected. It ;nosed with certainty except in those rare cases which alities to auscultation and percussion. In a certain ses not showing physical signs, the condition will be)entgen ray examination.

IS.—Slight degrees of pulmonary atelectasis are prob-)le with normal development. Such cases, however, cognized. In the majority of cases the children are ıd in babies born with such low vitality, the mortality A certain number die in the first days of life; others 'or several weeks. A few cases, even those in which been clearly recognized, survive the early weeks. They small, and delicate, and are difficult to rear. A few :ven these show backward growth and development.

NT.—At birth, the physician should make sure that strongly, and should see to it that this cry is repeated he various means of resuscitation employed in asphyxia adequately described in works on obstetrics. Feeble m atelectasis is suspected, should have the treatment)ntinued. During each twenty-four hours, they should their cribs a number of times, and thoroughly aroused, made to take a number of deep inspirations. This may out in various ways. In the milder cases shaking or pplying cool water to the face and chest, will suffice. s they should be dipped alternately into warm and cold)ne in asphyxia.

the treatment is that applicable to any premature, or eeble baby. They must be kept warm, often with in the form of hot water bottles. Sometimes their ill remain subnormal unless the "premature jacket" is bed for premature infants. They are often too feeble reast, yet breast milk is almost an absolute essential to ıtrition. In such cases the breast milk must be drawn)ump, and given to the baby with a medicine dropper, with a Breck feeder.

)RRHAGIC DISEASE OF THE NEWBORN

in the first few days of life is seen in connection with y of recognizable causes. The trauma of birth not roduces a lesion of the blood vessels, but in hemor-

rhages of simple traumatic origin, there is no tendency for the bleeding to be either unduly prolonged, or uncontrollable, and the purely traumatic origin of the hemorrhage is usually plainly apparent. The recognized infections of the newborn are frequently attended with hemorrhage. In these cases also, the symptomatic nature of the hemorrhage is usually apparent, and the clinical picture is comparable with that sometimes seen in cases of infection in later life, with a tendency to hemorrhage, such as occurs at times in measles, smallpox, and malignant endocarditis. There remains a class of cases in which the hemorrhages are not associated with any other recognizable process, and in which the bleeding does tend to be both unduly prolonged, and uncontrollable. A very few cases of this character represent manifestations shortly after birth of that particular congenital and hereditary hemorrhagic disease known as hemophilia. Hemophilia, however, is a disease which persists throughout the life of the patient, and the appearance of hemorrhage soon after birth is not a characteristic manifestation of the disease, but only accidental, occurring in comparatively few cases. In these cases, if there is traumatic injury sufficient to produce hemorrhage, the hemorrhage attracts attention by its persistent and uncontrollable character. Most cases of hemophilia do not manifest themselves at birth.

Finally, we have a class of cases in which prolonged and uncontrollable hemorrhages occur in newborn babies, and in which the disease is self-limited, the tendency toward hemorrhage ceasing permanently when recovery occurs. These cases, in the present state of our knowledge, are grouped under the term *Hemorrhagic Disease of the Newborn.*

ETIOLOGY.—The etiology of hemorrhagic disease of the newborn is one of the obscure problems of medicine. There are two principal possibilities as to the cause of these hemorrhages. The first is that the bleeding is due to some peculiarity in the tissues of the newborn infant. The second is that it is a manifestation of some obscure infection. Under the first possibility the tissue peculiarity cannot be a permanent defect, because hemorrhagic disease of the newborn is self-limited, and is thus distinguished from hemophilia, in which the probable cause is a permanent tissue defect. The defect in hemorrhagic disease of the newborn, if such a defect exists, must be developmental in origin, a condition incidental to the relative physiological incompleteness of the newborn baby. Such a defective, incomplete condition of the tissues might involve either the blood itself, or the walls of the vessels.

Under the other possibility, in which the hemorrhage is attributed to some obscure infection, the immediate cause of continued bleeding is also to be sought in some defect either of the blood itself, or of the

vessel walls. The only difference is that under this theory, the cause of the defect is not developmental incompleteness, but infection working upon the tissues through toxins.

The disease is not a very common one. The majority of reports show a percentage incidence of under 2%. Townsend's report from the Boston Lying-in Hospital showed 32 cases out of 5,225 births, or .57%. Green and Swift, in a more recent series from the same hospital, report 51 cases out of 4,455 births, or 1.14%. In Prague, Ritter observed 190 cases out of 13,000 births, or 1.46%. The highest record I have seen is 8% out of 740 births. The disease is more frequent in institutions than in private practice. In Green and Swift's series, there was a notable seasonal incidence, the disease being more frequent in the six months from November to April inclusive. Males are only very slightly more frequently affected than females. Syphilis is associated with hemorrhagic disease in only a small proportion of cases.

PATHOLOGICAL ANATOMY.—There is no constancy in the lesions found post-mortem in hemorrhagic disease of the newborn. In the majority of autopsies nothing is found except the hemorrhages in the various situations, and the consequent anaemia of the organs. The stomach and intestines frequently contain considerable blood, but usually show no lesions, except areas of congestion or ecchymosis on the mucous membrane. A few cases have been reported in which ulcers have been found on the gastric or intestinal mucosa. The proportion of these cases is small, and the cause of the ulcers obscure. The changes found in the blood have not been uniform, and have not been thoroughly studied.

SYMPTOMS.—The hemorrhage begins usually in the first week of life, and very rarely occurs after the twelfth day. The majority of the infants are two or three days old.

The hemorrhages are usually multiple. They occur from a great variety of situations, including the intestine, stomach, mouth, nose, umbilicus, skin, genito-urinary tract, eyes, meninges. In Green and Swift's series, the cases arranged themselves in three distinct clinical groups. The largest group is the sero-mucous, in which the bleeding comes from the mucous or serous membranes. In the second largest group the principal bleeding is from the umbilicus. The smallest group is characterized principally by the formation of subcutaneous purpuric patches or ecchymoses.

TABLE 17

Classification of Green and Swift's Series

Clinical Type	Cases	Recoveries	Deaths
Sero-Mucous	27	13	14
Umbilical	15	6	9
Purpuric	9	7	2
	51	26	25

Usually nothing abnormal is noted until the hemorrhage begins. In the sero-mucous group, the most frequent symptom is hemorrhage from the bowel. The blood is always dark colored, and usually thoroughly mixed with the feces. Clots are comparatively rare. In cases where there is also hemorrhage from the stomach, blood is vomited, usually not in great abundance. The blood vomited is usually dark brown, rarely bright red. In other cases there may be bleeding from the mouth, nose, or conjunctiva. When there is hematuria, the urine is blood-stained, and sometimes contains clots. Occasionally bleeding from the female genitals is severe.

The bleeding from the umbilicus is apt to occur somewhat later than the sero-mucous bleeding. It usually betrays itself first by a slight stain on the dressing, though occasionally it begins more profusely. Hemorrhage into the skin is most apt to appear in places exposed to pressure, but many occur anywhere.

The amount of the hemorrhage is very variable. Sometimes there is only a continuous oozing; at other times larger amounts of blood are lost. There is prostration, which is often marked from the beginning, and there is rapid loss of weight. The temperature may be elevated, or may be normal or subnormal. There is frequently diarrhea.

The hemorrhages continue until death or until recovery occurs. In the mild, favorable cases which recover, the duration is usually not more than one or two days. In fatal cases, death usually occurs in three days. Death may result either from the rapid loss of blood, or more frequently, from failure of vitality.

DIAGNOSIS.—It is sometimes necessary to make sure that the dark masses passed from the bowel are blood and not meconium. In such a case one should first examine the discharges microscopically for blood corpuscles, and if they are not conclusively found on account of their disorganization, a chemical test for hemoglobin should be made.

Accidental, or traumatic hemorrhages are usually easily recognized, and are not persistent. When it is established that a hemorrhagic condition is actually present, the causes to be excluded are hemophilia, and symptomatic hemorrhage in infectious disease of the newborn. Hemophilia is a rare manifestation at birth; males are affected oftener than females in the proportion of thirteen to one; there is usually a history of heredity, particularly in the maternal grandfather; finally the hemorrhage is usually from one situation only.

In infectious disease of the newborn, there is usually a persistent fever, and some signs of infection on physical examination, such signs being most frequently found at the umbilicus.

In a few cases the hemorrhage is into some internal organ, and

does not appear externally. In such cases the diagnosis is so difficult, that the disease usually escapes recognition.

PROGNOSIS.—The mortality of hemorrhagic disease of the newborn is high. It varies in the reports of different observers, the variations depending on the differences in the severity of the disease at different times, and on differences in the treatment employed. Schloss and Commiskey state that the mortality ranges from 35 to 87 per cent. For all forms of the disease 60 per cent. represents an average mortality. In Green and Swift's series the mortality was 60 per cent. in the umbilical type, 50 per cent. in the sero-mucous type, and 22 per cent. in the purpuric type.

In any single case the prognosis depends on the severity of the hemorrhage, the vigor of the child, and the treatment employed. There is no reason for ever giving a hopeless prognosis, for recovery can occur even in the most severe cases.

TREATMENT. The most effective weapon against hemorrhagic disease of the newborn is human blood. Favorable results of a convincing character have been obtained by the use of whole human blood given both subcutaneously, and intravenously, and by the use of human blood serum.

Of the methods of employing human blood, I believe that transfusion is the ideal one, because whatever the cause, transfusion restores to the infant's circulation all the elements needed for coagulation. It further corrects anemia by replacing the cellular elements which have been lost, and dilutes any toxin which may be present to the greatest possible extent. The only objection to transfusion is the difficult technic of the operation when performed on newborn babies.

In view of the convincingly favorable reports of Schloss and Commiskey and others on the subcutaneous use of whole human blood, and those of Welch, confirmed by many others, on the use of human blood serum, these methods of giving human blood deserve a place in the treatment of this disease. I believe that, as suggested by Vincent, a rational combination of all three methods is the best routine treatment which can be employed.

In the beginning the hemorrhages are often trivial, or the infant, when first seen, is not yet either severely exsanguinated or prostrated. In this stage, at the first injection, whole blood may be used to save delay. The donor having been obtained, about 60 c.c. of blood should be withdrawn with a sterile syringe and needle from the vein at the bend of the elbow. Of this, from 10 c.c. to 30 c.c., according to the severity of the hemorrhage, should be injected subcutaneously at once. The rest should be set aside to furnish serum for further treatment. In the subsequent injections, this serum may be used,

and from 10 to 30 c.c. may be given every four to eight hours, according to the course of the case.

If the patient, when seen first, is already so greatly prostrated or exsanguinated as to appear in a serious condition, or if the injections of whole human blood, or human serum are not followed by recognizable improvement, the safest treatment is by transfusion. Under these circumstances, whenever a surgeon is available who is familiar with the more formidable technic of this operation, direct transfusion should be performed.

FIG. 62

Dr. Beth Vincent's apparatus for transfusing. Consisting in paraffin treated tube, compression bulb, and needle

Transfusion is usually followed by immediate and permanent cessation of the hemorrhages. Whenever the hemorrhages once cease, they are not likely to recur. It should be remembered that cases in which hemorrhage is due to bacterial infection, to syphilis, or to ulcers of the stomach and intestine, are not so favorable for treatment with human blood. This may explain the failure of treatment in certain cases.

FIG. 63

)od in paraffin treated tube from the donor in transfusion

FIG. 64

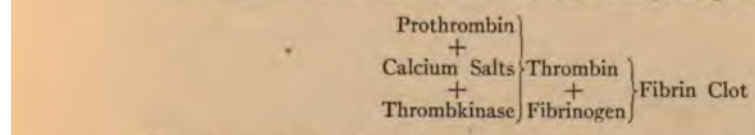
TRANSFUSION IN THE NEWBORN

)d from a paraffin-coated tube into the longitudinal sinus

There are other methods of treatment which have not, in my opinion, given evidence of value sufficiently convincing to warrant their discussion here. Of these rabbit serum has given the best reports. I believe the only indication for its use would be in a case in which human blood or serum was not obtainable.

PROBLEMS AND RESEARCH.—The obscure questions in the etiology and pathology of hemorrhagic disease of the newborn, have been the subject of considerable investigation and experimental research within the last decade. The particular problems at present engaging the attention of investigators are two in number. The first problem is, what are the changes or abnormalities in the tissues which are concerned with this tendency to hemorrhage in newborn babies? The second problem is, what is the cause of these changes or abnormalities? The first problem is intimately connected with the mechanism of the coagulation of the blood, for the tissue defect must involve either the blood, or the walls of the blood vessels.

The mechanism of blood coagulation, while still imperfectly understood, presents certain known features, interference with which might easily lead to prolonged and uncontrollable hemorrhage. The most generally accepted view of the coagulation of the blood is the following: Coagulation is due to the action of thrombin (fibrin ferment) upon the fibrinogen of the blood, or to the union of thrombin and fibrinogen. The term fibrin ferment is now not so much used as formerly, because it is doubtful if thrombin is actually a ferment; its mode of action is uncertain. It is fairly certain, however, that thrombin does not exist as such in the circulating blood, but as it appears shortly after the blood is shed, all its constituents must have been present. The mother substance from which thrombin is supposedly formed is called prothrombin, and this is supposedly derived from the blood platelets, but it has not been isolated. It is theoretically supposed to be held neutralized in the circulating blood by antithrombin (antifibrin ferment), which is believed to have the function of preventing the occurrence of an extending coagulum from the passage of thrombin from its original seat of formation. The other substances involved in the formation of the thrombin are the calcium salts normally present in the blood, and a substance called thrombokinase, which is liberated from the body tissues after injury. This kinase, with the calcium salts, generates thrombin from prothrombin. The mechanism is shown in the following diagram.



When all these elements are present in normal proportion, a normal

iminution or absence of any one of them will
ıt, or a delayed, or absent clot. If the calcium
r absent, it has been found that the coagulation
there is no clot. If the prothrombin is absent,
ulation time will be normal, but the clot will be
rm. If the kinase, or fibrinogen, are dimished
ıtion time will be delayed, with inferior clot, or
ı fluid.
estigation now in use, are the following: Meas-
ılation time; studies on the firmness of the clot;
f adding calcium salts; studying the effect of
ormal human blood; studying the effect of the
rmal blood; artificial extraction of thrombin in

e investigations have not been uniform, and do
diminution or absence of any one particular
morrhagic disease of the newborn. The most
are those of Schloss and Commiskey, and of
ıurg. In some cases the coagulation time has
delayed, and there is evidence of an inferior
be explained on a basis of deficiency of fibrino-
leficiency of thrombokinase. Other cases have
gulation times which are only slightly delayed
ses with normal coagulation time, but in which
is followed by excessive bleeding, it is possible
s in the prothrombin. In three of Schloss and
owever, the coagulation was normal, and there
punctures of the skin, hemorrhage being quickly
well contracted clot. The writers attribute the
cases to some localized vascular lesion, and
ıy possibly be a local absence or interference
rombokinase, which is normally liberated upon
all. There is no well supported evidence that
salts plays any part in this hemorrhagic disease.
whether the tissue defect, whether it lie in the
ıf the vessels, is due to incomplete development
susceptible of experimental investigation. The
the developmental theory, is that the tendency
ı only in the early days of life, and does not
wever, a certain tendency to obscure infection
en in the newborn. Those who believe in the
point to the absence in most cases of clin-
ıacteriologic signs of infection.
is an increasing tendency to attribute this
ction. It is known that hemorrhage is a com-

paratively common symptom of some well-recognized infections of the newborn. The evidence against the infectious theory is of a purely negative character, while on the other hand, considerable evidence has recently been accumulated tending to substantiate this infectious theory. The disease is most apt to be seen in institutions. Green and Swift have pointed out the tendency of the disease in their series of hospital cases to occur in groups, and believe that this, together with the seasonal incidence, tends strongly to confirm the theory of an infectious etiology. Moreover, a number of observers have isolated bacteria of various kinds from certain of these cases. A certain number of cases show a distinct febrile reaction. The self-limited character of the disease is often brought forward in support of the infectious theory, but goes very well with the theory of incomplete tissue development.

We can only look to further investigations for the solution of these problems. The attempt to determine the pathology of the blood in hemorrhagic conditions is beset with numerous obstacles, and it is improbable that the exact causation of hemorrhage in the newborn can be definitely ascertained until the physiologic processes of blood coagulation are more thoroughly understood. It is quite probable that, as in many functional disturbances, several different conditions may be concerned in the etiology, any one of which may be the cause of a particular case of hemorrhage.

A number of methods of treatment will be found recommended in the literature of hemorrhage in the newborn, each based on some particular theory of the etiology of the condition, and each with clinical evidence in its support. Among the various measures which have been recommended are the following:—Injections of gelatine; administration of calcium salts; administration of adrenalin; subcutaneous injection of horse serum; subcutaneous injection of rabbit serum. We have seen, however, that no exclusive theory of the pathogenesis of this disease is tenable. Clinical evidence in any single case can be explained by the fact that some cases will recover without treatment.

The use of gelatin is entirely empirical, and it has been finally proven to be inert in its power to increase the coagulability of the blood, either *in vitro* or *in vivo*. Adrenalin has also been found to be inert, having value only as a local hemostatic. Calcium salts have been found to increase the coagulability of the blood in test-tube experiments, but it has been found that only the ionizable calcium salts of the blood enter into blood coagulation, and these can be only very slightly increased by administering calcium salts to a patient. Deficiency of calcium salts, also, does not appear to play an etiologic rôle in this particular disease. Of the animal sera,

experiment has shown that horse serum is less potent than rabbit serum, but that rabbit serum is less potent than human serum.

ICTERUS NEONATORUM

Jaundice is a symptom seen with great frequency in newborn infants. In the majority of cases the jaundice is of so slight a grade, and is characterized by so entire an absence of pathological symptoms, that it is usually looked upon as representing a physiological process which is normal in the early days of life. This type of jaundice is called Icterus Neonatorum.

There are, however, a number of other conditions, of true pathological nature, which are characterized by the appearance of jaundice in newborn babies. These more serious conditions, though rare, must be carefully distinguished from the common icterus neonatorum. In the first place, jaundice is a not infrequent symptom of ***infection*** in newborn babies; it is also sometimes, though rarely, a symptom of ***congenital syphilis***. It is the chief symptom of a ***congenital cirrhosis*** of the liver, which has been found at autopsy, and of ***congenital obliteration of the bile ducts***. This last condition may represent a congenital malformation, or may represent a late stage of congenital cirrhosis. ***Catarrhal jaundice***, an obstructive form, in which the cause is swelling of the mucosa of the bile ducts, is very rare in newborn babies, but is still a possibility. Finally, certain cases of jaundice have been observed in newborn babies, in which all the above causes were absent, and in which no cause was found.

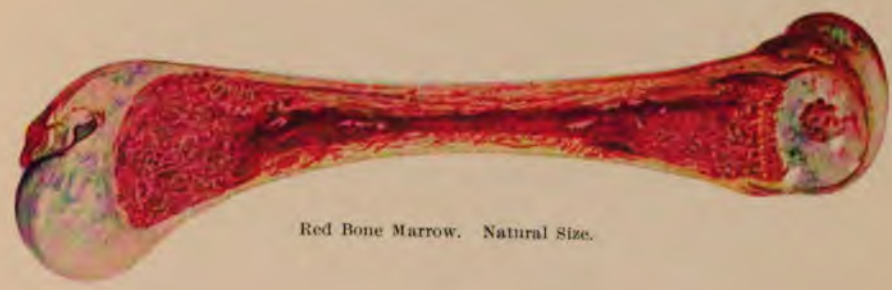
Icterus neonatorum is by far the commonest condition producing jaundice in the first days of life. It has been variously reported to exist in from 40 to 80 per cent. of all newborn infants.

ETIOLOGY.—The etiology of icterus neonatorum remains obscure, in spite of the fact that it has been the subject of much ingenious speculation. The most satisfactory explanation is that of Abramow, who considers the jaundice due to an anomaly of secretion of the liver cells. Shortly after birth, as the result of the rich supply of blood to the liver, the hepatic cells produce bile more rapidly than it can escape. The bile capillaries at birth are overfilled with a thick and tenacious bile, which obstructs its own flow as it is formed so rapidly. The bile, consequently, passes into the blood capillaries, and thence into the general circulation. This explanation is in accordance with the theory that icterus neonatorum is a physiological and not a pathological process.

SYMPTOMS.—The jaundice is not present at birth, but develops usually on the second or third day. It is first noticed on the face and chest, then in the conjunctivae, then on the extremities. It increases

PLATE II.

Icterus Neonatorum.



Red Bone Marrow. Natural Size.

Yellow Bone Marrow. Three-fourths Natural Size.

Erythema Neonatorum.

in severity for three or four days, then rather rapidly disappears. In rare cases it may last longer. The color is usually a pale yellow, but at times is more intense. The fecal discharges are not light colored, and contain bile pigment. The urine in most cases is normal, not showing bile pigment, though sometimes the presence of bile pigment can be demonstrated by special tests. There is no enlargement of the liver or spleen, and no rise of temperature. The general condition is not affected; there are no symptoms of indigestion, and icteric babies show no tendency to gain weight less rapidly.

DIAGNOSIS.—The recognition of the condition is very easy. The absence of all other signs of disease, together with the normal stools, serve to differentiate icterus neonatorum from the other conditions in which jaundice is seen in the newborn.

PROGNOSIS. The prognosis is good.

TREATMENT.—None is required.

PROBLEMS AND RESEARCH.—A number of theories as to the cause of icterus neonatorum have been advanced, and disproved, although references to them are still to be met with in medical literature. Among these disproven theories are the following:—Frank's theory that the cause of jaundice is plugging of the common bile duct with mucous and cast-off epithelium; Birch-Hirschfeld's assumption of an edema of Glisson's capsule; Bouchet's hypothesis of a hepatitis; Epstein's theory of a catarrh of all the bile ducts. Quincke's theory of a patent ductus venosus is improbable.

The hematogenic factor in the etiology of icterus neonatorum is still under discussion. The many supporters of this theory assume that there is in the early days of life a countless destruction of erythrocytes, which produces polycholia and jaundice. This theory has been refuted, the apparent destruction of red blood corpuscles being attributed to an increase in plasma. Still more recently, evidence has been brought forward to show that the destruction of erythrocytes actually does occur. Even if this evidence is true, it only furnishes plausible explanation of the increased bile formation in the early days of life, which must otherwise be attributed to a rapid awakening of the function of the hepatic cells.

The only experimental work which has been done in this condition is that of Hess, who investigated cases of icterus neonatorum with the duodenal catheter. He found that bile is excreted into the duodenum very rarely in the first twelve hours of life. In the next twenty-four hours, bile excretion is variable, being profuse in cases with marked jaundice, and scanty or absent in cases not jaundiced. He found that jaundice precedes the excretion of bile into the duodenum, and is more marked in cases with profuse secretion. Hess believes that the cause of icterus neonatorum is due to the fact that

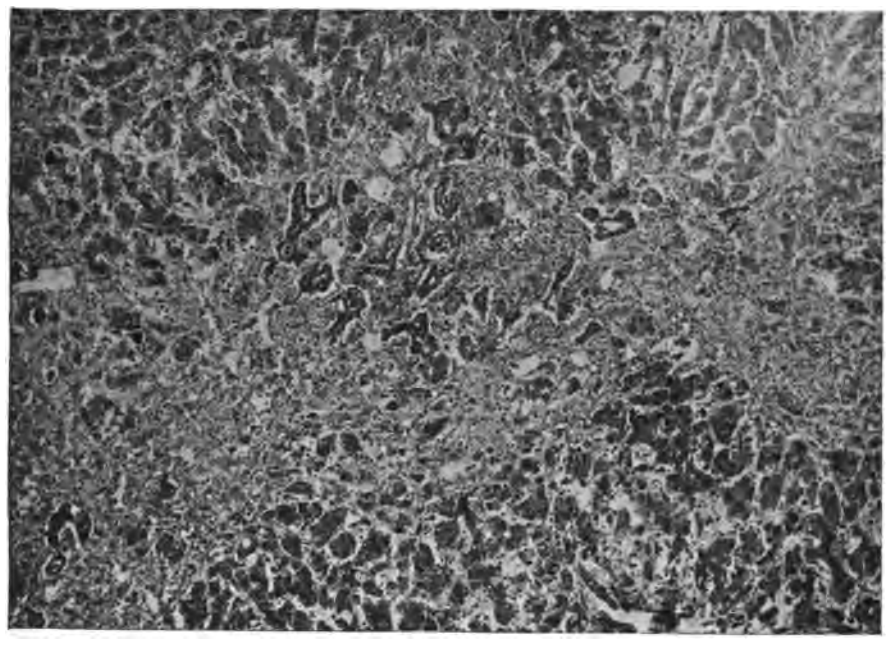
the mechanism of bile excretion into the duodenum is not well equipped in the earliest days of life. Excess of bile is secreted, whether or not the cause is an increased amount of available hemoglobin. Excretion becomes profuse, but not enough to take care of the excess, with resulting congestion of the bile capillaries.

CONGENITAL CIRRHOSIS OF THE LIVER,

CONGENITAL OBLITERATION OF THE BILE DUCTS

These conditions, while described by many writers as separate diseases, are here described together, because the preponderance of evidence points to so close an association between them, that many authorities believe them to be different stages of the same process, and because their clinical manifestations are practically identical.

FIG. 65



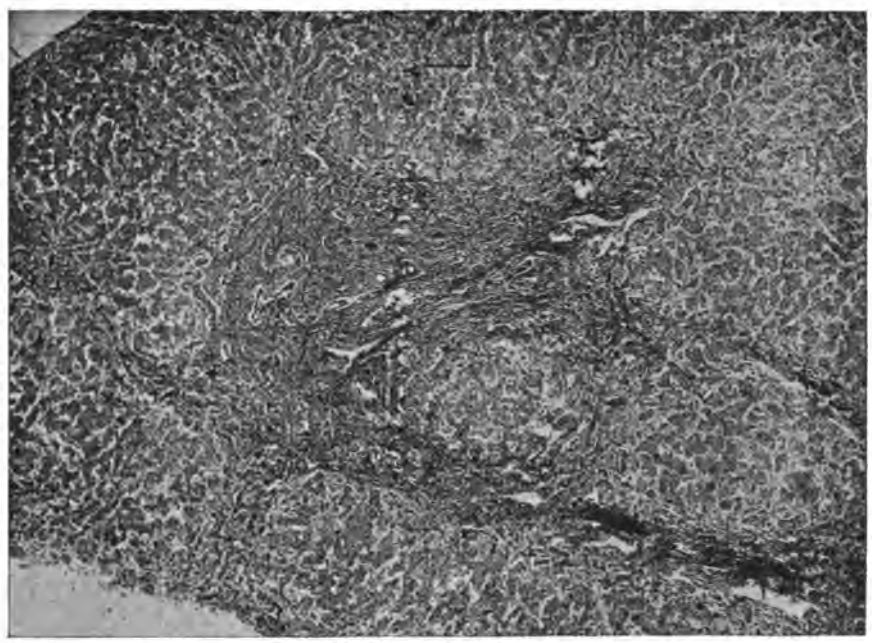
Congenital cirrhosis of the liver

PATHOLOGICAL ANATOMY.—In the majority of the reported cases, there is found at autopsy complete obliteration of some part of the extra-hepatic bile ducts. In the majority of cases the common duct is obliterated, usually down to and including the opening of the papilla of Vater. The obliteration usually also involves the

hepatic ducts. The cystic duct may be obliterated, or its lumen may be much narrowed. The obliterated ducts are usually represented by a fibrous cord, but may be entirely absent. The gall bladder is frequently very small, and is sometimes also obliterated, and replaced by fibrous tissue.

Accompanying this condition of obliteration of the bile ducts, there are always found marked changes in the liver. The principal lesion is a cirrhosis, of the biliary type, characterized by an increase in connective tissue, which is most marked in the region of the portal vessels, but which often occurs to a varying extent diffusely within

FIG. 66



Congenital cirrhosis of the liver. Section showing increase in fibrous tissue and invasion of the lobules

the lobules. The connective tissue varies in age, in some cases appearing comparatively newly formed, in other cases old and dense. The changes in the liver cells show marked variations in different cases. In some the cells remain arranged in orderly trabeculae, in others some of them are much increased in size, in still others they show marked atrophy or extensive destruction. The bile capillaries are usually much distended and many contain inspissated bile. There are often newly formed bile capillaries to be seen.

Two views are held as to the pathogenesis of the cases in which obliteration of the bile ducts is found. One view regards this lesion as developmental in origin, a congenital malformation. The other view regards the lesion as due to an inflammatory process, leading to fibrous tissue formation and obliteration. It is possible that in some instances the condition may represent a congenital malformation, but such instances are certainly extremely rare. It is probable that in the majority of cases the obliteration of the ducts is due to inflammation.

Two views are also held as to the relation of the cirrhotic process in the liver to the obliteration of the bile ducts. One view regards the bile duct process, whether inflammatory or developmental, as primary, and considers the cirrhosis of the liver as a secondary process. The other view regards the process in the liver, cholangitis and cirrhosis, as primary, and considers that the occlusion of the bile ducts is due to an extension of this tissue reaction down along the large bile ducts. In favor of this latter view is the fact that obliteration of the extrahepatic bile ducts is never found without an accompanying cirrhosis of the liver, but on the other hand many cases have been reported in which precisely the same cirrhosis was found in the liver, but in which the common and hepatic ducts were found patulous throughout their entire extent. While the changes in the liver do not resemble those generally described as occurring after sudden obstruction to the flow of bile, it is usually difficult to tell which process is primary. There is anatomical evidence on either side, and it is quite possible that either lesion may be the primary one.

The two conditions are certainly so closely connected that they cannot well be considered as distinct diseases.

ETIOLOGY.—The cause of the condition is unknown, and very possibly varies with the case. Congenital syphilis is probably not an etiological factor. If we exclude those cases, certainly rare, in which the obliteration of the bile ducts may represent a congenital malformation, the prevailing view is that the inflammatory process is due to the absorption of toxic substances from the mother, and this theory is unaffected by the question as to whether the liver or the bile ducts are first affected. This theory is of course, without proof.

SYMPTOMS.—The most striking symptom is jaundice, which is sometimes present at birth, but which usually develops on the second or third day, and sometimes not until the fourth day. The jaundice increases rapidly, and soon becomes intense, so that the skin appears of a greenish-yellow tint. The stools may or may not be composed of meconium at birth, according to the period of fetal development

when the obstruction occurred. Meconium is usually seen, but very soon the dejecta become white or clay colored. In the majority of cases, bile pigment is not to be demonstrated in the stools by a chemical test. In some cases, however, bile pigment has been found present, these cases being usually those conditions in which congenital cirrhosis exists without obliteration of the bile ducts. Nevertheless, I have recently seen a case in which no obliteration of the ducts was found at autopsy, with bile pigment constantly absent in the stools, while Hess has recently reported a case with complete obliteration of the common duct, in which the stools contained traces of bile pigment, which proves the possibility of excretion of bile from the circulation through the intestinal wall. The stools always show defective absorption of the fat given in the food.

The urine is always dark, and contains bile. The liver and spleen are usually enlarged, although not always, especially in cases where cirrhosis of the liver is very marked. Hemorrhages may be seen in the early days of life, or late in the course of the disease.

The babies lose steadily in weight and strength. Symptoms of indigestion may be present, but are usually notably less marked than one would expect. In fact the general condition, for quite a time after the development of the symptoms, remains much better than would be expected in a condition with such serious lesions. Finally, however, malnutrition becomes extreme, and death occurs from inanition, or from some intercurrent infection.

DIAGNOSIS.—Icterus neonatorum is easily excluded in these cases, on account of its mild and brief type of jaundice, and the fact that the stools and urine remain normal. The conditions to be particularly considered in diagnosis are infectious disease of the newborn with jaundice as a symptom, and catarrhal jaundice. When jaundice is a symptom of infection, the temperature is usually elevated, and not infrequently high and irregular. The general condition is usually more profoundly affected from the start. The physical examination frequently gives definite information as to the source and location of the septic process. The jaundice is often not quite so intense. The most important point, however, is that the movements in sepsis are not light colored, and contain bile.

It is more difficult to exclude catarrhal jaundice, although this condition is extremely rare in the early days of life. Catarrhal jaundice may show the same intense icterus, with absence of bile in the stools. Even when it is seen early in life, it usually develops at a somewhat later period after birth. In such a case, with comparatively late development, and with absence of enlargement of the liver, catarrhal jaundice may be suspected. The diagnosis can be confirmed only by the favorable course of the disease.

PROGNOSIS.—The prognosis of congenital cirrhosis and obliteration of the bile ducts is bad. Some cases die in the early weeks; others may survive for several months. Eight months is the longest recorded period of survival.

TREATMENT.—Careful feeding is the only treatment which can be employed in these cases. Milk modifications in artificially fed infants should be low in fat, high in protein.

In cases in which the diagnosis is fully established by the persistence of signs of complete obstruction to the flow of bile, I believe that a surgical exploration should be considered. Such an operation will probably shorten the life of the baby, if the diagnosis is correct. On the other hand, there is a possibility of the jaundice being due to some remediable condition, such as mechanical obstruction of the large ducts from pressure of glands, inspissated bile, or peritoneal adhesions. The absolutely hopeless prognosis makes it justifiable to take advantage of the possibility, even though remote, of a mistake in diagnosis.

PROBLEMS AND RESEARCH.—The problems of the cause of the tissue changes, whether they are due to a malformation, or to the intrauterine absorption of toxin from the mother, and of the location of the primary lesions, are not very promising for experimental investigation. Possibly the continued study of pathological material, together with thorough and continued observations on the presence of bile in the duodenum or stools throughout the life of the baby, may eventually give more light. The method of studying the contents of the duodenum by means of the duodenal catheter, as described and practiced by Hess, is particularly worthy of mention in this connection. Hess has further called attention to the rôle of the pancreas in this disease. He finds that the pancreatic duct of Wirsung is frequently obliterated at its opening, when the common bile duct obliteration involves the papilla of Vater, but that the pancreatic ferments may nevertheless be found undiminished in the contents of the duodenum obtained by the duodenal catheter. In these cases the obliteration of the duct of Wirsung is compensated by the presence of the accessory duct of Santorini, and the prognosis as to duration of life is probably better than in cases in which no accessory pancreatic duct exists.

SCLEREMA

Sclerema neonatorum is a rare condition, characterized by progressive hardening of the skin and subcutaneous tissues.

ETIOLOGY.—Sclerema is seen most frequently in premature or very feeble infants. While it is commonest in the early days of life, it is not exclusively a disease of the newborn, as it may be seen

at any time in the first few months in infants weakened by malnutrition, diarrhea, or some acute infection. In the newborn it is most frequently found among those who are born in exceedingly poor hygienic surroundings, and in cold weather. The cause is probably general, a number of factors being involved, such as great feebleness with weakness of circulation, loss of fluid from the skin, lowering of the body temperature, and hardening of the subcutaneous fat.

PATHOLOGICAL ANATOMY.—Although Northrup reported a case clinically typical, in which microscopic examination of the skin revealed nothing abnormal, the majority of writers have reported changes in the skin. These changes, however, show nothing peculiar to the disease, and may not be an essential feature. Various degrees of atrophy of the subcutaneous connective tissue appears to be the chief lesion described.

SYMPTOMS.—Soon after birth, spots of circumscribed hardness appear on the skin. These spots soon become diffuse, and the disease, starting, as it usually does, in the feet or the calves of the legs, passes up the thighs to the trunk. It may, however, first appear upon the face and upper extremities, though not commonly. The skin is waxy and glistening, and is hard and cold; the limbs become thick, stiff, and misshapen. The infant soon grows weak and somnolent, and refuses to take its food; the breathing becomes rapid and superficial, the voice is weak and whimpering, and the pulse small and retarded. The temperature is markedly subnormal and death takes place seemingly from inanition.

DIAGNOSIS.—Sclerema presents so definite a clinical picture, that the diagnosis is usually easy. It must be distinguished from the edema occasionally seen in newborn babies. In sclerema the skin is harder, and does not pit on pressure.

PROGNOSIS.—The outlook in sclerema is very bad, because of the grave character of the etiologic factors. It is usually, but not invariably fatal.

TREATMENT.—Artificial heat is the most important thing in the treatment. The incubator or incubator-bed must be used. The nutrition must be maintained as well as possible by careful feeding.

EDEMA

Edema is occasionally seen in early infancy. In a certain number of cases, edema is a symptom of some severe disease, such as septic infection, syphilis of the internal organs, pulmonary diseases, and severe gastro-intestinal disturbances. There is a form to which the name *Oedema Neonatorum* may be applied, which is not symptomatic of any recognizable disease process. This edema is seen

most frequently in premature or congenitally feeble infants, and appears usually on the first day of life. The edema appears first in the suprapubic zone, then involves the legs and sexual organs, and is occasionally seen on the backs of the hands. It is never general. There are two stages in its development; in the first the tissue appears overfilled with fluid, but does not pit on pressure; in the second, there are the usual signs of edema with pitting. It lasts usually but a few days; the longest recorded case lasted twenty days. Any deviation from the typical development is suggestive of symptomatic edema from one of the other causes. Edema neonatorum, however, may pass over into one of the other forms. Its prognosis and treatment is that of congenitally feeble and premature infants.

VI. INFECTIONS

INFECTIOUS DISEASE OF THE NEWBORN

The condition described under this name is one of general sepsis in newborn babies in which the infecting microörganisms enter the blood and internal organs of the newborn infant.

The infant, even in the early days of life, may suffer from any of the common infectious diseases. The manifestations of these infections in newborn infants do not differ in any essential particular from those seen at a later period of life. In addition to the recognized infectious diseases, such as pneumonia, influenza, typhoid, and so forth, there are other infectious conditions seen in the newborn which are caused by the various pyogenic microörganisms, such as the streptococcus pyogenes, the staphylococcus pyogenes aureus and albus, the gonococcus, the pneumococcus, the bacillus pyocyaneus, the colon bacillus, and other rarer forms. These microörganisms may form only localized lesions at the portal of entrance, without any general condition of sepsis supervening. Such infections show themselves in the form of abscess formation, cellulitis, and similar lesions, which differ in no essential from the same lesions seen at a later period of life. Omphalitis or inflammation about the umbilicus, and erysipelas are among the most noteworthy of these external infections. Even when the organisms penetrate within the body, they may still give rise to a localized inflammatory process of which the pathology shows no features peculiar to newborn infants. Among these lesions are to be numbered especially peritonitis, pneumonia and pleuritis, pericarditis, meningitis, gastro-enteritis, osteomyelitis, and septic arthritis. The clinical manifestations of these conditions are often obscure, so that the diagnosis is rather more difficult in newborn infants. Nevertheless the symptoms do not present any features sufficiently peculiar to the early days of life to warrant their separate description. The localizing signs may be less marked, so that the clinical picture is one of a general infection. On the other hand the presence of a general infection may be masked by the prominence of localizing signs.

A general condition of infection, a true bacteremia or pyemia, is a particularly common manifestation of infection in newborn infants. The manifestations of such an infection are so peculiar in the newborn, that they call for a detailed description, and it is to this condition that the term *Infectious Disease of the Newborn* is here applied.

ETIOLOGY.—The microörganisms concerned in infectious disease

newborn are the pyogenic types already mentioned. Infection when from any source these microörganisms are brought to the portals of entry which are open in a newborn child whose nce is not sufficient to prevent their entrance. I do not believe ıe low general resistance of the newborn infant is so great a in the occurrence of infectious disease as is generally stated. infant's general resistance to infection were so very low, infec- ould be relatively much more common.

most important factor is the relatively open condition in the rn infant of certain portals of entry, particularly the umbilical . It is true that in a large number of cases of infectious disease newborn the portal of entry cannot be discovered, but there is oubt that in most of these the infection is of umbilical origin. very newborn baby has an open wound, which is closely con- with the general circulation through the anatomical connec- of the umbilical vessels, is adequate explanation for the fre- and peculiarities of this type of infection in newborn infants. he umbilicus, the stump of the cord is undergoing a necrotic gration with which may be associated pyogenic organisms. ligated umbilical veins are formed thrombi which may easily e infected, and as a result may break down into purulent al. As a result of such a septic phlebitis the infected material nter the circulation and produce a general septicemia, while d emboli may be carried to any organ in the body. All these ses may go on without any lesion being noted at the umbilicus, erefore it is wise, in any case of infectious disease of the new- o regard the umbilicus as the probable portal of entry.

e are undoubtedly other portals of entry in certain cases. ganisms may enter through abrasions of the skin or mucous anes, through the lungs, through the mouth, and in rare es through the eye, ear, or genital tract.

probable that even in umbilical infection, the organisms are ose normally associated with the disintegration of the cord, at the umbilical cord is inoculated with organisms of a par- ly pyogenic nature. Such inoculation is also the probable of infection through the other portals of entry. The sources ı inoculation are the vaginal discharges of the mother, unclean of nurse or physician, improper care of the umbilical wound, ath water, dirty clothing, in short, any agent which may carry organisms to the portal of entry. In cases of infection through ıgs or intestinal canal, it is probable that infected mucus or amnii may reach these respective portals of entry in the in- first movements of respiration or of deglutition. In rare es septic infection may be transferred directly from an in- mother to the fetus through the placental circulation.

PATHOLOGICAL ANATOMY.—The lesions of infectious disease of the newborn show no essential peculiarities differing from pyogenic infection at other periods of life. Umbilical arteritis or phlebitis are the most frequent lesions. The liver is the organ which most frequently shows pathologic changes, as one would expect from the frequency of umbilical infection. In the liver there may be suppurative processes, or various degenerative changes. Similar degenerative changes are found in most of the parenchymatous organs, while individual foci of suppuration may have almost any distribution. Hemorrhages are common.

Associated with these generally distributed lesions, there are usually also localized lesions, the most common of which is peritonitis. Pneumonia, pleuritis, meningitis, encephalitis, pericarditis, osteomyelitis, and arthritis are also found.

There is a condition in which the particular group of lesions have lead to its description as a separate disease, under the name of *Fatty Degeneration of the Newborn* or *Buhl's Disease*. The pathological changes are essentially a parenchymatous inflammation, followed by fatty degeneration of the heart, liver and kidneys, together with multiple hemorrhages in the various organs. Although the nature and cause of this condition are unknown, it was first described before the advent of modern methods of bacteriological study. Both the lesions and clinical features of this disease show no essential difference from those of pyogenic infection, and it is probable that it merely represents a particular grouping of the lesions in infectious disease of the newborn.

SYMPTOMS.—The clinical manifestations of infectious disease of the newborn present great variations. The symptoms due to the various localized lesions seen in these infections are particularly dependent upon the portal of entry, and are often so marked as to obscure the general nature of the disease.

The most common mode of onset is seen in the umbilical infections. There is a rather sudden rise of temperature, followed by an irregular septic fever. The temperature curve is very irregular and can only be properly observed by a four-hourly chart. At some periods of the day it may be very high, at others may even be below normal. After a period of such fever, the temperature in some cases may remain permanently below normal.

Associated with fever, the two most common symptoms in general sepsis of the newborn are jaundice and hemorrhages. Jaundice is an early and prominent symptom, but is not always present. It is not of the complete obstructive type, bile being found in the stools, as well as in the urine. Purpura is the most common hemorrhagic manifestation; it occurs as a fine petechial rash, or as large, scattered,

ue blotches. Hemorrhage from the bowels is also seen at
nd more rarely, there are hemorrhages from the nasal, buccal,
r mucous membranes.

nervous system is usually profoundly affected. There is at
notable apathy, which rapidly increases into stupor leading
olete coma. Prostration is very marked, and a condition of
exhaustion is apt to come on rather rapidly. In other cases,
eal symptoms, such as restlessness, irritability, twitchings,
paralysis, or convulsions, are present.

hysical examination, the liver is usually found enlarged. One
or localized evidences at the portal of entry, which, as has
eady stated, often cannot be found. The umbilicus may be
l, the umbilical depression may be filled with pus, or pus may
e to ooze by pressure about the umbilicus. There may be
nal distention and tenderness, which are an aid to the diag-
umbilical infections where the umbilicus appears normal.

ne skin, or on the mucous membrane of the mouth, nose or
can sometimes be observed the ulceration or abrasion through
he infection has entered. A great variety of lesions may be
these situations.

igns of localized infection are sometimes the most prominent
manifestations in infectious disease of the newborn. These
not differ essentially from those of the same lesion when not
festation of this particular disease. One should look first
s of peritonitis; then for signs of pneumonia, pleuritis, arth-
teomyelitis, pericarditis, meningitis, and encephalitis.

GNOSIS.—The diagnosis of infectious disease of the new-
esents extreme difficulty, owing to the great variety of the
manifestations. When the case presents the clinical syn-
which may be called most typical, namely, that of septic
ture with jaundice, hemorrhages, and great prostration, the
is is comparatively easy. The other causes of jaundice in
n babies, namely, icterus neonatorum, and congenital cirrhosis
iver, are not associated with fever, nor do they show extreme
ion. Catarrhal jaundice often is associated with fever, but
rare in newborn infants, and the fever is not of a septic char-
nor is extreme prostration present. The other causes of
nage in newborn infants, namely, hemophilia and hemorrhagic
of the newborn, can usually be easily excluded, as in them
nage is practically the only symptom.

most difficult cases for diagnosis are those in which the syn-
described above is not present, but in which the symptoms
localized lesions as peritonitis, pneumonia, meningitis, or
nteritis are the prominent clinical features. It should be

remembered that such severe symptoms at this period of life are always strongly suggestive of a general sepsis.

In all cases, the finding of evidences of infection at the umbilicus, or at any other portal of entry, is a great aid to the diagnosis.

Laboratory methods are not of much aid. The blood examination usually shows a marked leucocytosis, but this is of no value in recognizing the general character of the infection. Only by blood culture can the diagnosis of infectious disease of the newborn be definitely proven. The technic of blood culture in newborn infants is so difficult, that, while it has been successfully done in hospitals, it is not very valuable as a routine method of diagnosis.

PROGNOSIS. The prognosis is bad. Nearly all the severe cases presenting the typical syndrome of fever, jaundice, and hemorrhage, die. Death may occur within a few days, or may be postponed for a longer period. It is probable that nearly all cases in which there is a general sepsis, or any important visceral lesion, die rapidly. A few cases of apparent umbilical infection recover,but it is probable that in these cases the septic process never became general.

PROPHYLAXIS.—That infectious disease of the newborn is preventable, is shown by the great diminution in its occurrence in institutions since the introduction of aseptic methods into obstetric practice. It may be compared to puerperal fever. The majority of cases are caused by the carelessness of some attendant, physician, nurse, or parent, having the care of the newborn infant. The essentials are cleanliness of the child's surroundings, and proper care of the umbilicus. The umbilicus should be treated like any clean, fresh wound. It should be cleansed and dressed with sterile dry dressings. Everything coming in contact with the wound should be sterile. Clean clothes, clean hands, and clean surroundings, are the other essentials. Care should be taken to prevent all excoriation or abrasion of the skin or mucous surfaces. In institutions all septic cases should be strictly isolated.

TREATMENT.—The treatment is purely symptomatic. Whenever any localized focus of suppuration open to surgical treatment is present, incision, evacuation, and drainage should be performed. Vaccine therapy with an autogenous vaccine can be tried, if the microörganism causing the disease can be isolated from the blood, or from a localized lesion. There are as yet no favorable reports from its use, and I do not think it offers much prospect of success.

OPHTHALMIA NEONATORUM

Ophthalmia neonatorum is an inflammation of the conjunctiva of newborn infants. There is a mild catarrhal form occasionally seen, which is caused by any slight irritation of the eyes of the infant.

It runs a very mild course, attacks chiefly the palpebral conjunctiva, and usually the only symptoms noted are a slight photophobia and a collection of secretion in the angles of the lids and upon their borders.

The usual condition known as ophthalmia neonatorum is a purulent conjunctivitis.

ETIOLOGY.—The gonococcus is the most frequent pathogenic microörganism in this disease. A few cases are due to other organisms, such as streptococci, staphylococci, and pneumococci. Infection most commonly takes place during birth from direct contact of the conjunctiva with the pus-forming organisms present in the vagina of the mother, who has a vaginitis due to gonococcus or other infection. Occasionally the infection may be carried to the eyes of the infant by the hands of the physician or nurse, or by contaminated linen or other material, and in such cases the infection may take place at a period subsequent to birth.

SYMPTOMS.—The disease begins as a redness of the conjunctiva, with a slight discharge from the corner of the eye. This is succeeded with startling rapidity by intense inflammation of the lids. In twenty-four hours the upper lid may become so much swollen as to overhang the cheek and render opening the eye impossible. On separating the lids, a little greenish pus, which may even be tinged with blood, wells up between them. At first the cornea is unaffected, but if the pus accumulates under the edematous lids it soon shows signs of ulceration. In the second twenty-four hours the ulceration may perforate the cornea and evacuate the aqueous humor, thus bringing the iris into contact with the posterior surface of the cornea. The swelling may extend around the eye and well over the forehead and malar prominence, but it does not persist in the latter region very long. All the symptoms disappear slowly, and recovery takes place, except in those cases in which the cornea has been permanently injured by ulceration.

DIAGNOSIS.—The diagnosis of purulent ophthalmia may be easily made from the examination of the eyes. A gonorrheal etiology may be suspected when a very violent inflammation is present, and from the history suggesting gonorrheal vaginitis in the mother. A positive etiologic diagnosis can be made only by bacteriological examination of the purulent discharge.

PROGNOSIS.—The earlier the pus appears, the more severe will be the course of the disease, and the more unfavorable the prognosis. Cases seen early and treated properly, terminate as a rule in complete recovery. Neglected cases and very severe gonococcus infections frequently result in corneal ulceration, which may go on to perforation and complete loss of vision.

TREATMENT.—Prophylaxis is the most important part of the treatment of this disease. In private practice, when the mother has no vaginal discharge, all that is required is washing the eyes after delivery with a weak solution of boric acid. When the mother has a vaginal discharge, or in institutions, Crédé's method of prophylaxis should be employed. This consists in dropping one or two minims of a two per cent. solution of nitrate of silver into each eye of the newborn infant. As even this has been known to cause a considerable amount of irritation, a solution of protargol, or argyrol, ten or twenty per cent., is often used in preference to the silver nitrate.

When one eye only has become infected, care must be taken to prevent the spread of the infection to the other eye. This is accomplished by carefully covering the sound eye with cotton and a bandage.

After the disease has once begun, two indications must be kept in mind: (1) To reduce the inflammation, and (2) to prevent the pus from accumulating behind the tightly closed lids. By far the best way of applying cold to the eye is by compresses of soft pieces of linen cut into small squares. Not more than two thicknesses are to be used at once. These compresses are to be cooled by laying them on a piece of ice or floating them in ice water. They must be constantly changed. To remove the pus, a gentle irrigation, such as can be easily obtained by using a medicine dropper, is sufficient.

In the irrigation of the eye one should first turn the child's head a little to the diseased side, and with the fingers of the left hand gently separate the lids as far as possible. Then, holding the dropper with the right hand, irrigate between the lids, directing the stream *from* the nose. After each irrigation vaseline should be applied to the edge of the lids. This should be done at least every half-hour, day and night, until the swelling has so far subsided as to preclude the danger of any secretion being retained. For irrigation many solutions have been advocated. The most simple, and perhaps the best, is a saturated solution of boracic acid, or one of bichloride of mercury in the strength of 0.05 gramme (1 grain) to 480 c.c. (1 pint) of distilled water. In addition to the irrigations, a daily instillation of a 2 per cent. silver nitrate solution should be used. In the later stages of the disease, in which all the tissues are relaxed, a solution of nitrate of silver, or still better, of protargol or argyrol, 20 per cent. in strength, painted on the conjunctiva with a camel's-hair brush once daily, is very effective in shortening the course of the disease.

TETANUS NEONATORUM

This disease differs in no way from tetanus occurring at any other period of life. The cause is the same, the bacillus of tetanus, which sometimes finds a portal of entry in the umbilical wound of new-

born infants. The symptoms, diagnosis, and treatment differ in no essential particular, whether the disease is seen in the newborn or at a later period of life.

DERMATITIS EXFOLIATIVA

In the year 1878 Ritter gave the first complete description of the disease *dermatitis exfoliativa neonatorum*. Previous to this date cases of this affection had been reported, but many of them were regarded as some rare or unusual manifestation of pemphigus. Ritter studied and reported the cases which he saw at the Foundling Asylum in Prague from 1868 to 1878. A careful review of Ritter's original observations of these cases has been made by Elliot. The majority of cases were in male infants, and the mortality was found to be 48.82 per cent. The disease is very rare. It occurs rarely before the end of the first week, and usually appears between the second and the fifth week of life. It was found to vary greatly in the intensity of its symptoms. In some cases a dry scaly condition of the skin preceded the subsequent lesions, which had apparently lasted after the physiological desquamation of the epidermis had taken place.

SYMPTOMS.—The first symptom noticeable in these cases was a diffuse redness, usually over the lower half of the face about the mouth, sometimes, however, beginning in some other portion of the body, and at times being universal from the beginning. This hyperemia of the skin spread rapidly, and in a few days became universal, the extremities, as a rule, being the last parts affected. The mucous membrane of the mouth and the nose was at times affected, and the conjunctivae usually participated in the hyperemia. The color of the efflorescence varied from a light to a dark purple-red. As the hyperemia extended to new surfaces, those which were first affected began to desquamate. This desquamation at times gave no evidence of exudation, the epidermis being simply thickened, and the loosened epithelium separating easily. At times other lesions appeared, such as milia, and sometimes the horny layer of the skin was raised above an intensely reddened base, and large, irregularly shaped bullae filled with fluid were formed. After the desquamation had taken place the skin recovered its normal condition, occasionally very rapidly, but it remained for some time rough and irritable. In the cases in which there was no exudation a longer time was necessary for the separation and regeneration of the epithelium.

Usually the disease was found to run its course in from seven to ten days. Relapses were sometimes observed ten or twelve days after the first attack, but were always mild.

In typical cases the process was unaccompanied by any fever or systemic disturbances unless some complication existed. The

functions were normal, and the weight of the infant remained stationary or was even at times increased. The fatal cases resulted either from the intensity of the attack or from some intercurrent affection or sequela, such as furunculosis. This disease is usually recognized as a local septic infection of the skin, and it would seem that it would be easily distinguished from the pemphigus which occurs in the early weeks of life.

INFECTIOUS HEMOGLOBINEMIA (Winkel's Disease)

Infectious hemoglobinemia is an affection which is met with in newborn infants, usually in the early days of life, and, as a rule, arises as an endemic disease in hospitals. The disease is characterized by extreme cyanosis, icterus, hemoglobinuria, somnolence, rapid collapse, and the absence of fever.

ETIOLOGY.—The etiology of this disease is obscure. A specific microörganism has not yet been discovered, yet the endemic character of the malady and the changes which are produced in the blood warrant us in supposing that it is an infectious disease. It would be described, not as a separate infection, but as a probable special manifestation of infectious disease of the newborn, were it not for its epidemic character, which suggests a probable undescribed specific infection as a cause.

PATHOLOGICAL ANATOMY.—A careful post-mortem examination of Winkel's cases showed that there was cyanosis of the external and internal organs. Except in one instance, no pathological condition of the vessels of the cord was described. The cortex of the *kidney* was found to be wider than normal, to be of a brownish color, and to present numerous minute hemorrhages. In some places the pyramids were entirely black-red in color, and in other places numerous black streaks were found which converged to the papillae. This color was caused by the filling of the straight tubules with granules of hemoglobin. Intact erythrocytes were never found. The *bladder* was found to contain greenish-brown urine. The *spleen* was strikingly enlarged and hard. Its length was about 7.5 cm. (3 inches), and its weight 25 grammes (5/6 ounce). It was black-red in color, and on section the surface was smooth. Microscopic examination showed a considerable accumulation of brownish coloring matter, partly free and partly in the pulp cells.

In addition to these appearances in special organs, minute hemorrhages were found in nearly all the organs, but especially in the pleura, pericardium, endocardium, mucous membranes of the stomach and small intestine, and kidney; they were also found in the dura and pia mater and under the capsule of the liver. The lymph-follicles were swollen, especially Peyer's patches and the mesenteric

lymph-glands. A microscopic examination showed fatty degeneration of many important organs, especially the liver, and at times of the muscles of the heart. The bacteriological examinations were, as a rule, negative, especially as regards the tissues of the intestine. Clumps of bacteria were found only once in the liver and once in the kidney.

SYMPTOMS.—The first symptoms were generally restlessness and cyanosis, not only of the face but also of the body and extremities, and especially of the back. The color increased progressively until it became a deep blue. To this was added an icteric color, which when death did not occur within twenty-four hours became very marked. The respiration was rapid; the pulse was not especially increased in rate. The rectal temperature never rose higher than 38.1° C. (100.6° F.). The skin generally felt cool. Vomiting and diarrhea occurred in some cases. The most striking symptom was the appearance of the urine. It had a pale brownish color, and was passed frequently, and often with considerable straining. An examination showed that the color was due not to bile, but to hemoglobin. In the sediment were found numerous epithelial cells from the walls of the kidney, granular casts with blood-corpuscles adherent to them, micrococci, masses of detritus, and urate of ammonia. A small quantity of albumin was present. Later in the disease convulsions occurred, followed rapidly by death.

In other cases besides those of Winckel's in which the blood was examined the condition was found to be one of hemoglobinemia. The percentage of hemoglobin was high, and free hemoglobin was found in the blood-serum, while the erythrocytes were greatly reduced in number, at times amounting to only 1,700,000 or even less.

DIAGNOSIS.—The resemblance of this disease to the form of infectious disease known as acute fatty degeneration of the newborn is very striking. Most of the symptoms are common to both diseases. Large hemorrhages are also not uncommon in this disease, but are not so marked as in acute fatty degeneration. The striking points of difference are the presence of hemoglobinuria and the fact that large numbers are affected at the same time in infectious hemoglobinemia, while these conditions have not been found in acute fatty degeneration.

TREATMENT.—The treatment should be the administration of oxygen and stimulants, and forced feeding by means of a dropper when the infant is too weak to suck.

VII. PREMATURE INFANTS

APPEARANCE AT BIRTH.—The picture of a premature infant in the early days of life is quite characteristic. Besides its very small size, as shown in the illustration, where the size is compared with the nurse's hand, it shows in varying degrees an absence of the life and vigor which is seen in the fully developed infant at term. It is emaciated, its skin being soft, wrinkled, and showing very little subcutaneous fat. Its head is large, its abdomen broad and dis-

FIG. 67

Infant premature at seventh month. Birth-weight, 1740 grammes. Age, 10 days; weight, 1540 grammes

tended, and its limbs puny. According to the stage of its development it may or may not have the remains of the hair (lanugo) on its body which was present in uterine life, and in like manner its nails may or may not be well formed. Its face has a senile expression and it is torpid and extremely somnolent. The eyes are closed. Its cry is very feeble. The surface temperature is usually cool, the extremities seldom move. The respirations are very superficial and irregular, often ceasing altogether for a few seconds. The power to suck and even to swallow is often slight. These signs evidently indicate that the vitality is very low, and if the weight is below three or four pounds, and the length less than eighteen or nineteen inches, that the functions and organs are not developed sufficiently for use, and that unless unusual care be taken in the treatment of such cases, they will soon die.

PROGNOSIS.—The majority of infants born before the completion of the seventh month of pregnancy die within a very short time, although I have seen one or two cases of survival in babies premature in the sixth month. A number of babies premature at the seventh

and eighth months survive, the outcome depending on the vitality of the individual baby, and the care with which it is treated. There are a number of factors which make the prognosis very uncertain in every case. Many premature infants have a varying degree of congenital pulmonary atelectasis, which generally lowers their vitality and resistance. Syphilitic infants usually die within a few days. Sclerema is a very unfavorable sign. The best prognostic indication in a premature infant is the temperature curve. If with artificial heat the temperature remains subnormal, or if the curve is very irregular, rising above the normal when the external heat is increased, and falling below the normal whenever it is slightly diminished, the probability is that the infant's vitality is very low. When the temperature begins to approach the normal line, and to show less irregularity, the prognosis becomes better. The weight curve stands next to the temperature curve as a prognostic sign. A steady gain in weight is a very favorable sign. Many premature infants, however, do not begin to gain in weight for a number of weeks, and yet may survive.

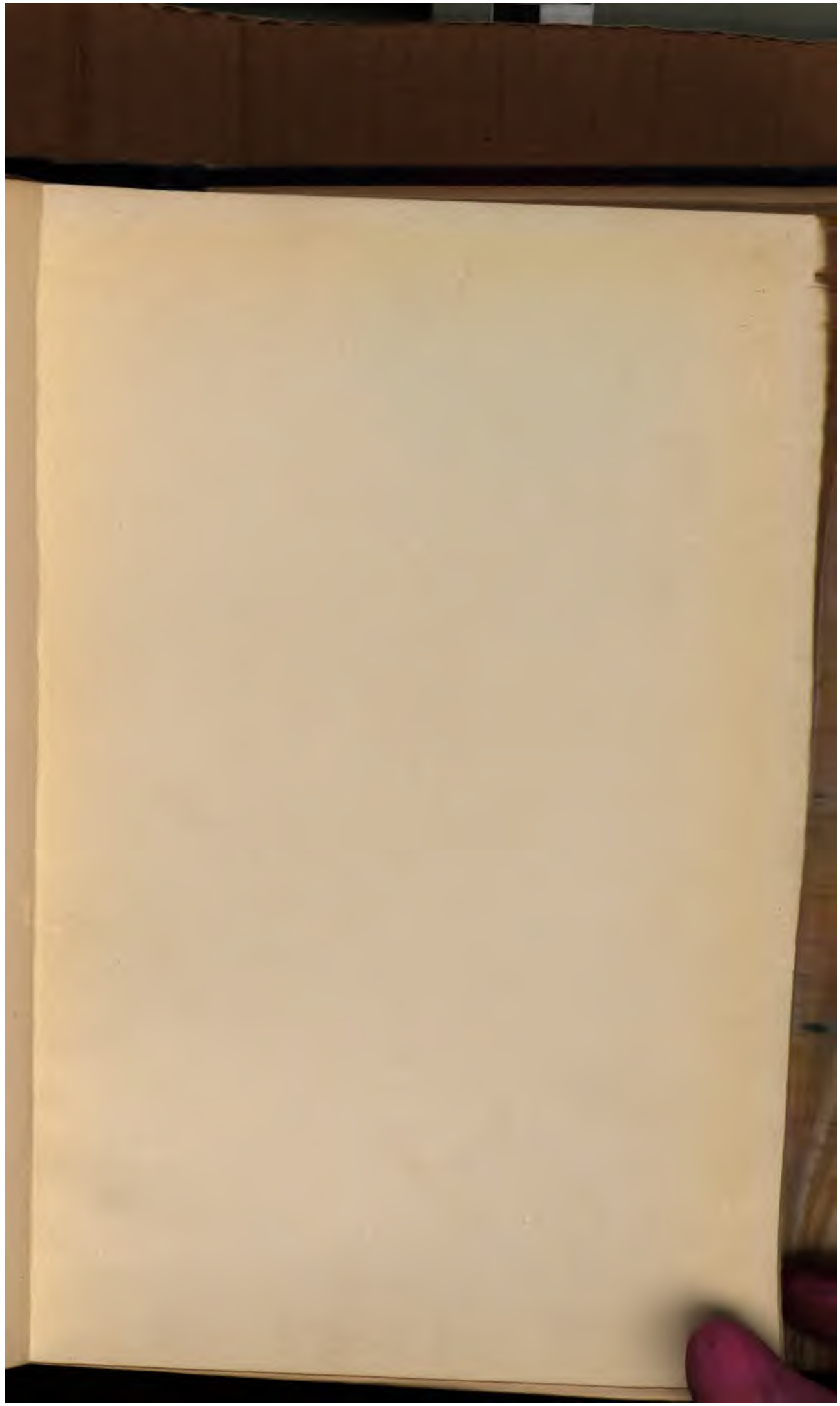
It must be remembered that even with the best care, premature infants are liable to sudden death from no apparent cause. This liability to sudden death is not passed when the infant reaches the period of full term, but continues for a number of months thereafter.

TREATMENT.—The same measures of care which are indicated for premature infants, are also applicable to infants who, although born at full term, show the same evidences of low vitality as are characteristic of infants prematurely born.

There are three principal points to be considered in the treatment of premature and feeble infants. These are (1) the maintenance of the body heat; (2) the modifications of hygiene and care which are necessary to protect the infant from the deleterious influences of the external world; (3) nourishment.

ARTIFICIAL HEAT.—For the maintenance of heat in premature infants, various forms of incubators and brooders have been recommended and widely used. My experience with incubators has been entirely unsatisfactory, and I cannot recommend their employment. An "*incubator bed*" has given the best results in my hands. This consists in an ordinary infant's crib, padded, and with the lower half covered with a blanket, sufficient space being left at the top for the admission of a plentiful supply of air.

The infant is wrapped in cotton. The best means of wrapping the infant is the "*premature gown.*" Such a gown is shown in the illustration. It is made of a thin layer of absorbent cotton, about a quarter of an inch thick, between two double layers of fine gauze. The layers of cotton and gauze are sewed together on the machine.



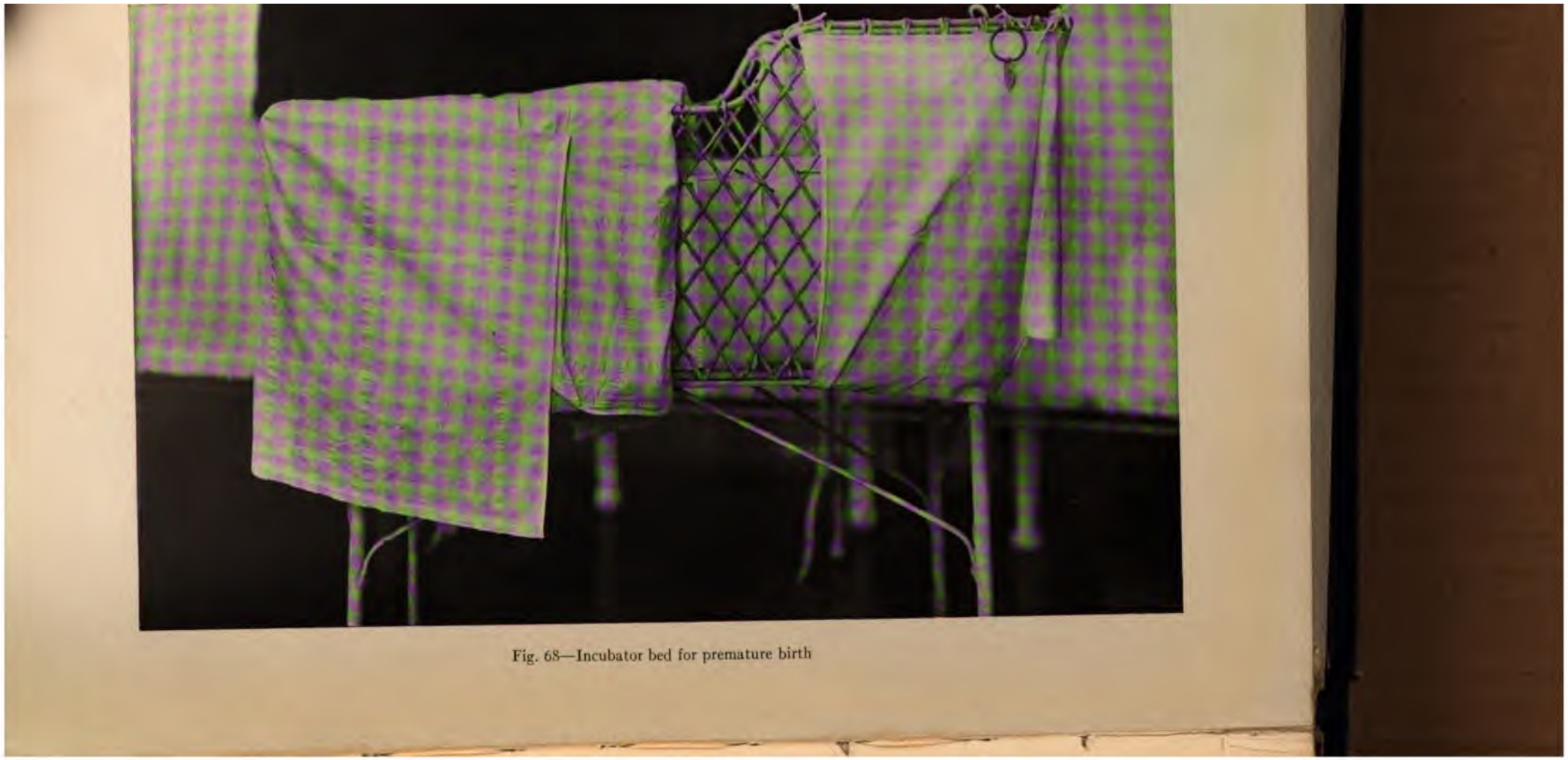


Fig. 68—Incubator bed for premature birth

both around the edges, and across in both directions, the lines of stitching being about five inches apart. The dimensions of the gown should be about twenty-six inches in length by twenty-three inches in width. To the top of the body of the gown is attached a hood, made of the same materials. The infant is wrapped in this gown, and laid in the incubator bed.

There must be in the bed with the infant some means of supplying artificial heat. An excellent device for this purpose is an electric pad or electrotherm. Just as good results can, however, be obtained by the use of hot water bottles. Earthenware bottles filled with hot water and wrapped in flannel retain their heat for a long time. One or two of these are placed in the bed alongside the infant. A thermometer wrapped in absorbent cotton is also placed in the bed beside the infant, and the whole, infant, bottles, and thermometer, are covered with a blanket. By changing the water in the bottles at proper intervals, a fairly constant temperature can be maintained in the incubator bed. Ordinarily with infants of fairly good vitality, the thermometer should register 85° to 95° F.; but the temperature which is maintained in the incubator bed must be regulated by the rectal temperature curve of the child. If this shows a tendency to remain subnormal, more external heat will be necessary, and the temperature of the bed should be kept at from 95° to 98° F.

HYGIENE AND CARE.—The premature baby must have a constant supply of pure fresh air. For this purpose, the infant must be kept in a room which has some proper communication with the outdoor air, the temperature of the room being maintained by ordinary heating methods. Under ordinary circumstances the temperature of the room should be from 70° to 72° F.

Most pediatrists believe that a great mistake has often been made in having the air of the room too warm. Nevertheless, I find that in many cases a warmer temperature is of distinct value provided that the air is kept fresh. When a premature infant in a properly devised incubator bed and in a room of 72° F., still shows a tendency toward constant subnormal temperature, they will often do better if the temperature of the room be kept at 80° F. In a number of cases I have seen premature babies who had not been gaining in weight, begin to gain immediately when they were transferred to a room at 80° F. The probable explanation of this is that they do not have to use so much of their nourishment for the maintenance of their bodily heat, and more food becomes available for tissue building. We have found at the Infants' Hospital that the "80° room" is a valuable measure in the treatment of certain premature and feeble infants.

The premature infant should be handled as little as possible. It

should not be bathed, but its body is kept oiled with olive oil. When it is necessary to change the diaper, this should be done rapidly and with as little exposure as possible. The morning and evening temperatures should be taken daily, as this record is necessary in regulating external heat, and also has a bearing on the problem of nutrition. But the taking of the temperature should be attended with a minimum of handling and exposure. The premature infant should also be protected from excessive light and from noise.

FIG. 71

Feeder for premature infants (reduced one-half)

FEEDING.—Breast milk should be used in the nourishment of premature infants whenever possible. Few premature infants are strong enough to take the breast. In the early weeks, a premature infant should be fed as often as every hour, but such frequent nursing has a deleterious effect upon the secretion of the mammary glands of the mother or wet-nurse. Consequently the infant should be put to the breast only every two hours. It will not be able to empty the breast and the remaining milk should be withdrawn by means of the breast pump. In this way, sufficient breast milk can be

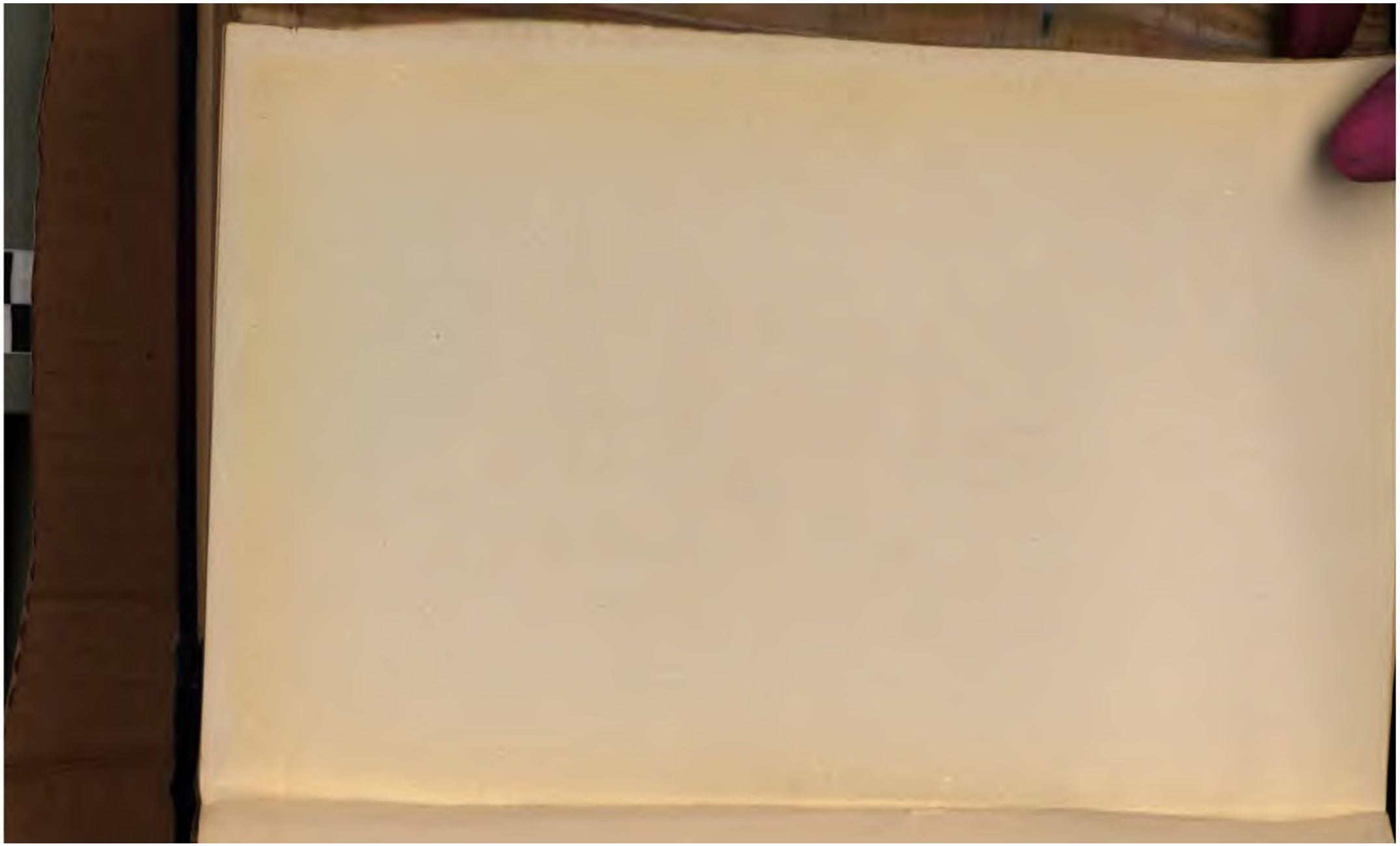


FIG. 69—Articles for premature infant

obtained for use in the alternate feedings when the infant is not put to the breast. When an infant is too feeble to take the breast, as is usually the case in infants born before the eighth month, and with feeble babies born in the eighth month, the infant's food can be obtained by pumping the breasts at the proper intervals.

The method of administering milk to a premature baby who is unable to take the breast is important. If the infant is able to swallow, the milk should be given with the Breck feeder. This consists in a graduated glass tube narrowed at the ends. Over one end is placed a proper sized nipple, and over the other end is placed a rubber compression bulb. The advantage of the Breck feeder over giving the milk with a spoon or medicine dropper is that the sucking reflex is not entirely abolished. There is evidence that the sucking reflex has an important influence upon the digestive function, and with the Breck feeder, even though the baby be unable to suck strongly enough to draw the milk from the bottle, it can make efforts at sucking, and can be helped by the pressure made by the nurse upon the rubber bulb. Whenever the infant is so feeble that it is unable to swallow, it must be fed by means of gavage. When, as often happens with premature infants, the mother has not a sufficient supply of milk, a wet-nurse should be obtained if possible. The wet-nurse should always be accompanied by her own infant, and should nurse it, as the requirements of the premature infant will not be sufficient to maintain the normal function of lactation in the wet-nurse.

The quantity to be given at a feeding to premature infants varies with the age and weight of the child. With a seven months' baby weighing from three to four pounds, one can begin with half an ounce every hour. Changes are regulated by the temperature and weight curves, the composition of the food, the appearance of the movements, etc. Usually after a few weeks the amounts can be increased and the intervals made longer, so that the baby will get an ounce every two hours. This amount will later be further increased in accordance with the ordinary principles of infant feeding.

When a wet-nurse cannot be obtained, artificial feeding becomes necessary. It is true that the outlook with artificial feeding is not so good as it is with human milk, but the prognosis is not so bad as some writers would lead one to suppose. In all premature infants we have to contend only with congenital weakness of the digestive powers, and such cases are often not so difficult to manage with artificial feeding as are older infants whose powers have been ruined by improper feeding.

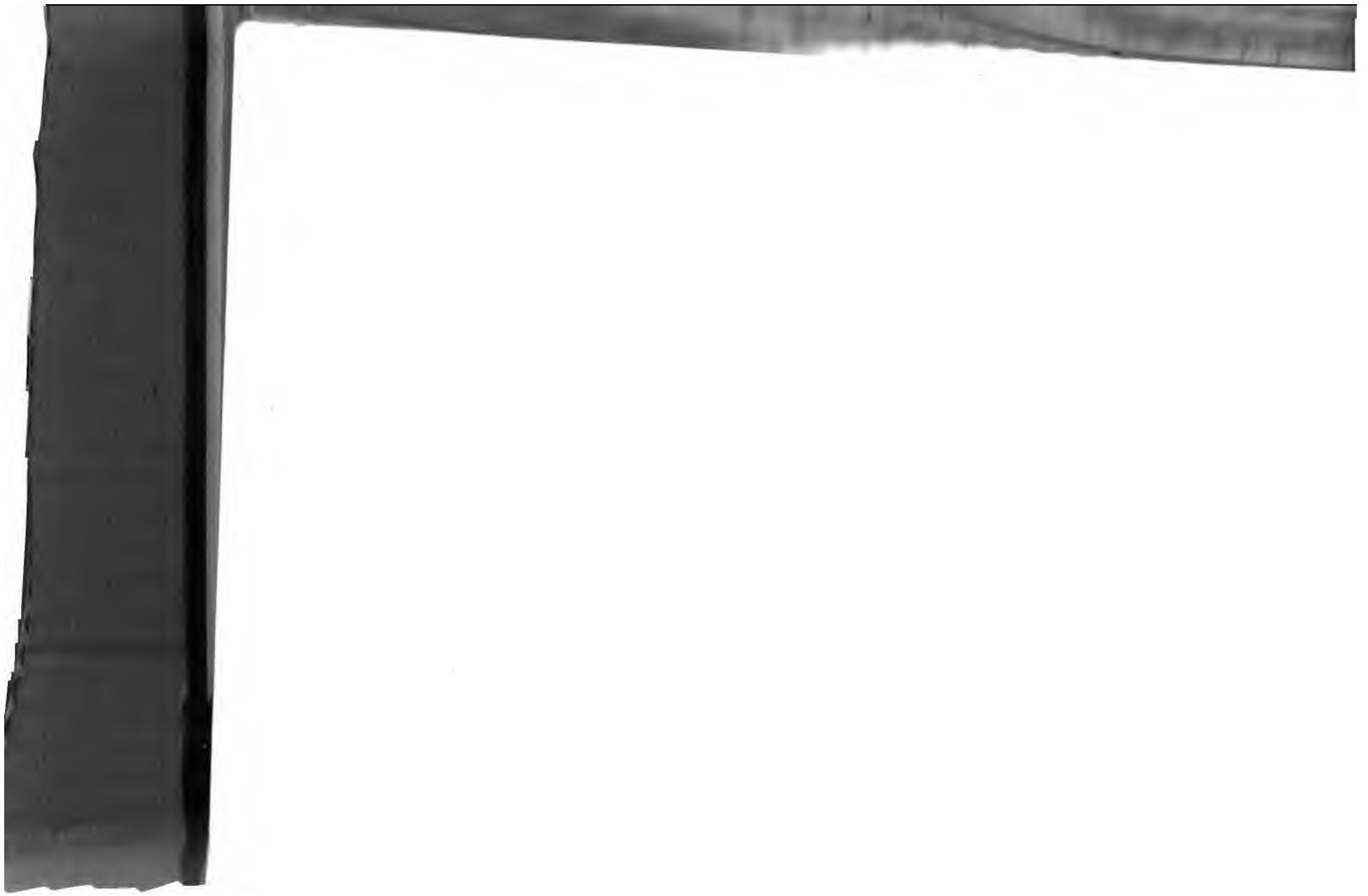
The amount to be given at a feeding and the intervals between feedings are the same with artificial feeding as when breast milk is used. The composition of the food, however, must be weak. In my experience premature infants have less difficulty in digesting

the fat than the casein of cow's milk, fat indigestion being a particular manifestation of infants who have been improperly fed. Consequently, I usually select for a premature infant a whey mixture or split protein formula. An average food to begin with is—

Fat, 1; lactose, 5; whey protein, 0.75; casein, 0.25; lime water, 20 per cent. of the milk and cream.

The methods of obtaining such a food are described in the division on Feeding. Further changes in the composition of the food will depend upon the weight curve and symptoms of the infant, the physician being guided by the general principles of infant feeding.

The signs of insufficient nourishment in a premature infant are, failure to gain in weight, constipation, but otherwise good stools, and a persistent tendency toward subnormal temperature. The signs that the food is too strong, or improperly adjusted to the digestive power of the infant, are, vomiting, green or undigested movements, and failure to gain in weight.



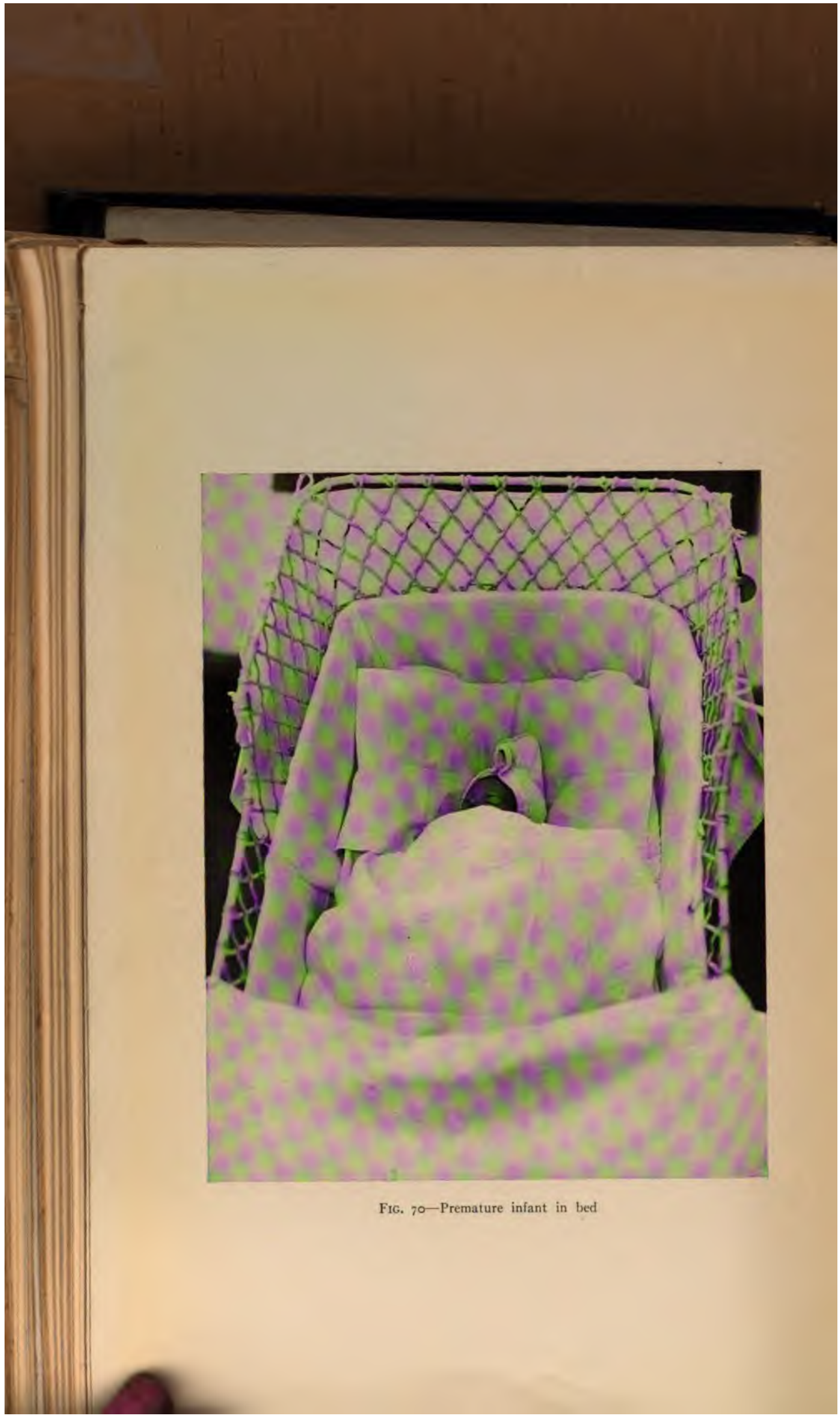


FIG. 70—Premature infant in bed

CHART 4

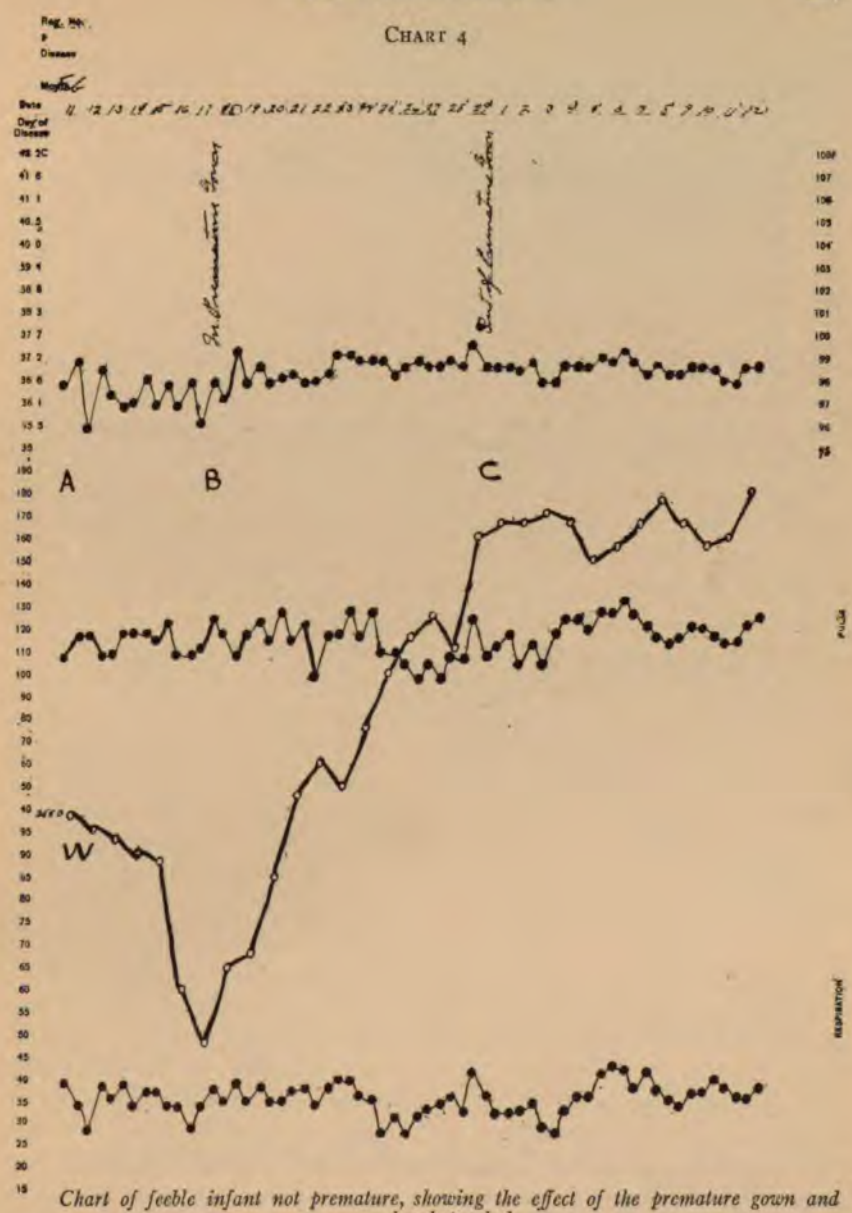


Chart of feeble infant not premature, showing the effect of the premature gown and incubator bed

The feeding was not changed during the period represented by this chart, which shows the temperature, pulse, respiration, and weight curves.

A. Infant admitted to hospital and placed in ordinary bed.
B. Infant placed in incubator bed and premature gown.
C. Infant taken out of incubator bed and premature gown.
W. Weight curve.

It will be noted that under ordinary treatment the temperature remained subnormal and there was a steady loss of weight. When no other change in the treatment than putting the baby into the incubator bed and premature gown was made, the temperature rose at once to the normal, and gain in weight was rapid. Later, when removed from the incubator bed, the infant maintained a normal weight but did not gain so rapidly. The artificial heat of the incubator bed enabled this infant to spare food from heat production and from the effort to maintain a normal temperature, and to utilize this food for tissue building.

CHART 5

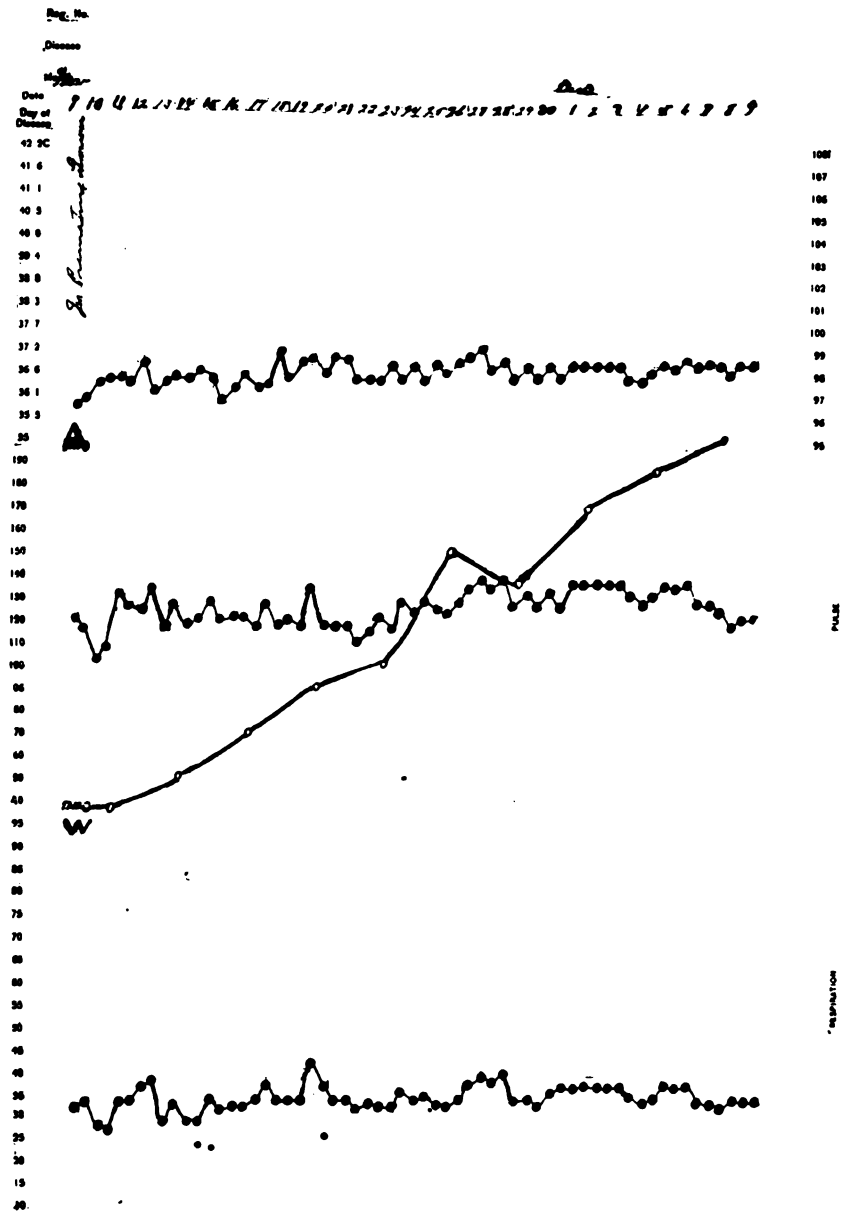


Chart of premature infant, showing temperature, pulse, respiration, and weight curves

A. Treatment begun.
W. Weight curve.

It is to be noted that at the beginning of treatment the temperature was subnormal, but the infant did well and steadily gained in weight. As improvement occurred, the temperature rose to the normal line.

DIVISION IV

FEEDING

I. GENERAL PRINCIPLES

Just as the highest aim of medical art should be directed to the province of preventive medicine, so the highest and most practical branch of preventive medicine should consist of the study of the best means for starting young human beings in life. They should be preserved from the perils which surround the early hours of their existence, and be given strength and vigor to resist the attacks which must inevitably be made on their vitality, which are greater and more dangerous in inverse proportion to their age. With these objects in view, the preventive medicine of early life becomes pre-eminently the intelligent management of the nutriment which enables young human beings to breathe and grow and live. In fact, it is a proper or an improper nutriment which makes or mars the perfection of the coming generations. The feeding of infants is, then, the subject of all others which should interest and incite to research all who are working in the domain of pediatrics, and in fact as a matter of public health, a practical knowledge of the modern methods of infant feeding becomes an imperative duty. The subject is a great one, and is worthy of the most careful study. It should be taken up carefully. It should be dealt with broadly.

In reviewing the immense amount of literature which has accumulated on the subject of feeding, we find that the superiority of human milk to all other kinds of infant food in the early months of life is acknowledged so generally, that it has become an axiom. On the other hand, the opinions expressed regarding artificial feeding in the past are so diverse and so opposed to one another that it is evident that much which has in former years been taught must be unlearned, or rather admitted to be untrue, before we can expect to make any decided progress in this most difficult subject.

The feeding problem is one which is surrounded with many difficulties on account of the great diversity of individual circumstances and idiosyncrasies. Certain infants thrive on peculiar mixtures which are not adapted to infants as a class. Many will not thrive on that food which nature has provided for them, and the well-being of an infant will depend much upon the circumstances by which it is surrounded, such as affluence or poverty, country or city life. The

constituents of the nutriment which nature has provided for the offspring of all mammals in the early period of their existence is essentially animal and never vegetable. It is therefore evident that an animal food, entirely and freshly derived from animal and not vegetable sources, has been proved to be the nutriment on which the greatest number of human beings live and the least number die.

THE MAMMARY GLAND.—The mammary gland, in its perfect state, uninfluenced by disease or nervous disturbance, or by the improper living of the mother, is a beautifully adapted piece of mechanism constructed for the elaboration and secretion of an animal food. When in equilibrium it represents the highest type of a living machine adapted for a special purpose,—mechanically, physiologically, and economically. The breasts of all mammals are elaborators and producers. They are not storehouses for preserving sustenance until it is needed. They are delicately constructed mills, turning out when demand is made for it, a product which has been directly formed within their walls from material which has been brought through their portals from various parts of the economy. The breast is a compound racemose gland, lined with glandular epithelium, which forms sugar, fats, and proteids, and these are mixed with water from the blood. The epithelial cells are so finely organized, and so sensitive with their minute nerve connections, that changes of atmosphere, changes in food, the emotions, fatigue, sickness, the catamenia, pregnancy, and many other influences, throw their mechanism out of equilibrium most readily, and change essentially the proportions of their finished product. Then again this delicate mechanism adapts itself to the quantity of its product, elaborating a smaller or a greater supply, according to the demand actually made upon it by the consumer. The same breast will either supply the proper amount of milk demanded for the requirements of the average age or a greater amount for the same age in case of a greater gastric capacity. Again, this machinery is regulated as to the time which it takes to produce the average food required for the different ages, a shorter interval of feeding being needed for the younger infant and a longer one for the older. This fact is made evident by the decided qualitative changes which result when the gland is called upon to produce its product at improper intervals. Moreover, the product of the mammary gland is clean, free from injurious bacteria, and is already warmed to the proper temperature demanded by the delicate mouth and stomach of the young infant. The analyses of large numbers of specimens of human milk at different periods of lactation show us that not only do the constituents vary from month to month, and even from day to day, but that this variation takes place as much in the early as in the later periods of lactation. We are not war-

ranted, therefore, in assuming that the milk grows stronger as its age increases, provided that it still remains in normal equilibrium. The mammary gland acts both as a secretory and as an excretory organ, so that it cannot be classed as a metabolic tissue in the limited meaning which we now attach to these words. Yet the metabolic phenomena giving rise to the secretion of milk are so marked, so distinct, and have so many analogies with the metabolism which we meet in adipose tissue, that we must look upon the mamma chiefly as a secretory organ (Foster). This, however, is only within certain limits, for we know that at times foreign elements may be excreted from the gland. This at once suggests the interesting question as to when the mammary gland is most likely to have what we might call its normal secretory function interfered with and to assume temporarily the function of an excretory organ. This seems to occur both before the gland has attained its equipoise, as during the colostrum period, and later when any of the above-mentioned influences occur which affect the general mechanism of the gland. In these instances we find the colostrum reappearing in the milk. Therefore in the beginning of lactation, during lactation when normal metabolism is interfered with, and as lactation draws to a close, we have analogous conditions in which the mammary gland instead of being a normal secretory organ becomes abnormal and more or less an excretory organ. During these periods of abnormal gland excretion we must remember that drugs can be eliminated by the milk more freely than when the gland is in equipoise. We assume, therefore, that the mamma during that early period of lactation, which essentially represents a condition of lack of equipoise, has a double function, partly secretory, partly excretory. The greater the excretory function of the gland is at any time in proportion to the secretory, the more abnormal will be the finished product; while the nearer the gland approaches to a purely secretory organ, the more perfect and normal will be its product. The mechanism of the mammary gland is therefore in its most perfect condition after the colostrum period has ceased, and at a time when the general organism, both physical and mental, is freed from causes detrimental to a perfect metabolism.

General principles are vital in their importance when we come to study the subject of feeding in all its phases, whether the nutriment to be provided for the infant is to come directly from its mother, a wet-nurse, or an animal, or indirectly from the product of the mammary gland. These principles are, (1) That nature throughout all ages has clearly indicated by means of natural selection what the source of supply should be; that is, that the mother should during some early period of its life supply food for her offspring from her mammary glands. (2) That when, owing to disease, over-civiliza-

tion, or any causes which prevent the offspring from receiving its sustenance directly from the maternal mammae, some nutriment must be substituted which will correspond as closely as possible to the natural food-supply. (3) That this substitution can be obtained most exactly through the product of the mammary gland of another woman. (4) That, owing to the strong analogy between human beings and all animals which suckle their young, we should in our efforts to copy good human milk make use not only of what we have learned from human beings, but also of what is known of lactation as it occurs in animals. This requires a knowledge of the investigations and experience of those who have studied commercially the breeding of animals and their food, and the production and modification of their milk.

II. MATERNAL FEEDING

Human milk is the only ideal food for infants. The statistical results of breast feeding are vastly superior to those of artificial feeding. In Berlin, in the years from 1900 to 1904 only 9 per cent of the infant mortality occurred in breast-fed infants. In Boston, statistics show that an artificially fed infant is six times as likely to die than is a breast-fed infant.

In spite of these facts, we frequently encounter mothers who prefer not to nurse their infants. They give as a reason that the study of artificial feeding has led to such perfecting of the methods employed, that the best artificial feeding directed by the most carefully trained physicians gives results sufficiently good to make this a safe method. These same mothers, in rearing their children, would hesitate to violate any other hygienic principle, when such violation would diminish six-fold the chance of their child surviving. Only gross ignorance of the facts can explain their desire to incur such a risk. It is the duty of every physician to impress the superiority of maternal feeding upon every mother, and to steadfastly refuse to countenance any other method of feeding unless absolutely necessary. The desire of the mother to be relieved of the trouble and confinement incidental to nursing, is not a sufficient reason for resorting to the bottle.

If human milk is the ideal infant food, a thorough knowledge of all its characteristics is essential to the study of infant feeding.

HUMAN MILK

PHYSICAL CHARACTERISTICS.—Human milk is of a bluish-white color and closely resembles cow's milk in appearance. It is odorless and has a somewhat sweeter taste than has cow's milk. The specific gravity averages between 1.030 and 1.032, the extreme variations being 1.020 and 1.038. The reaction is amphoteric, alkaline to litmus, and slightly acid to phenolphthalein. Microscopically are seen many minute fat droplets of an almost uniform size. Human milk shows a coagulation when acids are added. With the rennin ferment no change is observed until the milk is acidified, although the ultramicroscope shows that the rennin acts in neutral solutions. Human milk does not coagulate uniformly with rennin, and the casein is precipitated with greater difficulty with acids than is the case with cow's milk. The diminished coagulability of human milk has been explained by the relative alkalinity of the milk and by its low calcium content. The curd formed is very fine and often can only be seen with the microscope.

CHEMICAL COMPOSITION.—Milk contains fat, carbohydrate, and protein substances, together with certain mineral salts and water. A large number of analyses gives the following composition for an average human milk:

TABLE 18

Average Human Milk

Fat	3	to	4	per cent
Carbohydrate	6	to	7	per cent
Protein	1	to	2	per cent
Ash	0.2			per cent
Water	86.8	to	89.9	per cent

The fat in human milk is in the form of minute globules, which are held in a permanent emulsion by the fluid in which they are suspended. The fat is made up chiefly of the nonvolatile fatty acids, palmitic, stearic, myristic, and oleic acids. Among these oleic acid predominates. The volatile fatty acids are present in small quantities in human milk, constituting no more than about 2 per cent of the total fat. Among them have been demonstrated butyric, capronic, caprinic, and caprylic acids.

The carbohydrate of human milk is lactose, or milk-sugar. It is in solution.

The protein of human milk is of two chief forms, one of which is soluble in water, the other insoluble. The insoluble protein is casein, and is held in suspension in milk, probably because it is in chemical combination with the calcium phosphate. The soluble proteins consist of a number of substances, of which the predominating is lactalbumin. A globulin is also found, and possibly other nitrogenous substances. Lactalbumin much resembles the serum albumin of the blood.

Analyses differ widely as to the relative proportions of soluble protein and casein in human milk. König, in a total protein of 1.82 per cent estimated casein at 0.59 per cent, and the soluble protein at 1.23 per cent. The figures now most generally adopted are those of Schlossmann, who found that about 41 per cent of the total nitrogen in human milk is in the form of casein. There is in milk, however, a certain amount of so-called residual nitrogen contained in bodies which are not protein, such as, possibly, urea. This may be from 15 to 20 per cent, and if it is deducted, only 44 to 39 per cent is left for the soluble proteins. It is easiest to remember that in human milk the casein and soluble proteins are in about equal proportion. The composition of the *mineral salts* in human milk is not thoroughly known. Analyses differ widely and variations are great. The table shows some analyses from various observers:

TABLE 19

Percentage of Salts in Human Milk to 100 Parts of Ash

(From Engell and Schloss)

	BUNGE	BLACKHAND AND KRONHEIM		LANGE	SCHLOSS
	I	I	II		
K_2O	32.14	33.34	27.33	19.9	28.77
Na_2O	11.75	11.91	15.88	29.6	10.26
CaO	15.67	17.36	15.52	12.9	20.44
MgO	3.99	3.17	2.13	2.9	4.66
Fe_2O_3	0.27	0.63	1.75	0.25	
P_2O_5	21.42	14.79	11.75	17.9	22.0
CL	20.35	15.47	23.93	21.3	16.61

Gradually importance has been attached to the *ferments* or *enzymes* of milk, especially in the discussion as to whether raw or boiled milk is most favorable to the metabolism of the infant. All that can be said at present is that a large number of such ferments have been found in human milk, such as proteolytic, fat-splitting, and carbohydrate-splitting ferments, pepsin, trypsin, fibrinogen, and a number of others. The rôle of these ferments is still unknown.

VARIATIONS IN MILK.—The composition of human milk is by no means constant. Not only do the milks of different women differ widely from one another, but the milks of an individual woman vary from day to day, and even from hour to hour. The composition of the milk obtained at the beginning, at the middle, and at the end of a nursing shows considerable variation. The percentage of fat, for example, increases steadily from the beginning to the end of each nursing.

The normal variation in the milks of different women, or in the milk of the same woman at different times, is most marked in the fat. Extreme variations in the fat are from 0.1 per cent to 13.7 per cent. Normal variations are from 2 per cent to 6 per cent. The sugar varies least, extreme variation being from 4.22 per cent to 10.9 per cent, and normal variation being from 6 per cent to 7 per cent. The protein shows a gradual, but rather marked increase during lactation. Its normal variations are 1 to 2 per cent, and extreme variation from 0.2 to 3.4 per cent.

It does not follow from the fact of these variations in the composition of human milk, that a departure from the standard of an average milk is a cause of harm, or an explanation of any symptoms of disturbance which may be present. Variation is the normal condition, and the composition of an average milk does not constitute a standard of excellence in this ideal baby food. We should rather conclude that nature provides these variations for a purpose, to meet varia-

tions in the digestive powers and requirements of different babies. In attempting to make an artificial food, we should remember that variation is an essential feature, and that babies, in general cannot be fed on an arbitrary food of fixed composition.

COLOSTRUM.—The most marked variation in the milk is seen in the earliest days of the infant's life. When the secretion of the mammary glands first begins, the milk differs widely from that seen when lactation has become fully established. The milk secreted at this time is called *colostrum*, and it has a less sweet taste than the later milk, and is of a deeper yellow color. The color was formerly supposed to be due to the presence of colostrum corpuscles, but is attributed by Czerny to a coloring matter contained in the fat drops. Colostrum is coagulated into solid masses by heat. The specific gravity ranges from 1.028 to 1.072, the average being about 1.040. In reaction it is strongly alkaline. The chemical composition of colostrum differs markedly from that of the later milk. The analyses to be found in the literature of the subject differ very widely, and many of them are probably based on rather faulty chemistry. The analysis most frequently given in textbooks is based on the average of five analyses by Pfeiffer, and is as follows:

TABLE 20

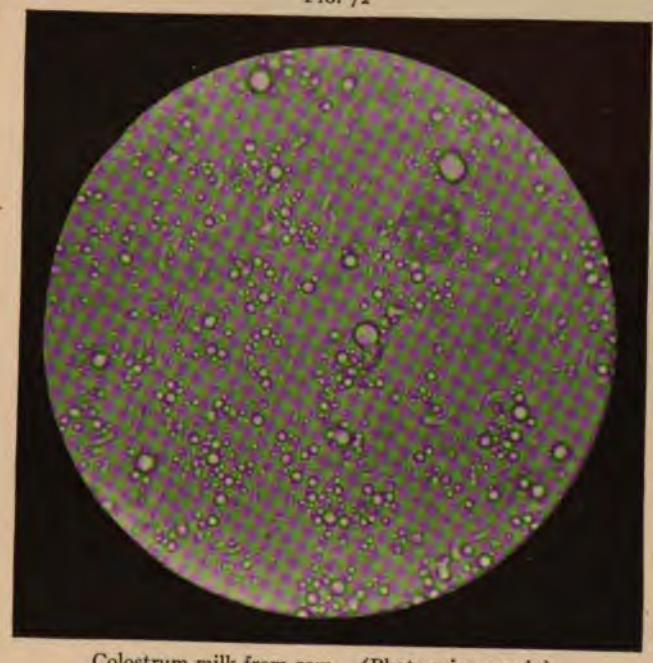
Composition of Colostrum

Fat	2.04 per cent
Lactose	3.74 per cent
Protein	5.71 per cent
Salts	0.28 per cent
Water	88.23 per cent

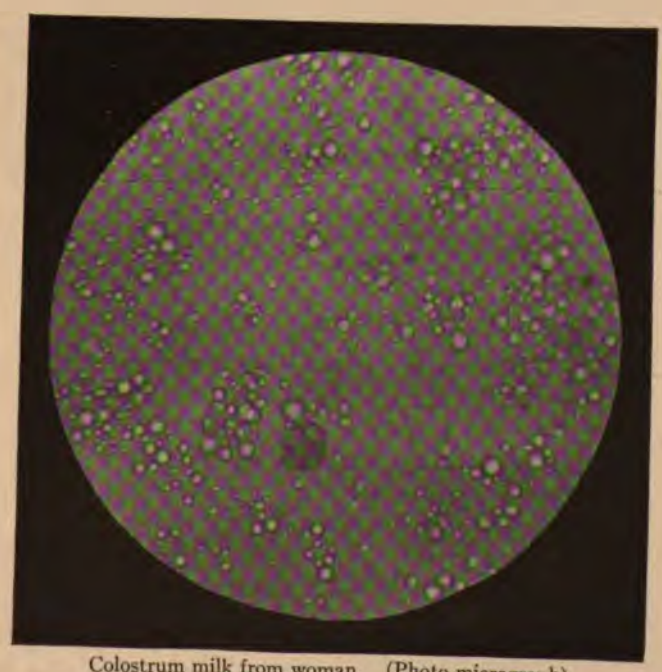
Microscopically the fat droplets in colostrum are more unequal in size than in ordinary milk. There are in addition certain bodies known as colostrum corpuscles, which are large, round, and granular, with a nucleus. They are probably leucocytes, whose cell membranes are completely filled with small fat drops. They persist in milk for a week or ten days, gradually disappearing.

The most recent theory on colostrum is that advanced by Czerny and Keller. According to their view, if the infant does not empty the breasts and they fill up again with secretion, there is a change in the composition of the milk as the result of absorption of its different components. This changed secretion they designate as colostrum, and explain its presence in the breasts during pregnancy and the first few days postpartum by the fact that the breast is at this time not completely emptied, and that re-absorption takes place. Under this theory they also include as colostrum all milk in which there has been any absorption such as would appear when the mammary secretion is in process of drying up, or when a feeble infant is unable

FIG. 72



Colostrum milk from cow. (Photo-micrograph.)



Colostrum milk from woman. (Photo-micrograph)

properly to empty the breast. It has been observed that the colostrum corpuscles always reappear in the milk when nursing is stopped and the milk is drying up, and that they are seen even when lactation is interrupted for a few days.

DAILY QUANTITY OF HUMAN MILK.—The quantity of secretion produced by the breast during the first days of life is very small. On the first day the quantity of colostrum secreted is only from 10 to 20 c.c., and on the second day from 80 to 100 c.c. The quantity of milk secreted daily after lactation has become established, varies within the widest limits. It is measured by weighing the baby before and after each nursing. The quantity differs in the case of each individual woman, and differs in the same woman at the different periods of lactation. The quantity also varies with the weight, vigor, and demands of the baby. In view of this variability, it would seem as if any figures showing the average quantity taken by normal babies from the breast at different times, would have comparatively little practical value. Unfortunately, this is the only method we have of arriving at any idea as to how much food the normal baby should take each twenty-four hours, and such a standard is necessary both in judging disturbances of breast feeding, and in planning a routine of artificial feeding. It has been shown that the quantity taken from the breast by normal infants is a better guide to their food requirements, than are post-mortem measurements of gastric capacity.

The table taken from Czerny and Keller gives Feer's calculation of daily quantity for babies of average weight (Camerer's figures).

TABLE 21

Average Daily Amount of Milk Drawn by a Baby

(From Czerny and Keller)

AGE IN WEEKS	AVERAGE WEIGHT OF BREAST-FED BABIES ACCORDING TO CAMERER, GM.	THE CALCULATED DAY'S AMOUNT OF MILK, GM.	AGE IN WEEKS	AVERAGE WEIGHT OF BREAST-FED BABIES ACCORDING TO CAMERER, GM.	THE CALCULATED DAY'S AMOUNT OF MILK, GM.
1	3,410	291	14	5,745	870
2	3,550	549	15	5,950	878,
3	3,690	590	16	6,150	893
4	3,980	652	17	6,350	902
5	4,115	687	18	6,405	911
6	4,260	736	19	6,570	928
7	4,495	785	20	6,740	947
8	4,685	804	21	6,885	956
9	4,915	815	22	7,000	958
10	5,055	800	23	7,150	970
11	5,285	808	24	7,285	980
12	5,455	828	25	7,405	990
13	5,615	852	26	7,500	1,000

BACTERIOLOGY.—Human milk is generally considered to be sterile, but investigations have shown that the milks of healthy women contain bacteria in the majority of instances. The organism most commonly found is the staphylococcus aureus, but others may be present. They are generally believed to enter the ducts from without. Under normal conditions, these organisms have no pathological significance for the healthy infant.

CONDITIONS AFFECTING LACTATION.—It has generally been held that the composition of the milk of a nursing mother is and can be much influenced by her *diet*. Much investigation has failed to reveal any very direct or important influence exercised upon the milk by the nurse's diet. It has been shown that the quantity of fat will diminish when nursing women are underfed, and will increase to some extent when more fat is given in the diet. Certain fruits and vegetables eaten by the mother occasionally produce a sudden disturbance of digestion in nursing babies. Such disturbances are however, an idiosyncrasy, and do not come under any general rule. The weight of modern opinion grants little influence to diet.

That certain *drugs* taken by the mother can be excreted in the milk has been definitely proven. Drugs known to have been so excreted, are, according to Talbot, potassium iodid, sodium salicylate, antipyrin, mercury, aspirin, calomel, arsenic, bromides, hexamethylenamin, and bodies soluble in fat. These drugs, however, are found in milk only in slight traces, and probably cannot do harm, except in the case of salvarsan, when sudden death of the infant has occurred after treatment of the mother. Alcohol is found in breast milk only in minimal amounts, and only after a very large quantity has been taken.

Vegetable poisons, and *toxins* formed in the mother, may be passed into the milk. *Antibodies* may go over, and it has been shown that *immunity* can be transmitted to the infant through the breast milk. This probably explains the immunity against infection possessed by the nursing baby, not seen in bottle fed babies. Much work is now being done on the transmission of various immune bodies.

Nervous impressions.—Excessive nervous disturbance, excitement, or violent emotions may produce a marked alteration in the composition of milk. The evidence of this phenomenon is mainly rather clinical than chemical, but there is some evidence that such nervous disturbance causes an actual chemical change, the protein becoming increased, through which the baby becomes ill.

Menstruation usually produces little chemical change. Some cases have been reported of marked change in the composition of breast milk during menstruation. The coming on of menstruation, is not a sign that a baby should be weaned because of this cause alone. *Pregnancy*, however, is a contraindication to nursing.

MATERNAL NURSING

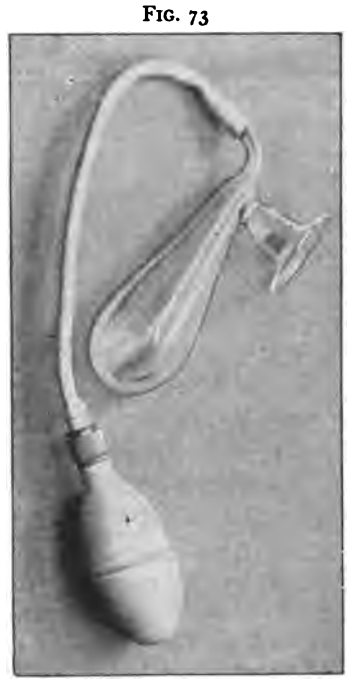
NORMAL MATERNAL CONDITIONS.—The assumption that the maternal is, when normal, the ideal source of infant food-supply presupposes many important conditions concerning the mother and the function of her mammary glands. She should be strong and healthy, of an even, happy temperament, desirous of nursing her infant, and have time to devote herself to this special duty during the whole period of her lactation. She should have a sufficient supply of milk, and should be willing to regulate her diet, her exercise and her sleep according to the rules which will best fit her for her task. These may be said to be the ideal conditions which we endeavor to obtain for an infant which is to be nursed under the most favorable circumstances. Because these ideal conditions cannot always be obtained does not mean that maternal nursing is not the best method of feeding the baby. When the failure of ideal conditions is due to such facts as that the mother is unhappy, has an uncontrollable temperament, is unwilling to nurse her infant, is hurried in the details of her life, is irregular in her diet and in her periods of rest and exercise, it does not necessarily follow that breast feeding will be a failure, or that the mother's breast milk will not be a better food for her baby than any artificial food. Under such circumstances breast feeding is not likely to be so successful as under the ideal conditions described, but nevertheless, it should always be tried, while every effort is made to remove, improve, or correct unfavorable features. At the outset, breast feeding should always be tried, unless the mother has absolutely no milk, or unless some positive contraindication for breast feeding exists.

CONTRAINDICATIONS FOR BREAST FEEDING.—The only positive contraindications for breast feeding are the following: 1. Tuberculosis of any form in the mother. 2. Epilepsy or insanity in the mother. 3. When the mother is very delicate and feeble, or is suffering from any serious chronic disease. 4. When the mother's pregnancy and labor have been attended by any serious complications, such as sepsis, hemorrhage, nephritis, or eclampsia. Syphilis is not a contraindication for the mother to nurse her own baby, but obviously should preclude her nursing any other baby.

SUCKING.—The natural method of feeding is by sucking. The infant should be placed in a comfortable position in its mother's arms, with its head and back supported. It should be made at once to understand that it is to begin its meal as soon as the breast is offered to it, and continue, of course with breathing-spells, until the meal is finished. The mother should herself preferably be sitting, as she can thus best manage and control the infant if it is inclined to be restless.

Sometimes babies are unable to nurse in the natural manner by sucking. Such inability of the infant to nurse may be caused by a condition on the part of the mother, in which the nipples are very small or depressed. Nipple shields will sometimes obviate the difficulty; when this device is employed, great care must be exercised in keeping clean the glass shield and rubber nipple. Inability to nurse may be due to certain conditions on the part of the infant, such as deformities of the sucking mechanism, great feebleness, or premature birth. In such a case, the breast pump should be employed to obtain the breast milk which the baby needs. Under the use of the breast pump, the secretion of the breasts is liable to diminish, and eventually to disappear. Sometimes it is well, if possible, to borrow a strong, healthy baby, to empty the breast once or twice a day.

FIG. 73



Breast-pump

The breast pump is an apparatus for obtaining milk by suction. A number of such devices are sold, but none is absolutely satisfactory. The main essential is to have a device which is easily kept clean.

CARE OF THE BREASTS.—The care of the breasts, if free from fissures and excoriations, requires nothing more than scrupulous cleanliness. They should be bathed before and after nursing with cold water which has been boiled. A piece of folded sterile linen or some other soft sterile absorbent material should be kept on the

nipples to protect them from contact with the clothing and to absorb any overflow from the breasts.

When the nipples are very tender and cause great discomfort to the mother during the nursing, their condition frequently becomes so serious an obstacle as to prevent nursing altogether. Any change, however, should not be thought of for at least several days, or until it is absolutely certain that the exquisite pain is more than the mother is willing or able to endure. It is often the case that after a brief continuation of great suffering from tender or excoriated nipples the whole difficulty will pass away and the mother be able to nurse her infant with comfort. Fissures of moderate severity should be treated by washing with sterile water after the nursing, and by applying boracic acid ointment, or the compound tincture of benzoin. In more severe cases nitrate of silver should be applied to the fissures, the healthy skin being protected. Care should be taken not to apply remedies which might injure the infant. Where the nipples show a tendency to be dry and hard, it is well to apply some simple ointment once or twice a day during the last few weeks of the pregnancy. Astringents, as a rule, should not be used. Bathing with cold water before and after the nursing, and thus keeping the tissues in a healthy condition, is a valuable prophylactic measure.

Another trouble which may arise during the nursing period is a disturbance of the mammary gland itself, sometimes amounting merely to a stasis in its milk production, but again going on to inflammation. The latter is a serious matter, and should at once be placed in the hands of a skilful surgeon. The former condition requires great care in its management. Gentle massage from the periphery of the gland towards the nipple, amounting in fact to merely a delicate stroking with the ends of the fingers, is an important part of the treatment. The breast should be withheld from the infant for about twenty-four hours, and the milk from time to time drawn in small quantities by means of a properly adjusted breast-pump. The breast should also be carefully supported by a swathe. If these measures are begun as soon as there are any indications of disturbance in the breast, these abnormal conditions soon disappear. The indications referred to consist in the appearance of hard swellings in place of the usual soft elastic condition of the milk glands. These swellings may occur without any especial pain, but on palpation they are usually tender to a greater or less degree.

When nursing must be temporarily interrupted on account of tender or excoriated nipples, the breast pump is indicated, or the nipple shield may first be tried.

NURSING IN THE NEWBORN.—The newborn baby should be put to the breast at the end of six to twelve hours, and every six

hours for the next twenty-four hours. After this period it should nurse every four hours for another twenty-four hours, and then should begin the regular routine which is to be followed throughout early infancy. The infant will obtain very little food during the first two days of its life, as the secretion of the breast is not yet established. The object of putting the infant regularly to the breast in these early days, is to stimulate the breast secretion. The sucking of a normal infant is the best possible stimulus to the flow of milk.

The infant does not need more food in these early hours of its life. If there were such a need, Nature would supply it. It is not necessary nor wise to give an infant sugar solution, nor artificial food during the first forty-eight hours. It does need water to flush out the kidneys, and one or two teaspoonfuls may be given every two hours. Most babies do not begin to show signs of hunger till after forty-eight hours, and at this time the breast secretion usually begins to be more profuse. If there is still delay, and very little milk in the breast, the bottle must be used temporarily, but every effort should still be made to encourage the mammary secretion, by putting the baby to the breast at proper intervals, and letting him get what he can by sucking. In the bottle feedings, care must be taken to give neither too much nor too rich food. The artificial food should be made very weak, and its strength should be increased slowly if the baby digests it well. A whey mixture is preferable for these early feedings. Two drachms may be given at first, at two-hour intervals, and this amount is increased if the baby is not satisfied.

Colostrum is supposed to have a laxative effect. If the bowels have not moved at all at the end of twenty-four hours, one teaspoonful of castor oil should be given.

MANAGEMENT OF NORMAL NURSING.—The great essential in the management of breast feeding, is to insure the most absolute *regularity* of the nursing. This must not only be recommended by the physician, but enforced. Infants rapidly tend to form habits, and when they are fed regularly, they soon get into the habit of expecting their feedings at regular times. They will then sleep more, and will only cry and show signs of hunger when their feeding is due. This gives the mother more peace, enables her to plan her work better, and under such happy conditions, she is free from nervous anxiety, and the quality of her milk remains good. Irregularity in nursing is the most common cause of disturbance in the quality of human milk. Such change in the milk tends to produce a greater or less degree of indigestion on the part of the baby. Mothers should be instructed not to nurse the baby before its regular feeding time comes due, even if it cries and seems hungry. On the other hand they must

be told to wake the baby up if it happens to be asleep at nursing time. Under such management the baby soon acquires regular habits.

THE INTERVALS BETWEEN FEEDINGS.—These are less important than regularity, provided they be not too short. There is considerable difference in opinion and practice in different parts of the country as to what the nursing intervals should be. The tendency in some localities is to use longer intervals, such as four hours. The clinical evidence brought forward in favor of long intervals is not very conclusive, nor is there evidence based on accurate scientific investigation of the emptying time of the stomach. Infants can be accustomed to longer intervals, but the process of training them is often troublesome, and there is no proved advantage to be gained. I believe the tendency toward long intervals, under present evidence, is rather a fad than a practice to be recommended. Individual variation also plays a part, some babies doing better on longer, and others on shorter intervals. The intervals which I am accustomed to recommend are the following:

TABLE 22

Intervals Between Feedings

AGE	NUMBER OF FEEDINGS IN 24 HOURS	NIGHT FEEDINGS	INTERVALS
First 4 weeks	10	1	2 hours
	or 8	1	2½ hours
4 weeks to 3 months	8	1	2½ hours
	or 7	1	3 hours
3 to 6 months	7	1	3 hours
6 to 9 months	6	0	3 hours
9 to 12 months	6	0	3 hours
	or 5	0	3 or 4 hours

The feedings, exclusive of the night feeding, are supposed to begin at 6 A. M. and end at 9 or 10 P. M. By night feedings are meant feedings given between 9 or 10 P. M. and 6 A. M. This is the only feeding at which an absolutely regular hour is not essential, but which can be given when the baby wakes up and cries. If however, the baby wakes up and cries a second time in the night, it is better to give the night feeding at a regular time in the middle of the period, about 2 A. M., in order that the baby shall not form the habit of wanting more than one night feeding. The figures showing night feedings in the table represent the maximum. Generally, the night feeding should be dropped as soon as possible at any age, and as soon as the baby

begins to sleep through the night until within one or two hours of his morning feeding, he should be accustomed to go the whole time without a night feeding. As a baby grows older, the last day feeding, or evening feeding, should also be dropped as soon as possible.

An alternative between shorter and longer intervals is given at some ages in the table. When a baby has been previously accustomed to shorter intervals, the shorter alternative should be used first, and then later the longer alternative should be substituted. With a baby previously accustomed to longer intervals, or who is under your care from the time of its birth, the longer intervals may be used from the start.

The baby should be nursed on alternate breasts at each nursing, if the mother has plenty of milk. If there is any tendency to deficiency of milk supply, both breasts should be used at every feeding after the first two morning feedings. The baby should not nurse more than twenty minutes; if he takes longer, it is a sign that something is wrong. On the other hand, if he takes his full supply in less than fifteen minutes, he should be made to take longer breathing spells. A baby gets half his meal in the first five minutes, and one-quarter more in the next five minutes. He should not go to sleep while nursing; if he does, it generally means that the baby is feeble, or that there is too little milk.

HYGIENE OF THE NURSING MOTHER.—The essentials in the hygiene of the nursing mother are, freedom from nervous excitement and worry, regular habits of life, plenty of sleep, a proper diet, and sufficient exercise.

The diet of the nursing mother should not essentially differ from what would be considered to be a suitable one for her at any time. There is no special diet which, under all circumstances, is best for all nursing women during the period of their lactation. In the early days of the puerperium there is, as a rule, more danger of overfeeding than of underfeeding the mother. A light and plentiful diet should be given to the mother while she is confined to her bed. This diet should consist of milk, gruels, soups, vegetables, bread and butter, and after the first week a small amount of meat once during the twenty-four hours. When the mother is able to go out of the house again, and has resumed her usual habits, the quality of the diet can be very much increased, and she can have the usual variety of food represented by meats, vegetables, milk, fruits, and cereals. There are no special kinds of food which are contraindicated, provided that the food be kept within the limits of the ordinary articles which commonly represent a plain but nutritious diet. It is very important for the nursing mother to have her meals at regular intervals, and during the early part of the lactation to take food somewhat more frequently

than when she is not nursing. The additional meals, as a rule, should be made up of milk or cocoa. There does not seem to be any advantage in adding any special beverages, such as beer, malt, or stimulants, to her diet. She should receive as much milk as is compatible with her digestion, and should drink a plentiful supply before retiring at night. Certain vegetables, and sometimes fish, will in individual cases affect the milk and cause discomfort to the infant. We must, then, in every case, seek to determine which article of diet may cause disturbance in the special woman's milk secretion, and eliminate that article. We should, however, be very careful not to exclude this special article of diet from the regimen of a large number of women to whom it might be of benefit rather than of harm, simply because it has affected the milk of a few women. For the average woman a plain mixed diet, with a moderate excess of fluids and proteids over what she is normally accustomed to, will, as a rule, give the best results.

Exercise has so constant an influence on the changes which take place in the daily secretion of the milk, that the mother should be encouraged to be out of bed and to walk about her room as soon after her confinement as is possible without injuring her physical condition. Exercise is so important for promoting the proper elaboration and equilibrium of the milk secretion during the entire period of lactation, that it should always be insisted upon, and regular hours for wa'king should be as definitely arranged during the day as the hours for eating. The exercise must, however, be in accordance with the strength of the special woman, for fatigue has the same deleterious influence on the production of the milk as has lack of exercise.

EVIDENCES OF NORMAL LACTATION.—In judging of the results of breast feeding in any given case, we must fully appreciate the fact that it is the equilibrium between the digestion and nutrition which constitutes success. A child may digest its milk perfectly and yet drop steadily behind in its weight development. On the other hand, it may make satisfactory gains in weight from week to week, in spite of persistent symptoms of gastric and intestinal indigestion. In either case the indications for regulation of the feeding are present.

It is desirable to keep careful records of the state of the digestion, and of the weekly gains in weight. A child in whom the equilibrium between digestion and assimilation is well established shows the unmistakable signs of good health. It is free from vomiting and colic. Its sleep is restful and regular. It is always eager to nurse and is satisfied at the end of the prescribed period of fifteen or twenty minutes. It cries only when disturbed by urination, defecation, or hunger shortly before nursing. The movements are regular, one or two a day, smooth, of a light-yellow color and mush-like consistency

and slight sour, but not foul or fecal odor. Its weekly gain in weight is regular but varies greatly, according to individual peculiarity. The importance of weight development as an indication of the nutrition of the infant is very great and furnishes us, both in breast and substitute feeding, with the most valuable evidence of all as to the success of the feeding in any particular case.

DISTURBANCES OF BREAST FEEDING

CAUSES.—In all disturbances of nutrition in infancy there is one factor which is constantly present. This is the relatively undeveloped digestive mechanism of the young infant. The degree to which the digestive power is undeveloped is not fixed and constant in all babies of the same age, but varies in different individual babies. This variation in digestive power, and in food requirement, is so constant, that it may be considered normal.

Disturbance of digestion and nutrition is due to lack of balance between the digestive power and requirement of the individual baby, and the food which is given to that baby. The two factors, peculiarity of digestive power on the part of the baby, and unsuitable composition on the part of the food, combine to produce the disturbance.

In the majority of normal breast-fed babies, the first factor plays very little part. The digestive mechanism of the baby, though undeveloped as compared with that of older children, is nevertheless fitted to digest human milk, and will digest human milk, unless for some reason the milk is particularly unsuitable in composition. Babies are seen at times, however, who fail to digest the mother's milk, even when the chemical composition of their food shows no wide variation from the normal average. In such babies we must assume that individual peculiarity of digestive power plays a considerable part. Such babies will usually digest human milk better than any artificial food. Only in very rare instances are babies seen who will digest a cow's milk modification better than human milk. Even in cases where the mother's milk does show a considerable variation from the normal average, individual peculiarity of digestive power may play a part, and the milk may be well digested by another baby.

It is best to regard this factor of individual variation in digestive power as constant and normal, and when disturbance of breast feeding is seen, to attribute such disturbance to unsuitable milk. As there is no constant standard for breast milk composition, our conclusion, in disturbance of breast feeding, is that this particular milk is not wholly suited to the digestion of this particular baby.

In seeking the cause of disturbed breast feeding, we must look for all the causes which may produce an unsuitable breast milk.

The violation of any of the requisites for normal lactation outlined

above may be the cause. The commonest is irregular habits of nursing. Worry, nervousness, excitement, overeating. constipation, insufficient sleep, insufficient exercise, menstruation, pregnancy, acute illness—any of these may be the cause. In many cases the cause cannot be found; the milk is or becomes either insufficient in quantity or quality, or abnormal in quality, without any apparent reason. In such cases, the cause is to be sought in heredity, and in the conditions of the life of our present civilization.

Several types of abnormal breast milk are particularly common. Perhaps the commonest is an over-rich milk, high in all the nutritive elements, having a composition of fat 5 or 6 per cent, sugar 8 per cent, protein 2 1/2 or 3 per cent. Such a milk is often seen in well-fed women who take insufficient exercise. Another type of abnormal breast milk is low in fat and sugar, high in protein, having a composition of approximately fat 2 per cent, sugar 5 or 6 per cent, protein 2 1/2 or 3 per cent. Such a milk is often seen in women who work hard and are underfed. A third type of abnormal milk is very low in fat and sugar, and very high in protein. Its composition approximates fat 1 to 1 1/2 per cent, sugar 3 per cent, protein 3 1/2 to 4 per cent. Such a milk is seen in "highly civilized" women with overdeveloped nervous systems.

SYMPTOMS AND DIAGNOSIS.—Disturbed breast feeding is recognized by certain symptoms. The babies may have vomiting or colic, or abnormal movements, or hunger-crying and restlessness, or constipation. Failure to gain, or loss of weight may be the chief symptom, but may be absent.

For purposes of practical diagnosis and treatment, cases of disturbed breast feeding may be divided into two classes. 1. Cases in which the trouble is due to an insufficient breast milk, and 2. cases in which the trouble is due to a bad breast milk. In the first class the milk is insufficient to meet the nutritive requirements of the baby, but does not cause symptoms of indigestion. In the second class the term bad means that the milk is so unsuited to the digestive powers of the baby as to cause symptoms of indigestion.

SYMPTOMS OF INSUFFICIENT BREAST MILK.—These are failure to gain, or loss, in weight, hunger-crying, with periods of excessive somnolence; constipation, with small stools of increased consistency, but otherwise well digested and of normal color. This set of symptoms always means insufficient milk. The milk may be deficient either in *quantity* or in *quality*, and for purposes of treatment it is important to determine which condition is present. When the baby seems satisfied after nursing, but wakes up and cries long before the next feeding time, the milk is apt to be normal in quantity, but of very weak composition. When the baby is not satisfied at a feeding, wants to nurse

a long time, finally going to sleep on the breast, the deficiency is probably in the quantity. The diagnosis however, cannot be made with any certainty on this evidence. Insufficient quantity may be recognized by the method of weighing the baby before and after nursing. No conclusion can be based on the amount of milk taken at a single nursing, but the baby must be weighed before and after each nursing for a period of twenty-four hours, and conclusions are based on the twenty-four hour quantity of milk taken by the baby. If no deficiency in quantity is found, we may assume that the deficiency is in the quality. This diagnosis can be confirmed by a chemical analysis of the milk, which usually shows a marked deficiency in one or more of the food elements.

SYMPTOMS OF UNSUITABLE BREAST MILK.—The babies show signs of indigestion, such as colic, vomiting, or abnormal stools. In mild cases they may continue to gain weight, even when these symptoms are present; in moderate cases the weight is stationary; in severe cases there is loss of weight. The existence of symptoms of indigestion is sufficient evidence for the diagnosis of unsuitable breast milk. The result of analysis may give us some idea as to which element of the milk is excessive, in proportion to the infant's digestive power, but conclusions drawn from such an analysis must not be accepted as too positive, as the peculiarity of the child's digestive power may be so great, that the element shown by analysis to be most excessive may not be the cause of the symptoms.

MANAGEMENT OF DISTURBED BREAST FEEDING.—These disturbances of breast feeding are never an indication for weaning. Every effort should be made to correct them before artificial feeding is even thought of.

The first step in the management of a case of disturbed breast feeding, is the removal of any possible cause in the hygiene of the mother. The most common cause is irregularity of nursing, and this is the cause easiest to remove. Absolute regularity must be insisted on, and any other bad nursing habit must be corrected. Every detail of the mother's daily hygiene must be scrutinized and all abnormal conditions, such as worry, excitement, improper diet, over-eating, constipation, insufficient exercise, must be removed. Some cause may be found which is not sufficient to indicate weaning, but which cannot be removed, such as menstruation.

After the hygiene of the mother has been placed on the best possible basis, a special effort should be made to affect favorably the character of the milk by other means. Such an effort will not always be successful, and the regulation of breast milk by means of diet and exercise cannot be carried to so great an extent as was formerly supposed. The following rules may be used as a guide, in such efforts:

1. *To increase the total quantity*, try to better the mother's physical condition by careful attention to all hygienic conditions. Increase the liquids in the mother's diet, moderately, not to excess. Encourage the mother to believe that she will be able to nurse her infant. Gruel is a good addition to the diet, but malt liquors do no good, and rich cocoa or chocolate should be avoided. It is probable that the special galactogogues mentioned in medical literature from time to time, such as injections of pituitary extract, or corpus luteum, or placenta, do no good.

2. *To diminish the total quantity*, restrict the fluid a little, diminish the food taken a little, and keep the bowels well open.

3. *To increase the total solids*, shorten the intervals between the nursing, and try to improve the mother's general condition in every possible way.

4. *To diminish the total solids*, lengthen the nursing intervals, and increase the food.

Additional rules are given in many text books for increasing or diminishing the particular food elements, fat, carbohydrate, and protein. Increasing the protein in the food was formerly supposed to increase the fat in the milk, but it is now believed that there is no relation between the protein in the mother's diet, and the fat in the breast secretion. If a woman is underfed, increased fat in the food will cause increased fat in the milk. If she is not underfed, the fat in her milk cannot be altered by any special dietary regulations. Too much protein in the milk may undoubtedly be caused by insufficient exercise, and also by fatigue, worry, and nervousness. In the treatment, nothing further can influence the protein than by generally correcting all unfavorable hygienic conditions. There is no way of affecting the carbohydrate.

When the disturbance of breast feeding is due to unsuitable milk, and the symptoms are those of indigestion, every effort should be made to aid the baby by relieving the symptoms with palliative measures, while waiting for the treatment of the mother to take effect. One of the simplest and most effective of these measures, is to try to dilute the breast milk at each nursing. This is done by giving the baby some diluent immediately before the feeding. Boiled water may be used, or still better, some slightly alkaline solution, for in the majority of instances, the symptoms of indigestion are due to excessive protein, and the alkalies have a favorable influence in disturbances due to protein. From two to four teaspoonfuls each of boiled water and lime water may be given immediately before each nursing, or a weak solution of sodium bicarbonate, 1 grain to the teaspoon, may be used instead of the water and lime water. If constipation is present, boiled water with about 10 drops of milk of magnesia may be used.

After all these measures have been instituted, continuance of the symptoms of indigestion may show that the breast milk is still unsuited to the baby's digestion, and that it cannot be made suitable either through regulation of the mother, or through palliative measures in the baby. In such a case, further treatment depends on whether or not the baby is gaining in weight, for many babies will gain weight on breast milk in spite of showing symptoms of indigestion. If the baby is gaining in weight, nothing further should be attempted, as the case is not severe enough to call for artificial feeding. Palliative measures should be continued, and often, as the baby grows older, its digestive power improves, or the composition of the milk improves, and the symptoms disappear. If the baby is not gaining, or is losing in weight, some artificial food must be given in addition to the breast milk. The baby must, however, not be weaned, and breast milk should still be the basis of its diet. The condition of indigestion is usually one calling for diminution of the total breast milk solids, and this is favored by lengthening the nursing intervals. The baby may now be fed at regular intervals, the breast and the bottle being used *at alternate feedings*. This tends to improve the breast milk, by lengthening the *nursing* intervals, without disturbing the child by lengthening the *feeding* intervals. Under such circumstances the breast milk frequently improves to such an extent that the bottle feedings may later be omitted. If the symptoms continue, but the baby gains weight, nothing further need be done. If in addition to the persistence of indigestion, there is no gain in weight, the physician should observe carefully whether the symptoms are most marked after the breast, or after the bottle feedings. If the bottle feedings give no lessening of the symptoms, the composition of the artificial food must be changed until improvement is obtained. If the symptoms are relieved by the bottle feedings, then for the first time, the physician should begin to consider passing over from maternal to artificial feeding. Such a change, however, must be made gradually, and the physician must assure himself that the artificial food is well digested, and sufficient to nourish the infant. Even when this is clear, the breast feedings should be dropped out one by one only, until the baby is gaining weight, and as many breast feedings as possible should be retained, in spite of their causing some indigestion. The activity of the breast should in the meantime be maintained by pumping, so that the breast milk will remain for the physician to fall back upon, if necessary. Only when it becomes clear that the breast milk is so bad that any given to the baby causes disturbance, and prevents gain in weight, should the breast be discarded. When mixed feeding is thus used in these cases of unsuitable breast milk, the selection of the first artificial food, and any

subsequent changes made in its composition, are to be guided by the principles described under Artificial Feeding.

When the disturbance of breast feeding is due to insufficiency of the breast milk, and when the regulation of conditions affecting the mother, and special efforts to increase her milk, have failed to relieve the condition, mixed feeding must be resorted to. The artificial food to be used in such a case should be that which would be given to a normal artificially fed baby of the same age, as described under Artificial Feeding. In insufficiency of breast milk, the bottle feedings should not be given to *alternate* with the breast feedings, but to *supplement* them. Short nursing intervals tend to increase the quantity and total solids of the breast milk, and the intervals must not be lengthened by alternating the breast and the bottle. If the deficiency is in the quantity, its amount may be judged by weighing the baby before and after nursing, and the amount of supplementary food to be given by the bottle at each nursing is to be regulated by the average amount of the deficiency. If the deficiency is in the quality of the milk, the baby must be allowed to take only a part of his feeding from the breast, and the amount which he ought to have should be made up from the bottle. The remaining milk in the breast may be removed by the breast pump, but the breast should not be completely emptied. However, the breast pump will never completely empty the breast. Only as much supplementary artificial food should be given as will enable the baby to gain weight. Deficiency in quality will often correct itself under this treatment, and bottle feedings may be omitted.

Deficiency in quantity may correct itself, but usually will not. As much breast milk as the baby can get should always be given, and breast feeding should be continued as long as there is any milk at all in the breasts.

WET-NURSING

A certain number of mothers are unable to nurse their infants, in spite of every effort on the part of the mother, and care on the part of the physician. In addition to the positive contraindications to maternal nursing enumerated above, many cases are seen, in which there is no milk, or in which the breast secretion soon becomes insufficient, and then disappears, or in which the breast milk, in spite of every effort, remains persistently unsuited to the child's digestive power. It is generally supposed that the mother's milk, as a rule, is more likely to be suited to her infant's digestion than the milk of another woman; but we have as yet too few cases where direct investigation by means of chemical analysis of the two kinds of milk has been made, to lay down actually as a fact what we can merely grant as a supposition, that an idiosyncrasy in the mother's milk will find an analogue in her infant's digestive powers. The reverse of this propo-

tion has also been held to be true, that at times some peculiarity in the mother's milk will make it radically unfit for her infant. The probability is that analyses will show either that these varieties of milk are poor ones, or that the infants have unusually weak digestive powers.

The fact that every mother cannot provide as good a milk for her infant as can be supplied by another woman finds its analogy in the inability of some Jersey cows to rear their own calves

When it has been demonstrated that it is impossible or inadvisable for the mother to nurse her infant, some other food must be sought. If the best possible food for the human infant is the milk secreted by the human breast, then the best possible substitute for the mother's milk, is the milk of another woman, and no imitation of human milk made by modifying the milk of an animal will approach the special characteristics of human milk.

Upon theoretical grounds therefore, and if we are obliged to take into consideration nothing more than the requirement of supplying the best food for the infant, wet-nursing would be indicated in every case in which maternal feeding cannot be used. There are, however, certain practical difficulties in wet-nursing. In the first place, in most communities, satisfactory wet-nurses are difficult to obtain, and the supply would by no means meet the demand, if every baby whose mother cannot nurse him were nourished by a wet-nurse. There are obvious inconveniences about the introduction of a wet-nurse into a family, which, while they would quickly be disregarded if the baby's welfare positively depended on a supply of human milk, are very real in many cases. Artificial feeding, while a less favorable means of nourishing babies, is free from such inconveniences, and is successful in a large proportion of cases. It is therefore becoming the custom to use artificial feeding in the majority of cases, and to save the few wet-nurses available in any community for the cases in which artificial feeding is attended by special difficulty.

In the selection of a wet-nurse, the physician should see that the woman is healthy, and free from any suspicion of tuberculosis or syphilis. She should be preferably under thirty years of age, and should be of pleasant personality. Her lungs should be examined, and her skin, glands, teeth, throat, scalp, and eyes should be carefully inspected. The nipples should be long enough to offer no obstacle to sucking. The shape and size of the breasts is no indication as to the amount of her milk. It is of no use to analyze her milk, as the normal standard of breast milk is not definite enough to permit the drawing of conclusions from its composition, as to whether it will meet the baby's requirements. The only way by which we can judge the probable quality of the milk is by the appearance of the wet-nurse's baby. The quantity can be determined by a series of weighings of the baby before and after nursing.

It is by no means as essential as was formerly supposed, that the age of the wet-nurse's baby be about the same as that of the baby she is to nurse. After the first month, the changes in the composition of breast milk, are not very important. In an infant from one to six months old, the milk of a wet-nurse whose milk is one to six months old will usually suffice.

In Boston there has been established a Wet-Nurse Directory, connected with the Infants' Hospital, of which the object is, on the one hand, to help poor mothers who are obliged to support their babies; through the Directory they can earn money, without being separated from their young infants. On the other hand, the Directory provides wet-nurses for physicians who need breast milk for their cases. The women are examined, and cared for medically by the staff of the Infants' Hospital, and those out of employment are used by the hospital. Thus a physician applying to the Directory, is supplied with a wet-nurse, in whom he can be sure of a plentiful supply of good milk, and that syphilis and all other diseases are excluded. The price paid is fixed by the Directory.

WEANING

By weaning is meant the discontinuance of breast feeding, and the substitution of some other form of nourishment. Weaning should be allowed for no other reason than for the mother's or baby's best good. It should never be permitted for the mother's convenience, nor for insufficient grounds, such as colic, cracked nipples, menstruation, or mild symptoms of indigestion.

Weaning is to be allowed under the following conditions:

1. In the normal course of things, when the baby has reached the age of between 10 and 12 months.
2. When the supply of breast milk completely gives out.
3. When the breast feeding is attended by symptoms of indigestion, which are sufficiently serious to cause continual failure to gain in weight, and when every effort to relieve this condition has failed.
4. If the mother becomes pregnant.
5. If the mother has a severe acute or chronic disease.

There is a tendency to wean babies upon insufficient grounds, especially to give up breast feeding before the breast milk has completely disappeared, or when breast milk causes comparatively mild symptoms of indigestion. Babies should not be weaned except under the conditions enumerated.

Whenever possible, weaning should be carried out gradually. Sudden weaning is apt to produce an acute disturbance of digestion. Gradual weaning may be used in all the conditions in which weaning is indicated, except pregnancy or severe illness. The method in which weaning is carried out when the supply of breast milk fails,

or when it is proved finally unsuited to the baby, has been described under the management of disturbed breast feeding. In many cases the breast milk begins to give out at the sixth month, and supplementary bottle feedings have to be used, and from this time on, weaning may have to be completed at any time.

In babies in whom there is no diminution in breast milk, weaning should be carried out between the tenth and twelfth months. With no apparent diminution in the breast milk in a child nine months old or over, a period of several weeks of stationary weight should be the signal for beginning weaning. It is not a good plan to continue breast feeding into the second year, just because the baby is doing well. The late months of lactation require close watching, as there is an increasing liability to sudden disturbance. The prejudice against weaning in hot weather is exaggerated, and babies may be safely weaned in summer, if they are given a pure, properly modified milk. Nevertheless a baby's digestive power is depressed by heat, and if a baby has just reached the age of ten months at the beginning of hot weather, I usually wait until cooler weather begins.

The gradual weaning is carried out by means of mixed feedings. First one bottle feeding is substituted for one breast feeding, and then the number of bottle feedings is gradually increased, while the breast feedings are gradually diminished, until the baby passes over wholly to the bottle. The choice of food to be given in the bottle is to be guided by the principles discussed under *Artificial Feeding*. Many well babies of ten to twelve months can be given whole milk at once. Usually at this age, it is best to begin with a dilution of whole milk with barley water, and if the baby has shown any signs of indigestion, the milk dilution must be weak. The strength of the mixture can be gradually increased until whole milk is reached.

In babies who are weaned suddenly because of pregnancy or illness in the mother, or at an earlier age because of symptoms of indigestion or failure of breast milk, the artificial food must be more dilute. Whey mixtures are usually best for very young babies.

It is sometimes difficult to get a baby accustomed to the breast, to take the bottle, especially when it has reached the age of ten to twelve months. Feeding sometimes requires much patience and time on the part of both physician and nurse. It is best to have some one other than the mother give the bottle, if possible. It is best not to waste time trying to teach infants from ten to twelve months old to take the bottle; it is better to teach them to drink from a cup, beginning with a spoon if necessary. Forcing, or too much coaxing are objectionable. If the food is offered at regular intervals, and taken away at once if refused, the babies often begin to take it after a time. The mother must be assured that the child will not starve, even if a period of thirty-six or even forty-eight hours elapses. In extreme cases, a few tube feedings may be necessary.

III. ARTIFICIAL FEEDING

PRELIMINARY CONSIDERATIONS

The resort to artificial feeding is usually a matter of necessity. It is the exception, not the rule, for an American mother to be able to feed her infant exclusively on breast-milk for the full twelve months. Supplementary feeding generally begins between the sixth and ninth months and often earlier. The fact that a mother does not nurse her infant is due not so much to her unwillingness from selfish reasons, but to the fact that she is a victim of the artificial conditions of modern life, and cannot nurse because of a deficiency either in the quantity or quality of her milk. The great majority of babies fed on substitutes for breast-milk represent cases of failure of breast feeding. Whereas, in exceptional cases, one may solve the problem by securing a competent and reliable wet-nurse, there is not in this country a sufficient number of the class from which wet-nurses are derived to meet the demand made by the enormous number of infants whose mothers, for one reason or another, cannot supply their natural food. It is, therefore, not a question as to the relative advantages of breast and artificial feeding, which must be considered, but one of how best to solve the problem of feeding, by artificial methods, the constantly increasing number of infants who must be reared on the bottle. There is little question that here in America the methods of artificial feeding have been more fully developed along scientific lines than in any other country, but the principles of the modification of cow's milk and percentage feeding have by no means been generally adopted by the profession as a whole. The scientific modification of milk, and percentage feeding, are followed for the most part by those whose practice is especially in the line of children. It is owing to the unfamiliarity of the general practitioner with these principles and their aversion to mastering the details of a somewhat difficult subject, that infant mortality in the first year has reached such alarming proportions. The introduction of the proprietary foods has not, and never will, solve the problem of substitute feeding of infants. Fresh, clean, cow's milk must be the basis of successful feeding, and the knowledge of the principles by which such milk may be modified in its composition and adapted to the varying needs of human infants, becomes of vital importance to the welfare of the race.

SOURCE OF FOOD

REQUIREMENTS.—Human milk, the food which nature provides for infants, contains certain food elements, which we must assume to be necessary. These food elements are fats, carbohydrates, proteins, and certain mineral salts. The digestion of the infant being fitted to utilize these food elements, any artificial food must contain the same food elements. The fat, sugar, protein, and salts should correspond as closely as possible chemically to the same elements as found in human milk, and should correspond also in their relative proportions. The food which contains these elements in a form most closely resembling those of human milk in their chemistry and proportions is the mammary secretion of another animal. The only animal milk which can be obtained in sufficient quantity for use as a food for infants is the milk of the cow.

Further requirements, are that the food must be fresh, clean, free from injurious bacteria, that the number of bacteria be not excessive, that it be free from preservatives, and that it be not skimmed nor diluted.

COW'S MILK. THE COW.—The breeds of cows which are considered most desirable for the general purposes of milk production are not the best for use in infant feeding. The most prized breeds are those which give the richest milk, such as the Jersey and Guernsey cows. These breeds yield a milk which contains a greater quantity of fat than the commoner breeds, such as the Holstein. Moreover the fat globules in these rich milks are larger than are the globules in the milks from the commoner breeds. One feature in which cow's milk differs most markedly from human milk is the chemical composition of the fats. Cow's milk contains a greater proportion of the volatile fatty acids than does human milk, and this difference is greater in Jersey milk than in the milk of the commoner breeds. Many infants who cannot digest Jersey milk in any modification, can take the milk of the commoner kinds of cows.

There is a theory prevalent in some communities, that the milk of one cow is preferable for the nutrition of an infant than the milk of a mixed herd. This is a false theory, and indeed, quite the contrary is true. The milk of a mixed herd of cows is much more stable in its composition, showing much less variation from day to day, than the milk of a single cow. Also, any single cow is liable to some sudden upset which changes the composition of her milk. An upset in one cow does not manifest itself to any great extent in the mixed milk of the whole herd.

COMPOSITION OF COW'S MILK COMPARED WITH HUMAN MILK.—Cow's milk, like human milk, contains fat in the form of an emulsion, and carbohydrate, protein, and mineral salts in solution. The milk

varies in its composition just as does human milk. The variations depend upon the breed of the cow, the methods of feeding, the health of the animal, the season of the year, the stage of lactation, the length of the interval between milkings, the portion of the milk withdrawn, and many other conditions.

The analysis of an average good cow's milk is shown in the table, compared with the average human milk.

TABLE 23

Average Cow's Milk Compared with Human Milk

	COW'S MILK	HUMAN MILK
Specific gravity	1.028 to 1.032	1.030 to 1.032
Reaction	Amphoteric or slightly acid	Amphoteric
Fat	4.00 per cent	4.00 per cent
Lactose	4.75 per cent	7.00 per cent
Protein	3.50 per cent	1.50 per cent
Salts	0.70 per cent	0.20 per cent
Water	87.05 per cent	87.30 per cent

Fat is contained in cow's milk in about the same average amount as in human milk. There is, however, a marked difference in the chemistry of the fat. In both, the greater part of the fat is in the form of neutral fat, although oleic acid is in greater quantity in human milk. The chief difference is that cow's milk contains a greater proportion of the lower or volatile fatty acids than does human milk. This difference is very important, because it cannot be corrected by any known method of cow's milk modification. The fat globules, also, are slightly larger in cow's milk.

The carbohydrate in both milks is lactose. The only difference is quantitative, cow's milk containing considerably less than human milk.

The protein of cow's milk shows both quantitative and qualitative differences, as compared with human milk. The quantity of protein is much greater in cow's milk, averaging 3.50 per cent. The proportion of casein to the soluble whey proteids (chiefly lactalbumin) is very much greater. The separation of the different forms of protein is so difficult that chemists are by no means agreed as to the relative proportions of lactalbumin and casein in either human milk or cow's milk. In general, the whey protein and casein in human milk are about equal, or possibly, the soluble protein is in excess. On the other hand, it is generally agreed that in cow's milk the casein is in excess. Some writers place the proportion of casein to whey protein in cow's milk at 4 to 1, others at 3 to 1. The casein of cow's milk is much more coagulable than is that of human milk. Whereas the human casein is only slightly coagulated with acids, and not regularly by rennet plus acid, the cow's casein is readily coagulated both with acids, and with rennet plus acid. The curd formed in gastric digestion

is tough, and firm, and is much less readily dissolved by the gastric juice.

The inorganic salts of cow's milk also show differences as compared with human milk. Their total quantity is more than three times greater than in human milk. There are great differences in the amounts of the various salts in the two milks. The accompanying table was compiled by averaging the observations of a number of writers on this subject.

TABLE 24

Composition of the Ash in Human and Cow's Milk, in per cent (grams per 100 grams)

	COW'S	WOMAN'S
Potassium oxide	.1720	.0750
Sodium oxide	.0431	.0217
Calcium oxide	.2117	.0346
Magnesium oxide	.0284	.0063
Ferric oxide	.0010	.0005
Phosphoric acid	.2513	.0471
Chlorine	.0949	.1428

While all the salts are in larger percentage in cow's milk than in human milk, the relative proportions of the different salts differ greatly. In general cow's milk contains relatively a very large amount of calcium phosphate, while the proportion of potassium salts and iron in cow's milk as compared with human milk is relatively small. There is a great difference in the form in which phosphorus is present in human and in cow's milk. In human milk three-quarters of the phosphorus is in organic combination, while in cow's milk only one-quarter is in organic combination. The iron in neither human milk nor in cow's milk is sufficient to meet the demands in the first year of life; the infant must depend on the iron stored during fetal life.

BACTERIOLOGY OF COW'S MILK.—It is impossible to obtain cow's milk free from bacteria, nor even as relatively free from bacteria as is human milk. Our efforts must be directed toward obtaining milk which is wholly free from pathogenic bacteria and which is as free as possible from the other forms of bacteria not usually harmful.

Of the pathogenic bacteria transmitted in milk, the most important is the tubercle bacillus. This organism has been frequently found in milk supplies, and comes from cows diseased with tuberculosis. Opinions may differ as to the exact amount of the danger of acquiring tuberculosis through milk, but there is no doubt that contaminated milk is a possible source of tuberculosis. I believe that the sale of milk from cows showing on physical examination evidences of tuberculosis should be forbidden by law. Whether or not the sale of milk from cows reacting to the tuberculin test should be forbidden is still an open question. I should advise, for use in infant feeding, only milk from tuberculin tested cows having negative reactions.

Streptococci and other pyogenic organisms may be found in the milk of cows having disease of the udder. These organisms have been shown to be the cause in some cases of gastro-enteric disease. The milk supply should be obtained from a source in which milk from cows diseased in this way is not used.

Certain recognized infectious diseases have been traced to milk infection. The diseases known to have been transmitted in this way are, in the order of frequency, typhoid fever, scarlet fever, diphtheria, dysentery, and cholera. The reason that dysentery occupies so low a place in this list, is that the order is based on the frequency with which the infection has been actually traced through the milk. Contaminated milk is probably the usual source of infection with the bacillus of dysentery, but the connection with a definite case of dysentery is less direct, and more difficult to trace.

The other bacteria found in milk come from a variety of sources, entering the milk chiefly during the operation of milking. They may come from the hands and clothing of the milker, from the dirt falling from the cow, from the dust of the stable, and from many similar sources. A great variety of organisms are found in milk. They are for the most part harmless, and it is possible that some of them exercise a protective action by keeping out more harmful forms. Most of them belong to the lactic-acid-forming group, and are concerned in the souring of milk. In too great numbers, it is probable that these organisms may produce changes in the milk which may cause harm to the infant.

CERTIFIED MILK.—In any locality, the physician should use in infant feeding, the best milk supply which can be obtained. Through the concerted efforts of physicians and others interested in the questions of public health and infant mortality, attempts have been made in many parts of North America to improve the milk supply. The means used have been various; in some places more or less stringent laws have been passed regulating the conditions of milk production and distribution; in other places milk commissions have voluntarily undertaken to inspect milk supplies, and certify milks which fulfil their requirements, the community being informed of the advantages of milk so certified. In the inspection of milk, one of the chief criteria by which milk supplies are judged, is the count of the number of bacteria contained in the milk. The bacterial count is valuable, not so much because a high bacterial count is necessarily a sign that the milk will harm the baby, as because it is a guide to the general cleanliness of the conditions under which the milk is produced.

Every physician interested in the subject of infant feeding, should be familiar with the present status of pure milk production. The details of this subject are too voluminous to be incorporated in a

text book of this kind, and the reader is referred to the reports of the various milk commissions.

In practice, a physician should recommend the use of certified milk whenever possible. When certified milk cannot be obtained, he should be familiar with the available milk supplies in his locality, and with the conditions under which each milk is produced, and should recommend the best.

STERILIZATION AND PASTEURIZATION OF MILK.—Cow's milk may be heated in preparation for its use as a food for infants. The object of heating is to lessen the danger of damage to the infant from the bacteria contained in the milk. The term "sterilization" is generally used to designate the heating of milk to a boiling temperature, while the term "pasteurization" is used to designate the process of heating at a temperature lower than boiling. Neither term is satisfactory. Milk is not rendered bacteriologically sterile by the process of boiling ordinarily employed, and pasteurization is a very vague term, unless it is definitely stated at what temperature and for how long the milk is heated.

THE EFFECT OF HEAT ON THE BACTERIOLOGY OF MILK.—The heating of milk at a temperature of 140° F. (60° C.) for twenty minutes will destroy the recognized pathogenic non-spore-bearing organisms, such as the bacilli of typhoid, diphtheria, and dysentery, and the vibrio of cholera. Heating at a higher temperature will destroy these organisms in a shorter time. Many of the organisms of putrefaction are spore-bearing, and their spores are highly resistant to heat; some of them have been shown to resist boiling for one hour.

Many of the lactic-acid-forming organisms are destroyed by heat, and it has been very generally believed that their destruction favors the development in heated milk of proteolytic spore-bearing organisms, which may form highly toxic products. Ayers and Johnson, however, have recently shown that many acid-forming organisms are not destroyed below 168° F. (75.6° C.) and that in milk pasteurized at a temperature lower than this, the normal process of souring takes place, though somewhat delayed. No difference was found in the relative numbers of the acid-forming, and proteolytic groups in pasteurized milk, from that seen in clean raw milk, nor in the rate of multiplication. There is at present no definite evidence to prove that the heating of milk favors the growth of any harmful organisms.

The general belief is that the heating of milk has no effect on any bacterial toxins which may already have been produced. This is only partly true. True soluble bacterial toxins are thermolabile, many of them being destroyed at a temperature of 140° F. (60° C.). The endotoxins are for the most part resistant to heat.

The general effect of heating milk at 140° F. (60° C.) or over, may be summarized as follows: The most common pathogenic spore-bearing organisms, including those of specific infections, are destroyed, and the total number of bacteria is lessened. In other words, heating brings milk bacteriologically into the same condition as raw milk produced under the most careful and cleanly conditions. The subsequent development of toxin-forming spore-bearing organisms is possible, if the milk is not kept at a temperature sufficiently cold to inhibit their growth.

THE EFFECT OF HEAT ON THE COMPOSITION OF MILK.—The boiling of milk produces a scum upon the surface, and alters the taste and smell of the milk. These changes are not marked at a temperature lower than 158° F. (70° C.). Prolonged boiling changes the color to brown from caramelization of the sugar. Heating at 150° F. (65° C.) for over thirty minutes delays or prevents the rising of the cream.

When milk is boiled, some of the mineral salts are precipitated, and the inorganic phosphorus is increased at the expense of the organic compounds. Certain changes are produced in the chemistry of the casein, through which it becomes less easy of coagulation by rennet plus acid, and less readily acted on by the proteolytic digestive ferments. The curd formed in gastric digestion is softer and more flocculent. The soluble lactalbumin is entirely precipitated.

The data as to the temperature at which the precipitation of the soluble albumin begins are not in agreement, and there are no satisfactory data as to the temperature at which the other changes begin. Hippius concludes that heating at 149° F. (65° C.) for thirty minutes produces no notable changes in the chemical composition of cow's milk, and the conclusion is probably not far wrong.

The evidence as to the effect of heat on the ferments of cow's milk is very inconclusive. It is probable that some of them are destroyed by heat, while it has been shown that others resist various temperatures.

THE EFFECT OF HEAT ON THE DIGESTIBILITY AND FOOD VALUE OF COW'S MILK.—The more or less general belief in this country has been that babies fed continuously on cooked milk do not thrive as well as babies fed on raw milk. Recently there has been a tendency to examine more critically the evidence on which this assumption is based, with the result that more or less doubt has been thrown upon its truth. The evidence has been derived from three sorts of experiments, first, in artificial digestion, second, on animals, and third, on babies. Artificial digestion experiments have given contradictory results, some writers concluding that cooking increases, and others that it diminishes, the digestibility of milk. Animal

experiments are mainly in agreement that young animals thrive better on the raw than on the cooked milk of their own species. The experiments on babies have been for the most part clinical and statistical, consisting of comparing the clinical results of raw and cooked milk in the feeding of large series of babies. The results with cooked as compared with raw human milk resemble the results in animals, that the babies do better on the raw milk. The results with raw and cooked cow's milk are open to the objection that clinical and statistical evidence of this kind is apt to be misleading. In general, the majority of investigators have failed to detect any difference in the digestibility and food value of cooked and raw milk.

As to the particular diseases of nutrition, there is no conclusive evidence that rachitis is more frequent in babies fed on cooked milk. Scorbutus, on the other hand, has been more generally associated with the use of cooked food. The evidence in favor of the connection between scurvy and the heating of milk is much stronger than any other evidence as to the value of heated milk. The evidence, however, is almost wholly of a clinical and statistical rather than an experimental nature, and evidence of this kind can never be conclusive. In general, the evidence is that in all large series of cases of scorbutus, a considerable proportion of the patients have been fed on heated milk, more often on milk which has been boiled or scalded than simply pasteurized. On the other hand scurvy develops at times in babies fed on raw milk, and even in breast-fed babies. This evidence will be considered at greater length when scorbutus is described. That the heating of milk may be a more or less important cause of scorbutus is possible if not probable, but is not proven.

STERILIZATION OR PASTEURIZATION.—The higher the temperature at which milk is heated, the greater are the changes of chemical composition produced. While it is still an open question how great an effect these changes have on the well-being of the infant, it is obviously better to avoid them if possible. The object of heating the milk is to produce the destruction of certain pathogenic organisms, and a diminution in the total number of bacteria. It has been shown that this result can be attained by subjecting the milk to a much lower degree of heat than the boiling temperature. Therefore pasteurization should be preferred to sterilization. The temperature of the pasteurization should be as low as is consistent with the object to be attained. Pasteurization at a temperature lower than 140° F. (60° C.) is not efficient. At this temperature there is no change in the color, taste, or odor of the milk, and but little change in chemical composition; most ferments and the bactericidal action of milk, are unaffected, while most bacterial toxins and all the patho-

genic non-spore-bearing organisms are destroyed. Therefore, when heating is indicated, pasteurization at 140° F. (60° C.) for twenty minutes should be chosen.

INDICATIONS FOR PASTEURIZATION.—Whether or not milk should be pasteurized is a question which must be decided in each individual case. Three factors must be considered in arriving at a decision: 1, the source of the milk supply; 2, the season of the year; and 3, the time taken in delivering the milk. With every source of milk supply except the very cleanest, pasteurization should be employed as a routine measure. With such a clean milk supply as, for instance, certified milk, pasteurization should be employed whenever the milk has to be transported for any considerable distance, or when considerable time elapses between milking and delivery. In summer it is always safer to pasteurize, whatever the milk supply; occasionally an exception can be made when a milk is used which is known to be produced and delivered under the most perfect conditions. In general, pasteurization should be employed as a routine measure, except with the cleanest milk supply, delivered under the most favorable conditions.

When an infant does not seem to thrive on pasteurized milk, it must not be at once assumed that the heating of the milk is the cause. Only when other measures have been thoroughly tried, the pasteurization may be omitted for a time as an experiment. The only risk supported by any real evidence in the use of pasteurized milk is the possible development of scorbutus. Scorbutus is a much less serious danger, than is bacterial infection or intoxication, and if it develops, it is easily cured.

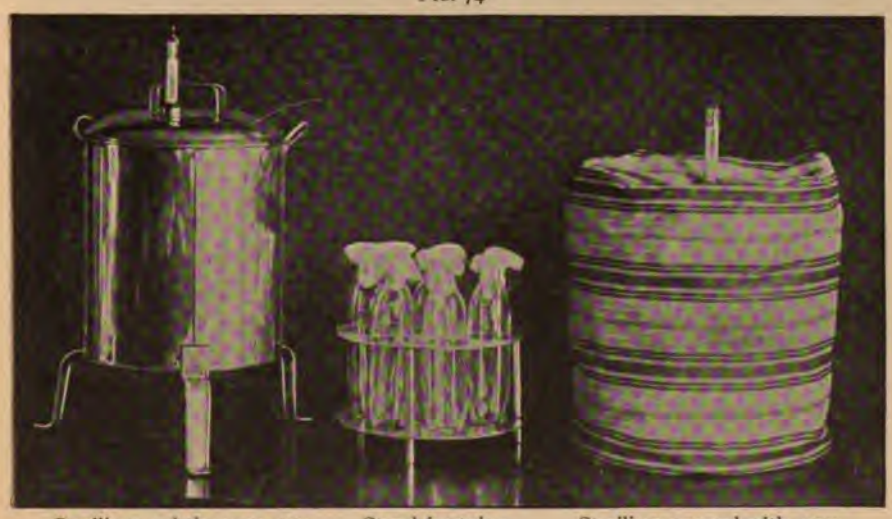
TECHNIQUE OF PASTEURIZATION.—It is better to pasteurize milk for each feeding in a separate bottle, than to pasteurize the whole twenty-four hours' food supply in one large receptacle. It is difficult to maintain so large a quantity of milk at the desired temperature for such a length of time as twenty or thirty minutes, and the results bacteriologically have been shown to be not as good as when the milk is pasteurized in smaller quantities.

Each feeding should be put in a separate clean bottle which has been boiled, and the bottles should be stoppered with non-absorbent cotton. The bottles are then placed in a dish, which is filled with cold water up to a level with the milk in the bottles. The dish is then placed on the stove and heated until the thermometer suspended in the water reaches 145° F. (62.7° C.). The dish with the contents is then removed from the stove, covered with a blanket, and allowed to stand for thirty minutes. At the end of this time the bottles are taken out and quickly cooled in running tap water, after which they are placed on ice, or in a cold place, until used.

The pasteurizers on the market, designed for home use, are all good. They are somewhat more convenient, though not more efficient, than the method described.

THE EXAMINATION OF MILK.—It is often important or desirable for the physician to determine the composition of milk. In the case of human milk, the analysis occasionally throws some light on the cause in disturbed breast feeding. In the case of cow's milk, it is often important to determine how closely a certain milk used in modifying approaches in composition the milk taken as the basis for calculating percentages. A thorough and accurate analysis of milk

FIG. 74



Sterilizer and thermometer | Stand for tubes | Sterilizer covered with cozy after removal from heat

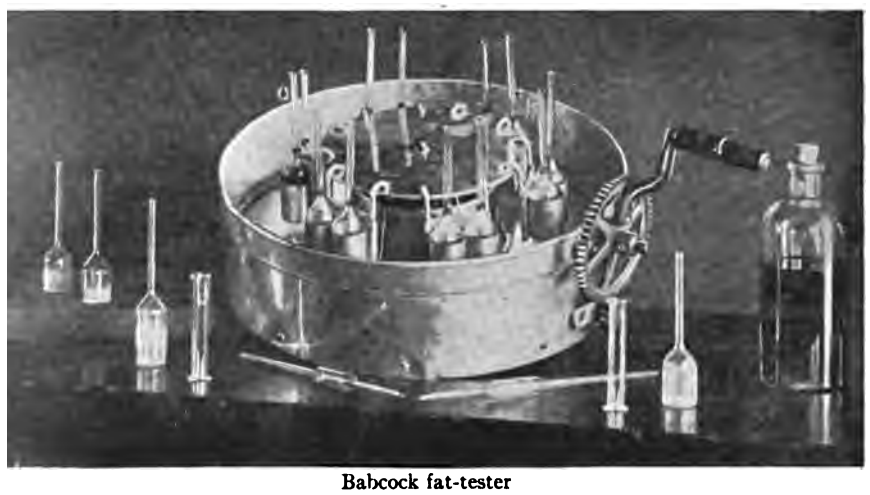
can only be carried out by an expert chemist. When an expert chemist is not available, the physician himself can frequently carry out an analysis of clinical value. The method of analysis is the same for both human milk and cow's milk.

THE SPECIFIC GRAVITY is obtained by means of any ordinary hydrometer, graduated from 1.010 to 1.040.

THE FAT.—The estimation of the fat is the most important in clinical milk analysis. The fat can be measured with practically absolute accuracy by means of the Babcock Fat-Tester, or by some similar centrifugal apparatus. Most institutions, and many physicians treating many infants, keep this instrument at hand. Its operation is very simple. To a given volume of milk is added a certain amount of sulphuric acid, the quantities varying with the exact model of the machine used. Pipettes for measuring the milk and

acid are sold with the machine. The milk and acid are mixed in a test-bottle with a long graduated neck. The bottles are then whirled in the centrifugal machine at a high speed for five minutes. Hot water is then added to bring the mixture up to the neck of the bottle, and then the bottle is centrifuged for two minutes. Hot water is again added to bring the separated fat up into the graduated neck of the bottle, the machine is again whirled for a short time, and the length of the column of separated fat, as measured on the graduated bottle-neck, is read, the reading giving the per cent of fat in the mixture.

FIG. 75



Babcock fat-tester

For those who have no Babcock machine at hand, a simpler method of estimation, of approximate accuracy, is by means of Holt's cream guage. This instrument is simply a graduated stoppered tube, which is filled with fresh milk to the zero mark, and is then allowed to stand, corked, at room temperature for twenty-four hours. The reading is taken from the cream line, and three-fifths of this reading is taken as the percentage of fat in the milk.

THE PROTEIN.—No chemical method of estimating the protein is very satisfactory. The most accurate is that in which the protein is precipitated in the Esbach tube by a solution of phosphotungstic and hydrochloric acids, the reading being taken at the end of twenty-four hours. An approximate idea as to whether the protein is high or low may be found by considering the known specific gravity in connection with the known percentage of fat. In this estimation we assume that the percentages of sugar and mineral salts in milk are constant; while not constant, these percentages are more constant than those of the other elements. The way in which this approximate conclusion as to protein is made is shown in the table:

TABLE 25

Approximate Estimation of Protein from Known Fat and Specific Gravity

SPECIFIC GRAVITY	FAT		PROTEIN
High	High	=	High
High	Low	=	Normal
Low	High	=	Normal
Low	Low	=	Low

THE CARBOHYDRATE AND SALTS.—These elements of milk cannot be estimated by any simple chemical method. If examination by an expert chemist is not necessary or available, they may be considered constant.

THE MICROSCOPIC EXAMINATION OF MILK.—This should be part of the routine in the examination of a specimen of milk. Both the cream and the centrifugalized sediment should be examined. In the cream one should look for colostrum corpuscles. In the sediment one should look for dirt, pus, and blood. A few leucocytes are normal, but any excess of dirt, pus, or blood, should cause us to reject the milk. If pus be present, the sediment should be stained for bacteria, and streptococci should be sought.

THE MODIFICATION OF COW'S MILK

We must now consider the most important part of the subject of the artificial feeding of infants, which concerns the adaptation of cow's milk to the nutritive requirements of the infant. While cow's milk is the only available food which in composition at all approaches the food designed by nature, it is very far from being an ideal baby food. We have seen how greatly its chemical composition differs from that of human milk. That these differences are a very frequent cause of disturbances of digestion and nutrition is a fact proved by years of clinical experience. The existence of such disturbances in babies fed upon cow's milk, has led to a great number of investigations which have been made with the object of discovering whether there are any means by which the digestibility and nutritive value of cow's milk as a food for infants may be increased.

THE SOURCES OF DIFFICULTY IN ARTIFICIAL FEEDING.—There are two factors responsible for the difficulties encountered in the artificial feeding of infants. The first is to be found in the chemical composition of cow's milk, which makes it a less suitable food for infants than is human milk. This may be called the chemical difficulty. The second is to be found in the variation in the digestive powers and nutritive requirements of different babies. This may be called the individual difficulty. The relative importance of these two factors has been very largely misjudged, not only by the public, but even by the physicians engaged in the very study of the feeding problem. Only in this country has the individual factor in infant

feeding been appreciated, and then only by comparatively few physicians. In Europe the entire attention of investigators has been centered upon the chemical factor, and the individual factor has been, and still is, almost entirely neglected, yet it is the individual factor which is really the more important practical source of difficulty in artificial feeding.

One great obstacle to advance in our knowledge of the principles of artificial feeding, has been the generally prevalent idea, existing not only in the minds of the public in general, but even in the minds of physicians and of specialists in infant feeding, that there is somewhere, yet to be discovered, a *baby food* of transcendent value which will solve the problem of infant feeding, and which can be fed successfully to babies in general. It is possible that in the minds of those who have studied the subject, this idea takes a little different form. Such men will look upon one method of modifying cow's milk as better than another method. To regard one method of modifying cow's milk as better than another, must mean that it is regarded as better for *babies in general*, and that it is the best method which has been found. This opinion carries with it the idea that there is a best method of modifying cow's milk, and that is only the ideal baby food expressed in other words.

The nature of the feeding problem is such that an ideal baby food can never be attained. The factor of individual variation plays too great a part, and we shall never be able to feed babies as a class, nor to find a food or method which will be best for babies in general. The modification of cow's milk which is best for one baby will always be worst for some other.

Great advances have been made from time to time in the attempts to solve the chemical difficulty of infant feeding. The most careful and painstaking researches on the physiology and pathology of digestion in infancy, on infantile metabolism, and on the effect upon digestion and metabolism of various methods of modifying cow's milk have been made. These investigations have given us additions to the sum of our knowledge of these subjects, the value of which cannot be overestimated. Usually the results of each research of this kind has suggested some new practical method by which cow's milk can be modified to greater advantage. There has been an unfortunate tendency to hail each new method of modifying milk suggested by researches of this kind, as the best, if not the ideal method of feeding babies. The new method is regarded not as ranking equally in value with previous methods, but as being a step in advance which will supersede older methods in the feeding of babies. Thus we have passed through successive stages in which modification with whey, with cereal diluents, with various alkalies, with maltose, with lactic acid ferment, with precipitated casein has each in turn been regarded

as a better method of modifying cow's milk than its predecessors. Even if the particular investigator has not himself regarded his addition to the methods of milk modification as a new method of feeding which shall supersede others, the readers of his reports have so regarded it. The way in which the results in practice of any new method of modifying milk have been studied and reported has been such as to perpetuate the idea of its superior value. The new method of feeding is applied to a large series of babies, the clinical results are studied, and a statistical report is published. Such reports are very misleading, because, studied in this way, every new method of modifying milk gives good statistical results. The individual variation in babies is so great, that in any series a large number of babies will do well, and a considerable number of babies will do better with the new method of feeding than with any other.

This tendency to overestimate the value of the new methods of modifying milk suggested from time to time by research, and to apply them to the feeding of babies in general, has been most unfortunate. There has never been any logical ground for believing that any one of them is better for babies as a class than any other. Each method is based on some actual discovered fact in infantile digestion and metabolism. Each older method has been in its day just as well supported by clinical and statistical evidence as is the new method. The old facts, if brought out by proper research, remain, and the newly discovered facts are only additional, but do not contradict the older ones. There are babies who do better on one method, and babies who do better on another. We should not discard any method of modifying milk based on some discovered fact about the infantile digestion, simply because some additional fact has been discovered which suggests a different method. *We should regard every method of modifying milk based upon adequately demonstrated facts concerning the digestion and metabolism of the infant, as having its own particular value, as being particularly fitted to meet the digestive peculiarities of individual babies. We should regard all such methods as constituting our stock of weapons, which we need to combat the many difficulties springing from the peculiar chemistry of cow's milk, and from the limitless variation in the baby's digestive powers and requirements. We should hail each new research and the practical application to feeding suggested by it, not as a new or better method of feeding babies, but as a new addition to our stock of weapons, a new resource, which will have its particular use in a problem, the nature of which is such, that we need all the resources at present available, and more.*

CALORIC REQUIREMENTS AND DIGESTIVE REQUIREMENTS.—One of the most important efforts to devise a method of feeding babies in general, based upon research, is that which is some-

times called the *caloric* method of feeding. This is based on the so-called minimum caloric requirement of a normal baby. A number of infants who were thriving and gaining normally in weight were investigated, and the energy value of the food taken each twenty-four hours was estimated in calories. The quantity of food taken varied with the weight of the baby, and the food required to maintain proper nutrition was measured in terms of calories per kilogram of body weight. From the averages of large series of babies, was deduced the minimum caloric requirement, which represents the minimum number of calories per kilogram of body weight which each baby must receive daily in order to thrive.

THE MINIMUM CALORIC REQUIREMENTS.—As estimated in this way the minimum caloric requirement is as follows:

TABLE 26

Minimum Caloric Requirement

Birth– 6 months	120 cal. per kilo
6–12 months	100 cal. per kilo
12–24 months	90 cal. per kilo

PROTEIN REQUIREMENT.—Protein is that element of the food which is used in the building up of the body tissues, while it is the fats and carbohydrates which are concerned with energy production. From metabolism experiments has been deduced a protein requirement for the normal baby. The protein requirement thus established is open to the same objections as caloric requirement, as it is based on the method of averaging variations. I believe, however, that variations are not so extreme, and that the protein requirement is often a useful guide. It is from 1.0 to 1.5 grammes of protein daily per kilogram of body weight. Most milk modifications, unless extremely low in protein, more than fulfil this requirement.

CALORIC REQUIREMENT AS A BASIS FOR FEEDING.—This study of caloric requirement led to the idea that here at least was a definite and satisfactory basis for the artificial feeding of babies. Under this system the physician reckons the minimum daily caloric requirement from the weight of the baby, and then chooses the food with the primary object of at least meeting this requirement. The amount of fat, carbohydrate, and protein is designed to produce the requisite calories. In case of indigestion, if one element in the food must be reduced, the others must be proportionately increased, in order to maintain the caloric value of the food.

Caloric requirement as a basis for feeding,—a caloric method of feeding—is open to numerous very serious objections. In the first place, the figures for minimum caloric requirement were obtained by the method of averaging a given number of observations, which

method gives a very misleading result whenever individual variation plays much part. The caloric requirement even of normal babies has been shown to be very variable. This variation is still more extreme in sick babies, or with babies in poor condition. Moreover, caloric requirement varies on account of a number of other factors, such as the surface area, and the activity of the baby. Finally, and most important of all, the nutrition of the baby depends upon the quantity of food assimilated, not upon the quantity ingested, and the net caloric value of the food cannot be estimated. In babies with deficient power of digestion, less food is absorbed and utilized, and indigestion does more harm in preventing a child from thriving than anything else. Babies cannot be fed as a class on a caloric basis, because the variation in their power of digesting and absorbing food is too great. Clinical experience has shown the widest extremes, some babies gaining weight on a caloric intake far below the so-called minimum, and others failing to gain on a caloric intake far in excess.

DIGESTIVE REQUIREMENT.—In choosing an artificial food for a baby, our first aim must always be to give the baby a food suited to its power of digestion. Our principal difficulty is in meeting variation in digestive power. We are also confronted by a variation in caloric requirement. Consequently we must assume that every baby has digestive and caloric requirements such that some particular combination of the food elements in milk, modified in some particular way, will be the best food for that baby. Our problem is to find that combination, or at least, if not the best combination, one that will be suitable. To solve this problem, we must consider first all the chemical difficulties in digesting cow's milk, and the methods of milk modification which have been devised to solve these difficulties.

Of course the great majority of babies have not such defective powers of digestion as call for any special measures. For such babies, certain general rules for the modification of cow's milk in the preparation of their food, can be stated. The difficult cases call for all our knowledge, and all our resources.

VALUE OF CALORIC ESTIMATION.—To estimate the caloric value of an infant's food, and to compare it with the theoretical minimum caloric requirement, is a procedure which is often useful in the conduct of a case where artificial feeding is employed. Sometimes, for instance, we may be in doubt whether failure to gain in weight is due to defective digestion and assimilation, or to insufficient food ingestion. In such a case, caloric estimation may be a great help. Always, however, the calculation of calories should be used as a check, not as our principal guide.

PERCENTAGE FEEDING.—The world owes to Professor Thomas Morgan Rotch a great debt, for elaborating the theory of *percentage feeding*, which is now very generally accepted by the leading teachers and writers in America as the basis of the modern scientific feeding of infants. Percentage feeding is often wrongly spoken of as if it were some special method of feeding infants, which is considered by its users as superior to other so-called methods. Those who thus misjudge the theory appear to think that it involves some particular formula or set of formulae for the feeding of infants, some special combination of percentages of superior value.

Percentage feeding is not at all a method of feeding in the sense that it chooses any particular method of modifying the composition of the infant's food. It is primarily simply a *method of recording* the characteristics of the food given to the infant. Instead of recording the food in terms of mixtures of various ingredients such as cream, milk, lactose, barley water, and so forth, the food is *recorded in terms of the percentage of the various elements:* fat, protein, lactose, maltose, or starch, etc. contained in these mixtures. Even when a baby is fed on a simple dilution of cow's milk, the record is made in terms of the percentage of fat, lactose, and protein contained therein.

The advantage of such a system of record is that it gives a common terminology in infant feeding, and a common basis by which various methods of modifying milk may be compared, both as to theory, and as to practical results. A physician may feed a baby, for example, on a mixture of milk and barley water, and change the food from time to time by gradually increasing the milk and diminishing the barley water. If he thinks of his food in terms of the relative ounces of milk and barley water, he has no way of comparing his method of modifying milk with other methods; and, if he publishes his results, they will have no value to other physicians until they have estimated just what quantity of the various food elements the baby was getting in that food. To advance in our knowledge of infant feeding, we must be able to compare our results one with another, and must also be able to compare our work with the published work of others. Such a comparison necessitates a common basis, a universal manner of record. It is the tolerance or intolerance of the infant for the various food elements, which is the final result in infant feeding. Therefore we must know the amount of *each food element* given, as recorded in percentage form.

As record in terms of percentages became customary, physicians naturally came to think of foods as combinations of percentages of the essential elements, rather than in terms of mixtures of ingredients. The essential thing in choosing a food for an infant is to arrange the quantity and kind of fat, sugar, and protein in such a way as to meet the digestive and nutritive requirements of the

infant. As our knowledge of digestion and nutritive requirements is derived from percentage records, we have come to think first of the percentages we wish to give, and which we record. Then we give the proper directions for obtaining and preparing the food.

THE MODIFICATION OF COW'S MILK—THEORY

To utilize all the resources of artificial feeding, the physician must be familiar with every method of modifying cow's milk which is "in good use." Those methods are in good use which are based on adequately proven facts, or on plausible theories, regarding the infantile digestion and metabolism, or which are supported by accepted clinical evidence. In some methods a combination of two or more principles is used. The methods of modifying milk depending on a single principle, which are at present in good use, are ten in number. They are the following:

1. The dilution of milk or cream with the addition of lactose.
2. The addition of starch to cow's milk mixtures.
3. The addition of alkalies to cow's milk mixtures. Of the alkalies there are three—
   a. Lime water.
   b. Sodium bicarbonate.
   c. Sodium citrate.*
4. The peptonization of cow's milk mixtures.
5. The addition of whey to cream dilutions—the so-called "split protein."
6. The addition of other soluble carbohydrates than lactose to cow's milk mixtures.
7. The addition of the lactic acid ferment to cow's milk mixtures.
8. The addition to the food of precipitated casein.
9. The cooking of cow's milk mixtures.
10. The use of "homogenized" milk mixtures.

These various methods may be combined in any variety of ways. A cow's milk dilution may be peptonized, or have lime water or barley water added. Two combinations of principles have been so widely known as methods of modifying milk, that they deserve special mention. They are—

1. "Malt soup" modifications, a combination of maltose, starch, and cooking.
2. Albumin milk, a combination of lactic acid milk and precipitated casein.

In order to use to best advantage these various methods of modifying milk, it is necessary to know the scientific basis of each one. In

* It may be objected that sodium citrate is not really an alkali. In the physiology of digestion it acts in a manner so like the lime water and sodium bicarbonate, that it is most conveniently classified with them.

other words, we must know exactly how each method of modifying affects the composition of cow's milk, what effect such modification of the composition has on the infant's digestion and assimilation, and how the milk is fitted to meet individual variation. The following is a summary of the present state of our knowledge of milk modification.

MILK AND CREAM DILUTION WITH THE ADDITION OF LACTOSE.—This is the method of modifying cow's milk to which the term *percentage modification* was first applied. The origin of this method was an effort to produce a cow's milk mixture, the composition of which would approach as closely as possible to that of human milk. The chief difference between human milk and cow's milk is that the latter has a higher proportion of protein as compared with fat. In cream the protein is lower in proportion to the fat, and if cream be diluted, the proportion found in human milk may be imitated. For example, the percentage formula of an average human milk is—

Fat 4.00 Lactose 7.00 Protein 1.50

and of an average cow's milk is

Fat 4.00 Lactose 4.50 Protein 3.50

A cream can be obtained which has an approximate formula of—

Fat 8.00 Lactose 4.00 Protein 3.00

If this be diluted one-half with water, the resulting mixture has a composition of—

Fat 4.00 Lactose 2.00 Protein 1.50

If now sufficient dry lactose be added to this mixture to make a 5% solution, the resulting mixture will have a formula of—

Fat 4.00 Lactose 7.00 Protein 1.50

which is that of an average human milk.

It must not be supposed that the above figures are absolutely accurate. They are given only as an example of how the composition of cow's milk may be altered, by dilution and the addition of lactose, to imitate human milk. The actual modification would involve certain fractions, which have been omitted.

The modification of cow's milk to imitate an average human milk by no means solved the problem of infant feeding. Certain essential chemical differences remained, such as the composition of the fats and mineral salts, and the relative proportion of soluble protein and casein. Moreover, the imitation of an average human milk did not meet the important factor of individual variation in digestive powers

and requirements. In this method, of dilution with the addition of lactose, we are by no means confined to the imitation of an average human milk. By diluting creams of various fat percentages we may alter at will the proportion of fat to protein. By diluting skimmed milk we may alter at will the percentage of protein without affecting the fat. By adding varying amounts of dry lactose, we may alter at will the percentage of lactose. Thus, by this method of diluting in various proportions milks of various fat percentages, with the use of dry milk sugar, we may alter at will the percentage of fat, lactose, and protein in our food, within very wide limits.

There are certain limits to the variety of percentage combinations which we can obtain. Any percentage of fat whatever is possible. The minimum protein depends upon how strong a cream we can obtain. A cream of higher fat percentage can be obtained by centrifugal separation than by gravity separation, and the former would give a lower minimum protein. The maximum protein is 3.50, the percentage in undiluted milk. The maximum lactose percentage has no limit, but the minimum varies with the percentage of protein, as all milk and cream contains lactose. The following table shows the practical limits of milk modification, by this method, with home and laboratory modification, assuming that 16 per cent is the highest fat obtainable in cream by the gravity method.

TABLE 27

Practical Limits of Milk Modification—Minimum Protein, with Varying Fat Percentages

	MINIMUM PROTEIN	
FAT PERCENTAGE	HOME MODIFICATION, GRAVITY CREAM, 16%	LABORATORY, CENTRIFUGAL CREAM, 32%
.50	.10	.05
1.00	.20	.10
1.50	.30	.15
2.00	.40	.20
2.50	.50	.25
3.00	.60	.30
3.50	.70	.35
4.00	.80	.40

TABLE 28

Practical Limits of Milk Modification—Minimum Lactose, with Varying Protein Percentages

PROTEIN PERCENTAGE	MINIMUM LACTOSE
.50	.70
1.00	1.40
1.50	2.10
2.00	2.80
2.50	3.50
3.00	4.20
3.50	4.50

This method of modifying cow's milk makes no attempt to solve the chemical difficulties involved in the composition of the cow's milk fat and mineral salts and in the high proportion of casein, except by simple dilution. If any of these elements is the cause of trouble, the quantity of that element can be reduced, and if necessary, other elements can be increased. The chief value of this method of modifying cow's milk lies in enabling us to meet the factor of individual variation in digestive power and requirements. It provides us with an almost unlimited number of combinations of the three main elements of an infant's food. The variations in the infant's digestive powers are primarily variations in its ability to digest and assimilate fat, sugar, and protein. Consequently, this method of modifying milk, by which the relative amounts of these elements can be varied at will, is *fundamental* in infant feeding, and is the basis of all efforts to solve any given problem.

MODIFICATION WITH STARCH.—The value of using some cooked cereal preparation as a diluent in cow's milk modification has long been recognized. When a solution of starch, such as is found in diluents like barley water, is present in a cow's milk modification, the curd formed by the precipitation of the casein in the normal process of digestion, is more finely divided, and is easier of digestion. The action of the starch in preventing the formation of large casein curds is not chemical, but mechanical, due to the colloidal nature of the starch solution.

If the starch possesses this colloidal action, there must be some minimum quantity of starch necessary to produce it. This quantity is that necessary to give a sufficient concentration of the starch solution, and it must therefore be proportioned to the total quantity of the food mixture. The minimum quantity necessary to obtain in full the colloidal action of the starch, is .75 per cent.

It must be remembered that starch is a readily fermentable carbohydrate, and that if it is not split by the amylolytic action of the pancreatic secretion, it will ferment. Babies vary in their power of digesting and absorbing starch. It is not known at just what age the amylolytic function develops, as this is probably very variable. The power of starch digestion may be present at birth, or may develop at some subsequent period. It is usually established at six months. Starch indigestion and fermentation is more likely to occur in younger babies.

MODIFICATION WITH ALKALIES.—The original basis for the use of alkalies in cow's milk modification was a further attempt to imitate the composition of human milk. It was believed at that time that, cow's milk being acid and human milk alkaline, the addi-

tion to cow's milk mixtures of 5 per cent lime water would cause them to resemble human milk in alkalinity. It has since been learned that human milk is not alkaline but amphoteric, and that the addition of lime water to cow's milk does not cause it to resemble human milk in reaction. In the meanwhile, however, clinical evidence had been accumulating, which was strongly in favor of the value of lime water in certain cases of difficult digestion.

A theoretical explanation of the value of lime water was sought in the fact that many cases of disturbed digestion are accompanied by symptoms suggesting acidity, such as " sour stomach," and acid stools. There is no experimental evidence in support of the theory that the alkalies act by neutralizing the products of acid fermentation, although such action is quite possible, and may explain some cases in which benefit is clinically quite evident.

The only positive and scientific data which we have, on which to base our use of the alkalies in infant feeding, is to be found in the work of Hamburger and Slicka, who published the results of an extended study on the action of the alkalies in gastric digestion. The truth of their conclusions has been confirmed by subsequent investigators. They have shown that the alkalies combine chemically with the casein of cow's milk, and that as a result of such chemical union, the precipitation of the casein by the rennin and acid of the stomach, is delayed, and the character of the curd is altered. The curd, when thus modified, is easier of digestion by the gastric juice. Furthermore, if sufficient alkali be present, the acid of the gastric juice is so neutralized, that the precipitation of the casein is entirely prevented. On this basis, our indication for the use of the alkalies is when we wish to delay curd formation with modification of the curd, or to prevent it entirely.

If the alkalies have this action, there must be some definite quantity of alkali which gives the action at its best. As the action of the alkali is a chemical one, the alkali entering into actual chemical union with the casein, this optimum quantity of alkali must be in direct proportion to the quantity of casein, with which the alkali combines. As milk and cream both contain casein, and are assumed for practical purposes to contain the same percentage of casein, the quantity of casein in a cow's milk modification varies directly with the quantity of milk and cream in the mixture. The optimum quantity of alkali, that needed to produce the desired result, is, therefore, expressed as a percentage of the quantity of milk and cream used in the preparation of the food.

The quantities of alkali which produce the various results which we must have in view when using the alkalies on this basis have been satisfactorily determined by experiment, and are shown in the table.

TABLE 29

Quantity of Alkali Expressed in Terms of Per Cent of the Milk and Cream

	TO DELAY CURD FORMATION AND MODIFY THE CURD	TO PREVENT PRECIPITATION OF THE CASEIN CURD
Lime water.....	20.00%	50.00%
Sodium bicarbonate..	0.68%	1.70%
Sodium citrate.....	0.20%	0.40%

As far as the different alkalies, lime water, sodium bicarbonate, and sodium citrate are concerned, all three have the same general action. They differ one from another only in the character of the casein curd formed under their influence. With one alkali the curd is more porous, with another more gelatinous, and so forth. Clinically, certain individual cases are found in which some one of the alkalies works best, but there is no guide as to which we shall choose. This work on the alkalies is the only definite basis for their use established by experimental evidence. In the majority of cases our use of the alkalies should be upon this basis. *We should order alkali with a definite purpose, to produce a known result, in the quantity which best accomplishes our purpose, and which is expressed in per cent of the milk and cream in the mixture.*

There is another basis for the use of the alkalies, not definitely established by experimental evidence, but suggested by clinical results. The work of Hamburger and Slicka is confined solely to the action of the alkalies in gastric digestion. Clinical evidence suggests that the alkalies may have some influence after the food leaves the stomach, possibly by neutralizing the acid products of excessive carbohydrate fermentation in the intestine. While such influence is not proven, it is sufficiently possible to warrant the use of alkali on this basis. In such a case, however, the physician should have a definite reason for using alkali. The amount of alkali used on this basis is not to be proportioned to the milk and cream, for the theoretical action of the alkali involves no chemical union with the casein. We do not know the amount of alkali necessary to neutralize excessive acid fermentation. All we can do is to order alkali in large excess. In the case of lime water, such an excess would be expressed as about 25% of the total mixture.

PEPTONIZATION.—Cow's milk modifications may be peptonized through the agency of a pancreatic extract obtained from an animal. The object is to effect a pre-digestion of the protein, which is converted into peptone by the active ferment trypsin. The amount of peptone formed varies with the duration of the process. If milk is peptonized for ten minutes, only a part of the protein is converted into peptone, and the milk is not altered in taste. If milk is peptonized for twenty minutes, most of the protein is converted, and the mixture has a bitter taste. Peptonization represents one method of

overcoming the difficulty presented by the high percentage of protein in cow's milk. With the development of other methods of dealing with protein indigestion, the use of peptonization has become more and more limited.

WHEY MIXTURES. THE SPLIT PROTEIN.—This method of modifying cow's milk is based on a further attempt to imitate Nature. When cow's milk is modified by the method of milk and cream dilution, with the addition of lactose, the relatively high proportion of casein to the lactalbumin and other soluble proteins persists. In cow's milk about four-fifths of the protein is casein, whereas in human milk, only about half is casein. Thus if the formula of cow's milk be expressed in terms of the two forms of protein, four-fifths of the total protein will be casein, and in a cow's milk mixture made by milk and cream dilution with the addition of lactose, designed to imitate human milk, the same proportion will hold. The table shows such a formula compared with human milk.

TABLE 30

	FAT	LACTOSE	TOTAL PROTEIN	WHEY PROTEIN	CASEIN
Cow's milk	4	4.50	3.50	.70	2.80
Modified cow's milk	4	7	1.50	.30	1.20
Human milk	4	7	1.50	.75	.75

The older ideas on the difficulties of digesting cow's milk in artificial feeding laid especial stress on the high casein content of cow's milk as the probable cause. The three methods of modifying cow's milk just described, namely, modification with starch, with alkalies, and with peptonization, were all designed to meet this difficulty presented by high casein. As will be seen from the table, in any formula made with cow's milk, the baby is required to digest much more casein than he would have to digest with the same total amount of human milk protein. The method of modifying with whey was designed to overcome this disadvantage, in another way, by altering the relative amounts of whey protein and casein in such a way as to imitate more closely the relative amounts in human milk.

If a cream of high fat percentage be diluted, the protein is much reduced. If, for example, a cream having a formula of—

Fat 16 Lactose 4 Protein 3.00

be diluted one to four with water, the formula of the mixture will be—

Fat 4 Lactose 1.00 Protein .75

If this formula be expressed in terms of soluble whey protein and casein it will be—

Fat 4 Lactose 1.00 Whey protein .15 Casein .60

whey be used as a diluent instead of water. Whey
ıt, and no casein, but only lactose and whey protein.
of whey be considered as approximately—

Lactose 5	Whey protein 1.00	Casein 0

e used as the diluent, and if one-half of the mixture
ıve added to our mixture with the whey the following—

Lactose 2.50	Whey protein .50	Casein 0

ether the percentages obtained by diluting our cream
nd making up the remaining three-quarters with whey
r one-quarter, we have to add together—

Lactose 1.00	Whey protein .15	Casein .60
Lactose 2.50	Whey protein .50	Casein 0

—

Lactose 3.50	Whey protein .65	Casein .60

add enough lactose to raise the total sugar percentage
—

Lactose 7.00	Whey protein .65	Casein .60

ar from the formula of human milk.

given above are not accurate, but are given as an
e method by which the relative proportions of whey
sein may be altered by the use of whey as a diluent.
ıod of modifying cow's milk, we are not confined to
)f an average human milk. By using various propor-
or milk and whey, we can obtain various proportions
rotein, and casein, and are thus provided with a great
centage combinations, fitted to cope with the factor
ariation in digestive powers and requirements.
rtain limits to the percentage combinations obtainable
whey. The percentage of whey protein in whey is
cent, and a whey mixture cannot have a whey protein
ch higher than this. The low limit of casein is deter-
amount of fat required. In general, .90 per cent is
upper limit for the whey protein, and .50 per cent
limit in home modification, while in laboratory feeding
ıgal cream used as a basis, the casein may be reduced
per cent.
remembered that the primary object of whey modifi-
luce the casein in the food without reducing the total
an use a food of high nitrogen content, without im-
den on digestion which the high casein of cow's milk

would otherwise involve. It must further be remembered, however, that such modification would only be useful in cases in which the casein is the cause of digestive trouble. Also, the minimum lactose obtainable with whey mixtures is much higher than with ordinary milk and cream dilution, because the whey used as a diluent in place of boiled water has a high lactose content. The high percentage of the mineral salts of cow's milk, which is ordinarily reduced by the dilution of milk and cream, is not reduced in whey mixtures, as whey contains a high mineral content. In cases where lactose, or the high salt content of cow's milk might be the cause of trouble, whey mixtures would do harm. On the other hand, the split protein method is the best theoretical method of dealing with casein intolerance, as it is the method used by Nature as exemplified in human milk.

MODIFICATION BY THE ADDITION OF SOLUBLE CARBOHYDRATES OTHER THAN LACTOSE.—The majority of cow's milk modifications require the addition of a certain quantity of lactose, because the lactose content of cow's milk is low in comparison with human milk, and is often still further lowered by dilution. The substitution of some other form of soluble carbohydrate for this extra lactose, did not originally spring from the results of any scientific investigation of the infantile digestion and metabolism, but was adopted upon purely empirical grounds. Indeed, this method of modifying cow's milk owes its adoption to the marked success sometimes seen in an individual case, attending the use of some patent baby food. It sometimes happened, that a baby fed most carefully on scientific principles, with every kind of milk modification suggested by our knowledge of the subject of artificial feeding, would fail to thrive; finally the mother, caught by the lure of some advertisement, or by the suggestion of a friend or neighbor, would try some patent baby food, with the most startling success. Such cases, of course, did not mean that the patent food was a better food for babies than all the resources of scientific feeding, but that it embodied some principle in the modification of cow's milk which was suited to the digestive peculiarities of that individual baby. It is the aim of scientific artificial feeding to contain in its stock of weapons every principle which may bring success in an individual case. It therefore became necessary to study the principles involved in the various patent foods. This study showed that most of the patent foods are mainly carbohydrate preparations, and that their essential feature is that they contain other carbohydrates than lactose, such as maltose, dextrine, dextrose, or cane sugar. Therefore it was obvious that there were babies whose individual peculiarities made them better fitted to utilize these other carbohydrates, and

the substitution of these sugars for lactose had to become one of our stock of resources in dealing with the problem of artificial feeding.

More recently, light has been thrown on the probable scientific explanation of the value of a change in carbohydrate. The various carbohydrates usually employed in milk modification are disaccharides, differing in the character of the component monosaccharides into which they are split in the course of digestion. Lactose, for example, is split into dextrose and galactose, saccharose is split into dextrose and levulose, while maltose is split into dextrose and dextrose. These monosaccharides differ in the readiness with which they are absorbed, and in the readiness with which they undergo bacterial fermentation. Dextrose is the substance most easily absorbed, and is, moreover, the only sugar which can be immediately utilized for energy production, without undergoing the process of glycogen-storing in the liver. Galactose and levulose, on the other hand, while less easily utilizable for energy production, are much more readily fermentable. In any case, therefore, in which there was difficulty in digesting and absorbing sugar, or in which there was difficulty in obtaining from other food elements, such as the fat, a sufficient supply of fuel for energy production, maltose would be the preferable sugar. This explains the good results often seen when maltose is used in cases of severe atrophy.

On the other hand, Nature does not provide for the infant the easily utilized maltose, but the more fermentable lactose. There must be a reason for this. Kendall has shown that the normal bacterial processes in the infant's intestine are those of carbohydrate fermentation, and that a sufficient supply of fermentable carbohydrate must remain behind in the intestine, in order to maintain the normal character of the intestinal flora. It is probably for this purpose that lactose is provided by Nature, and under this theory, lactose would be preferable to maltose in babies whose powers of digestion and assimilation showed no very wide deviations from the normal, or in babies whose digestive disturbances are traceable to abnormal bacterial processes.

This explanation, while not conclusively proven, is sufficiently satisfactory to serve as a basis for our choice of carbohydrate in feeding, and there is no other. Our choice must be made upon this basis, or else empirically without any other guide than clinical experience, which is very deceptive.

MODIFICATION WITH THE LACTIC ACID FERMENT.—This method of modifying milk originated from the same idea as gave us the use of starch, the alkalies, the split protein, and peptonization, namely the difficulty in digesting the large tough casein curds which are formed from cow's milk during the process of gastric digestion.

When the lactic acid bacillus, or any preparation containing its active ferment, is added to cow's milk, part of the lactose is by fermentation converted into lactic acid. This is an artificially produced souring of the milk, much like the natural souring which takes place in milk from the action of other organisms of the lactic-acid-forming group normally present in milk. The lactic acid bacillus and its product are known to be harmless. In the presence of the lactic acid formed in this way, the casein of the milk is precipitated in the form of a curd which is especially finely divided, and easily digested. There is no formation of large curds in the stomach, and lactic acid milk seems to be relatively quite digestible.

There are two reasons for using lactic acid milk as a mere feeding method. One is that described, to obtain the casein already precipitated in a finely divided, and easily digested form. The other is that lactic acid milk is relatively low in carbohydrate in proportion to the protein, and may be useful in cases in which carbohydrate is not well tolerated.

There is another reason for using lactic acid milk, under which it is not strictly a resource in artificial feeding. This use is based on the influence of the lactic acid bacillus on certain bacterial processes in the intestine, and will be described in greater detail in the division on the gastro-intestinal diseases. To meet this indication it is necessary to use a culture of the lactic acid bacillus of known anti-putrefactive power, while, if lactic acid milk is used simply as a feeding method, any preparation of the lactic acid bacillus which will form lactic acid may be used.

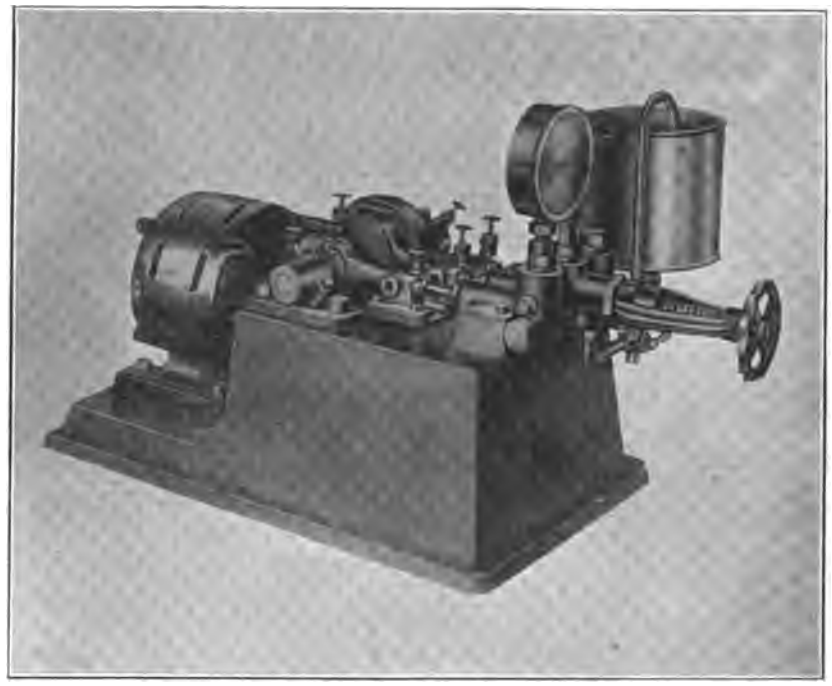
MODIFICATION WITH PRECIPITATED CASEIN.—The use of precipitated casein in infant feeding, is to be traced to the researches and theories of Meyer and Finkelstein. Their original theory ascribed to the carbohydrate of cow's milk, the lactose, a very important rôle in the production of severe malnutrition and atrophy. In their first publications, lactose was considered quite deadly, while the fats and mineral salts were not absolved from partial blame. To fit their theory, they prepared their celebrated " Eiweiss Milch," or albumin milk. The only new feature in albumin milk was the use of precipitated casein.

The casein is precipitated with a rennin preparation, washed, ground fine through a sieve, and then is added to some food mixture. The idea underlying its use was originally, to increase the casein content of the food, without a proportionate increase in the lactose or mineral salts. It has been found recently that a good part of the mineral salts remain with the casein instead of going out with the whey. The lactose, however, can be gotten rid of. The casein is in a finely divided form, which is probably easy of digestion

in the majority of cases. The use of precipitated casein does not show very much difference in its probable effect, from the use of lactic acid milk. Only, the possible benefit from the anti-putrefactive properties of lactic acid milk, is not to be expected with precipitated casein.

THE COOKING OF COW'S MILK MIXTURES.—The effect of the cooking of cow's milk on the digestibility of the milk has never been determined. There is some evidence that cooking makes milk more digestible, other evidence that it makes it less so. Cooking produces certain changes in the chemistry of the milk which are known, but the relation of these changes to digestion has never been established,

FIG. 76



Apparatus for homogenizing fat for infant feeding

It is known that cooking coagulates the lactalbumin, precipitates some of the mineral salts, and produces certain chemical changes in the casein. These changes make the casein less coagulable, and the curd formed with rennet is softer and finer than in raw milk. On the other hand, the casein curd is less readily acted on by the proteolytic ferments of gastric digestion. It is probable that in some cases, cooking has a favorable effect. The best established favorable action of cooking, is that seen in cases in which large casein curds are found

in the stools of the infant. In such a case, the trouble may often be relieved by boiling the milk.

HOMOGENIZED MILK MIXTURES.—This is a very recently advocated method of modifying cow's milk. It is based on the fact that many of the most serious cases of atrophy and malnutrition in infancy have been shown to be characterized by inability to digest and absorb the cow's milk fat. The difference in chemistry between the fat of cow's milk and of human milk is not affected by any of the methods of modifying milk described above. The homogenizer is a complicated and expensive apparatus for breaking up fat into the finest molecules, much finer than the emulsion seen in milk. This is done by forcing the fat through the apparatus under heavy pressure. Other fats than cow's milk fat may be used, and thus the unfavorable chemistry of cow's milk fat may be avoided. Those now interested in homogenized milk advocate the use of olive oil, which is added to a milk mixture, and then forced through the apparatus. The olive oil comes out in a form so finely divided as to be supposedly easy of digestion.

The method of feeding with homogenized milk is a very newly suggested method, and its possible value is as yet unsupported by conclusive evidence. If it be shown in the course of time that it has a value in infant feeding, its use will probably be confined to cases of very resistant difficulty, with inability to take as much fat as they require.

ALBUMIN MILK. (PROTEIN MILK; FINKELSTEIN'S EIWEISS MILCH).—This is a modification of cow's milk recommended by Finkelstein. The theory underlying its use was originally that the lactose in cow's milk is not well borne in certain cases, and is a frequent source of difficulty in artificial feeding. The original object of Finkelstein's preparation was to secure a food which was low in lactose, and high in protein. The most definite directions were given as to the preparation of this food, which will be described when the subject of home modification is discussed.

Albumin milk is essentially a combination of lactic acid milk with precipitated casein. When made according to the prescribed directions it has an average composition of about—

Fat 2.50 Lactose 1.50 Protein 3.00 Salts .50

The fat percentage is rather variable.

Albumin milk was recommended to meet a definite theory as to the etiology of chronic gastro-intestinal disease. The theory has been later much modified and changed. In particular, the original theory held that the lactose in cow's milk mixtures is one of the most frequent sources of danger. This has not been corroborated by other investi-

gators. That lactose may, through excessive fermentation, be cause of indigestion, is undoubtedly a fact, but that it is a seriou disturber of metabolism, giving rise to intoxication, is probably no true. This idea was probably borrowed by Finkelstein from som work published by Grosz, later elaborated by Langstein and Steinitz on sugar elimination in the urine in severe cases of gastro-intestina disorder. It has been contradicted by the work of later investiga tors. While there may be cases in which there exists an intoleranc of sugar sufficiently severe to call for an amount of sugar in the foo no greater than that in albumin milk, it is probable that they are ver rare, and do not explain the favorable results obtained by many in vestigators in using this food.

Another theory advanced in favor of the value of albumin milk was that the high salt content of cow's milk was a source of damage and that in the precipitated casein used in making Finkelstein's food the salts are got rid of. This also has been disproved. It has bee shown that much of the mineral matter goes over with the casein and that the salt content of albumin milk is quantitatively but littl diminished.

The principal respects other than the low proportion of lactos in which albumin milk differs in composition from an ordinary cow' milk modification, of the same fat and protein content, is, first, tha the casein is precipitated beforehand in a very finely divided form and second, that the food contains lactic acid. The favorable result reported from its use must be due to one or both of these features That both features are of value, is quite probable. That lactic aci milk has a favorable effect on certain fermental processes in the intes tine, has been fairly well established, although Finkelstein entirel disregarded this as a possible mode of action, in his effort to fit hi food to his theory. There is also evidence, that foods containing th casein already precipitated in a finely divided form, are more digestibl in certain cases.

It must be remembered that the evidence on which the value o albumin milk in infant feeding is based, is of the highly unreliable statistical kind, obtained by feeding a series of babies on this food and making a compilation of the various results observed. Almos any method of feeding investigated in this way will give favorabl results. The method of studying individual cases, under condition approaching as nearly as possible those of an experiment, has no been employed. No great effort has been made to determine whethe either of the two principles combined in albumin milk, namely, lacti acid fermentation, and the addition of precipitated casein, will no give equally good results, when used separately. No great effor has been made to determine the clinical type of case in which albumi milk works better than other methods.

That each of the two principles combined in albumin milk is of great value in certain cases, is undoubtedly true. That there are cases in which a combination of these two principles, as realized in albumin milk, is the best method of feeding, is also probable. Therefore these principles, and their combination, must have a place in artificial feeding, among our stock of resources. What is the exact indication for their use remains to be determined.

It must be remembered that albumin milk being so widely spoken of as a food, or method of feeding, there is danger of its coming to be regarded as an ideal baby food, as a food or method of feeding of superior value for babies in general, and not as a resource to be used in special cases. If we are to judge by the precedents of other widely advertised methods of feeding, there is danger that the general or indiscriminate use of albumin milk may do harm in the future. This danger is now heightened by the fact that in Europe Finkelstein's albumin milk is a secret, patent preparation, sold by a manufacturing company in sealed cans, and advertised as a baby food. The same company, or its branch, has introduced its sale into this country. It is true that we know enough about the composition of albumin milk to order its preparation in our homes and hospitals, without resorting to the patented form. Is it not better, however, in using and studying albumin milk, to consider chiefly the two main features in its composition, and to order them separately, or in combination? We are not confined to making albumin milk according to the original formula, but may combine lactic acid milk with precipitated casein in any proportions we see fit. We can thus use albumin milk, while avoiding the name, and will not be perpetuating the idea of its superior value as a baby food. We can extend our investigations as to its value, by combining its principles with varying amounts of different sugars, and of fat. Like all other methods which have given clinical evidence of good results, albumin milk or the principle it embodies, will probably in the end occupy its definite and particular place among our stock of resources.

MALT SOUP MODIFICATION OF COW'S MILK.—This modification of cow's milk was introduced by Keller, as a food to be used in cases of difficult feeding. Wheat flour is cooked in the milk, and then malt extract is added, and the whole is heated for about five minutes. This food has given clinical evidence that in certain difficult feeding cases, it gives better results than ordinary modifications. It must therefore receive a place among our stock of methods of modifying cow's milk.

Malt soup is rich in carbohydrate. Keller believed that through its use, less protein was lost by intestinal fermentation, but this has never been definitely proved. Malt soup is essentially a combination

of three methods of modifying milk already described, namely, modification with starch, modification with maltose, and the cooking of milk. Furthermore, a certain amount of the starch is dextrinized.

We have seen that all three of the methods of modifying combined in malt soup, have their particular favorable action on the digestion of cow's milk. To combine them is probably desirable in certain cases.

The dextrinization of the starch in the malt soup is a new element. Dextrinization has been advised as a good method in preparing a food for infants, and *dextrinized gruels*, made by heating a cereal decoction with thick malt extract, have been employed. We have no essential facts in digestion or metabolism to support their use, although they may have a value in certain cases. The evidence in their favor is of the unreliable clinical variety. It seems best to include them under malt soup.

HOW THE RESOURCES OF COW'S MILK MODIFICATION ARE REALIZED IN PRACTICE

Familiarity with the various principles on which the modification of cow's milk is based, is not the only essential in the intelligent artificial feeding of infants. The physician must know exactly how he can utilize in practice the various methods at his disposal, so that any method which he believes to be indicated in an individual case may be employed.

PREREQUISITES.—It is essential that the physician should be able to utilize the resources of artificial feeding to their fullest extent. It is undoubtedly true that the majority of babies which he will be called upon to feed, will not require any of the more complicated methods of modifying milk. Simple combinations of milk, diluent, and lactose, is all that is required in these cases. Consequently, many so-called simplified methods of feeding babies have been devised and published, which give the impression that the artificial feeding of babies is not in reality a very difficult or complicated problem. The success of such simplified methods, when judged by clinical statistics, is evident, and is to be expected from the comparatively normal digestive powers of the majority of infants. These methods, however, as is well-known, will fail to solve the occasional difficult problems which arise. Knowledge of the methods used in the more difficult cases is essential to their proper treatment, and does not preclude the use of foods of simple composition in comparatively normal cases. The difficult cases are numerous enough to demonstrate that the problem of artificial feeding is not a simple one, nor is it to be solved by simple methods.

We must assume that the competent physician must be prepared

to use every method. Therefore no method of prepari
fications can be considered which does not fulfil thi

THE PATENT FOODS.—There are a number of
foods on the market, which embody principles known
in artificial feeding. Their use has given repeated cli
of the special value of some one of them in certain in
We know that their good results come from the partic
such as change of carbohydrate, which they exemplif
that their use as foods for babies in general would n
feeding problem, and most of us have seen how grea
done by their indiscriminate employment. The quest
we not justified in using the patent foods, not indiscr
as general baby foods, but intelligently, in a particular
we believe that just the particular modification exem
patent food, is indicated. Used thus intelligently, i
that the patent food would do no harm, and it may be
venient means of realizing the particular feeding reso
wish to employ.

I do not believe we are justified in thus using the
Whatever their value in individual cases, the fact
they are advertised, and sold as general baby foods
they are thus advertised, their use cannot but help
the idea that they are baby foods of superior valu
should avoid perpetuating this harmful idea. Every
bodied in them can be realized without their use. If
dextri-maltose, it is better to use some preparation adv
as a specially pure form of dextri-maltose, than a pr
taining the same dextri-maltose but advertised as a b

MILK LABORATORIES.—The object of the milk
to afford to the physician a means of utilizing every l
of milk modification employed in infant feeding. In
of scientific rational medicine physicians all over the
first, means of saving time, and second, exact methods
in themselves soon become time-savers. In every bra
the tendency is growing year by year to systematize th
laborious work of the individual for the common pract
profession at large. The two chief advantages of t
ratory are, first, the saving of time for the physician
exactness in the composition of the foods prepared
The physician's time is saved because, thinking of his
of the percentages of the various food elements whicl
give, he does not have to calculate the various comb
gredients which will give the required food compositio
to nurse or mother detailed directions for the prepa

food. This is done at the laboratory. Exactness is obtained from the fact that the various ingredients used in preparing the food, such as cream, milk, barley water, and so forth, are standardized, and contain exactly the percentages of the food elements demanded by the method of calculation employed. The calculations are of course, exact.

In using the milk laboratory, the physician writes a prescription, calling for any food which he wishes to give. He specifies the percentages of fat, sugar, and protein he desires, states the variety of extra sugar to be used, states, if he wishes, the relative quantities of whey protein and casein. He may order any alkali to be added, may order starch in any amount, may have the food fermented with lactic acid, cooked, pasteurized, dextrinized, or peptonized. He may order albumin milk or precipitated casein. All he has to do is to specify the composition of the food he wants, and the laboratory puts up this food. These laboratories have been placed under the control of educated, intelligent men in whom we should have the same confidence that we have conceded to the pharmacist, and we can write directions for infants' foods and send them to these laboratories just as, in the treatment of disease, we write our pre scriptions for the division of one drug or the combination of several. As the pharmacist has nothing to do with the various methods of treating disease, so the milk-modifier is simply required to carry out the directions and ideas of the physician, and the laboratory assumes no responsibility as to the success or failure of the food. The result attained depends wholly upon the physician's intelligence in the use of the resources of feeding.

In addition to carrying out the physicians' prescription calling for a food of a certain composition, the milk laboratory provides a good and clean milk supply, and the modification and delivery of the milk is carried on under the cleanest conditions.

To illustrate the use of the laboratory, a prescription blank from the Walker-Gordon Laboratory is reproduced on the opposite page: It will be seen how all the more ordinary milk modifications can be ordered very simply from these blanks. For some more complicated preparations, the physician must give a more detailed description of what he wants.

The filled prescription calls for a food having a fat percentage of 2, a sugar percentage of 6, the extra sugar being maltose, a whey protein percentage of .75, and a casein percentage of .50. Lime water is to be added in the amount of 20 per cent of the milk and cream. The food is to be pasteurized at 145° F., and delivered in eight feeding bottles, each containing six ounces.

The prescription blank is arranged for the use of all the more ordinary resources of infant feeding. When the physician wishes

FIG. 77

WALKER-GORDON LABORATORIES

℞

		Per Cent
Fats		2
(a) **Carbo-hydrates**	Lactose (Milk Sugar) Maltose (Malt Sugar) Sucrose (Cane Sugar) Dextrose (Grape Sugar) Starch	6
(b) **Dextrinize**		
(c) **Proteids**	Whey	75
	Casein	50
(d) **Peptonize**		
(e) **Sodium Citrate**	% of milk and cream	
	% of total mixture	
(f) **Sodium Bicarb.**	% of milk and cream	
	% of total mixture	
(g) **Lime Water**	% of milk and cream	20
	% of total mixture	
(h) **Lactic Acid Bacillus**	1—To inhibit the saprophytes of fermentation	
	2—To facilitate digestion of the proteids	

Heat at ..... 145°F.....

Number of Feedings ..... 8.....

Amount at each Feeding ..... 6 ..... oz.

ORDERED FOR

.....

ADDRESS .....

DATE ..... 19.....

..... M. D.

NOTE—See back of pad. (OVER)

EXPLANATORY

(a) It requires 75% starch to make the precipitated casein finer.

(b) One hour completely dextrinizes the Starch.

(c) In case physicians do not wish to sub-divide the proteids, the words "Whey" and "Casein" may be erased.

(d) Twenty minutes renders the mixture decidedly bitter.

(e) It requires 0.20% of the milk and cream used in modifying to facilitate the digestion of the proteids; i. e., the formation of a soft curd. 0.40% to prevent the action of rennet; i. e., the formation of tough curd.

(f) It requires .68% of the milk and cream used in modifying to favor the digestion of the proteids. 1.70% of the amount of milk and cream used suspends all action on the proteids in the stomach. 1.7% of the total mixture gives a mild alkaline food.

(g) It requires 20% of the milk and cream used in modifying to favor the digestion of the proteids. 50% of the amount of milk and cream used suspends all action on the proteids in the stomach. 5% of the total mixture gives a mild alkaline food.

(h) Percentage figures represent the per cent of Lactic Acid attained when the food is removed from the thermostat. When the Lactic Acid Bacillus is used to facilitate digestion of the proteids, this is the final acidity, as the process is stopped by heat at this point. When the Lactic Acid Bacillus is used to inhibit the growth of saprophytes, the acidity may subsequently increase to a variable degree, as the bacilli are left alive. .25% Lactic Acid just curdles milk. .50% gives thick curdled milk. .75% separates into curds and whey.

WALKER-GORDON LABORATORIES

1106 Boylston Street

Boston

And all Large Cities

to use certain special methods of modifying milk, such as albumin milk, modification with precipitated casein, malt soup mixtures, and homogenized fat mixtures, he must transmit to the laboratory more specific directions as to what he wants. The laboratory is prepared to carry out any wishes of the physician in regard to the modification of cow's milk for infant feeding.

HOME MODIFICATION.—In many cases, probably the majority, a milk laboratory cannot be utilized to obtain the desired milk modifications for the infant. In these cases the food has to be prepared at home, by the nurse or mother. To be successful in all cases, the physician must be able to utilize by home modification, all the methods of milk modification available in artificial feeding. This can be done, with less accuracy than at the laboratory, but with

sufficient accuracy for practical purposes. All the methods of modifying milk described here except homogenization can be carried out at home, although some of them are rather troublesome and laborious, and in some cases, the widest extremes of percentages cannot be obtained.

TECHNIQUE OF HOME MODIFICATION

The following implements are required:—As many feeding bottles as there are feedings in twenty-four hours; the milk, in quart jars;

FIG. 78

Home modification of cow's milk. Apparatus

lactose or other carbohydrate; a pitcher large enough to hold the whole of the mixture; an empty quart milk jar; a graduate, holding one pint; a tablespoon; a teaspoon; sterile cotton or albumin caps to stopper the bottles; a cream dipper. All the implements should be clean, and sterilized by boiling.

The lactose is measured with a tablespoon, put in the empty milk jar, and the boiled water measured in the graduate is added while still hot. The jar is then shaken until the lactose is dissolved, and put aside to cool. The top ounce of cream is removed with a teaspoon, to allow the insertion of the cream dipper without overflow. The rest of the cream, down to the cream line, is removed with the

cream dipper, and put into the graduate. The excess of cream in the graduate is poured off, leaving in the graduate only the quantity of cream required by the formula.* This is then placed in the pitcher. The skimmed milk remaining in the milk jar is mixed by shaking, and the quantity required is measured off in the graduate, and added to the pitcher. When cool, the boiled water and lactose are poured into the pitcher. The quantities to be given at each feeding are measured off in the graduate, and placed in the feeding bottles. The

FIG. 79

Home modification of cow's milk. Obtaining the cream

bottles are stoppered, and are then pasteurized, if pasteurization is to be used. (See technique of pasteurization, p. 274.) The bottles are finally placed on ice, until used, when each bottle is warmed and given to the baby.

THE CALCULATION OF HOME MODIFICATION

The physician using home modification, thinks of the food he wishes to give in the general terminology of the percentages of its component elements. He must be able to translate this percentage formula into definite directions for obtaining and mixing certain

* If the amount of cream obtained from one quart is less than the amount required in the formula, two quarts must be used in obtaining the cream.

ingredients. This involves a certain amount of mathematical calculation. A great variety of methods of calculating have been devised and published. Some of them have a certain time-saving merit, but most of the abbreviated methods are difficult to remember, if not in constant use. The method given here is based on the fundamental mathematical principle involved, from which all abbreviated methods are derived. Once learned, it is not forgotten.

FIG. 80

Home modification of cow's milk. Mixing the ingredients

MILK AND CREAM DILUTION WITH THE ADDITION OF MILK SUGAR.—This is the fundamental method by which we vary at will the percentages of the three principal food elements, the fat, sugar, and protein. Certain ingredients are obtained, and we must calculate the number of ounces of each one, which must be diluted with water to produce the required food.

To simplify the calculations, we assume a definite composition for the ingredients used. The formulae thus assumed are by no means accurate, but the calculation of home modifications may be sufficiently simplified to avoid very troublesome fractions, and to shorten the process of calculation, provided that the error be not too great. The actual average composition of the milk ingredients most commonly used in home modification is shown in the table.

TABLE 31

Average Composition of the Milk Ingredients Used in Home Modification

	FAT	LACTOSE	PROTEIN
Whole milk	4.00	4.75	3.50
10% cream	10.00	4.45	3.27
16% cream	16.00	4.20	3.05
32% cream	32.00	3.40	2.50
Skimmed milk	1.00	5.00	3.55
Separated milk ("fat free")	.25	5.00	3.65
Whey	.25	5.10	0.90

The ingredients used are the following:—

1. Gravity cream, which is obtained by taking off with a cream dipper from a quart jar of milk all the cream which rises after eight or more hours' standing.

2. Skimmed milk, which is the milk left in the jar after all the cream has been removed.

3. Dry milk sugar.

For purposes of calculation, we assume the following composition for these ingredients:

	FAT	LACTOSE	PROTEIN
Gravity cream	16.00	4.50	3.20
Skimmed milk	0.00	4.50	3.20
Dry lactose	0.00	100.00	0.00

The calculation is based on a fundamental mathematical proportion, by which all problems in dilution may be solved. The proportion is as follows:

The quantity of an ingredient :
The total quantity of the mixture =
The per cent of any element in the ingredient:
The per cent of that element in the mixture.

If this is expressed in algebraic terms,—

Let x represent the ounces of the ingredient (ounces required).

Let a represent the total ounces of food desired.

Let m represent the per cent of any element in the mixture (per cent required).

Let n represent the per cent of that element in the ingredient (16% for fat; 3.20% for protein; 4.50% for lactose).

$$\text{Then} \quad x : a = m : n$$

$$x = \frac{m}{n} \times a$$

The ounces of an ingredient required to give a certain percentage is found by *dividing the percentage required by the percentage of the element in the ingredient, and multiplying by the total number of ounces in the mixture.* Or the percentage required may first be multiplied by the total number of ounces in the mixture, and then divided by the per cent of the element in the ingredient.

For example, suppose we wish to calculate the home modification of a food containing—

Fat 3 Lactose 6 Protein 2

the baby to have 8 feedings, of 4 ounces each—
The total mixture, a, is 32 ounces, the percentage of fat required, m, is 3, and as always, the percentage of fat in our first ingredient, gravity cream, is 16,

$$x = \frac{3}{16} \times 32 = 6 \text{ ounces of cream.}$$

Protein is contained in both the cream and the skimmed milk, and is assumed to have the same percentage in each, namely 3.20. The percentage of protein required being 2.00,

$$x = \frac{2}{3.20} \times 32 = 20 \text{ ounces of cream and skimmed milk.}$$

The cream having been already determined as 6 ounces, the skimmed milk must be—

20 — 6 ounces, or 14 ounces.

Before determining the amount of dry lactose needed to bring the lactose percentage up to the required 6%, we must determine what percentage of lactose is added by the milk and cream. The same formula—

$$x : a = m : n$$

is used, but now x, the number of ounces of the ingredient is known, the unknown quantity being m, the percentage of lactose in the mixture after dilution of the cream and skimmed milk—

$$\text{If } x : a = m : n$$

$$m = \frac{x}{a} \times n$$

The percentage given by diluting an ingredient is found by dividing the ounces of the ingredient used, by the total ounces in the mixture, and multiplying by the percentage of the element in the ingredient. Therefore:—

$$m = \frac{20}{32} \times 4.50 = \frac{5}{8} \times \frac{9}{2} = \frac{45}{16} = 3 \text{ approximately, i. e., } 3\ \%.$$

To determine the ounces of dry lactose needed to give the additional required sugar percentage of 3, we use the original form of the proportion, the percentage of sugar in dry lactose being, of course, 100.

$$x = \frac{3}{100} \times 32 = .96 \text{ or approximately, 1 ounce.}$$

In home modification it is troublesome to require the nurse or mother to weigh the sugar on a scale. The common domestic measure, the tablespoon, is usually employed. As a basis of calculation, we consider that,

2 rounded tablespoonfuls of sugar = 1 ounce.

A rounded tablespoon is obtained by gently shaking a heaping tablespoon, until the extra sugar has fallen off. In our calculation, the sugar required being one ounce, we would in our directions call for two rounded tablespoons.

The complete directions which we would issue to the mother would be as follows:

From a quart jar of milk, on which the cream has "set" (after eight or more hours' standing), remove with a cream dipper all the cream down to the "cream line." This is "gravity cream." The milk left in the jar mixed by shaking is skimmed milk. Mix as follows:

Gravity cream, 6 ounces
Skimmed milk, 14 ounces
Boiled water, 12 ounces
Milk sugar, 2 rounded tablespoons

The milk sugar should always be *dissolved first in the boiled water while the latter is still hot.*

The steps in the process may be summarized as follows:—

1. Determine the ounces of cream needed to give the required fat percentage (calculation of x). Divide the percentage of fat required by 16 and multiply by the total number of ounces in the mixture.

2. Determine the ounces of cream plus milk needed to give the required protein percentage (calculation of x). Divide the percentage of protein required by 3.20 and multiply by the total number of ounces in the mixture.

3. To determine the ounces of skimmed milk needed, subtract the ounces of cream used from the ounces of cream plus milk.

4. Determine the percentage of sugar given by diluting the cream and milk (calculation of m). Divide the ounces of cream and milk by the total number of ounces in the mixture, and multiply by 4.50. Subtract this from the percentage of sugar required, to find the additional sugar percentage needed.

5. Determine the ounces of milk sugar required to give the *additional* sugar percentage required (calculation of x). Divide the additional sugar percentage required by 100 and multiply by the total number of ounces in the mixture.

6. Reduce the sugar from ounces to rounded tablespoons by multiplying by 2.

Suppose now we wish to calculate the following formula—

Fat 2 Lactose 7 Protein 1.50

the baby to have 8 feedings of 5 ounces each.

The steps as enumerated above, would be as follows:

1. $\frac{2}{16} \times 40 = 5$ ounces gravity cream.

2. $\frac{1.5}{3.20} \times 40 = \frac{1.5}{.08} = 18.7$, or 19 ounces cream and milk.

3. $19 - 5 = 14$ ounces skimmed milk.

4. $\frac{19}{40} \times 4.50 = \frac{19}{40} \times \frac{9}{2} = \frac{178}{80} = 2.22$, or 2% sugar added by the cream and milk. $7 - 2 = 5\%$ additional sugar required.

5. $\frac{5}{100} \times 40 = 2$ ounces of dry milk sugar required.

6. $2 \times 2 = 4$ tablespoons dry milk sugar required.

Omitting the preliminary directions for obtaining the ingredients, the directions would be as follows:

Gravity cream, 5 ounces
Skimmed milk, 14 ounces
Boiled water, 21 ounces
Milk sugar, 4 tablespoons

It is necessary at times to eliminate various troublesome fractions of ounces or percentages which occur during the calculation. The error involved in thus eliminating fractions is not very great, and the various errors are very apt to offset one another. It is best to have some definite rule of procedure in eliminating fractions. The following is a good rule:—With fractions involving percentage of fat or ounces of cream, take the nearest quarter per cent or quarter ounce; with fractions involving percentage of lactose or protein, or ounces of skimmed milk or milk sugar, take the nearest half per cent or half ounce.

Troublesome fractions may also be eliminated automatically, if we take for our total mixture required some figure easily divisible by other numbers. For instance, if we wish to give 7 feedings of 4 ounces each, we can take for our total mixture 32 ounces, instead of 28. There will only be a waste of 4 ounces, and the figure 32 is much easier to handle in calculating. Similarly, if we wish to give to the baby 7 feedings of 5 ounces each, we can calculate for a mixture of 40 ounces instead of 35, with a waste of only 5 ounces.

This use of round numbers has the additional advantage that it provides extra milk to allow for spilling or other accident.

There are two ways of varying the relation of protein to fat percentage. We may choose for dilution creams of different fat percentages, each having a different proportion of protein to fat. Or we may choose for dilution a cream of maximum fat percentage, having the lowest proportion of protein to fat, and may then vary the percentage of protein by adding varying amounts of fat-free (skimmed) milk. The former way has the advantage that the mother or nurse is required to work with fewer ingredients, but the latter way is preferable. Creams of different fat percentages are obtained by taking from the top of the milk a various number of ounces after a various number of hours' standing. In home modification, there is always an error, due to the variations in cow's milk, so that our ingredients never represent the exact percentages which we assume to be their composition. The most essential thing in home modification is, not that the food shall contain the exact percentages that we wish, but rather, when we change the food, that the new food shall bear an exact relation to the old one. If we are constantly changing our method of obtaining our ingredients, this unknown initial error comes in all over again with each change of food. If we work with two constant ingredients, always obtained in the same way, the error is constant, and no new error is introduced. Furthermore, it has been found that the gravity cream obtained by the method described of removing all the cream down to the cream line, is more apt to be close to the assumed fat percentage, and is less subject to variations, than creams of other fat percentages obtained in different ways. Another advantage of using the ingredients described here, is that they are constant, and the directions for obtaining the ingredients when once given to the mother or nurse, hold for all formulae in milk and cream dilution, and do not have to be changed or repeated when the composition of the food is changed.

THE CALCULATION OF STARCH.—Barley water is the usual ingredient used to modify cow's milk with a solution of starch. The percentage of starch in barley water varies according to the manner in which the barley water is made. Barley water made according to the directions accompanying most of the barley flour preparations on the market, contains about 1.50 per cent of starch. As the minimum percentage of starch required in a milk modification to give at its best the colloidal action of the starch is .75 per cent, it is obvious that if a barley water containing 1.50 per cent of starch be used as the ingredient, half the mixture must be barley water. As a protein of 1.60 per cent requires half the mixture to be milk and cream, we cannot have half our mixture barley water with a protein percentage higher than 1.60. As we frequently wish to order a higher protein than this, it is better to use as our stock ingredient a barley water having a starch percentage of 3.

Such a barley water may be obtained from any of the barley flour preparations, by giving to the mother or nurse the following directions:—To one pint of water add two level tablespoons of barley flour; boil for twenty minutes; replace the water boiled away with enough water to make up the full pint; strain through three layers of cheese cloth.

The calculation is very simple, the same formula

$$x : a = m : n$$

being used, as for milk and cream, n being 3.

If we want a .75 per cent starch solution, in a 32-ounce mixture,

$$x = \frac{.75}{3} \times 32 = 8 \text{ ounces of barley water.}$$

It is easier to remember that if we are working with a barley water containing 3 per cent of starch, and if we desire .75 per cent of starch in our mixture, one-quarter of the total mixture must be barley water.

THE CALCULATION OF THE ALKALIES.—As shown above, the alkalies should usually be prescribed in definite relation to the number of ounces of milk and cream in the mixture. If we wish to use lime water in the amount of 20 per cent of the milk and cream, we divide the ounces of milk and cream by 5, to obtain the ounces of lime water required in the mixture. If we wish to use lime water in the amount of 50 per cent of the milk and cream, we divide the ounces of milk and cream by 2. It must be remembered that lime water, being a liquid, and not a dissolved dry, ingredient, enters into the total mixture, and the boiled water, or other diluent must be proportionately diminished.

In the case of the other alkalies, the calculation is as follows:—Divide the figure representing the percentage of sodium bicarbonate or sodium citrate required by 100, and multiply by the ounces of milk and cream in the mixture. This gives the alkali required in fractions of an ounce. To reduce to drachms multiply by 8, and to reduce this to grains, multiply the result in drachms by 60.

For example, in a formula requiring 5 ounces of cream and 13 ounces of skimmed milk, it is desired to add sodium bicarbonate to delay curd formation in the stomach, and to alter the composition of the curd. The amount of alkali required for this purpose is .68 per cent.

Then $.68 \div 100 = .0068$.
$.0068 \times 18 = .1184$ ounces.
$.1184 \times 8 = .9472$ drachms.
$.9472 \times 60 = 56.8320$ or 57 grains.
approximately one teaspoonful.

As an example of the calculation of both starch and alkali, let us suppose that in the formula calculated on p. 308, we desired to give .75 per cent of starch, and enough lime water to prevent precipitation of the protein in the stomach. The usual directions would be given for obtaining gravity cream and skimmed milk. Further directions would be given for making a barley water containing 3 per cent of starch. The mixing directions would be as follows—

Gravity cream,	5	ounces
Skimmed milk,	14	ounces
Barley water,	10	ounces
Lime water,	9½	ounces
Boiled water,	1½	ounces
Milk sugar,	4	tablespoons

THE TECHNIQUE OF PEPTONIZATION.—Pancreatine (Extractum pancreatis) is an official preparation. It acts only in an alkaline medium, and the amount required varies with the protein, and hence with the ounces of milk and cream in the mixture. For each ounce of milk or cream in the mixture, we should use 1/2 a grain of pancreatic extract, and 2 grains of sodium bicarbonate. There are various commercial pancreatic preparations usually in the form of pastes put up in tubes, and usually called peptonizing tubes. These are very convenient, and as the directions as to the quantity accompanying them apply to whole milk, the determination of the amount required in dilutions of milk and cream is easy. They do not require the addition of bicarbonate of soda.

The extract and sodium bicarbonate, or the paste, is dissolved in about 4 ounces of the boiled water, which has been allowed to cool. This is then added to the milk mixture, which, in its jar, is set in a vessel of water at a temperature of 107° F. The time varies according to the degree of peptonization desired. The usual time is from seven to ten minutes.

CALCULATION OF WHEY MIXTURES.—The calculation of split protein formulae is much like the calculation of milk and cream dilutions, except that a new food element, the whey protein, enters into consideration, and an additional ingredient, whey, is used. In the instructions given to the mother or nurse, in addition to the directions for obtaining gravity cream and skimmed milk, we must give the necessary directions for obtaining whey. They are as follows:—

To make the whey, use skimmed milk. Add liquid rennet, or essence of pepsin, or a junket tablet in the proportions of two teaspoons or one tablet to the pint. Warm to about body temperature (100° F.) and let stand until the curd has solidified. Break up the curd with a fork or spoon, and strain three times through cloth or fine cheese cloth. Then heat the whey to 150° F. to kill the active rennin.

The average actual percentage formula of whey is—

Fat 0.25 Lactose 5.10 Whey protein 0.90 Casein 0.00

For purposes of calculation, the formula is assumed to be—

Fat 0.00 Lactose 4.50 Whey protein 1.00 Casein 0.00

A further assumption is made for the purpose of simplifying the calculation. A certain proportion of the protein of cow's milk is whey protein, this proportion lying between one-fifth and one-fourth of the total protein. If this fact were taken into consideration in the calculation of whey mixtures for home modification, the mathematical difficulties would be much increased. The small proportion of whey protein is disregarded, and it is assumed that all the protein added by the milk and cream is casein. The error involved in this assumption is not very great, and is partly offset by the assumption that the whey protein percentage in whey is 1.00 instead of 0.90.

The various steps in the calculation of split protein formulae are the following:

1. Determine the ounces of cream needed to give the required fat percentage (calculation of x). Divide the percentage of fat required by 16 and multiply by the total number of ounces in the mixture.

2. Determine the per cent of protein contained in this cream dilution (calculation of m). Divide the ounces of cream by the total number of ounces in the mixture, and multiply by 3.20. This protein is considered as being all casein, and represents the minimum casein which can be obtained. If the percentage of casein desired is below this minimum, the formula desired should be readjusted to require the minimum obtainable casein.

3. This third step is omitted, unless the per cent of casein contained in the cream is *lower* than the per cent of casein required. If so, determine the ounces of skimmed milk needed to give the additional casein (protein) percentage required (calculation of x). Divide the additional percentage of casein required by 3.20, and multiply by the total number of ounces in the mixture.

4. Determine the ounces of whey needed to give the required whey protein percentage (calculation of x). Multiply the per cent of whey protein required by the total number of ounces in the mixture.

5. Determine the percentage of sugar given by the cream, milk, and whey (calculation of m). Divide the ounces of cream + milk + whey by the total number of ounces in the mixture and multiply by 4.50. Subtract this from the percentage of lactose required to find the additional sugar percentage needed.

6. Determine the ounces of milk sugar required to give the *ad-*

dilional sugar percentage required (calculation of x). Divide the additional sugar percentage required by 100 and multiply by the total number of ounces in the mixture.

7. Reduce the sugar from ounces to rounded tablespoons by multiplying by 2.

The following example illustrates the calculation of a split protein formula:

We wish to give

Fat 2.50 Lactose 6 Whey protein .75 Casein .50

8 feedings of 4-1/2 ounces.

In this case, I should assume the total mixture to be 40, instead of 36, in order to avoid fractions.

1. $\frac{2.5}{16} \times 40 = 6\frac{1}{4}$ ounces gravity cream.

2. $\frac{6.25}{40} \times 3.20 = .50\%$ minimum casein obtainable.

In this case, the minimum casein obtainable is just what is required. Therefore the third step is omitted.

4. $.75 \times 40 = 30$ ounces whey.

5. $\frac{36.25}{40} \times 4.50 = \frac{36}{40} \times \frac{9}{2} = \frac{9}{10} \times \frac{9}{2} = \frac{81}{20} = 4$ per cent of sugar added by the cream and whey. $6 - 4 = 2\%$ additional sugar required.

6. $\frac{2}{100} \times 40 = .8$ ounces.

7. $.8 \times 2 = 1.6$ tablespoons, called $1\frac{1}{2}$ tablespoons.

The mother or nurse would consequently be told to mix as follows:

Cream,	$6\frac{1}{4}$ ounces
Whey,	30 ounces
Boiled water,	$3\frac{3}{4}$ ounces
Milk sugar,	$1\frac{1}{2}$ tablespoons

CALCULATION OF OTHER CARBOHYDRATES THAN LACTOSE.—Most of the other carbohydrates are obtainable in dry form. The carbohydrate most commonly used is maltose, or a combination of dextrin and maltose. There are a number of preparations of "dextri-maltose" on the market which are excellent, and are, I believe, the best form in which to use maltose as the extra sugar in cow's milk modification. These dry sugars are simply used as substitutes for the lactose added to ordinary modifications. In using maltose, the physician does not usually think of a definite percentage of maltose which he wishes to give, but rather of the total sugar, the extra sugar

to be maltose. The relative percentages of lactose and maltose this total carbohydrate are brought out during the calculation. we wish to give 6 per cent carbohydrate, extra sugar maltose, wh we determine the per cent of sugar added by the milk and cream the mixture, this represents the per cent of lactose, while the add tional carbohydrate percentage required represents the per cent maltose in the mixture.

Some of the carbohydrates used in milk modifications are sold liquid form. Examples of these are the various malt extracts. Whe these are used, it is necessary to know the percentage of maltose i the preparation employed. This figure is substituted for the figu 100, in calculating the extra sugar required, and the result is le in fluid ounces, forming a part of the total mixture.

LACTIC ACID MILK IN HOME MODIFICATION.—The making c lactic acid milk for therapeutic use against intestinal putrefactio will be described elsewhere. In making lactic acid milk as a metho of modifying cow's milk for the purpose of infant feeding, any prepa ration of the lactic acid bacillus, such as lactone tablets, lacto bacilline, or a bouillon culture of the lactic acid bacillus may b used. When the last is employed, a little of each day's milk, one o two teaspoons, may be added to ferment the milk for the next day

The ferment is added to fat-free milk, which should previously have been sterilized. If fat-containing mixtures are fermented, th fat globules may fuse together in the form of butter, which may cause indigestion. After adding the ferment, the milk is allowed to stand in a warm place, the exact temperature not being essential. A little experiment will show how long standing in a given place will suffice to cause souring of the milk with fine precipitation of the casein, without separation of the curd from the whey. When this point is reached, the milk is bottled and put on ice. If it cannot be continuously kept on ice till used, it should be heated to 155° F., in order to kill the ferment.

Lactic acid milk thus obtained is considered to have a formula of

Fat 0 Lactose *below* 4.50 Protein 3.50

The carbohydrate is very variable, depending on how far the fermentation has gone. This food may be diluted in various ways to meet digestive idiosyncrasy. If it be desired to give fat, it is usually better to use alternate feedings of the lactic acid milk and some fat-containing formula, although often a lactic acid milk containing a little cream may be given with safety.

PRECIPITATED CASEIN IN HOME MODIFICATION.—This is obtained as follows: To one quart of skimmed milk add half an ounce of essence of pepsin or liquid rennet, or two junket tablets. Heat to

about body temperature (100° F.), and let stand until the casein has coagulated. Strain off the whey through muslin. The curd is now rubbed through a fine wire sieve. During the process small quantities of water are added to the casein in the sieve. When all the casein has gone through, enough more water is added to bring the quantity of the casein suspension up to 10 ounces. This forms the stock precipitated casein solution.

The calculation is made as follows:—It has been found that the quantity of casein obtained in this way from a quart is 2.60 per cent of the quart. Ten ounces is very little less than one-third of a quart, and therefore the percentage of casein in the 10-ounce stock solution is represented by 2.60 × 3 or 7.8 per cent, which for purposes of calculation is considered to be 8 per cent. If the stock solution contains 8 per cent of precipitated casein, the calculation for obtaining any desired percentage of precipitated casein in any mixture is as follows:—

Divide the percentage of precipitated casein required by 8, and multiply by the total number of ounces in the mixture. This gives the number of ounces of the stock solution to be used.

COOKING MILK MIXTURES.—This procedure requires no specific directions in home modification. The food is simply brought to a boil. It is best to use a double boiler.

HOMOGENIZED MILK.—This very recent method of modifying milk is not available at present in home modification. It is necessary to resort to a hospital or laboratory possessing a homogenizer.

MALT SOUP.—The proportions are one ounce (by weight) of wheat flour to ten ounces of milk. The flour is rubbed smooth with cold milk, and the milk and flour are cooked for twenty minutes, and then allowed to cool. In another vessel three ounces of one of the thick malt extracts are dissolved, with 15 grains of potassium carbonate, in 20 ounces of lukewarm water. This is added to the milk and flour, and the mixture is kept warm for a few minutes, and then boiled for five minutes. After cooling it is ready for use.

This mixture has an approximate percentage formula of

Fat 1.30 Lactose 1.50 Maltose 7.50 Protein 1.00

There is also an amount of starch which is very variable, as some of the starch is dextrinized by the malt extract during heating.

The essential features of malt soup being the cooking of the flour with the milk and the warming with malt extract, any milk modification of known composition may be treated in this way.

ALBUMIN MILK; PROTEIN MILK; EIWEISS MILCH.—Precipitated casein is obtained by the method described under that heading, The amount of water added is up to one pint instead of 10 ounces.

Lactic acid milk is obtained by the method described under that heading, but from whole milk instead of skimmed milk, and one pint is mixed with the casein suspension.

Albumin milk thus made has a constant formula, which can only be altered by dilution, or the addition of sugar. Any combination of percentages, however, can be obtained by combining the 8 per cent precipitated casein solution with lactic acid milk in various proportions.

LABORATORY FEEDING AND HOME MODIFICATION; RELATIVE ADVANTAGES AND DISADVANTAGES.—In a discussion of the factors which guide the physician in choosing whether the infant's food shall be prepared at a milk laboratory or at home, it is necessary to state at the outset, that as far as practical results are concerned, there has never appeared to be any marked superiority on either side. It appears to be an undoubted fact in the experiences of most physicians who have had a large experience with both methods, that most infants can be fed with food prepared at home just as successfully as with food prepared at a milk laboratory. This fact applies to the majority of even the most difficult cases, provided that the home modification be properly carried out. The fact that the laboratory employs centrifugalized cream instead of gravity cream has been urged as an objection to laboratory feeding, on the ground that separated cream is less digestible. No basis has been found for this belief, and it is quite certain that the obtaining of cream by centrifugalization in no way impairs its digestibility.

Accuracy cannot be realized with home modification as with the laboratory. In a food prepared at the laboratory the physician can have the same assurance that the composition of the food is exactly what he ordered, as he has in the case of his drug prescription filled at a reliable pharmacy. That this superiority in the exactness of the percentage formulae has not given evidence of superior clinical results, only demonstrates that such absolute accuracy, while desirable, is not essential for practical purposes. Successful clinical results are due, not to exactness of the composition of the food given, but to the physician's knowledge of the resources at his command in artificial feeding and of the scientific principles on which these resources are based, and to his skill in the diagnosis of the conditions he has to treat, and in the choice among the methods at his disposal.

Accuracy is essential for purposes of research. If the physician is interested not only in obtaining good practical results, but also in adding to his knowledge of the relation of clinical manifestations to various quantities of the food elements, or if he is treating cases experimentally with the idea of publishing his findings, results based on home modification cannot be used, because of inaccuracy.

The principal practical advantage in laboratory modification is its convenience. It spares the physician all the trouble of the mathematical calculations involved in home modification, and spares the mother or nurse the trouble of obtaining and mixing the ingredients. In any case in which there is any doubt as to the willingness or ability of nurse or mother to modify the milk exactly as directed, under conditions of perfect cleanliness, laboratory modification, if available, should be preferred. Furthermore, laboratory modification as carried out by the best milk laboratories in this country, insures that the cow's milk used is of the best, although in many places an equally good milk may be secured for home modification. In short, laboratory feeding secures accuracy, convenience, and cleanliness.

The practical disadvantage of laboratory modification is the cost of the product; this has to be considered in many families. Sometimes the laboratory is available, but is situated at such a distance, that the cost is increased by the expense of transportation.

The wishes of the mother, even if founded on prejudice, must sometimes be considered in making a choice. One often encounters the idea that a skillful mother or nurse, whose "heart is in her work," who is in daily contact with "dear little Tommy," and who is familiar with his "cunning ways," is less likely to make mistakes than a mere paid laboratory clerk who has never even seen the baby. Such an idea, even if false, calls for sympathy, and if the available milk supply is good, if the mother or nurse is skillful, and if special accuracy is not desired, home modification may be chosen. In such a case when greater accuracy is desired, the physician can order standardized cream and fat-free milk from the laboratory, and let the interested nurse or mother do the mixing. The physician should not allow himself to be driven to home modification by any such false prejudice as that the laboratory makes mistakes, or that it dispenses some particular kind of food, but should explain that the purpose of the laboratory is the same as that of the pharmacy, to carry out the physician's prescriptions with accuracy, cleanliness, and convenience. Of course, in very many places, the need of a choice does not exist; no milk laboratory is available, and home modification must be employed. When there is a choice, the physician must take into consideration all the circumstances. He must consider the milk supply available for home modification, the financial and geographical situation of the family, the internal conditions of the household, the complexity of the feeding method to be employed, and whether the particular case calls for especial accuracy.

CALCULATION OF THE CALORIES.—The estimation of the daily calories per kilogram of body weight, for the purpose of com-

parison with the so-called minimum caloric requirement, is often useful in infant feeding, though its utility is rather as a check than as a guide. This calculation is made from the known composition of the food expressed in percentages. The "caloric coefficients" are 9.3 for fat, and 4.1 for carbohydrate and protein. This means that one gramme of fat has a value of 9.3 calories, and one gramme of carbohydrate or protein has a value of 4.1 calories. The calculation is made as follows:

1. Multiply 9.3 by the figure representing the percentage of fat and divide by 100.

2. Multiply 4.1 by the sum of the percentages of carbohydrate and protein, and divide by 100. This gives the caloric value of the carbohydrate and protein in one gramme of food.

3. Add these two results together. This gives the caloric value of one gramme of food.

4. Multiply the food taken in 24 hours, if expressed in ounces, by 30, to reduce to cubic centimeters. Multiply this by the caloric value of one gramme of food. This gives the daily number of calories ingested.

5. To reduce the weight of the baby from pounds to kilograms, multiply the weight in ounces by 30, and divide by 1000.

6. Divide the daily calories taken, by the weight of the baby in kilograms. This gives the daily calories per kilogram of body weight.

As an example of this calculation, let us suppose that a baby weighing 9 lbs. 8 oz. is taking daily 32 ounces of a food, of which the composition is represented by the formula fat 2, lactose 6, protein 2. The steps of the calculation are:—

1. $9.3 \times 2 \div 100 = .186$ Ca. from fat in one gramme of food.
2. $4.1 \times 8 \div 100 = .328$ Ca. from carbohydrate and protein in one gramme of food.
3. $.186 + .328 = .514$ Ca. in one gramme of food.
4. $32 \times 30 = 960$ grammes of food in 24 hours.
   $960 \times .514 = 493.44$ calories ingested daily.
5. 9 lbs. 8 oz. $= 152$, weight of baby in ounces.
   $152 \times 30 \div 1000 = 4.56$, weight of baby in kilograms.
6. $493.44 \div 4.56 = 108$, daily calories per kilogram of body weight.

CALCULATION OF PERCENTAGE COMPOSITION IN A FOOD OF KNOWN INGREDIENTS.—In order to treat intelligently a case of difficult feeding, it is necessary to know all the foods previously used, and how well the baby thrived, and what symptoms it manifested with each food. In order to draw from such a history conclusions which shall serve as a guide in choosing the method of feeding to be employed, it is necessary to know the composition of each previous food as expressed in percentage. In the history of the case, the previous foods are often described, not as percentage formulae, but as mixtures of ingredients. These mix-

tures have to be reduced to a formula representing the composition of the food in percentages of the principal food elements.

To reduce mixtures to their percentage formulae requires that we shall know the composition of each ingredient used. Many of the ingredients met with are creams of different fat percentages, or whole milk, or whey. We can only judge the fat percentage of a cream or milk used, by ascertaining the procedure employed by the mother or nurse in obtaining these ingredients. The percentage of fat in a cream or "top milk" varies with the number of ounces removed from the top of the jar.

The following table shows the average composition as expressed in percentages, of top milks obtained in various ways, and of the other ingredients most commonly met with in the history of cases of artificial feeding.

TABLE 32

Percentage Composition of Various Ingredients Used in Preparing Food for Infants.

TOP MILK

NUMBER OF OUNCES TAKEN FROM QUART	FAT	CARBOHYDRATE	PROTEIN
4 (or less)	20.00	4.00	2.80
6 (or all the cream)	16.00	4.20	3.05
8	12.00	4.36	3.18
11	10.00	4.45	3.27
16 (upper half)	7.00	4.55	3.41

OTHER MILKS AND CREAMS

	FAT	CARBOHYDRATE	PROTEIN
Whole milk	4.00	4.75	3.50
Skimmed milk	1.00	5.00	3.55
Separated cream	32.00	3.40	2.50
Separated fat-free milk	0.05	5.08	3.60
Whey	0.25	5.10	0.90
Commercial buttermilk	0.50	4.06	3.60
Fat-free lactic acid milk	0.00	4.06	3.60
Whole lactic acid milk	4.00	4.00	3.60
Albumin milk	2.00	2.50	3.00
Malt soup with whole milk	1.30	9.00	1.00
Condensed milk	9.61	54.94	8.01

OTHER INGREDIENTS

	FAT	CARBOHYDRATE	PROTEIN
Dry carbohydrate	0.00	100.00	0.00
Malt extract	0.00	75.00	0.00
Barley water	0.00	1.50	0.00

At times some one of the widely advertised infant foods has been used in the previous feeding of a baby. These foods are mainly carbohydrate, though some of them have evaporated or condensed milk as a basis, which gives them a considerable fat and protein content. The composition of condensed milk appears in the table of ingredients.

Of the 54.94 per cent of carbohydrate contained in condensed milk, 42.91 per cent is cane sugar, and 12.03 per cent is milk sugar. Most of the other infant foods are in dry form. Their composition is shown in the following table, taken from Holt.

TABLE 33

The Composition of the Infant-foods. (Holt)

	NESTLE'S FOOD	MELLIN'S FOOD	ESKAY'S FOOD	MALTED MILK	RIDGE'S FOOD	IMPERIAL GRANUM	CARNRICK'S FOOD
	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent
Fat	5.50	0.24	1.16	8.78	1.11	1.04	7.45
Protein	14.34	11.50	5.82	16.35	11.81	14.00	10.25
Cane sugar	25.00	....	....	....	....	....	....
Dextrose	....	....	53.46	....	0.52	0.42	....
Lactose (milk sugar)	6.57	....					
Maltose	27.36	60.80	....	49.15	....	....	....
Dextrins		19.20	14.35	18.80	1.28	1.38	....
Total soluble carbohydrates	58.93	80.00	67.81	67.95	1.80	1.80	27.08
Insoluble carbohydrates (starch)	15.39	....	21.21	....	76.21	73.54	37.37
Inorganic salts	2.03	3.59	1.30	3.86	0.49	0.39	4.42
Moisture	3.81	4.73	2.70	3.06	8.58	9.23	3.42

The calculation of the percentages in the mixture given to the baby from the known composition of the ingredients, is made from the same proportion as was used in home modification—

$$x : a = m : n$$

In this case, the unknown quantity is m, its value being expressed by

$$m = \frac{x}{a} \times n$$

Divide the number of ounces of the ingredient used by the number of ounces in the mixture, and multiply by the percentage of any element in the ingredient. This is repeated for each food element, and for each ingredient.

For example, a mother is preparing her food as follows:—She takes the upper 11 ounces from a quart of milk, and mixes as follows:

Cream, 8 ounces
Skimmed milk, 10 ounces
Lime water, 2 ounces
Barley water, 20 ounces
Dextri-maltose, 4 tablespoons

The total mixture is 40 ounces, and, as will be seen by reference to table 32, the cream is 10 per cent fat.

$\frac{8}{40} \times 10 = 2\%$ fat from the cream.

$\frac{10}{40} \times 1 = .25$ fat from the skimmed milk.

$\frac{8}{40} \times 3.27 = .65$ protein from the cream.

$\frac{10}{40} \times 3.55 = .88$ protein from the skimmed milk.

$\frac{8}{40} \times 4.45 = .89$ lactose from the cream.

$\frac{10}{40} \times 5 = 1.00$ lactose from the skimmed milk.

4 tablespoons dextri-maltose $\div$ 2 = 2 ounces.

$\frac{2}{40} \times 100 = 5\%$ maltose.

$\frac{20}{40} \times 1.50 = .75\%$ starch.

$\frac{2}{18} \times 100 = .11\%$ of the milk and cream for lime water.

Therefore this food represents in percentages—fat 2.25, lactose 1.89, maltose 5.00, protein 1.53, starch .75, lime water 11 per cent of the milk and cream.

PRACTICAL MANAGEMENT OF ARTIFICIAL FEEDING

GENERAL PRINCIPLES

There are certain general principles which govern the management of all cases of artificial feeding.

ENDS TO BE ATTAINED.—The first object in artificial feeding, which should be kept in view as the principal end to be attained, is *to secure the normal development of the infant.* The most useful measure of development is the *progressive gain in weight* which is seen in normal infants, and the absence of which is the most frequent, early and important sign seen in any disturbance. The second object desired in artificial feeding is to keep the infant *free from all symptoms and signs of disturbed digestion.* This second object usually goes with the first, as babies who show signs of disturbed digestion do not gain weight normally. Sometimes, however, a baby may fail to thrive without showing recognizable symptoms of disturbed digestion; while conversely, some babies will continue to gain nor-

mally in weight, even when digestive symptoms are present. The third object is *to develop the digestive powers of the infant.*

CONDITIONS OF THE PROBLEM.—The difficulties encountered in attaining these objects are due mainly to the variation in the digestive powers and nutritive requirements of different babies. To meet these difficulties we are provided with the various methods of modifying cow's milk which constitute our stock of weapons. The problem is to choose our methods so as to attain the desired object. Success depends on our knowledge of the tools to be employed, of the principles underlying their use, and the skill with which we choose among them.

Feeding a baby is always an experiment. The artificial feeding of infants is not, and never will be, an exact science. We know at the start, only that the baby must be fed on some combination of fats, carbohydrates and protein. We know that this food must be given in proper amounts, and at proper intervals. We know that the milk supply used must be as clean as possible. We must decide whether or not the milk is to be pasteurized or sterilized, and whether we shall employ a milk-laboratory or home modification.

The principles which guide us in the decision as to these two last points have already been described. The principles underlying the decision as to the quantity of food to be given each twenty-four hours, and as to the feeding intervals are comparatively simple. The principal problem is to choose the composition of the food.

GENERAL CONDUCT OF A CASE OF ARTIFICIAL FEEDING.—The various steps taken in carrying on a case of artificial feeding are the following:

1. A careful history of the case is obtained. Such a history should include the entire past feeding of the child; if bottle feeding has been used, the composition of each food should be translated into percentages. The regularity, intervals, and amounts of feeding should be inquired into. The reaction of the child to each food used should be ascertained. In particular, such questions as to whether or not the child gained in weight, whether or not it seemed satisfied or hungry, whether or not the bowels moved properly, should be recorded in detail. All the possible symptoms of indigestion, such as vomiting, regurgitation, sour eructation, colic, excoriated buttocks, should be inquired into. The daily number, and character of the bowel movements should be ascertained. From such a history may often be deduced important facts as to the digestive idiosyncrasy of the baby.

2. The initial food, on which the baby is to be started, is now chosen. The basis of choice is any deductions which have been made from the past history, together with the knowledge and experi-

ence of the physician in artificial feeding. If the baby has been previously breast-fed, or if the past history is not of such a character as to permit any deductions influencing the choice, the only guide is the age, weight, and apparent development of the baby.

3. The initial food is always a sort of trial formula. The greater the knowledge of the physician, the more apt is the first food chosen to give a good result. It is, however, mainly on the results observed with this initial food, that the further conduct of the case is based.

4. The composition of the food is changed from time to time. The changes are based on the reaction of the baby to the trial formula first chosen. If the baby shows symptoms of indigestion, and failure to gain in weight, changes are made in the composition of the food, with the purpose, first, of obtaining a gain in weight, and second, relieving symptoms. If the baby gains weight, but shows symptoms of indigestion, the changes in the food are made in the effort to relieve the symptoms. If the baby shows no symptoms but fails to gain, the object of changing the food is to make the baby gain.

5. In artificial feeding, even when a baby is free from symptoms of indigestion, and is gaining weight, we should not rest satisfied indefinitely, but should still change the composition of the food from time to time, with the idea of increasing the burden laid upon the infant's digestive powers, without overburdening them. This is done, because one of the principal objects to be kept in view in artificial feeding, is the strengthening of the infant's digestive power. Any function deteriorates when not exercised. In general, it is always best within certain limits to work a baby up to taking a food of as strong a composition as it can digest.

6. The various changes made in the composition of the food should be carried out in such a way as to enable the physician to learn as much as possible about the digestive peculiarities of the baby. It is often best to change the quantity of just one food element at a time, in order to draw conclusions as to the baby's power of digesting each one.

THE FEEDING OF NORMAL INFANTS

There is no such thing as a normal infant with digestive powers and nutritive requirements which conform to any standard. What is meant by a normal infant, is a baby who has previously had no symptoms of indigestion. Babies who have been previously breast-fed, without signs of indigestion, or babies in whom bottle feeding has failed to reveal any weakness of digestive power, or babies who are artificially fed from birth, are considered normal, when the first choice of artificial food is made. They continue to be considered normal when the first artificial food chosen causes no disturbance. In such a case, after having decided as to home or laboratory modification, and as to pasteurization, we must decide

three points; first, the quantity of the food; second, the intervals of feeding; and third, the composition of the food.

THE QUANTITY OF THE FOOD TO BE GIVEN.—The most important thing is the quantity to be given in the twenty-four hours. The guide in beginning artificial feeding is the age and development of the baby; the latter is measured chiefly by the infant's weight. Table 34 shows the twenty-four hour quantities in ounces for babies of average weight at different ages. The figures are based on the quantities taken by healthy, breast-fed infants. Big babies require more food than small babies.

The guide as to making changes in the quantity after artificial feeding has been begun, is the behavior of the baby. When a baby is not satisfied with the quantity of food given, he shows symptoms of hunger. Such symptoms may be relieved either by increasing the quantity of the food, or by strengthening its percentage composition. Either increase may cause indigestion. If the baby seems satisfied immediately after feeding, but begins to cry and fuss too long before the next feeding is due, the deficiency is more apt to be in the quality of the food, while if the hunger symptoms are seen as soon as he has finished his bottle, increase in quantity is more apt to relieve them. This guide is, however, by no means positive. Generally a baby should be given from the start the full quantity for his weight and age. Then, as a normal baby should preferably have as strong a food as he can digest, the quality should be increased first, until he is either satisfied, or begins to show signs of indigestion. Only when the limit in increase in quality is reached should the quantity be raised.

The amount of food in ounces to be given at a feeding is the next thing to be decided. It would seem at the first glance that the most scientific basis for determining the quantity to be given at a feeding is the gastric capacity of infants at various ages. Two methods have been employed in estimating gastric capacity. The first is by post-mortem measurements of actual capacity; the second is by measuring the quantity of food taken at a feeding by a healthy breast-fed infant. Both have proved rather unsatisfactory as practical guides. The technique of post-mortem measurements is complicated, and the results vary, not only with individual babies, but according to the technique employed. Moreover, it has been clearly shown that the quantity of fluid a baby takes in nursing is not limited by gastric capacity, as a portion of the feeding is passed into the duodenum before the nursing is completed. It would seem that the capacity as estimated by the second method, measuring the quantity taken from the breast, should be a more logical basis for artificial feeding. Here again, however, individual variation plays a very

important part. Also, the standard for breast-fed babies is not applicable to the artificially fed, who are taking a food less ideally suited to their needs. In general, in determining the quantities to be given in each bottle feeding, the figures on gastric capacity based on post-mortem measurements, should be taken as a *minimum*, and those based on the quantities taken by breast-fed babies, should be taken as a *maximum*.

Some rule is necessary as a guide in beginning artificial feeding. The important factors are the twenty-four hour quantity, and the intervals between feedings. The quantities given in table 34 have been based on all the various forms of evidence on the subject. They tend to represent minimum, rather than maximum, quantities.

THE INTERVALS BETWEEN FEEDINGS.—Artificial feeding does not require any different arrangement of the feeding intervals than that of breast feeding. The subject of the feeding intervals has been already discussed. They are shown in the table—

TABLE 34

Quantities and Intervals for Healthy Babies of Average Development

	NUMBER OF FEEDINGS IN 24 HOURS	NIGHT FEEDINGS	QUANTITY AT A FEEDING	QUANTITY IN 24 HOURS	INTERVALS
Premature	24–18	3	⅛- ¾ oz.	3-18 oz.	1-1½ hours
At term	10	1	½ oz.	5 oz.	2 hours
First week	10	1	1 oz.	10 oz.	2 hours
1- 2 weeks	10	1	1¼- 1½ oz.	12-15 oz.	2 hours
2 weeks-1 month	10	1	1½- 2 oz.	15-20 oz.	2 hours
1- 2 months	10	1	2 -2½ oz.	20-25 oz.	2 hours
2- 3 months	8	1	3 - 4 oz.	24-32 oz.	2½ hours
3- 4 months	8 or 7	1 or 0	4- 4½ oz.	28-36 oz.	2½ hours
4- 5 months	7 or 6	1 or 0	4½- 5½ oz.	30-33 oz.	3 hours
5- 6 months	6	0	5½- 6 oz.	33-36 oz.	3 hours
6- 7 months	6	0	6 - 6½ oz.	36-40 oz.	3 hours
7- 8 months	6	0	6½- 7 oz.	40-42 oz.	3 hours
8- 9 months	6	0	7- 8 oz.	42-48 oz.	3 hours
9-10 months	6	0	8 - 8½ oz.	48-52 oz.	3 hours
10-12 months	5	0	9 -10 oz.	45-50 oz.	3 hours

The table is not intended for arbitrary or literal application, but rather as a general guide. For this reason alternatives in arranging the quantities and intervals have been omitted, in order to make the table as simple as possible. In general, the intervals of feeding, and the quantities to be given at a single feeding should be considered as minimum figures. In other words, the change from 2-1/2 to 3-hour intervals, and the omitting of the night feeding, may often be advantageously made at ages earlier than those given in the table. When this is done, the amount given in the 24 hours should be kept constant, and the quantity given at a single feeding should be proportionately increased.

For example, a baby in the second month is to be artificially fed. The table gives 10 feedings of 2 or 2-1/2 oz. each at 2-hour intervals, with one night feeding as the proper routine. At any age the night feeding should be omitted as soon as possible. If the night feeding can be omitted, the baby will have nine feedings of 2-1/2 oz. each, which will give him 22-1/2 oz. in 24 hours, which comes within the limits of the quantity he should take in 24 hours. Often at this age, 2-1/2 hour intervals are better. If the baby is fed at 2-1/2 hour intervals with a night feeding, he will get 8 feedings, of 2-1/2 oz., and 20 ounces in 24 hours, and if he does not need a night feeding, he will get 7 feedings, only 17-1/2 oz. in 24 hours. In such a case the quantity given at a single feeding should be raised to 3 oz.

PERCENTAGE FORMULAE FOR STARTING AVERAGE WELL BABIES.—In choosing the food with which to start artificial feeding, three main principles should be kept in mind.

1. The feeding of normal infants does not require any of the more complicated methods of cow's milk modification. The first method, of milk and cream dilution, with the addition of lactose, usually suffices. The whey modification, however, may often be advantageously used in starting newborn or very young babies.

2. The general relation between the quantities of fat, carbohydrate, and protein, should be based on the relation found in human milk. This means that the food element having the highest percentage in the formula should be the carbohydrate, with the fat next, and protein last. *The percentage of fat should never exceed 4%, and in home modification, to allow for error, 3.50% is a safer limit. The carbohydrate should never exceed 7%* in well babies, nor in sick babies except under the most exceptional circumstances. *The protein should not exceed 3.50%.*

3. It is better to begin with a comparatively weak food, which will probably be digested by the infant, even if such a food does not meet the caloric needs of the infant. It is easy to work up from a weak food to a strong one, if necessary. When indigestion has once been produced, it is not so easy to find the right combination. Infants of the same age show such variation in their power of digesting cow's milk, that if we want the food to produce no symptoms, it is better to begin with a minimum.

The table shows the percentage formulae with which artificial feeding may be begun at different ages.

In the table the unmodified protein and split protein are alternatives. Some very young babies will take a split protein better than an unmodified protein. The probability of advantage is not great enough to make the more troublesome preparation of a split

TABLE 35

Formulae for Starting Artificial Feeding

AGE	FAT	LACTOSE	PROTEIN (UNMODIFIED)	PROTEIN IF WHEY PROTEIN	SPLIT CASEIN	STARCH
First food (after birth).....	1.00	5.00	0.50	0.50	0.25	....
1 week-1 month...	1.50	5.50	0.75	0.75	0.25	....
1- 2 months...	2.00	6.00	1.00	0.90	0.25	....
2- 4 months...	2.50	6.50	1.50	....	....	....
4- 6 months...	3.00	7.00	1.75	....	....	....
6- 8 months...	3.50	7.00	2.00	....	....	0.75
8-10 months...	4.00	6.50	2.50	....	....	0.75
10-12 months...	4.00	5.50	3.00	....	....	1.50

protein formula actually indicated in these young babies. In home modification the minimum casein is not so low as in laboratory modfication, and I usually do not order the split protein in young babies when home modification is to be employed. In laboratory feeding, I usually begin babies under two months with the split protein, and then gradually increase the casein and diminish the whey protein, until the composition of unmodified protein is reached.

In babies over the age of six months, I usually add starch to the modification. At this age, babies require a comparatively high percentage of protein, and they can usually digest starch. Therefore, there is no reason why the favorable colloidal action of the starch should not be taken advantage of. The appearance of the teeth at about the age of six months is Nature's sign that the time is approaching for some other food beside milk. The next addition to the infant's diet is naturally to be starch.

The reason that the percentage of lactose is diminished in the formulae given for babies in the last four months of the first year, is that at this age one is beginning to work up the food of the baby from modified milk to whole milk. This is done by increasing the protein and diminishing the carbohydrate, and babies at this age are started on slightly diminished carbohydrate in preparation for whole milk.

Table 35 does not represent the strength of the food which should be *taken* by average well babies at the different ages, but only the formulae for *starting* them with safety. If these initial formulae cause any symptoms of indigestion, such babies are no longer considered normal, and the future management of artificial feeding comes under the treatment of disturbances of digestion. If the initial formula does not cause symptoms, we should not rest content, even if the baby is gaining in weight, but should increase the strength of the food. The limit of such increase is the digestive power of the infant. But one does not wish to pass this limit and produce symptoms of indigestion. The limit is unknown. There are, however, certain formulae which can usually be taken by the average well baby, on

which the baby would have been started if we had not wished to be on the safe side in beginning artificial feeding.

PERCENTAGE FORMULAE FOR FEEDING AVERAGE WELL BABIES.—This table represents the strength of the food which we should try to give to average well babies, after artificial feeding has been successfully started.

TABLE 36

Formulae for the Feeding of Average Well Babies

AGE	FAT	LACTOSE	PROTEIN	STARCH
1 week-1 month	2.00	6.00	0.75	....
1- 2 months	3.00	6.50	1.00	....
2- 4 months	3.50	7.00	1.50	....
4- 6 months	4.00	7.00	2.00	....
6- 8 months	4.00	7.00	2.50	0.75
8-10 months	4.00	6.00	3.00	1.50
10-12 months	Whole milk with cereal jelly.			

INCREASING THE STRENGTH OF THE FOOD.—The indications for increasing the strength of the food are three. 1. If the baby, being free from symptoms of indigestion, is not gaining, the strength of the food should be increased, provided that the microchemical examination of the stools does not show an excessive amount of fat. If the stools show excessive fat elimination, the baby cannot be considered an average normal infant, and the management of such a case is guided by the principles described under disturbances of digestion. 2. If the baby is gaining, but if the strength of the food is below that usually taken by an average baby of that age, the strength of the food should be increased. 3. If the baby, already taking a food of as great strength as is usually taken by a baby of that age, shows signs of hunger, but no signs of indigestion, increase is indicated.

In well babies, when increase in the strength of the milk modification is indicated, it should be gradual. It is not necessary, however, to increase only one of the food elements at a time. A slight increase, such as for example, an increase of .50 per cent in the fat and sugar, and .25 per cent in the protein, may be made in all three food elements. If indigestion develops, there is always the previous food to fall back upon.

Between the eighth and the twelfth months, the changes in the composition of the food are made with the express purpose of accustoming the baby to taking whole milk instead of modified milk. The percentages of the food elements should be gradually altered in such a way as to approach those in whole milk. For example, if a baby ten months old is taking a food having a composition of—

Fat 3.50 Lactose 7 Protein 1.50

the change should be somewhat as follows:—

1.	Fat 4	Lactose 6.50	Protein 2.00
2.	Fat 4	Lactose 6.00	Protein 2.50
3.	Fat 4	Lactose 5.50	Protein 3.00
4.	Whole milk		

When a baby goes onto whole milk, the milk sugar usually has to be reduced in greater proportion than the protein is increased. This is compensated for by introducing starch into the food, or by adding some cereal to the diet.

In strengthening the food of well babies who are gaining satisfactorily in weight, the changes need not be made oftener than once a week. They are continued until the baby is satisfied, or reaches a food of full strength for his age. If the food is being strengthened because the baby is not gaining, the changes should be made more frequently, twice a week or every three days.

THE FEEDING OF INFANTS HAVING DIFFICULTIES OF DIGESTION.

To this group belong infants who have previously shown symptoms of indigestion, and infants in whom the management of the feeding as recommended for normal infants reveals some abnormality of digestive power. In such cases the choice of food is much more difficult, and there is a demand for a wider use of the resources of milk modification. The various digestive disturbances which may arise in the course of artificial feeding are described and discussed in detail in the division on diseases of the gastro-enteric tract. Certain general principles of treatment having a definite relation to the various methods of cow's milk modification, may, however, be advantageously discussed here.

CAUSES OF DIGESTIVE DISTURBANCE.—The manifestations of difficulty in digestion in an infant are due to a lack of balance between the digestive power of the infant and the composition of its food. The food contains fat, carbohydrate and protein, and the digestive idiosyncrasy of the infant is usually manifested against one or more of these food elements. In certain cases, the quantity of food as a whole may be too great for the infant's digestion. Disturbances of digestion, therefore, are ultimately traced to fat, carbohydrate, or protein, or to overfeeding as a whole.

SYMPTOMS OF DIGESTIVE DISTURBANCE.—Various symptoms manifest themselves when digestion is abnormal. Among the most important are vomiting, colic, sour eructation, irritated buttocks, constipation, diarrhea, abnormal stools, and failure to gain in weight.

DIAGNOSIS.—It is often difficult or impossible to know from the symptoms what is the cause of the trouble. If we could

deduce from the symptoms that the difficulty lies in the digestion of fat, of carbohydrate, or of protein, the choice of the modification promising the best results would be comparatively easy. The only laboratory method of value available is the micro-chemical examination of the stools. This is invaluable, and should be carried out at frequent intervals in every case of digestive difficulty. More valuable than the symptoms in making a diagnosis, are the previous feeding records of the case, including the behavior of the infant under various methods of feeding. From all the evidence, it is often impossible to make more than a guess as to which is the form of indigestion present. The final diagnosis of fat indigestion, carbohydrate indigestion, protein indigestion, and so forth, can often only be made *from the results of our own experiments in the feeding of the case.*

In the meantime we have to treat the case. We wish, as soon as possible, to relieve symptoms and bring about a gain in weight. The only thing we have to go upon is the clinical symptoms, but we must nevertheless choose our initial formula, and our further changes. To do this, it is of value to know what experience has shown to be the best method of guiding the feeding in the face of certain symptoms.

CLINICAL TYPES OF DISTURBANCE SEEN IN ARTIFICIAL FEEDING.—It is convenient to know what methods of milk modification to try, and in what order to try them, in certain types of cases met with in artificial feeding. The following clinical types will be considered:

1. Vomiting the only symptom.
2. Undigested movements the chief symptom.
3. Green or discolored movements the chief symptom.
4. Failure to gain weight the only symptom.
5. Food idiosyncrasy against milk.

VOMITING CASES.—This clinical type may be due to indigestion from any one of the three food elements. Usually when carbohydrate is the cause of vomiting, there are other symptoms beside. Vomiting alone is most apt to be caused by fat or casein. The vomiting of large curds immediately after nursing points toward the casein. Sour vomiting during the interval points toward the fat. In many cases it remains always impossible to recognize the original cause of the vomiting, because the symptom, once started, is apt to persist after the original cause has been removed. The stomach has been left in such an irritable condition, that the ordinary precipitation of the casein in the stomach, or even the taking of any food into the stomach, will cause vomiting. The micro-chemical examination of the stools for fat is of no value in cases of this type.

In feeding a case of this type, try first a food with a fat percentage of zero. For example, a formula containing—

Fat 0 Dextri-maltose 6 Proteid 1.50

If the vomiting is relieved, it is a case of fat indigestion. If vomiting is not relieved, it is either a case of protein indigestion or of habitual vomiting from irritable stomach. In young babies, who have not a long history of vomiting, it is more apt to be due to casein. Even with habitual vomiting which may originally have been caused by fat or carbohydrate indigestion, the precipitating cause is usually the casein. The various methods of milk modification influencing protein digestion should be tried one after the other, in the following order:

1. A split protein formula, such as, fat 1, lactose 6, whey protein .90, casein .25.
2. The same with lime water 50% of the milk and cream.
3. Some other alkali, such as sodium citrate. For example, fat 1, lactose 6, protein 1.50, sodium citrate .40% of the milk and cream.
4. Lactic acid milk.

In making experimental changes in any case of artificial feeding, the changes should not be made oftener than *every three days*, unless some change produces immediately new symptoms of indigestion.

Some cases will do well with small quantities at shorter intervals. If all these measures fail, the case is of the obstinate, resistant type.

As additional measures in cases with vomiting as the chief symptom, daily gastric lavage is of great value. I have seen a few cases of this type relieved by the passage of the duodenal catheter. In cases in which there is vomiting of large curds immediately after nursing, the alkalies often act as a specific.

UNDIGESTED MOVEMENTS.—This clinical type is usually due either to fat, or to protein. The examination of the stools is of the greatest diagnostic value in these cases. If the stools show excessive fat, the treatment is that of fat indigestion; the baby may be started on a formula containing—

Fat 0 Dextri-maltose 6 Protein 2.00

and carried on according to the principles described under indigestion from fat. If the stools do not show an excess of fat, the various methods of influencing protein digestion may be tried in the following order:

1. The use of starch. Fat 2, lactose 6, protein 1.50. Starch .75 may be given.

2. The same formula may be boiled.
3. Lactic acid milk, precipitated casein, or a combination may be tried.

The alkalies are less valuable in this type of protein indigestion.

GREEN OR DISCOLORED MOVEMENTS.—This clinical type is usually due either to fat or to carbohydrate indigestion. The former diagnosis may be confirmed by the results of the micro-chemical examination of the stools for fat. The latter diagnosis is often confirmed by the other symptoms of carbohydrate indigestion, namely, vomiting, colic, sour eructations, and irritated buttocks. In either case, dextri-maltose is generally to be preferred as the extra sugar. Cases due to fat should be started on some such formula as—

Fat 0 Dextri-maltose 6 Protein 1.50-2.00,

and the fat increased according to the principles of treatment in fat indigestion. Cases due to carbohydrate should be started on some such formula as—

Fat 2 Dextri-maltose 4 Protein 1.50-2.00,

and the further changes made in accordance with the principles laid down for the treatment of indigestion from carbohydrate.

If these measures fail, whether the trouble be due to fat or carbohydrate, lactic acid milk, or precipitated casein, or a combination (albumin milk) should be tried next.

NO SYMPTOMS.—In this type the baby neither vomits, nor shows any marked abnormality in the microscopic appearance of the stools. It simply loses weight, or fails to gain. It is necessary to make sure that the loss of weight does not come from insufficient food. If not, these cases are usually due to fat indigestion. They are best started on a formula containing a low fat, a high protein, and with the extra sugar maltose, such as—

Fat 0 Dextri-maltose 6 Protein 1.50-2.00

The further treatment is carried on as for a case of fat indigestion.

FOOD IDIOSYNCRASY AGAINST MILK.—It is very common to hear of cases of infants who supposedly cannot take cow's milk in any form. In the majority of these cases it is not true that cow's milk cannot be digested and assimilated. Most of them are really cases which have some marked peculiarity of digestive power which has defied the ordinary resources of cow's milk modification.

There are, however, cases of true food idiosyncrasy against cow's milk. These cases probably represent a condition in which the unsplit cow's milk protein is for some reason absorbed into the circulation, and the disturbances caused thereby represent an anaphy-

lactic phenomenon. Various symptoms are observed in these milk poisoning cases. The commonest symptom-complex is vomiting and urticaria; sometimes diarrhea is seen. The positive diagnosis of the idiosyncrasy against cow's milk can be made by means of the cutaneous reaction. If the skin is abraded in the manner used for the performance of the von Pirquet tuberculin test, and inoculated with milk, a positive reaction will be obtained. In such cases artificial feeding is impossible, as substitutes for cow's milk are unsatisfactory. Breast milk should always be obtained.

THE FEEDING OF DIFFICULT CASES.—The suggestions given for the management of artificial feeding in infants showing difficulty of digestion, will not solve all the problems presented. The formulae given for starting cases of the various clinical types encountered, are only trial formulae, and even if they relieve the symptoms, often do not meet the nutritive requirements of the infant to an extent sufficient to permit a satisfactory gain in weight. Even after the use of the trial formula considered most available in a particular clinical type, the nutritive value of the food often has to be increased by raising the percentages of some of the food elements. Increasing the quantity of a food element, even of one which has previously been well digested, may cause some new type of indigestion to develop. Thus there are always a certain number of difficult cases which remain to tax the knowledge and resources of the physician.

In managing such cases, every effort should be made to reach the ultimate diagnosis. The changes in the food should be made in such a way as to give the physician new information as to the digestive peculiarities of the infant. For this reason it is usually best in such cases, to *alter the percentage of only one food element at a time*, or to *introduce only one new method of cow's milk modification at a time*, in order that the deductions based upon the reaction of the infant to the changes shall be warranted.

In trying the various available methods of milk modification in these difficult cases, the physician must depend upon his knowledge of the underlying principles of artificial feeding. Success will depend on the skill with which he applies this knowledge to the peculiarities of the case.

At all times in the course of a resistant feeding case, the physician knows that breast milk will in all probability be the best means of meeting the difficulties with which he is confronted. How soon he shall resort to breast milk depends on the severity of the case, and the availability of a wet-nurse.

In a difficult case, the physician must not be wholly guided by the clinical symptoms. There are cases in which the symptoms of

indigestion cannot be relieved at all, or can be relieved only by the use of a food the composition of which is not of sufficient strength to fulfil the nutritive requirements of the infant. Nevertheless in such a case it may be possible to find a combination of food elements on which the infant will gain in spite of the symptoms. Under these circumstances the efforts of the physician to relieve the symptoms should not involve changes in the composition of the food, but should be confined to those methods of milk modification which aim at making the cow's milk more digestible. At some period in the course of every resistant feeding case, it is well to try a comparatively strong formula without reference to the clinical symptoms. This will sometimes save time that would otherwise be wasted in the effort to render the infant symptom-free.

CLINICAL INDICATIONS FOR THE VARIOUS METHODS OF MODIFYING COW'S MILK

The subject of difficult artificial feeding has been discussed from the point of view of the clinical symptoms, with suggestions as to the methods employed in meeting the difficulties encountered. It is often convenient to consider the subject in the opposite order, from the point of view of methods of modification, with suggestions as to the clinical indications and contraindications for each. Such a summary is given here, in the hope that it may prove of service to physicians, in their employment of the resources used in artificial feeding.

The following are the indications and contraindications for the various methods of modifying cow's milk.

1. MILK AND CREAM DILUTION WITH THE ADDITION OF LACTOSE.—This is the fundamental method of varying the quantities of the three food elements. It is indicated in all cases in which some special method, such as split protein or lactic acid milk, is not indicated. It is the basis of the feeding of normal infants, and of cases of fat or carbohydrate indigestion.

2. STARCH.—Starch may be advantageously introduced as a routine into the food of well infants in the second six months. The exact age when starch feeding should be begun, cannot definitely be stated. It is best not to use it as a routine before the age of six months.

With sick infants, starch is theoretically indicated only in cases of protein indigestion. It is the method of first choice in dealing with protein indigestion in the latter half of the first year. It is more valuable in the type of protein indigestion characterized by undigested movements, than in the form characterized by vomiting.

When fat or carbohydrate indigestion is the fundamental condition present, there is usually need to give more protein. Starch is often

useful in such cases, especially in the clinical types characterized by undigested movements, or by green or discolored movements.

3. THE ALKALIES.—The use of lime water as a routine in well babies is no longer considered to be indicated. The alkalies should be used only to meet certain definite indications in disturbances of digestion.

The alkalies are most valuable in protein indigestion with vomiting. In the particular type in which the vomiting of curds follows very shortly after feeding, an alkali will often immediately relieve the symptoms. In protein indigestion, it should first be given in the amount necessary to delay curd formation and modify the character of the curd; if it fails to relieve the vomiting in this amount, it should be given in the amount which prevents precipitation of the casein in the stomach.

The alkalies are also very useful in other forms of indigestion, when vomiting is a prominent symptom. In such cases the vomiting often becomes persistent, through irritability of the stomach. The alkali, given in sufficient amount, will prevent the irritation of a sensitive gastric mucosa by the precipitated curd. The alkali may be used in combination with either split protein formulae, or starch-containing formulae.

There are no indications for the use of any particular one of these alkalies. In some cases lime water seems to work best, in others sodium citrate, in still others, sodium bicarbonate. One cannot tell beforehand which will work best.

In some cases, with green irritating acid stools, an excess of lime water, as 25 per cent of the total mixture, has given clinical evidence of good results.

The alkalies have no contraindications, but have not given evidence of favorable action in other types of digestive disturbance.

4. PEPTONIZATION.—This method of modifying milk constitutes a resource to be tried only in a resistant case, in which other more promising methods have failed. The type of case in which it is occasionally of benefit, is that characterized by persistent vomiting.

5. WHEY MIXTURES.—In very young well babies, whey mixtures are often of advantage. They enable us to give more protein without risk of causing symptoms. Whey mixtures should also be used in the feeding of premature babies.

Split protein formulae are indicated in protein indigestion. Protein indigestion is much commoner in young babies than in older ones. The split protein is the method of choice in dealing with protein indigestion in young infants. It gives the best results in cases characterized by vomiting.

In other forms of indigestion it is often necessary to give more

protein to compensate for deficiency in the power of assimilating fat or carbohydrate. In young babies, the increased casein is apt to cause vomiting. The whey mixtures enable us to increase the protein without increasing the casein.

In cases of persistent vomiting from gastric irritability at any age, the split protein combined with alkali should be the first measure chosen.

The split protein has given no evidence of having any value in those types of indigestion characterized by undigested movements, or by the absence of symptoms other than loss of weight.

There are two important *contraindications* for the use of the split protein. These are, first, acute diarrheas, and second, that type of indigestion characterized by green or discolored movements. Clinical experience has shown repeatedly that these conditions fare badly when fed with whey mixtures. In both conditions, any tendency toward intestinal fermentation appears to be increased, possibly because the soluble protein is a favorable culture medium, or because of the high lactose and salt content of the whey. The explanation is not clear.

6. THE CARBOHYDRATE.—Lactose is indicated as the extra carbohydrate to be added to milk modifications in normal babies, and in all cases with comparatively slight disturbance of digestion. It is indicated in infectious diarrhea due to the bacillus of dysentery, and in the form of acute diarrhea due to abnormal intestinal fermentation, with protein decomposition and toxic symptoms. It should be used in all cases in which there is no indication for maltose.

Maltose is indicated in very difficult feeding cases, and in severe cases of malnutrition and atrophy. It is part of the routine in the treatment of chronic indigestion from fat. Carbohydrate indigestion is most frequently seen in cases fed on lactose or on cane sugar; in such cases, maltose is indicated. In some cases, carbohydrate indigestion develops in babies fed on maltose, usually in excessive quantity. In such cases, changing the carbohydrate to lactose is usually beneficial.

7. LACTIC ACID MILK.—This is indicated as a therapeutic measure in two forms of diarrhea,—infectious diarrhea due to the gas bacillus, and acute diarrhea with toxic symptoms due to intestinal putrefaction.

As a feeding method, lactic acid milk is a valuable resource in certain difficult types. It should be tried in cases of fat indigestion, when the usual treatment with low fat, high protein, and maltose, is not successful. It is often of value in severe carbohydrate indigestion. It is most useful in the clinical type characterized by green or discolored movements, and should be the first method tried

in such types, when the usual methods of modifying cow's milk reveal difficulties.

It is usually not well borne when vomiting is a prominent symptom, or when habitual persistent vomiting has developed.

PRECIPITATED CASEIN.—This is used only in difficult feeding cases. It is an alternative to lactic acid milk, or may be used in combination with it (albumin milk). The indications for precipitated casein are the same as those of lactic acid milk.

COOKING.—This measure has only one definite indication, namely, the appearance in otherwise normal stools of large casein curds. It may be tried in any very resistant case.

HOMOGENIZED MILK.—The indication for this method is still only theoretical. The method was developed to meet certain resistant cases of fat indigestion, in which the baby cannot through any known method of modifying milk digest and absorb enough cow's milk fat to meet his nutritive requirements.

INABILITY OR REFUSAL TO TAKE FOOD FROM THE BOTTLE

Certain babies are unable to take their food, or sufficient food, from the bottle, on account of great weakness. In these cases feeding must be forced. If the difficulty involves their power of sucking, but not their power of swallowing, the food is best given with the "Breck Feeder." This consists of a graduated glass tube, drawn out at one end so as to be small enough to be fitted with a small rubber feeding-nipple. A compressible rubber cap goes over the large end after the tube is filled. The nipple is put in the baby's mouth, and its presence encourages efforts at sucking. If these efforts are ineffectual, the nurse compresses the rubber cap, gently forcing the milk through the nipple into the baby's mouth, and thus helping out his feeble efforts. It is always best to continue the sucking reflex in these cases.

If through greater weakness, or unconsciousness, or some other cause, the baby is unable to swallow, he must be fed by means of gavage.

In some cases, not at all uncommon, babies refuse their food. They are perfectly able both to suck and to swallow, but they do not want the bottle, and resist any effort to make them take it. They may refuse either the whole or a part of a feeding, and may refuse some or all of their daily feedings. This symptom may of course be due to the coming on of one of the common acute diseases. Apart from such recognizable diseases, I have found three common causes for this condition. These are, first, dentition, stomatitis, or any localized sore mouth; second, early scorbutus; and third, the baby may become tired of the taste of his food. Stomatitis and scurvy

call for the usual treatment of those diseases. In dentition, applying cold water to the gums immediately before feeding will often cause the baby to take his feedings better. If the baby is tired of his food, some minor change in the formula will often satisfy him; in particular, changing the extra sugar from lactose to maltose, is often completely curative.

In some cases of refusal of food, the cause may not be discoverable, or the condition may prove to be not easily remediable. The physician must then decide to what extent he shall allow the baby to refuse feedings without interference. The mother is often much worried by this symptom, and she should be cautioned against tempting the child with food in the intervals between his regular feedings. To what extent the child shall be allowed to refuse his feedings depends on a number of circumstances, such as the cause of the condition, the amount of food refused, and the general condition of the child. In general, temporary conditions of short duration require no interference. When the condition is of longer duration the child's nutrition must not be allowed to suffer too much. Sometimes one tube feeding will relieve the condition. Usually, when the child's nutrition begins to suffer, I am accustomed to order that the child shall take a certain minimum quantity of food daily. If he has not taken the right proportion of this minimum by a certain hour each day, tube feedings are to be used to the extent of insuring the taking of the desired minimum.

In dentition, acute disease, and similar conditions, the digestive power of the infant is frequently lowered, and his refusal to take his feedings is Nature's way of guarding his digestive system from an overload. Great harm can be done in such cases, by forcing the feeding.

IV. FEEDING IN THE SECOND YEAR

FEEDING OF HEALTHY INFANTS

There is no subject about which so much ignorance prevails, as the feeding of infants during the second year of life. It has become fairly well known that cow's milk has to be carefully modified and prepared for infants in their first year, but as soon as the twelfth month has been passed, and the infants are able to digest whole milk, there seems to be a very prevalent idea that they can digest almost anything. A few children are underfed in the second year, but the great majority are overfed, with badly prepared, unsuitable food carelessly given at improper intervals. The acute diarrheas so prevalent in the summer months, are very common in infants between one and two years of age, so much so that the "second summer" has come to be a much dreaded period. The dangers of the second summer are mainly due to improper feeding, and can be entirely prevented by proper management.

Among the mistakes most commonly made at this period of life, is the practice of allowing the infants to eat between meals. As long as the feeding of the infant involves the labor of administering the breast or the bottle, mothers are less likely to err in this particular; as soon as the baby is able to take food in its hand, and eat it, this involving no labor on the part of the mother, it begins to get crackers, cookies, or a slice of bread and butter at all times, this being regarded as a legitimate means of keeping it quiet. Not only crackers, but drinks of milk are given between meals, under the idea that drink is not food. As a result of all this, the child begins to take less food at meals, and its feeding becomes a continuous nibbling, which keeps its digestive system in a state of constant stimulation, with most ruinous results. Children in the second year should never be allowed to eat between meals. Even the drink of milk, or the "educator" cracker is harmful.

A second mistake, is irregular hours for meals, or improper intervals between meals. This error is sometimes committed through carelessness, and sometimes in the effort to make some of the infant's meals coincide with those of the parents, so that the infant can come to table, sitting in its "high chair." Beside the tax on the infant's digestive power which such irregularity of feeding intervals entails, the fact that the baby comes to table leads to the practice of giving it "tastes" of the food provided for the rest of the family. The idea that little tastes of these various foods can do no harm, is a very

mistaken one. In this practice the father is usually a more frequent offender than the mother, a father often being very proud that his boy of fifteen months likes the same articles of diet as himself. Another evil in having the infant take its meals with its parents, is that special care is not devoted to the preparation of the food for the infant, but many articles of diet suited only to the adult are given to the infant under the mistaken theory that if he is healthy, they can do no harm.

An infant in the second year should have his meals by himself, at regular and proper intervals. There is no reason why he should not eat with older children, when their feeding time falls at the same hour. The infant's food should be specially prepared for him. If the family cannot manage a separate dietary for their young children and the older members, it is better that the older members should eat what is good for the children, than that the children should be forced to eat what the older members like.

One common mistake made in the feeding of infants in their second year, is that they are not given sufficient milk. As they begin to take other foods, some of which they often like better than milk, the quantity of milk in their diet is gradually and often unconsciously cut down. *Milk should be the basis of the infant's diet throughout the second year.* The popular idea that many children cannot take milk, has no foundation in fact, food idiosyncrasy against milk being rare. The other articles of diet should always be additional, and should never take the place of milk.

Another fault in the feeding of infants is the giving of sweets. This I believe to be one of the commonest, if not the commonest cause of disturbed nutrition in all periods of childhood after the end of the first year. The symptoms of excessive sugar ingestion develop so insiduously, that they are rarely recognized as pointing toward the digestive system. Candy, even at meals, should never be given to infants in the second year. I am inclined to go much farther than this, and do not believe that sugar should be added to the diet at all. It may be true that the majority of children can take reasonable quantities of cane sugar without harm, but sugar is by no means a necessary component of the child's diet. The child gets all the carbohydrate it requires in the lactose of its milk, in the starch of its bread and cereals, and in the fruits which are added later. Sugar can be absolutely dispensed with, without harm. The trouble with sugar is that young children like it, and once having tasted it, want more and more of it. Under these circumstances, it is very difficult to keep it in moderation, for the young child well knows how to get what it likes, and is an adept at bullying its parents into giving it what it likes. Once having tasted sweets, it is very apt to begin to refuse to take its other arti-

cles of diet, and the feeding of the child becomes a struggle, a conflict in which the child is apt to win. If children do not *know* the taste of sugar, much trouble is avoided.

Underfeeding is sometimes met with in the second year. The commonest cause is keeping the infant too long on the breast only, or on an exclusively milk diet.

The various solid articles of diet given in the second year should be added gradually, one at a time, and at first in very small quantities. Sometimes the expansion of the diet list is attended with considerable difficulty. The children refuse to chew and swallow solid foods, and are thoroughly satisfied with milk. In such a case, it is often well for a time for the child to have its meals with its parents or with older children, and to see them eat. Children learn rapidly by imitation. They should be given every opportunity to eat solid food, but should not be forced. Milk, for a time, must be kept in the background, and the more solid articles given first. Often much time and patience are required before they will eat.

WEANING FROM THE BOTTLE.—At the beginning of the second year, weaning from the bottle should be started. This is sometimes a matter of considerable difficulty, the infants refusing absolutely to take milk from a cup. It can only be accomplished by patience. It is best to begin with the early morning feeding, and then gradually to extend the use of the cup to the other feedings. The cup should be offered, and if refused, the bottle should not be given till after an interval of fifteen or twenty minutes. It is advisable not to begin to add much solid food to the diet until weaning from the bottle has been started. The use of the bottle at the 10 P. M. feeding may be continued, for convenience, until this feeding is omitted. Except for this feeding, weaning from the bottle should be completed before the fifteenth month.

PREPARATION OF FOODS FOR INFANTS IN THE SECOND YEAR. MILK.—In the majority of cases the milk used as the basis of the diet in the second year, does not require modification. The same precautions should be taken as to the quality of the milk used. The decision as to whether the milk shall be pasteurized is to be made on the same grounds as in the first year. Cream should not be given, except in special cases, such as constipation.

ZWIEBACK.—This is somewhat more digestible than bread. The unsweetened variety should always be used. The zwieback made by the National Biscuit Company is excellent.

CEREAL JELLIES.—Oatmeal, four ounces, is added to one pint of water, and boiled for three hours in a double boiler. Enough water is added to form a thin paste when cooking is completed. While

hot, this is forced through a colander, and a semi-solid mass is formed. Only the regular Scotch oatmeal, not rolled oats or any similar preparation, should be used.

Barley jelly and wheat jelly are made in the same way, using barley flour, or cracked wheat instead of oatmeal.

Salt should always be added to cereal jellies, but they should be given without sugar.

BEEF JUICE.—Broil a round steak very rare, cut into small pieces, place in a lemon-squeezer or meat press, and press out the juice; add a little salt.

BROTHS.—Take one pound of meat free from fat, and cook for three hours in a quart of water, adding a little water from time to time. When the cooking is completed, there should be a pint of broth. Let the broth cool, remove the fat, strain, and add salt.

SCRAPED BEEF.—Broil a round steak very rare. Split the steak, and scrape out the pulp with a dull knife.

CEREALS.—Oatmeal and hominy should be strained to make a jelly, until toward the end of the second year. Farina, cream of wheat, and wheatena should each be cooked two hours, and need not be strained. All cereals should be cooked with salt, but served without sugar.

EGG.—Soft boiled, poached, or coddled are the only ways of preparing egg permissible for young children.

JUNKET.—To a pint of milk add one tablespoon of essence of pepsin or liquid rennet, or a junket tablet. Heat to 100° F. Allow to stand until the curd is set, then place and keep on ice.

DIET FROM THE TWELFTH TO THE FIFTEENTH MONTH.—The following schedule may be adopted for the feeding of an infant from the twelfth to the fifteenth month:

TABLE 37

Feeding from the Twelfth to the Fifteenth Month

FOUR OR FIVE MEALS DAILY

6:30 A. M.	(Minimum)	Milk, 8 ounces. Zwieback, gradually increasing to one whole piece.
	(Maximum)	Stale bread and butter, one piece, in place of zwieback.
9:00 A. M.	.....	The juice of one-half orange.
10:00 A. M.	(Minimum)	Milk, 8 ounces. Zwieback, increasing to one whole piece.
	(Maximum)	Oatmeal or barley jelly, one tablespoon, increasing to two tablespoons.

TABLE 37—*Continued*

2:00 P. M.	(Minimum)	Milk, 8 ounces. Zwieback, increasing to one whole piece.
	(Maximum)	Beef juice, one increasing to two ounces, or beef, mutton, or chicken broth, one increasing to four ounces. Scraped rare beef mixed with stale bread crumbs, and moistened with beef juice, beginning with one teaspoon, and increasing to a tablespoon.
6:00 P. M.	(Minimum)	Milk, 8 ounces. Cereal jelly, one increasing to two tablespoons.
	(Maximum)	Zwieback, or stale bread and butter.

The first two additions to the child's diet are zwieback and orange juice. I have found zwieback the most digestible form of starch-containing food which can be given at this time. It can be soaked in milk at first, but later the children usually prefer to hold it in the hand and nibble at it. Only a small piece should be given at the beginning, and later increased. During the last part of the first year, babies are weaned from the bottle, or are gradually brought from modified milk to whole milk. While they are still on modified milk, starch should be given in small quantities by using barley water as a diluent in their milk. Zwieback may be given even then, if they are a year old, or seem hungry. In any case, zwieback should be begun when they go onto whole milk, provided that they have teeth.

Orange juice may often be advantageously given before the child is a year old. As a routine, it should be begun at one year.

The schedule provides for a minimum and a maximum diet at each meal. The minimum diet refers to what is added to the diet in the beginning, the zwieback being given first, and then the cereal jelly at supper. The maximum diet shows the diet reached at the end of the period by gradual additions. The morning cereal should be added first, then the beef juice or broth, then the bread and butter, finally the scraped beef.

Many authorities begin to give egg during this period, substituting it for the scraped beef. In my experience scraped beef is less likely to cause disturbance than egg at this period of life.

DIET FROM THE FIFTEENTH TO THE EIGHTEENTH MONTH.—The following schedule may be used during this period:

TABLE 38

Feeding from the Fifteenth to the Eighteenth Month

FOUR MEALS DAILY

6.30 A. M.	.....	Milk, 8 to 10 ounces. Zwieback, or bread and butter.
9:00 A. M.	.....	The juice of one orange.
10:00 A. M.	(Minimum)	Milk, 6 to 8 ounces. Cereal (oatmeal or hominy) strained, two to three tablespoons, with milk. Zwieback, or stale bread and butter.
	(Maximum)	Egg, one (soft boiled, poached, or coddled).

TABLE 38—*Continued*

2:00 P. M.	(Minimum)	Milk, 6 to 8 ounces. Beef juice, or mutton, chicken, or beef broth with rice or stale bread broken in. Scraped rare beef with bread crumbs, one tablespoon. Bread and butter.
	(Maximum)	Stewed prune pulp, baked apple, or apple sauce.
6:00 P. M.	.....	Milk. Bread and butter, or bread and milk. Cereal, (farina, cream of wheat, or wheatena), one increasing to three tablespoons.

The only new articles not added to the maximum diet of the preceding period, are the egg, the cereal instead of cereal jelly at supper, and the fruit. The cereal and egg may be added first, but the fruit should be tried very cautiously.

DIET FROM THE EIGHTEENTH TO THE TWENTY-FOURTH MONTH.—The following schedule may be used during this period:

TABLE 39

Feeding from the Eighteenth to the Twenty-fourth Month

FOUR MEALS DAILY

7:00 A. M.	.....	Milk, 8 to 10 ounces. Bread and butter. Cereal. Egg.
9:00 A. M.	.....	Orange juice.
11:00 A. M.	(Minimum)	Milk. Bread and butter. Chicken, beef, or mutton broth with rice or stale bread.
	(Maximum)	Custard, or cornstarch, or plain rice pudding, or junket.
2:30 P. M.	(Minimum)	Milk. Bread and butter. Scraped beef, or the heart of a lamb chop cut up fine, or chicken. Baked apple, or apple sauce.
	(Maximum)	Spinach, or squash, or stewed carrots, or mashed cauliflower. Baked potato added toward end of the second year.
6:00 P. M.	.....	Milk. Cereal. Bread and butter.

Certain changes in the arrangement of the meals are made in this schedule, in preparation for the child having later three principal meals daily. Babies at this age are apt to sleep later in the morning, and the first meal becomes a regular breakfast at seven o'clock. The second meal is made relatively light, and the third meal will later become the dinner. The new articles should be only gradually added to the diet. Potato should not be given before the twenty-first month, and sometimes cannot be digested till the end of the second year.

FEEDING IN DIFFICULT CASES

The tendency to chronic digestive disturbance and malnutrition has not been outgrown when the infant reaches the second year of its life. Its digestive apparatus is still relatively undeveloped, and requires the most digestible foods. While the number of cases of difficult nutrition met with in the second year is smaller than in the first, such cases are by no means uncommon. Some of them are seen in infants whose nutrition has been a matter of difficulty from birth, and who have been brought through the first year only by the exercise of the greatest care. Others have had their digestive power badly damaged by improper feeding. Still others enter the second year apparently normal, and with every promise of normal development, but soon after weaning begin to do badly.

The factor of individual variation in digestive power still plays an important part in the second year. A diet which is exactly suited to the majority of infants may be wholly unsuitable to an infant with impaired digestive power. Infants may enter the second year with impaired digestive power, and although given an average diet, may yet be relatively overfed. On the other hand, many of the cases of difficult nutrition in the second year entered this period of life with normal digestive power, and their trouble is due to injudicious feeding after weaning. It is often difficult in a given case to say which factor predominates, weakness of digestive power, or faulty feeding.

There are two clinical types of chronic disturbance which are especially common at this period. In the first, the symptoms are very insidious, and do not point very directly at the digestive apparatus. In these cases, loss of weight is not marked, but the children become generally run down; they become pale, and their muscles become flabby. Loss of appetite is a prominent symptom. There is often a chronic cough, which, with the other symptoms, leads the parents to suspect tuberculosis. In other cases of this type, attacks of abdominal pain are a prominent feature. These patients usually have coated tongues, prominent abdomens, and clay colored stools.

The cause of this type is relative overfeeding with carbohydrate. It is the type of indigestion seen in the candy eaters, or in children who eat an excess of sweets. In some children any sugar in the diet may be an excess and cause this clinical picture to develop. In other children, no sugar may be given, but the carbohydrate foods may be in relative excess. Children, who, on taking solid food, take too little milk, or children who eat between meals, are common subjects of this type of nutritional disturbance.

This type is not really difficult, or resistant to proper treatment. The difficulty lies in recognizing it. All sugar should be cut out of

the diet, and the carbohydrate should be reduced to a minimum. I usually cut out all cereals, the diet being milk, beef juice, scraped beef, a little bread or zwieback, and in older babies, egg. Small doses of tincture of nux vomica are often of service in combating the persistent refusal to take food, which is sometimes a troublesome feature in these cases.

The second type is much more resistant and difficult to treat. First there is only failure to gain in weight, then increasing loss. Sometimes these cases may even go on to severe atrophy. The symptoms of gastro-intestinal disturbance are not marked. The stools are often abnormal in color, but are usually large, smooth, and rather dry, with a peculiarly foul odor. Microchemical examination usually reveals a deficiency in the absorption of the fat. The babies having this disorder are apt to be very irritable and cross, and this is increased when more food is given. They often have a slight fever, which disappears when their diet is restricted.

The cause of this condition is overfeeding. The original damage cannot usually be traced to any one food element. Cases of this type are seen most frequently in children who have been overfed in all respects, who have a highly varied diet, including tastes of rich foods. The disease manifests itself in a general deficiency of digestion and absorption, which appears most striking in the case of the fats. Carbohydrates also are poorly borne.

This type of disturbed nutrition is difficult to treat. When the diet is restricted, the infants may be freed from the irritability and slight fever, and the character of the stools may improve, but they lose weight rapidly. When additions are made to the diet, the symptoms return, without gain in weight. The main principle of treatment is restriction of fat and carbohydrate. To what extent this shall be carried depends on the severity of the case. In a severe case of this type, all carbohydrate foods, such as bread, zwieback, and cereals, must be prohibited, and milk must for a time be stricken from the diet, which consists of beef juice and scraped rare beef. Many cases improve on this diet, but it cannot be continued long. Some cases seem to become worse. The next addition is maltose, given first in 6 per cent solution. Then fat-free milk containing 6 per cent maltose is added. The rest of the treatment is the very gradual adding of fat to the milk, and starch to the diet, proceeding very slowly and cautiously, with frequent examinations of the stools.

Occasionally a definite intolerance toward fat or protein of cow's milk, like that seen in the first year, occurs in the second year. In such cases the milk must be modified as in the feeding of similar cases in the first year.

V. FEEDING AFTER THE SECOND YEAR

The diet after the expiration of infancy, gradually approaches that of the adult. It must be remembered, however, that during the entire period of childhood, the digestive functions remain undeveloped in comparison with the adult. Therefore throughout childhood certain kinds of food, which lay the greatest tax on the powers of digestion should be avoided. As the child grows older, the diet should be gradually enlarged, but must always be restricted. Many parents believe that children need a great variety in their diet. This is not true, but restrictions are difficult, if too much variety has once been allowed. The following table shows in a form convenient for reference, the foods which are allowed, and those which are forbidden during the greater part of childhood. Of course, toward the end of childhood, the restrictions can be gradually relaxed.

TABLE 40

Diet List for Children

ALLOWED	FORBIDDEN
Milk	Tea
Cream	Coffee
Stale bread	Cocoa
Graham bread	Soda water
Crackers and biscuits	Hot bread and rolls
Eggs (not fried)	Griddle cakes
Cooked cereals	Sweet cakes
Butter	Ready-to-serve cereals
Plain soups	All fried food
Beef juice	Ham
Beefsteak	Pork
Roast beef	Sausage
Roast lamb	Salt fish
Mutton chop	Fried fish
Chicken	Corned beef
Fish, baked or boiled	Dried beef
Potatoes (not fried)	Meat stews
Rice	Meat dressings
Spinach	Kidney
Asparagus tips	Liver
Stewed celery	Goose
String beans	Duck
Carrots	Fried potatoes
Fresh peas	Cabbage
Squash	Raw or fried onions
Mashed cauliflower	Tomatoes
Boiled onions	Beets
Junket	Egg plant
Custard	Green corn
Cornstarch pudding	Lettuce
Rice pudding (without raisins)	Cucumbers
Ice cream (rarely)	Raw celery
Oranges	Radishes
Baked apples	Salads

TABLE 40—*Continued*

Apple sauce	Candy
Stewed prunes	Pies
Peaches	Tarts
Pears	All pastry
Grapes (without seeds)	Preserves
	Nuts
	Dried fruits
	Raw apples
	Bananas

Many authorities believe that four meals should be given daily during the third year, and even until the sixth year. I believe that with most healthy children, three meals are better. The children have better appetites, digest better, and their daily schedule of exercise, airing, and naps, is more easily arranged. Nevertheless, especially in the third year, many children will thrive better, if a light lunch be given at about 3 or 3.30 P. M. With delicate children, this light fourth meal often has to be continued until the seventh year. This lunch should consist of a glass of milk, and some crackers, or a cup of broth and zwieback.

The meals for the earlier years of childhood should be arranged as follows:

Breakfast—7 to 8 A. M. Cereal with milk; eggs; bread and butter, or biscuit and butter; a drink of milk. On certain days a lamb chop or some hashed chicken may be substituted for the egg.

Dinner—12 o'clock. Soup or broth; meat, or when meat is given for breakfast, fish or egg; potato; vegetable; bread and butter; baked apple, apple sauce, or prunes; milk.

Supper—5.30 to 6 P. M. Cereal; zwieback or bread and butter; junket, custard or pudding; milk.

DIVISION V

DISEASES DUE TO DISTURBANCES OF METABOLISM OR TO CONSTITUTIONAL ANOMALIES

In this division are grouped a number of diseases of which the etiology is not definitely known. They are probably due to various abnormalities of the chemistry of the body, and consequently might be defined as constitutional diseases. The most probable cause of chemical abnormality is disturbance of metabolism, and this is the probable pathogenesis of the majority of the diseases of the group described in this division. In the case of hemophilia, however, the probability is that the disease is due to the direct inheritance of a constitutional abnormality, which involves the chemistry or structure of certain of the body tissues. In the other conditions, the cause of the chemical abnormality being unknown, it is attributed to disturbed metabolism. Whether or not an inherited constitutional abnormality plays a part, and if so, to what extent, and whether or not the chief part is played by causes acting from outside the body, and if so, to what extent, are unknown. Whatever the cause, these diseases present manifestations which strongly suggest that the tissue lesions, or functional disturbances produced, are due to disturbed metabolism.

Abnormality of metabolism produces two kinds of effects. The first effect is *disturbance of nutrition*. This may affect the body as a whole, or may affect certain particular tissues of the body. The result of disturbance of nutrition in childhood is an abnormality of development, with characteristic tissue lesions. The most conspicuous example of this effect of disturbed metabolism is the disease rachitis.

A second effect of an abnormality of metabolism is the formation of toxic products, resulting in a *toxemia*. The results of toxemia are again two-fold, one being the production of definite tissue lesions from the action of the poison, as in scorbutus, while the other result is a disturbance of function, as seen in spasmophilia.

The following scheme illustrates the probable pathogenesis of the diseases of this group:

RACHITIS

(Rickets)

Rachitis, or rickets, is a constitutional disease, with definite anatomical lesions, which certainly represent a disturbance of nutrition. This in turn is almost certainly due to a disturbance of metabolism. All the organs and tissues are involved to some extent in the nutritional disturbance, but the principal lesions are in the bones. The nutritional disturbance produces the characteristic skeletal lesions by affecting the anatomical growth and development of the osseous tissue. As the lesions are the central feature of the disease, and as a knowledge of the tissue changes are essential to an understanding of the theories of the etiology of the disease, the pathological anatomy and chemistry of rickets will be described first.

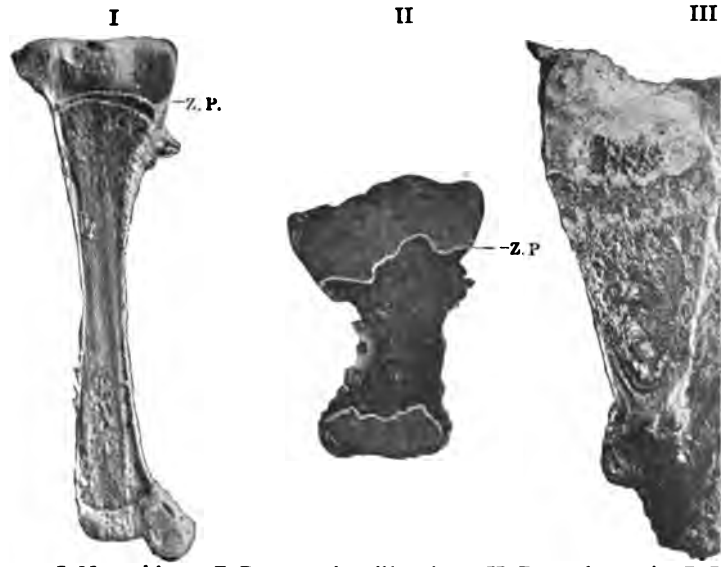
PATHOLOGICAL ANATOMY. BONE LESIONS.—The pathological lesions of rachitis are represented chiefly in the bones, and occur during the period in which the normal processes of ossification are most active,—that is, during the first year and the first part of the second year of life.

The normal growth of bone depends upon four conditions: (a) multiplication of cartilaginous cells in definite lines, followed by (b) calcification of the intercellular spaces; (c) the formation of medullary spaces by the penetration of the blood vessels, with subsequent absorption of tissue; and (d) finally, the concentric deposition of calcium within the medullary spaces. The bones grow in length by the production of bone-tissue in the cartilage between the epiphysis and the diaphysis, and in thickness by the growth of bone from the inner layers of the periosteum. At the same time the medullary canal is enlarged in proportion to the growth of the bone by the absorption of its inner layer. These processes progress in definite order and in clearly defined zones.

In rachitis the chief microscopic features are the changes which occur in the zones of growth and the asymmetrical character of the proliferative processes. The cartilaginous and sub-periosteal cell-growth which produces ossification goes on with increased rapidity and in an irregular manner both between the epiphysis and the dia-

physis and beneath the periosteum. If we examine micro
the region between the epiphysis and the diaphysis, usua
the zone of proliferation, we find that the cartilaginous cell
regularly arranged in rows around a definite zone in advan
ring of ossification, as in normal tissue, but there is an
heaping up of cartilaginous cells, sometimes in rows, somet
covering an ill-defined irregular area. This zone of prolifera
instead of being narrow and sharply defined, is quite lackir
formity. It presents a broad, reddish-gray appearance, wit
thickening and hyperemia. The medullary spaces are mu
vascular than normal, and are so increased in area as to ex

FIG. 81



I. Normal bone: Z. P., zone of proliferation. II. Bone of a cretin: Z. P
of proliferation. III. Rachitic bone: Z. P., zone of proliferation

the zone of calcification, and sometimes through it. Th
of calcium within these spaces is, however, either absent
irregular, and is for the most part replaced by a soft, fri
stance, consisting of a bone-tissue that is very lacking in l
with cells of various kinds embedded in a fibrillated ground-s
This tissue is called "osteoid," and is similar to that fo
osteoblasts.

In the region of ossification (ends of diaphysis and e
there is microscopically a pronounced increase of blood-ve
cartilage-cells, with lengthening of cell columns, and distu
calcification of the intercellular substance. Calcification, i
may be isolated in the region of the proliferating cartilagin

or may be altogether absent over considerable areas. The subperiosteal layer of cells, which is normally thin and scarcely noticeable macroscopically, becomes hyperemic and thickened, with an appearance similar to that of spleen-pulp. Beneath this periosteum is also to be found the "osteoid" tissue seen in the zones of proliferation.

The medulla of the bone is more hyperemic even than normal. Its tissue is rich in cells, and like the fetal medulla, contains dilated

FIG. 82

Spindle-shaped rachitic bone

vessels and fat. The intercellular substance may show mucoid degeneration or even be of fluid consistency. In such a condition it does not seem that lime is dissolved from the bone tissue by the blood, but it is the resorption of such bone *in toto* that is the important factor in the process. Resorption at the age at which rachitis occurs is normal. In rickets, rapid cases of softening certainly

show increased resorption. Ordinarily, with a resorption not greatly increased, the formation of fresh bone containing but little lime results in loss of strength. In the skull, in some places, absorption predominates (occiput); in other cases accretion of osteoid tissues (frontal and parietal eminences). Deficient bone-growth is the simple cause of enlarged fontanelles. In convalescence, lime is deposited in the previous limeless osteoid tissue, and the result is a thick and heavy bone. In fractures at this period callus-formation is intense and excessive.

The excessive proliferation of cells in the inner layers of the periosteum, the irregular calcification which occurs about them, and the absence of uniformity in the elaboration of the structure of the bone, produce an irregular, spongy bone-tissue instead of the compact lamellated tissue which is so necessary for the uniformity of the structure. The increased cell-growth between the epiphysis and the diaphysis produces the peculiar knobby swellings which are characteristic of rachitis. At the same time the medullary cavity

FIG. 83

Male, 3 years old. Rachitis, with enlarged spleen

increases rapidly in size, and the inner layers of the bone become spongy. The result of these processes is to diminish the solidity of the bones so that they cannot resist the strain of the muscles or outside pressure, and consequently become bent and curved. After a time the rachitic process may stop and the bones may assume a more normal character. The porous bone-tissue becomes compact, and even unnaturally dense, so that in later childhood the rachitic bone is unusually hard, like ivory, a condition noticed by those who have to operate on these bones.

VISCERAL LESIONS; SPLEEN.—Investigations have shown that in cases of rachitis which come to autopsy, the spleen is invariably enlarged, with the exception of the cases of clearly pronounced atrophy, in which the spleen is of normal size and weight or below normal. A

second marked characteristic is the more or less significant thickening of the capsule and the increase in the consistency of the organ. The third peculiarity of the rachitic spleen is the anemia of the organ and the diminution in the number of Malpighian bodies, which in children are otherwise well developed, but in these conditions are hardly perceptible. On section, the spleen has a blood-red color, the trabeculae are clearly marked by interlacing threads, and on drawing the knife over the cut surface there remains upon the edge blood and pulp tissue. The characteristics described hold true in all cases of rachitis, and, in general, indicate approximately the intensity of the changes in the bones.

The microscopic appearances are those of an interstitial splenitis. Whether the inflammatory appearances are directly dependent upon the rachitis or are due to the complicating diseases which caused the death of the patient has never been definitely determined. The uniform appearances in all the cases irrespective of the cause of death, seem to give weight to the opinion that the lesions are directly connected with rachitis.

PATHOLOGICAL CHEMISTRY.—The rachitic bone and cartilage is abnormally poor in mineral constituents, that is to say, in calcium and phosphorus. The ash content of the ribs and spine may be reduced to from sixty to twenty per cent of the normal. Metabolism experiments up to the present time have only succeeded in establishing the fact that the diminished calcium content of the skeleton in rachitis is due to diminished calcium retention—a negative calcium balance, and that in the healing stage the calcium balance is reversed. This is no more than would be expected from the character of the condition. Up to the present time, metabolism experiments have been unable to throw further light on the pathological chemistry of rachitis.

ETIOLOGY. THE NATURE OF THE DISEASE.—The cause of rachitis is unknown. The disease is almost certainly a disturbance of nutrition, and the bony lesions being the most prominent feature of the disease, it has been assumed by many writers that rachitis represents a disturbance of the calcium metabolism. That the calcium balance is disturbed in rachitis is an undoubted fact, but this does not mean that the disturbance of calcium metabolism is the primary factor in the etiology of the disease. That rachitis is due to insufficient calcium in the food, or to insufficient absorption of calcium from the intestine, has never been proven, and is certainly contrary to the evidence. There remains the possibility that the disturbance involves the utilization of calcium by the tissues of the body. Sufficient calcium is absorbed, but not being used, is excreted again into the intestine. Under this theory, inability to use calcium

leads to the formation of lime-poor bone, which interferes with endochrondral ossification. The other characteristics of the osseous lesions are considered secondary to this, the new cartilage being irregularly formed, while the medullary spaces, and an excessive number of blood vessels, project into the defective cartilage.

Another possibility is that the formation of defective bone tissue precedes and is the cause of the abnormality of the calcium metabolism. If this is the case, some other cause must be sought for the osseous lesions. The choice between these two theories must be a matter of opinion. I believe the weight of evidence to be against the theory that a specific disturbance of calcium metabolism is the primary factor. I believe that the calcium metabolism is disturbed only in this one respect, that the bony tissue is so affected by the disease that it is unable to seize and utilize the lime salts which are abundantly present for its use.

This does not, however, explain why the bony tissue is unable to utilize the lime salts. One theory, advanced by some writers, is that there is a special pathological peculiarity of the bony tissue which prevents ossification in rickets. Against this is the fact that rickets is not a disease limited to the bones, but affects all the organs and tissues of the body, which suffer in a manner which suggests disturbance of nutrition. If the bony tissues suffer with them, as a result of such disturbance, the effect in growing bone would quite naturally manifest itself by inability to carry on the most important function of such tissue, which is the storing of calcium. Moreover, histological and microchemical researches have given no evidence of any primary anatomical peculiarities of the bony tissue of the rachitic child, the lesions being suggestive of impaired nutrition.

The theory that a disturbance of metabolism in rickets causes an increased quantity of acid in the circulating body fluids, and that decalcification occurs through their influence, has not been supported, and is contrary to the preponderance of the evidence at hand.

From present knowledge, it can only be concluded that the disease rachitis represents a general disturbance of nutrition, in which all the tissues of the body suffer to some extent. Among them the bony tissue suffers, and the disturbance of nutrition so affects the growing bone, as to produce the characteristic osseous lesions of the disease. The effect of these lesions is impaired power of utilizing lime salts, and this leads to diminished calcium retention.

THEORIES AS TO ETIOLOGY.—In the voluminous literature of rickets will be found four principal theories as to the etiology of the disease. These are that the disease is caused, first, by an infection, second, by a disturbance of function of some of the ductless glands, third, by an unsuitable diet, and fourth, by bad hygienic surroundings.

The evidence in favor of the infectious nature of rachitis is very slight, and inconclusive. The weight of evidence points toward disturbance of metabolism as the most probable cause of the impaired nutrition.

The recent attempts to prove that some of the disturbances of metabolism are due to an impairment of the function of some of the ductless glands, such as the thyroid, parathyroid, thymus, and adrenals, have not been successful.

The two principal theories of the etiology of rachitis are the dietary, and the hygienic. According to the former, the disturbance of metabolism in rickets is caused by an improper diet. In favor of this theory are the facts that rickets is commoner in artificially-fed infants than in the breast-fed, and that it is also commoner when artificial feeding is badly conducted than when it is rationally conducted. Against it is the fact that the occurrence of rickets has never been conclusively associated with any particular dietary error, nor with any particular form of gastro-intestinal disturbance. Indeed, rickets is comparatively uncommon in the well-known gastro-intestinal diseases which lead to interference with growth, and atrophic infants are very infrequently severely rachitic. It is often seen in fat children, who have never had any digestive disturbance, and indeed, the disturbances of digestion in rickets seem to be usually secondary to the disease. The occurrence of rickets in fat children might partly be explained by diminution in their bodily activity.

It was formerly supposed that the most probable error in diet responsible for rickets was insufficient fat in the food. This was based on the statistical evidence obtained from the study of the previous diet of rachitic patients. The majority had been fed on foods high in carbohydrate, and low in fat, such as the various proprietary infant-foods. Such foods, however, are most commonly in use among the poorer members of society, where children are subjected to the worst errors of hygiene. Consequently no positive conclusion as to the influence of diet can be drawn from such statistics.

Metabolism studies on the effects of fat and carbohydrate on calcium metabolism, have suggested just the reverse theory as to the dietary error which causes rachitis. It has been shown that overfeeding with fat causes a negative calcium balance, while carbohydrate favors calcium retention. This phenomenon would only be of value in the etiology of rachitis, if the theory that the disease *is due* to a negative calcium balance were proven. It is possible, however, that overfeeding with fat may be a contributing cause.

No influence of diet on rachitis has yet been conclusively proven by any experimental method. We cannot, on the other hand, deny that the food, and its digestion, have any influence. How great a part the diet plays must be a matter of opinion. I believe that the

feeding certainly does not play the chief part in the etiology of rachitis. I believe, however, that improper feeding, and the nutritional disturbance which it causes, probably plays a contributory rôle in many disorders, among which is rachitis. This would explain the general frequency of rickets in the artificially-fed, without any relation to any particular dietary error. In breast-fed infants, rickets is seen at times, especially in babies with whom lactation is unduly prolonged, and also in Negro and Italian babies.

The fourth theory attributes rachitis to hygienic errors, and to faulty hygienic surroundings. This, I believe, is the view of the etiology of rachitis which is best supported by the evidence at hand. Rickets is commonest in the poorer classes; it is particularly common among the children of the proletariat of the larger cities. The housing conditions are of the first importance, and probably the most important factor is lack of fresh air. Confinement in small rooms, with overcrowding, gives lack of sufficient air space, and the absence of fresh air causes deficient oxygen intake. It is easily conceivable how serious disturbance of metabolism might follow such conditions. There is evidence also that among the unfavorable hygienic conditions contributing to rickets are lack of sufficient sunlight, and lack of sufficient bodily exercise. The probable influence on metabolism of both these factors is also obvious; lack of exercise, besides its direct influence on metabolism, would also lead to deficient oxygenation. Under this theory, rickets appears as a disease caused by the "domestication" incidental to civilization. All the conditions of civilization which differ from those of the natural life of the human animal, probably play some part. I believe this factor is far more important than are infections, or alimentary injuries or intoxications.

The evidence in favor of this view of the etiology of rachitis is both statistical and experimental. The remarkable investigation of Finlay in Glasgow is almost conclusive from the statistical standpoint. The frequency of rickets under crowded, unhygienic conditions, and its greater frequency of development in the winter and spring, when confinement to the house is common, are very significant. Also, the frequent development of rickets in animals confined in cages in zoological gardens, forms a very striking analogy. The only evidence against this theory is the infrequency of rickets among the inhabitants of polar countries, who live in close dwellings, deprived of sunlight for much of the year. Also, rickets is as common among the negroes of the South who live out of doors, as among our Northern negroes. This can be explained by the fact that another factor, that of race, is probably important in rickets. Such a factor must be assumed to explain the geographical distribution of the disease. The fact that rickets seen in breast-fed babies is common only among the Negroes and Italians, is further evidence of a racial

factor. There is some evidence also of the existence of a hereditary factor, a constitutional predisposition.

There is considerable experimental evidence on animals which supports the etiological importance of insufficient fresh air; exercise, and sunlight.

SUMMARY OF ETIOLOGY.—Rachitis is exclusively a disease of early life. It is only seen in infancy, the commonest period of its occurrence being that between the ages of six months and eighteen months. It may appear earlier, but is probably not congenital, the so-called congenital rickets representing a different disease (chondrodystrophy). It is a disturbance of nutrition, principally affecting the bones, causing definite lesions. It is accompanied by a marked disturbance of the calcium metabolism, but this is probably not the primary factor. Impaired nutrition of the bony tissue, which accompanies impaired nutrition of other tissues, causes the anomalies of calcium metabolism which are seen in the disease. The cause of the impairment of nutrition is probably a disturbance of metabolism, of which the nature is unknown.

The two chief causes of this are probably constitutional peculiarities, and injuries from without. Among the constitutional causes, the best established is the racial factor. There is some evidence in favor of a hereditary factor, but this probably is of very little importance. Of the external causes, the most important is probably faulty hygienic surroundings, including lack of fresh air, lack of exercise, and lack of sunlight. An unsuitable diet, various acute infections, and possibly chronic diseases, may act as contributory causes.

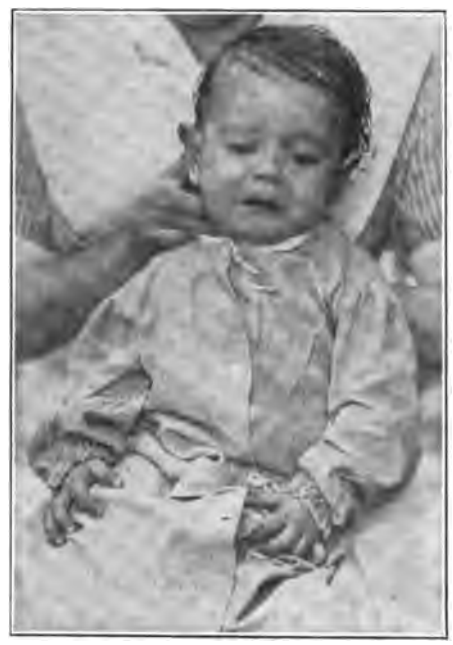
SYMPTOMS. CONSTITUTIONAL.—The symptoms of rachitis are those of a slowly developing constitutional disease. The early symptoms are the same as may occur in a number of diseases in which the nutrition is affected. The most common early symptoms are restlessness at night, profuse sweating, especially of the head, when asleep, and constipation. The appetite is impaired and capricious; the infants are fretful, the abdomen becomes prominent, and although they often grow fat they are anemic and their muscles are soft. The infants do not learn to walk as early as they should, their fontanelles do not close at the usual time; dentition is delayed and irregular, and soft areas appear in the cranial bones, especially in the occiput.

At this stage the characteristic tenderness of the body may occur, but many cases never present this symptom. This is usually due to a periosteal tenderness at the insertions of the muscles, and is sometimes confined to the bones. It is manifested only on trifling pressure, while at other times the muscles are tender and the gentlest effort to lift the child may cause him to shriek with pain. The

so-called paralysis of rachitis, which is an accompaniment of this stage, and, as a rule, precedes any marked osseous change, is generally brought to notice by the child's inability or disinclination to walk or stand. At other times it may be more severe and take the form of inability to use the arms as well as the legs.

After these general premonitory symptoms have continued for some time, the characteristic changes in the osseous system become prominent and are found in those parts of the bones which are in the most active stage of development. In cases beginning in earlier infancy, the skull undergoes the most marked changes.

FIG. 84



The rachitic head

HEAD.—The typical head of rachitis has a high, square forehead, with a decided prominence of the lateral parts of the frontal bones (frontal eminences), and sometimes there are also eminences on the parietal bones. The normal thickness of the bones is increased by means of a large amount of new periosteal soft growth between the periosteum and the bones. The head is somewhat lengthened and is usually larger in circumference than normal. The anterior fontanelle remains widely open, and may not ossify until the third year, or even later. The closure of the posterior fontanelle may be delayed in cases which develop rachitis very early in life. The sutures also remain open longer than normal, and in such cases may result in

leaving a depression in the course of the sagittal suture. Sometimes, however, a prominence is found instead of a depression. Flattening of the back and top of the head and asymmetry of the head may result from softening of the bones. The normal shape is usually regained when the disease is cured. The bones may be soft, porous, and hyperemic, while at their edges there may be rough, bony projections beneath the periosteum.

The name *craniotabes* is applied to an abnormal thinness of portions of the parietal and occipital bones, which are filled only with a fibrous membrane, and which yield to gentle pressure and give a sensation of crackling parchment. This condition of the bone may be only temporary, and the areas of thickening are often absorbed; but if there is much deposit under the periosteum it will sometimes remain, and where calcification takes place quickly, the thickened areas of the bone will remain unabsorbed throughout life. At times the jaw-bones are affected; the upper jaw is then found to spread behind, and to be pointed in front, while the lower jaw is flat in front and bent in at the sides, making an angle at the site of the canine teeth.

THORAX.—The rachitic thorax is narrow and is compressed laterally,—that is, there is a tendency to a flattening of the sides of the chest and to an increase of the antero-posterior diameter. The forces which produce deformities of the thorax are dependent on the muscular action on the soft bones by pulling, atmospheric pressure from without, and the pressure exerted on the bony structures by growing organs.

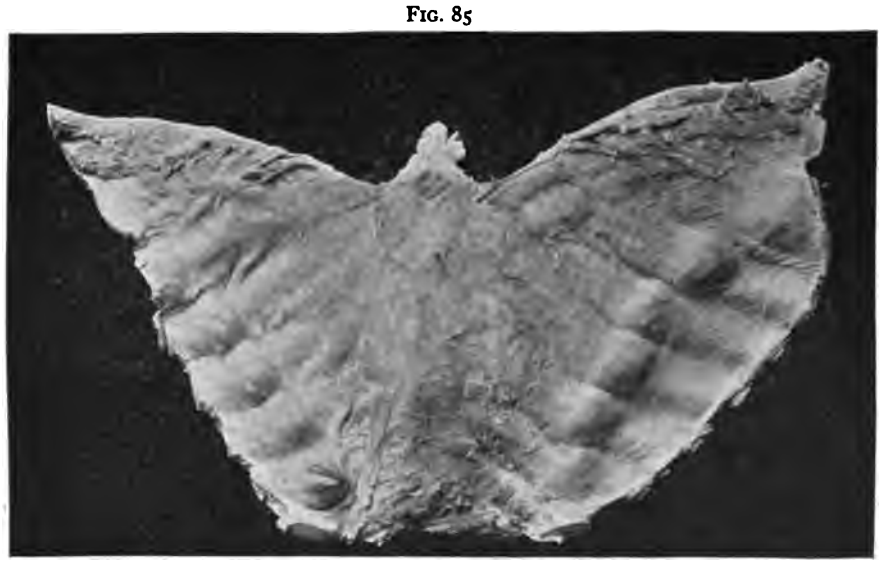
A transverse depression may also occur, starting at the junction of the ensiform cartilage with the sternum, extending laterally on the thorax, and corresponding to the insertion of the diaphragm. This is called Harrison's groove. The lower ribs may be elevated by the underlying distention caused by the prominent abdomen and the liver. Softening of the ribs is said to occur after the changes in the skull and before the changes in the extremities. In a typical rachitic thorax the clavicles are shorter and more curved than normal, and the clavicular deformities may be unilateral. Fractures of the clavicles in rachitis are not uncommon at the forward curve, and may possibly occur when the infant is being dressed.

When there is unusual lateral compression and narrowing of the thorax, the sternum is made to project forward, and this is called *pigeon-breast*, or *pectus carinatum*. The weakest part of the thorax is at the junction of the cartilages and ribs, and the sternum is thus naturally pushed forward. In another series of cases, in which the ribs are pushed together laterally and the sternum depressed, as where there is interference with the entrance into the lungs by adenoid

growths and enlarged tonsils, the condition of *funnel chest* is produced. Again, there may be greater compression on one side than on the other, with a resulting prominence or depression on one side of the sternum.

The costal cartilages are frequently enlarged at their junction with the ribs, and can be felt and often seen as a line of rounded prominences. These prominences are called the *rachitic rosary*, and, though most commonly occurring in the latter part of the first year, have also been met with in the early weeks of life. The rachitic rosary is the earliest of the physical signs of rachitis to develop, and is the most common abnormality in rachitis. It does not occur in normal children, and its presence justifies the diagnosis of rachitis.

FIG. 85



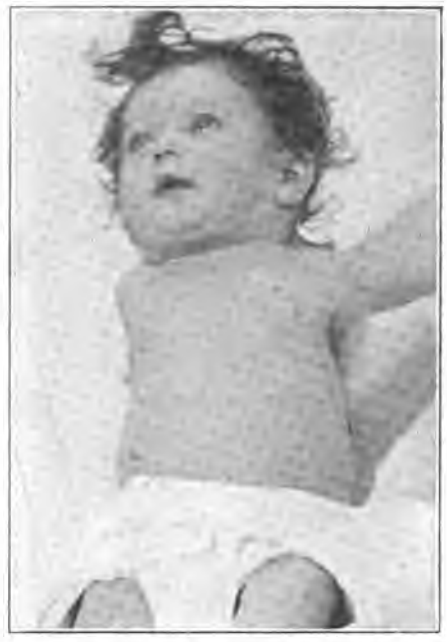
Inner surface of sternum, with cartilages and portions of ribs attached, showing rachitic rosary

As the pathological process is more pronounced in the lower ribs, especially the lower five, than the upper, the rosary is most distinct in the lower ribs.

SPINE.—Deformity of the spine is quite constant in rachitis and results mostly from muscular weakness, but the vertebrae may be affected by the rachitic process, and in cases of recovery may be found thickened. The most common deformity is *kyphosis*, which consists of a gradual bowing backward in the dorsal and lumbar regions.

The prominence of these vertebral spines is often quite sharp, and simulates Pott's disease, but the rachitic spine should be flexible to passive manipulation. *Scoliosis* (lateral curvature) and *lordosis* (forward curvature), are also common deformities occurring in rachitis.

FIG. 86



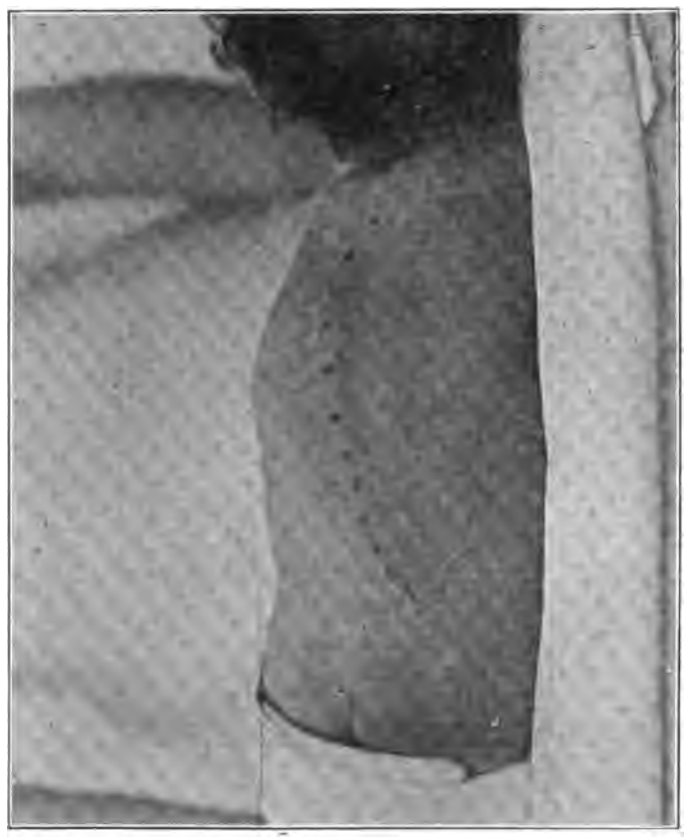
Position of the rachitic rosary

FIG. 87

Rachitic kyphosis. Female, 3 years old

EXTREMITIES.—Deformities of the long bones may arise in the epiphyses and in the shafts; the former show enlargement, the latter bending. Enlargement of the epiphyses appears especially at the wrists and at the anterior ends of the ribs; enlargement of the lower end of the radius and ulna is practically universal, and is second to the rosary in frequency of occurrence and in early time of appear-

FIG. 88



Rachitic deformity of the spine and chest

This particular deformity was caused by the fact that the patient had been cared for by an older child, who always held it with the same arm.

ance. Enlargement of the lower end of the tibia and fibula occurs next in frequency. The increase of the epiphyses at the wrists is greater than elsewhere. These enlargements do not involve the joints. In the deep-seated epiphyses, like the hip and the shoulder, the changes are not noticed so readily. The proliferating layer between the epiphyses and the bone may become so thick and softened that consequent deformity and separation of the epiphyses may

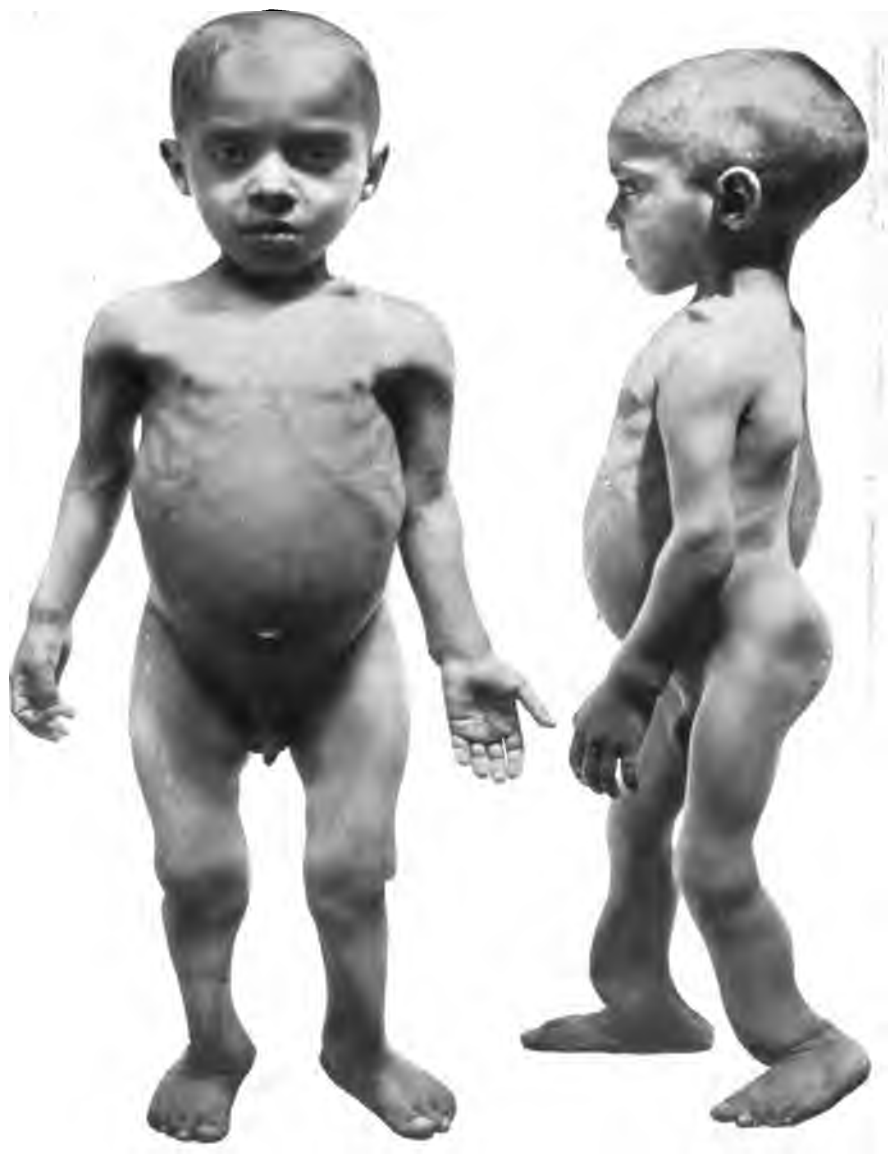
occur, but such an event is uncommon. When fractures occur they are on the concave side of the bone. In rachitic deformity the curve of the forearm may be due to the pull of the muscles, and there may be a special bend of the radius about the ulna, probably secondary to the anteroposterior curve. It prevents full supination. The humerus rarely bends. Fractures are not rare. Any considerable increase in the ankle epiphysis is rare. The curve of the femur is forward, and may involve only the anterior surface, so that there may be no curve on the posterior surface. Fractures may occur with excessive callus formation later. In the lower leg, fracture takes

FIG. 89

Rickets

place most often with anterior bowlegs, and the resulting callus is deeper than in the thigh. Fractures of the long bones, however, are uncommon, but their arrested development may cause permanent shortening. Bowing of the legs, knock-knee, and flat-foot, are all very common symptoms of rachitis. Coxa vara may be present. Localized rachitis, as of the legs, is possible and quite frequently met with in children with no symptoms of general rachitis.

Signs of previous rachitis are suggested by a caput quadratum with a thick skull, irregularity of the teeth, eversion of the lower edges of the thorax, pigeon-breast, pelvic deformity, deformities of the clavicles and extremities, and thickened epiphyses, which may



I II

FIG. 90—Rachitis. Age, 6 years

persist, as thickened epiphyses in general are slow in disappearing and outlast the active process.

The existence of flat-foot in children over two years old should lead to an examination for knock-knee. The combination of these two conditions will in most cases be found to be dependent upon present or previously existing rachitis.

MUSCLES.—There is a weak and relaxed condition of the muscles and ligaments, but microscopically the muscles are only pale and their fibres infiltrated with fat, although in some cases there may be atrophy from disuse.

FIG. 91

Showing deformities of the humeri in a case of rachitis of an infant of 10 months, due to the habit, on the part of the mother, of lifting the child by the arms

NERVOUS SYSTEM.—The nervous system is in an exceedingly unstable and sensitive condition. Convulsions are quite common. Rachitic children have a marked tendency toward spasmophilia, with its characteristic symptoms of tetany and laryngospasm.

LUNGS.—Owing to deformities of the thoracic walls there is a tendency to atelectasis of portions of the lung from pressure, with surrounding areas of emphysema. There is also a great tendency to bronchitis and bronchopneumonia.

HEART.—The heart sometimes shows signs of mechanical irritation, represented by irregularity and caused by deformities of the thorax.

BLOOD.—Anemia is often seen in rachitis. The blood shows the characteristics of a secondary anemia.

LYMPH-NODES.—The lymph-nodes are very frequently found to be enlarged from simple hyperplasia.

STOMACH AND INTESTINES.—There is often, though not necessarily, functional disturbance of digestion. The symptoms vary very much and diarrhea and constipation alternate. The distended abdomen results from weakness of the muscles of the abdominal wall and of the intestine, and a resulting umbilical hernia is not rare. In like manner atonic constipation is common.

DIAGNOSIS.—The diagnosis of rachitis cannot be made by the premonitory symptoms, as the disease is so often the result of impaired nutrition arising from many causes, that it is difficult to determine when the rachitic symptoms begin. The differential diagnosis has to be made from a number of diseases in which the general nutrition of the child is profoundly disturbed, these diseases being especially represented by functional disorders connected with the gastro-enteric tract. When the pathological changes in the bones have progressed sufficiently for physical detection, and the disease is fully developed, the diagnosis is not difficult. In its early stages, therefore, the manifestations of rachitis may be so slight that the diagnosis must often be held in abeyance. The most significant signs are, in the order of frequency of occurrence, the rosary, the enlarged radial epiphyses, the rachitic head, delayed dentition, and the deformities of the chest. Prominence of the abdomen, relaxation of the ligaments, and enlargement of the spleen, are important confirmatory signs.

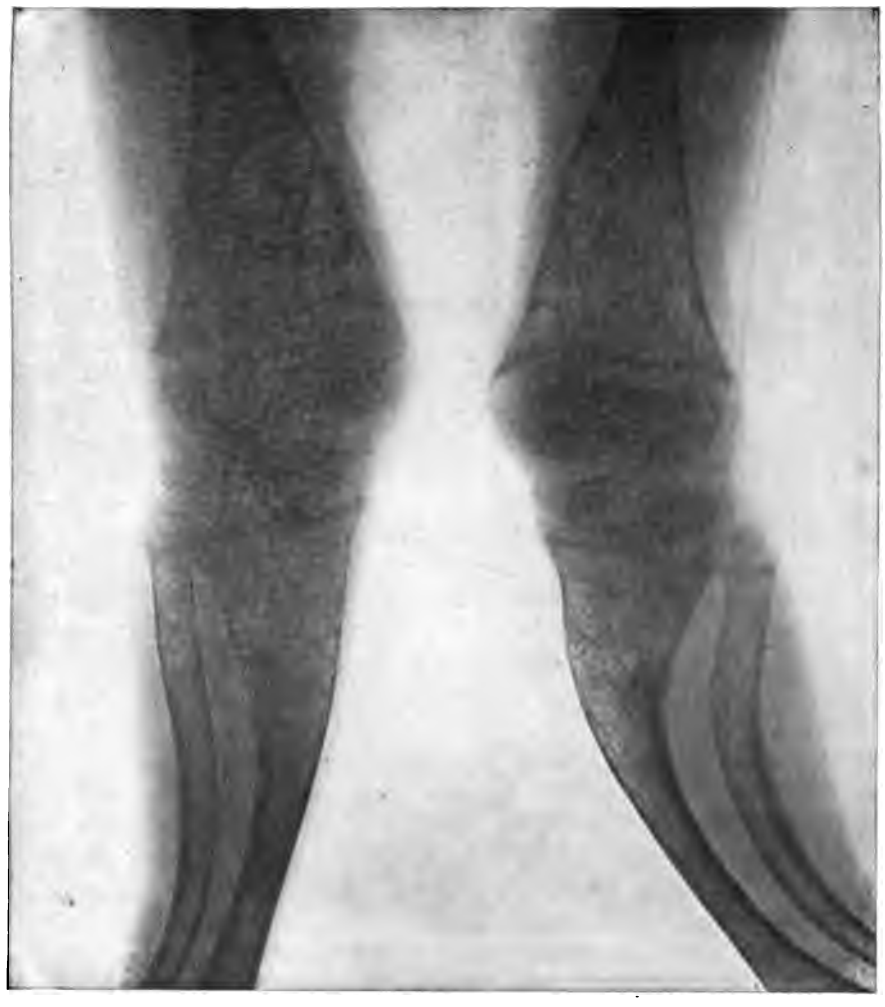
Examination with the roentgen ray is often of great value in the diagnosis of rachitis, and should be used in every case of doubt. The appearances of the bone structure as differentiated by means of the roentgen rays, from certain of the chronic conditions of childhood, may be summarized as follows: The characteristic curves of the long bones are easily apparent. There is great irregularity at the epiphyseal lines, with much hyperplasia of the osteoid tissue and hypertrophy of the epiphyseal cartilages. These pathological changes usually give the appearance of a great disproportion between the bony epiphysis and the epiphyseal ends of the diaphysis. The curves in the shafts of the bones are often sharp.

The differential diagnosis of rachitis is to be made from chondrodystrophy, scorbutus, rheumatism, osteomalacia, osteomyelitis, syphilis, paralysis of central origin, Pott's disease, and from the disability to use the limbs, due to simple weakness in infants who are not rachitic.

CHONDRODYSTROPHY, OR ACHONDROPLASIA, is distinguished from rachitis in the following characteristics: The trunk is normal, while the extremities are short and deformed. The head is large and the

bridge of the nose flattened. The enlargements at the ends of the long bones are due to overgrowth of the periosteum, instead of to changes in the ephipyseal cartilage, and the zone of proliferation is narrower than that in rachitis. A radiograph will usually bring out the differences in the pathology of the chondrodystrophy and all other lesions of the bones which resemble it. In this condition the epiphyseal lines, instead of being irregular, are straight, although much narrowed. The shafts of the bones are often thickened and

FIG. 92



Case of rachitis, the radiograph showing the marked deformity (knock-knee), the "zone of proliferation" at each epiphyseal line, as well as the marked rarefaction of the shadows cast by all the bones due in all probability to the deficiency in lime salts as well as to the change of function as to weight bearing.

stunted, while the apparent overgrowth of the bony epiphysis is so marked, that its shadow often appears to overlap that of the epiphyseal line.

SCORBUTUS is eliminated in the diagnosis by the presence of the various osseous lesions which have just been described as symptoms of rachitis, and by the absence of the characteristic features of scorbutus, represented by tenderness and swelling just above the joints, due to subperiosteal hemorrhages, and by stomatitis ulcerosa. The X-ray in scorbutus shows the raising of the periosteum by the characteristic subperiosteal hemorrhage. A "white line" at the epiphyseal zone of growth is considered characteristic by some authorities. The characteristic changes of rachitis are absent.

RHEUMATIC FEVER in its articular form would present such marked symptoms of acute tenderness, swelling, and pain in the joints, combined with a heightened temperature, that it could easily be distinguished from the general tenderness of the bones without much fever, together with the subacute or chronic course and the characteristic enlargement of the epiphyses in rachitis.

OSTEOMYELITIS.—The clinical symptoms of an acute infectious disease, with sudden onset, and represented by severe constitutional disturbance, heightened temperature, pain, localized tenderness, and rapid exhaustion, serve to distinguish osteomyelitis from the slow development and slight amount of fever met with in rachitis. In osteomyelitis, moreover, various foci of infection will appear in one or more bones with tenderness at these points and a tendency to suppuration which is not met with in rachitis. In roentgenograms the abnormal shadows are to be seen in the diaphysis, and are generally due to irregular necrotic areas, with or without sequestra. There is usually no thinning of the shadows of the involved bone or surrounding bones.

OSTEOMALACIA.—In very rare cases osteomalacia occurs in children, but it can seldom be differentiated from rachitis during life, except by X-ray examination. The lack of lime salts, as is shown by the "quality" of the shadow, is generally well-marked. The deformities of the bone-shafts are characterized by bendings rather than sharp curves.

HEREDITARY SYPHILIS.—The diagnosis of rachitis from hereditary syphilis is, as a rule, not difficult. While in rachitis the enlargement of the long bones is limited to the epiphyses, in syphilis it is not so limited, but involves the ends of the diaphyses. This enlargement is often accompanied by a condition which closely simulates a callus, and there is a distinct tendency to fracture in syphilis rather than to the bending which is common in rachitis. Lesions of the mouth and lips, and lesions of the skin are distinctive of syphilis.

CEREBRAL PARALYSIS AND POLIOMYELITIS.—In certain cases of rachitis, especially in the earlier stages, the so-called *paralysis of rachitis* occurs, and must be distinguished from paralysis of *central* origin, especially from poliomyelitis of the cord. The differential diagnosis must be made by the absence of the symptoms described under cerebral paralysis and poliomyelitis, and by an examination of the child in a recumbent posture, which in rachitis will show that the muscular movements are but little impaired, that the apparent inability to move the limbs and the disinclination to walk are caused by tenderness of the bones and muscular weakness, that the electrical reaction is normal, and that the reflexes are not affected. This pseudo-paralysis is certain to pass off if the child lives. Cases of rachitis which do not walk until late, on account either of muscular weakness or of tenderness, may resemble cases of organic nervous disease with true paralysis. The diagnosis must rest on the presence of the general signs of these nervous diseases.

POTT'S DISEASE.—When rachitis causes kyphosis of the spine, it may simulate Pott's disease very closely. A prominence may be present at the dorso-lumbar junction, which is a frequent seat of deformity in Pott's disease, and which, involving several vertebrae, may or may not be obliterated when the child lies on its face and is lifted by its feet from the table. The spine is held rigidly in severe cases, just as in Pott's disease, and the deformity may be angular rather than the usual gradual curve. The coexistence of enlarged epiphyses and other rachitic conditions makes it very probable that the affection is rachitic; but both diseases may coexist. The von Pirquet reaction is usually present in Pott's disease, and its absence is in favor of rachitis. In roentgenograms, abnormal shadows attributed to tuberculosis are to be seen most often involving the joints, while the appearances in the adjacent bones are characteristic.

In general, the age of the child, under eighteen months, the absence of much pain, and the existence of other signs establish the diagnosis of rachitis. Rachitis is, moreover, in children under two, much more common than Pott's disease. In doubtful cases the diagnosis can be made only after several examinations and a period of two or three weeks of recumbency, under which conditions the rachitic spine becomes somewhat more flexible. In doubtful cases time alone will establish the diagnosis.

WEAKNESS.—In certain infants who are not rachitic the power of walking is lost for variable periods, and is due to *weakness* following any disease, whether acute or chronic, which for a time may interfere with the infant's vitality. This condition is difficult to distinguish from rachitis, but must be differentiated by the absence of the other symptoms. Delay in learning to walk should lead us to

carefully examine for other symptoms of rachitis, as it is quite often one of the manifestations of this disease. An open fontanelle after the nineteenth or twentieth month suggests rachitis, and delayed dentition is also significant. If there are no teeth at the ninth or tenth month, the infant should be carefully examined for rachitis; at one year absence of teeth almost always indicates the disease.

Children with rachitis have weak muscles as well as weak bones, and the condition of such a child approaches that of one who stands and walks with the least expenditure of muscular force.

HYDROCEPHALUS.—The diagnosis between the rachitic head and the hydrocephalic is usually not difficult. The former is irregularly enlarged, flattened on top, square-looking, due to the prominence of the frontal bosses, and has a normally tense or depressed fontanelle. The latter is regularly enlarged and rounded, with prominent parietal bosses and a tense and bulging fontanelle.

PROGNOSIS.—The prognosis of rachitis is favorable, provided no complications arise. When left untreated the disease will, after more or less deformity has occurred, be arrested spontaneously, the pathological process in the bones will cease, and the bones will harden in their deformed condition. In these untreated cases the younger the child the more unfavorable is the prognosis as to permanent deformity. A spontaneous arrest of the disease may take place in any of its stages, but, as a rule, if the affection is pronounced, serious deformities are usually produced. Rachitic children are more liable to die than other children when they are attacked by such diseases as pneumonia or bronchitis. As a rule, the epiphyseal enlargements diminish with growth, but to a certain degree remain through life. In favorable cases the craniotabes, laryngismus stridulus, bronchitis, diarrhea and paralysis gradually pass away Rarely death may occur in attacks of laryngospasm or convulsions

When properly treated, the health of rachitic children improves steadily, and, unless the deformities which have occurred in the bones have advanced too far, complete recovery usually takes place by the third year. The arrest of the disease at an early stage is important. The persistent deformities are the curving of the long bones, the deformities of the spine, and the more advanced deformities of the chest.

TREATMENT. HYGIENIC.—The essential feature of the treatment of rachitis is removal of the cause. The treatment is therefore hygienic, and the earlier the treatment is begun, the better are the results. After the eighteenth month the harm has been done and the tendency is toward recovery on account of the changes in the mode of life of children at that age. Most of the cases seen in

private practice are comparatively mild and are often unrecognized until spontaneous recovery has already begun.

The degree to which faulty hygienic conditions can be corrected depends on the circumstances in the individual case. The ideal treatment only can be described, and this should be approached as closely as circumstances will permit. The essentials are fresh air, sunlight, and exercise. The rachitic child should be outdoors as much as possible. When indoors, he should be kept in well-aired rooms, which are not occupied by other children, or other persons. He should sleep in a room with well-opened windows. Exercise can only be attained with children who are able to walk. With babies unable to walk, the patient should be undressed daily in a warm room, and encouraged to roll and kick about. Massage is an extremely valuable substitute for exercise in babies unable to walk. Sponge baths in the morning, gradually made as cold as the vitality of the child will permit, are also valuable. In large cities it is advisable if possible, to send the patient to the country, because in cities, outdoor treatment is difficult to carry out, and sunshine difficult to obtain.

With the children whose domestic conditions prevent the full realization of proper hygienic surroundings, much can be done through social service organizations. Faulty hygienic conditions in the home can be remedied to a great extent. Frequent outdoor country excursions can sometimes be arranged, and the children can be kept as much as possible in the parks and open squares.

DIETETIC.—I do not believe that any specific modifications should be made in the diet of rachitic infants. There is no proven relation between diet and the disease to serve as a basis for such modification. Disturbances of digestion must, of course, be corrected. The evidence obtained by metabolism experiments of the influence of the various food elements on calcium metabolism is not, under present theories of etiology, sufficient to form a basis for treatment. As rachitis is most frequently seen in artificially fed infants, human milk is probably the best food for rachitic babies, as it is for babies in general. When artificial feeding is necessary, the next best food is cow's milk, and sufficient calcium to meet all requirements is present in all ordinary cow's milk modifications. In selecting the composition of the food, it must be borne in mind that the diet should be made as suitable as possible, the physician being guided by the general principles of infant feeding. When the diet best suited to the normal infant of the same age and development has to be modified, it is usually on account of the digestive peculiarities of the individual infant, and the changes which are made are designed to meet the clinical indications.

MEDICINAL.—No drug has been proven to have any influence on the essential pathology of rachitis. The discussion of the etiology showed the theoretical uselessness of giving calcium, and there is no convincing clinical evidence of its value. Cod liver oil and phosphorus, alone or in combination, have been widely recommended in the treatment of rachitis. In the case of cod liver oil there is no convincing evidence that it has any specific value. In view of certain experiments on the effect of fat on calcium metabolism, there is a possibility that cod liver oil might even do harm. I do not believe, however, that any specific harm from cod liver oil is established by these experiments. It provides a form of fat which is often easier of assimilation than cow's milk fat, and in any disturbance of nutrition, it is valuable as an adjunct to the diet. I do not think it should be used until the second year.

Phosphorus was first recommended by Kassowitz in the treatment of rachitis, and its effects have been the subject of much experimental and clinical investigation. It still holds first place in German therapeutics. The evidence is very conflicting, some investigations appearing to prove the value of phosphorus, and others exactly the reverse. While I believe the preponderance of evidence is against the value of phosphorus, I do not believe that it does any harm when properly used. The best preparation is phosphorated oil, one minim of which contains 1/115 of a grain of phosphorus. The dose for infants is from one-half to one minim three times a day, after meals. It may be given in combination with one-half to one drachm of cod liver oil.

The only symptom in rachitis which often requires treatment is the anemia. The treatment is with iron, as in any secondary anemia. Profuse sweating may sometimes be relieved by atropine in doses of 1/800 of a grain.

TREATMENT OF RACHITIC DEFORMITIES.—This is for the most part a matter for the orthopedist. For the deformities of the chest, breathing exercises and gymnastics are employed. The kyphosis can usually be overcome by postural treatment, massage, and exercises. The deformities of the extremities are either outgrown, or surgical measures are required.

PROBLEMS AND RESEARCH.—The problems connected with the study of rachitis have been suggested in the consideration of the etiology of the disease. At the present time, there is so much activity in experimental research devoted to the subject of rickets, and medical literature is so full of the discussion of the subject, that a summary of the most important recent investigations cannot fail to be of interest to the student. The chief reason for the activity of investigation on this disease, is the fact that research has given

to the investigator a definite measure, by which he can estimate the results of his experiments. The fact of a negative balance in the metabolism of the bone forming minerals—calcium and phosphorus—in the active stage of rachitis, has been proven beyond doubt. The great increase in calcium retention above the normal seen during convalescence has also been established. Consequently the experimental investigator is not dependent only upon the doutful results of clinical observations, but can measure the results of his experiments by the effect on the retention of calcium.

One of the first steps in experimental research was the attempt to produce rickets in animals by using a food deficient in calcium. In many instances the animals became clinically rachitic, but the microscopic and chemical examination of their bones disclosed a condition differing from true rickets, to which condition Miwa and Stoeltzner gave the name of pseudorachitic osteoporosis.

Another series of investigations was devoted to the problem as to whether the lesions of human rickets are due to a primary calcium deficit in the food. This led to the establishment of the normal calcium requirement in infants, and also established the fact that in the cow's milk modifications used in artificial infant feeding, there is practically never a calcium deficit.

Investigators now turned their attention to the other possibility, that the lesions are due to deficient calcium absorption. The loss of calcium in such a case must be due to abnormal digestion. A great number of metabolism experiments showed that variations in the diet have an influence on calcium retention. In particular, a diet rich in fat increased calcium loss. None of the experiments have warranted the conclusion that decreased calcium absorption is responsible for the decreased retention. This led to the theory of Stoeltzner, outlined above, that sufficient calcium is absorbed, but that the bone, being for some reason unable to utilize it, the calcium is excreted into the intestine.

In connection with the problem of ultimate etiology, most of the theories are supported by clinical and statistical evidence, rather than by experimental evidence. Klose and Vogt, and Stoeltzner, support the theory of deficient internal secretion, particularly of the thymus. A rachitic condition has been produced experimentally in animals by ablation of the thymus. Mettenheimer, Matti, and Howland repeated the experiments and were unable to produce rickets in animals by removing the thymus. There is at present certainly no sure foundation for a thymic origin of rickets.

Some writers have expressed an opinion that the lesions of rickets suggest the action of a bacterial toxin, rather than that of a disturbance of nutrition. Marfan pointed out that probably all rachitic children have some focus of infection, and suggested the theory

of an intestinal autointoxication. Ribbert also advances the theory of a toxemia from bacteria, believing that all attempts to connect rickets with a special anomaly of metabolism, and all attempts to make it amenable to specific diet, have failed. The relation of rickets to improper feeding is explained by a lowering of the general resistance against infection. There has been some experimental work supporting the infectious theory. Koch injected the streptococcus longus into young animals, and an acute infection followed at once, gradually entailing a chronic disease of the bones which apparently was the same as rachitis in man. He believes, however, that domestication favors the development of the disease, as the dogs allowed to run about freely did not develop so marked a degree of disturbance of ossification as dogs kept in stalls. His experience confirms further the injurious influence of secondary infection after the process of normal ossification has once been upset by the acute infection. The ossification centres are left in a condition of lessened resistance, and an intercurrent whooping-cough, intestinal infection, or the like, adds fuel to the flame. He believes the injurious influence of infection alone, without calling on derangement of metabolism, is a sufficient explanation of the occurrence of rickets. In order to confirm Koch's evidence, further research is necessary, directed at showing whether other organisms beside the streptococcus longus can exert this malign influence upon ossification. Study of the pathological changes in the skeleton after acute infectious diseases in children might also throw some light upon the subject. The chief objection to conclusions drawn from experimental rickets in animals, is that the possible etiological influence of lack of fresh air, exercise, and sunlight, cannot be excluded in the conditions under which such experiments are usually carried out.

There has been considerable experimental work done in connection with the dietetic theory of the etiology of rickets, which attributes the disease to improper feeding. This work has been chiefly calcium and phosphorus metabolism experiments, showing the effects of various diets upon the retention of those minerals. An undoubted influence of diet upon calcium and phosphorus metabolism has been demonstrated, but the result still lacks the links necessary to positively connect it with rachitis. The production of experimental rickets in animals by any specific method of feeding, has not been conclusively attained. Orgler's conclusions are perhaps the most noteworthy; he believes that there are two important conditions which regulate calcium assimilation, (1) the ability of the tissue to assimilate it, which depends on the intermediary metabolism concerning which nothing is known; and (2) the composition of the food.

In favor of the "hygienic" or "domestication" theory of the

etiology of rickets, in which the factors of overcrowding, insufficient air, lack of exercise, and deprivation of sunlight are considered essential the evidence is mainly statistical. While statistical evidence is usually of less value than experimental evidence, some of the reports, such as Findlay's statistical investigation of from 400 to 500 rachitic children in the city of Glasgow, are very convincing. In pursuing this study Findlay investigated the possible rôle of the following factors: (1) the length of the time the child was breast-fed; (2) the order of the child in the family; (3) the condition of the bowels previous to the onset of the trouble; (4) the amount of air space in the house allowed to the child; (5) the number of stairs to be mounted to reach the house, and finally (6) whether or not the child has been taken much out doors. There is also experimental evidence in favor of the hygienic theory. Kochmann and Petzsch have recently demonstrated experimentally that under otherwise normal conditions of life, lack of exercise prevents the retention of a sufficient amount of calcium. Raczynski has shown also that less calcium was retained in dogs deprived of sunlight.

A great quantity of experimental work has been done on the therapeutic value of the "phosphorlebertran," the combination of phosphorus with cod liver oil so widely used in Germany. Most prominent is the work of Schabad. All these investigations consist in metabolism experiments on rachitic children, in which was studied the influence of phosphorus alone, cod liver oil alone, the two combined, and other oils substituted for cod liver oil, upon the metabolism of calcium, phosphorus, nitrogen, fat, and other salts. The results of all this work have been very contradictory. Kissel found in animal experiments, that phosphorus had no influence on the osseous tissue. Birk and Schabad both concluded that while phosphorus has no beneficial action in healthy children, the combination of phosphorus and cod liver oil increases the retention of phosphorus and calcium in rachitic children. Later work showed apparently that the effects are variable, or that there was no influence at all in acute rickets. The most recent investigation is a very elaborate series of metabolism experiments by Schloss of Berlin, which were carried out in rachitic breast-fed infants in order to exclude the possible disturbing influence of diet. He found that the phosphorlebertran had no favorable influence on the rachitic metabolism, except when combined with calcium acetate, when the favorable effects on the calcium and phosphorus balance were very striking. It does not seem probable that all this work is likely to lead to any very positive results, but it is probable that the literature of the next few years will continue to be largely occupied by the results of metabolism and animal experiments.

SCORBUTUS

(Scurvy) (Barlow's Disease) (Die Möller-Barlowsche Krankheit)

Scorbutus is a constitutional disease closely associated with nutrition. The identity of the disease as it occurs in infancy with the scurvy of adults was not recognized in the older writings, and infantile scurvy was usually described as acute rickets. Our modern knowledge of the pathology of infantile scurvy is based on the observations of Barlow and Cheadle.

Infantile scorbutus is a hemorrhagic disease. The disease is primarily characterized by a tendency to hemorrhage, especially under the periosteum of the bones, into the gums, into the skin, and from various mucous membranes. Anemia and cachexia are secondary manifestations.

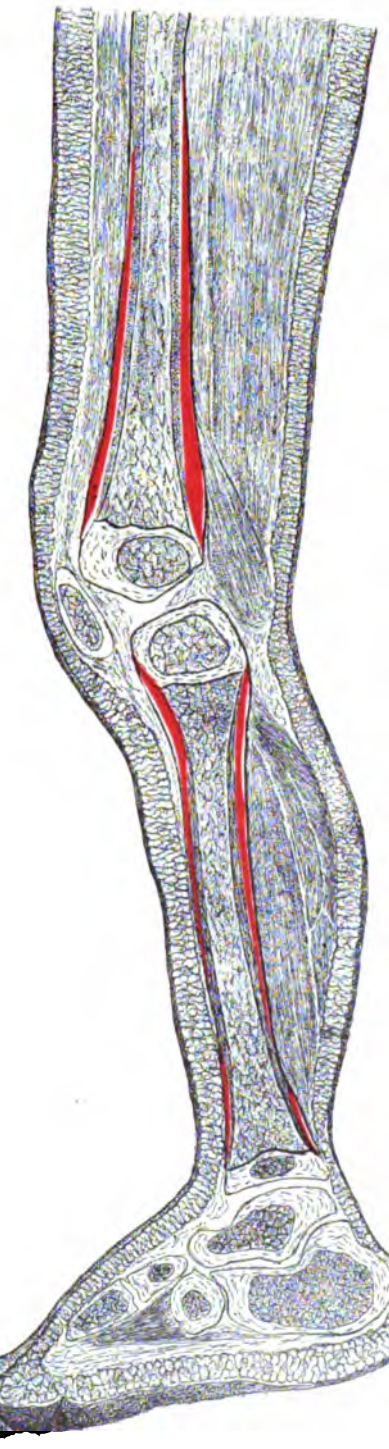
ETIOLOGY.—The exact cause of scurvy is unknown. A number of facts based mainly on clinical observation, though partly on experimentation, are known as to the etiology of the disease.

Leaving aside the scurvy of adults, such as develops among the members of polar expeditions, scurvy is exclusively a disease of infancy, and is never seen in older children. It occurs most frequently between the ages of six months and fifteen months, though a few cases are seen earlier than six months. It is probable that the presence in the infant at birth of antiscorbutic substances accounts for the time of appearance of the disease, as the disease frequently develops at seven to ten months after feeding with the same diet from the time of birth. The period when the disease occurs is just when antiscorbutic substances might be expected to be exhausted, and just before they are renewed by a more varied diet.

Clinical observations suggest the following conclusions: First, the hygienic surroundings have no influence whatever in the production of scurvy. The disease is fully as common, if not more common, in the infants of well-to-do parents brought up under the best hygienic conditions. Second, there is not the slightest evidence that previous disease, either acute or chronic, has any influence as a predisposing cause, and there is no evidence of any association with bacterial infection. Third, there is no evidence that digestive disturbance is a predisposing cause. Fourth, clinical experience shows a distinct association between the occurrence of scurvy and the diet, and a very rapid recovery under exclusively dietetic treatment.

It therefore appears that scurvy is due to a dietetic error. Furthermore, clinical experience suggests that the error in the diet is prolonged, and that it is not of such a character as to disturb the function of digestion. This strongly suggests that the improper feature

PLATE III



Vertical section of leg in a case of infantile scorbutus. The red areas around the femur and tibia represent subperiosteal hemorrhages. (Specimen preserved in the Museum of the College of Physicians and Surgeons, New York.)

of the diet is not an excess of any food element, but rather the *lack of some essential element in the food.*

Scurvy is very much more common in artificially-fed infants than in breast-fed. It is seen, however, in breast-fed infants. The occasional occurrence of the disease in breast-fed infants, and the fact that it does not always occur in the artificially-fed, proves that the disease is not due to the lack of some element found only in human milk, and the frequency of the occurrence of scurvy in the artificially-fed is probably to be explained by the great variety of artificial food-combinations in use, which allows a greater opportunity for the lack of a sufficient amount of some essential antiscorbutic substance. The analysis of a series of cases of scorbutus with reference to the diet has been undertaken from time to time, notably by a committee of the American Pediatric Society in 1898, and since then by various observers. These studies have shown no definite and constant relation between scurvy and any particular dietary error, which would point to the particular element which is lacking, but they are nevertheless very suggestive.

There is no suggestion of any connection between scurvy and a lack of fat, carbohydrate, protein, or any mineral salt. Scurvy occurs most frequently in infants fed for a long time on proprietary foods prepared without milk. It occurs next in frequency in infants fed on foods containing milk, but which have been prepared by the use of heat. This suggests that the substance of which the lack causes scurvy, is contained in milk, and that it is unfavorably influenced by heat.

The statistical evidence as to whether the heating of milk is a factor in the production of infantile scurvy is conflicting and inconclusive. In general, the weight of evidence points toward the conclusion that heating is an important etiological factor. In most series, the statistics show that the larger proportion of cases occurred in babies fed on heated milk. Nevertheless, while scurvy occurs more frequently in babies taking condensed milk, or boiled milk, or pasteurized milk, it also occurs in breast-fed babies and in babies fed on raw milk. This suggests that the heating of milk, even if it is of importance, is not the sole etiological factor. The fact that scorbutus does not always appear when babies are fed on heated milk, suggests that the antiscorbutic elements are not all completely destroyed by heat. There is evidence that the degree of heat, the length of the heating, and the time which elapses after heating before use, all have an influence. Several series show that more cases are *seen in babies fed on milk sterilized, boiled, or scalded, than on milk simply pasteurized.* Plantanza observed that scurvy developed more frequently with heated milk, but not when fresh milk was heated and used at once.

There have been some animal experiments on the effects of raw and heated milk, but they are few and inconclusive.

If heat is a factor, we must assume that it may wholly or partially destroy the antiscorbutic element or elements in the milk, and thus produce a deficiency. The occurrence of scurvy in breast-fed babies, and in babies fed on raw milk, can be explained on the ground of a deficiency of the same essential element from some other cause. In breast-fed infants such deficiency must be due to an anomaly of lactation. It is possible that dietetic error in the mother may account for the deficiency of her milk, but there is as yet little evidence in support of such a theory. In artificial feeding, the methods of cow's milk modification consist largely in dilution, and it is easily conceivable how a deficiency of some essential element can thus be brought about. In my own clinical experience, scurvy has occurred in a number of babies fed on raw milk, but in every case, the digestive peculiarities of the baby required a particularly dilute food, or the over-anxiety of a nurse or mother caused her to dilute the food without sufficient reason. I believe that ***to dilute a cow's milk modification is an important etiological factor*** in the occurrence of scurvy, and that dilution as well as heat may cause a deficiency of the essential antiscorbutic substance.

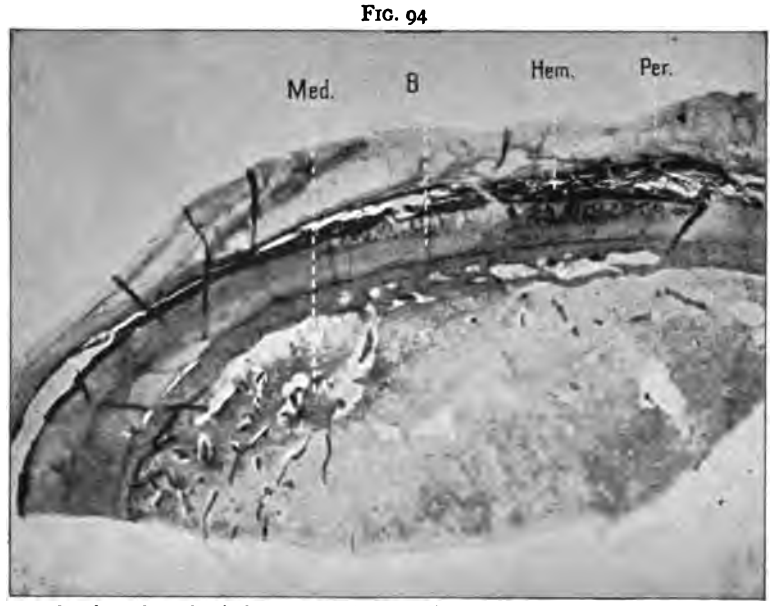
The nature of the essential element or antiscorbutic substance, the lack of which probably causes scurvy, remains unknown. There have been a number of investigations on animals, in which scorbutus has been produced experimentally. They have shown conclusively that scurvy is not due to simple starvation, nor to the prolonged use of a single article of food. They strongly support, however, the theory of the dietetic origin of the disease, and also strongly suggest that when scurvy develops as the result of the continuous use of any single food, it is ***not the presence in the food, but the absence in the food,*** of some substance which causes the disease. These experiments also suggest that this substance can be partially or wholly destroyed in various foods by heating or drying. They throw no light on whether there are one or more antiscorbutic substances, or on their nature.

One factor remains to be considered. Scurvy does not develop in all babies, in which the same prolonged error in diet is present. This suggests that there must be a constitutional predisposition which renders the production of scurvy easier in some individual babies. Such a factor probably plays some part. Hess' recent investigation suggests a connection between a predisposition to scurvy and the so-called "exudative diathesis" of Czerny. This has not yet been confirmed by other writers.

PATHOLOGICAL ANATOMY.—The most striking gross lesion

is hemorrhage, and the most characteristic site of hemorrhage is under the periosteum of the bones. The subperiosteal hemorrhage may affect any of the bones of the body, but the most commonly affected are the long bones of the lower extremities. These hemorrhages are often very extensive, and may run the entire length of the bone. The periosteum in severe cases may be entirely separated from the shaft by the extravasated blood, remaining attached only to the epiphyses. The epiphyses may also be separated from the shaft. While subperiosteal hemorrhage is so common as to be practically constant, hemorrhages may occur in other parts of the body. The most common location is in the gums of infants having teeth. Hemorrhage may also occur into the skin, between the muscles, and into the cellular tissue around the joints, although the joints themselves are usually normal. Hemorrhage may also occur in any of the internal organs; hematuria is the commonest form. Hemorrhage into the cellular tissue of the orbit, pushing the eye forward, is seen at times, as are also small hemorrhages beneath the pleura, pericardium, or peritoneum.

FIG. 94



Section of scorbutic bone. Med., medulla; B. bone; Hem., hemorrhage; Per., periosteum

Microscopic changes are confined mainly to the bones. The lesions differ from that of rickets in that it is the tissue of the bone marrow which is mainly affected. The myeloid tissue proper does not develop, so that the bone marrow is poor in cellular elements. The connective or supporting tissue increases at the expense of the

myeloid cells and blood vessels, and forms a homogeneous ground substance which contains numerous stellate and spindle-shaped cells. These changes are most marked at the ends of the diaphysis of the long bones, and at the ends of the ribs. As a result, the function of the osteoblasts is interfered with, calcification is diminished, and the cortex of the bone becomes thinner and more brittle. The density of the bone is particularly diminished near the epiphyseal line, but there is also an area of increased density at this point, forming the "white line" seen in roentgenograms.

The weight of American and English opinion differs from that of many European authorities on the interpretation of the lesions of scorbutus. Most European authorities regard the changes in the bone marrow as primary, leading to interference with the hematopoetic function of the tissue, and consider that this leads to progressive anemia and the "hemorrhagic diathesis." This view regards the hemorrhage as a secondary lesion. American opinion regards the hemorrhage as an early and essential effect of the cause which produces the disease. Scurvy is thus essentially a hemorrhagic disease, and studies on the blood suggest that the lesion is not due to any change in the blood, but to a change in the blood vessels. Under this view, the same cause produces changes in both the bone marrow and the blood vessels.

SYMPTOMS.—The symptoms of infantile scorbutus are those of a slow and progressive cachexia. The infants become pale, and show more or less gastro-enteric disturbance of a functional type. Profuse sweating, especially about the head, at times slight feverish attacks, and lessened appetite, are among the early symptoms. The temperature may be from time to time slightly raised, but not significantly so. The first symptom, however, which especially attracts the attention is a sensitive condition of the bones. The infant cries when the affected parts are touched. It does not seem to suffer pain when it is allowed to remain quiet, but as the disease advances the expression of its face indicates the fear of being handled. At this stage, pressure over the legs or thighs will usually elicit signs of tenderness.

As the disease progresses, more marked symptoms develop. Swellings of the limbs, usually of the diaphyses just near the epiphyses, appear. These swellings are most common and most prominent in the legs, but may also appear in the bones of the forearm. They are usually pyriform and symmetrical in shape, the skin over the swelling being more or less *tense, but not fluctuating*. There is commonly marked tenderness on pressure, but, as a rule, no especial heat of the affected part. The pain and swelling do not seem to be in the joint, but in the diaphysis and epiphysis.

The symptom second in frequency to tenderness and swelling over the bones of the legs, is hemorrhage into the gums. This is only seen when an infant has teeth, or when the teeth are just beneath the surface. The mucous membrane of the gums at the free margin of the teeth becomes reddened and soon begins to swell. The normal curve of the gum becomes almost a straight line and covers

FIG. 95

Infantile scorbutus in an infant of 9 months, showing extreme sub-periosteal hemorrhages of both femora, with slight affection of the lower ends of both tibiae

the lower part of the teeth. The gums in the space between the teeth remain unaltered at first. The mucous membrane then begins to change in color and becomes purplish. Extreme congestion and softening of the tissues allow hemorrhage to take place from the slightest pressure. Even when there are no teeth, when a tooth

is pressing on a gum and is almost through, or when a small portion of a tooth has penetrated the gum, small areas of congested mucous membrane appear, and are of great aid in the diagnosis.

The symptom next in frequency is the hemorrhage into the skin. These may occur in any part of the body, in any number or distribution. The commonest lesions are scattered small bluish macules. These may involve larger areas, forming at times extensive ecchymoses, and occasionally the eyeball may be pushed forward by hemorrhage into the orbit.

The commonest symptom of internal hemorrhage is hematuria. This is seen in quite a large proportion of severe cases. Occasionally it is the earliest and most prominent symptom.

Anemia is slight in the early stages of the disease, but often becomes marked in advanced cases. The blood shows the ordinary characteristics of a secondary anemia.

FIG. 96

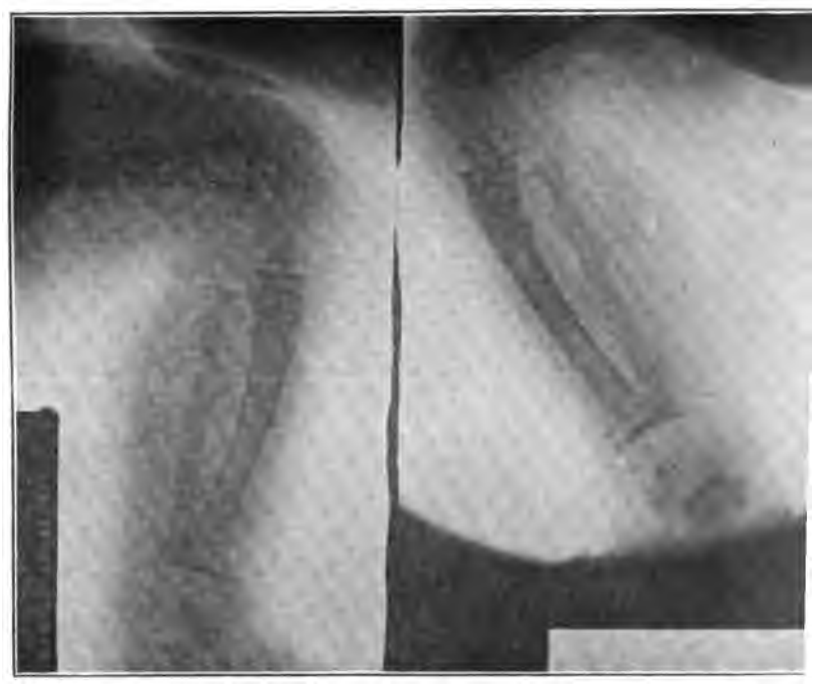
Infantile scorbutus. (Second month of disease.) Female, 10 months old

In addition to the symptoms of pain and tenderness, the infant keeps the affected limb perfectly still, so that, unless it were understood that it is pain which prevents it from moving the limb, it might be supposed that it was paralysis; in fact, this symptom in scorbutus has been termed pseudo-paralysis. It has nothing to do with true paralysis, and corresponds to what is seen in rheumatic affections of the joints.

DIAGNOSIS.—The diagnosis of infantile scorbutus is usually not difficult, if the characteristic symptoms of the disease be kept in mind. The chief cause of errors in diagnosis, is the fact that the frequency, or even the very existence, of infantile scurvy is often forgotten. The existence of tenderness over the bones, especially over more than one bone, in an infant between six and eighteen

months, is always suspicious of scurvy. If there is evidence of hemo rhage in any other part of the body, the probability of scurvy greatly strengthened. The association of tenderness over the limb with reddened, swollen gums is diagnostic. When the characterist lesion of the gums is absent, the differential diagnosis of scurvy mainly based on the absence of the characteristic signs of oth conditions for which scurvy might be mistaken.

FIG. 97



Scorbutus. The plate shows the "white line"

The X-ray findings in scurvy are also characteristic. The earli sign is the so-called "white line," an area of increased density at t junction of the epiphysis and the diaphysis. This is almost c stant in scurvy, but is also seen in some other conditions. Usua the thickening of the periosteum, and its separation from the sh by hemorrhage, can be made out in roentgenograms.

The diagnosis of infantile scorbutus is to be made from rheuma fever, rachitis, purpura, syphilis, acute anterior poliomyelitis, tub culous joint disease, osteo-myelitis, sarcoma, and acute nephritis

RHEUMATIC FEVER.—Scurvy is most frequently confounded w rheumatic fever. In view of the extreme rarity of rheumatism infants, especially under one year, pain, tenderness, and swell

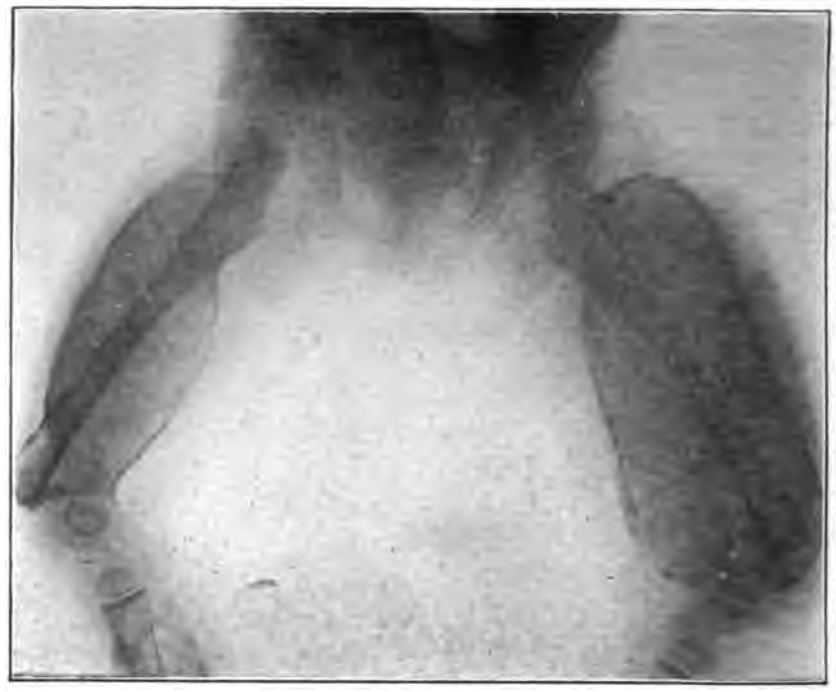


FIG. 98—Scorbutus. The plate shows enormous subperiosteal hemorrhages

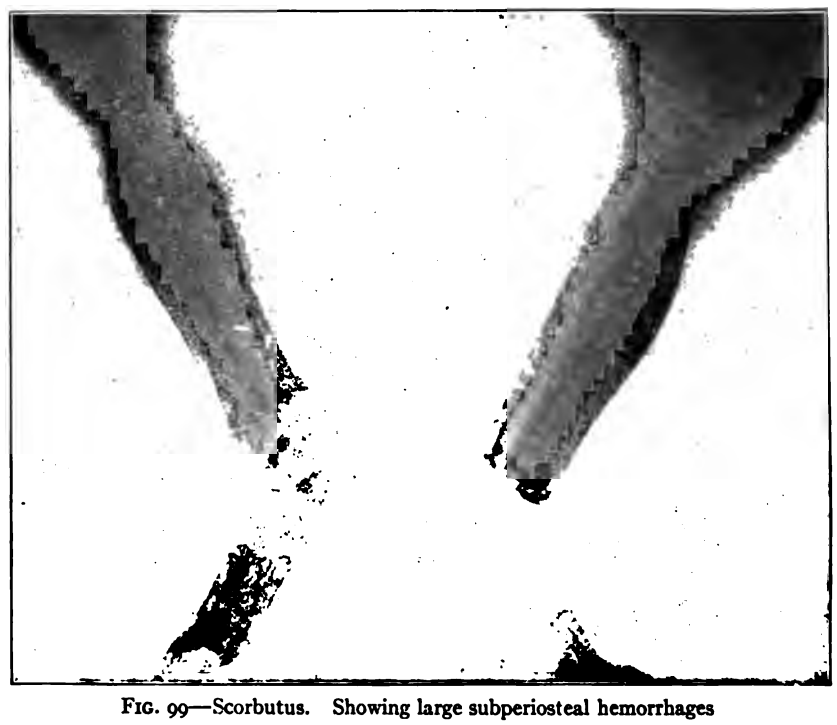


FIG. 99—Scorbutus. Showing large subperiosteal hemorrhages

of the limbs in an infant should always suggest scurvy. In scurvy, careful examination will usually show that the tenderness is connected with the bones rather than with the joints.

RACHITIS. —In cases of scorbutus not complicated by coexistent rickets, the diagnosis is based on the absence of the characteristic signs of rickets, such as the rosary, enlarged epiphyses, rachitic head, and so forth. In cases in which rickets is also present, the recognition of scurvy is sometimes more difficult. If the characteristic lesion of the gums is present, or if there are ecchymoses on the skin, or hematuria, the existence of scurvy can be recognized at once.

FIG. 100

Scorbutus. Showing small subperiosteal hemorrhages

If these confirmatory signs are absent, the physician should remember that tenderness is a rare symptom in rickets, but always constant in scurvy. The swelling near the epiphysis in scurvy can usually be distinguished from the enlargement of the epiphysis in rickets. Doubtful cases should be treated for scurvy, and the rapid response to treatment will confirm the diagnosis. The question can almost always be positively settled by X-ray examination.

PURPURA.—This disease is characterized by hemorrhage, particularly into the skin. It is very rare in infants. It can usually be excluded by the tenderness of the shafts of the long bones in scurvy,

SYPHILIS.—Occasionally cases of syphilis are seen in infancy, characterized by painful swellings about the ends of the long bones, due to syphilitic osteo-chondritis. This condition can usually be excluded, either by the absence of other signs characteristic of syphilis and described under that disease, or by the presence of the characteristic lesions of the gums or of hemorrhages in some other part of the body. Examination by roentgenogram will also make clear the distinction between the two conditions. It should also be remembered that syphilitic osteo-chondritis usually occurs before the fifth month.

POLIOMYELITIS.—The unwillingness of infants to move the limbs has led to scurvy being mistaken for poliomyelitis. Careful examination will show that in scurvy the condition is only a pseudo-paralysis. Poliomyelitis is a febrile disease of acute onset, but this stage is often overlooked. There may be pain on motion in poliomyelitis, but there is usually no tenderness to pressure as in scurvy. When any other signs of scurvy are present, there should be no difficulty in recognizing the condition.

TUBERCULOUS JOINT DISEASE.—This condition shows pain on motion, with muscular spasm, but usually no tenderness to pressure over the bone. Swelling, when present, is in the joints in tuberculosis, not over the bone, as in scurvy. The presence of hemorrhage elsewhere is diagnostic of scurvy. Roentgenograms will settle the diagnosis in doubtful cases.

OSTEO-SARCOMA.—I have seen cases of scurvy in which the swelling was mainly confined to one limb and very large, mistaken for osteo-sarcoma. The other signs of scurvy were present, but overlooked. The extreme tenderness of the swelling is the characteristic feature of scurvy.

OSTEOMYELITIS.—Scurvy is frequently mistaken for osteomyelitis by those who forget the possibility of the former. Osteomyelitis is usually confined to one limb, is characterized by a continued high febrile reaction, and by leucocytosis, and lacks the tendency to hemorrhages in other parts of the body. The diagnosis can be settled by X-ray examination.

ACUTE NEPHRITIS.—Albumin is often found in the urine in scurvy, and I have seen cases in which hematuria was the only symptom, mistaken for acute nephritis. The absence of edema, the absence of a diminished quantity of urine, and the absence of many casts, all point to scurvy. The rapid disappearance of the hematuria under antiscorbutic treatment, will finally confirm the diagnosis.

PROGNOSIS.—Scorbutus is very variable in its duration. If left untreated, all the symptoms may become more pronounced and the infant may finally die of exhaustion. When properly treated,

and uncomplicated by any other disease, the prognosis is very favorable. There is no disease in which the favorable reaction to treatment is more rapid. Tenderness disappears almost at once, and the hemorrhages into the gums, skin, and urine, clear up very rapidly. The swelling over the bones caused by subperiosteal hemorrhage persists somewhat longer, but recovery is always finally complete. The only danger in scorbutus comes from the disease remaining so long unrecognized, that the vitality of the infant has been greatly lowered. In such cases, death may occur from gastro-intestinal disease or intercurrent infection.

TREATMENT.—The treatment of infantile scorbutus is very simple and consists in two things, plenty of *raw milk*, and *orange juice*.

Fresh cow's milk should be used, modified to suit the peculiarities of the infant's digestive powers and requirements. It is advisable that the infant should take as much milk as he can digest, and therefore the modification should be made as strong as is possible without producing signs of indigestion. Slightly undigested or green movements should not be a contraindication to giving a strong food, provided that these symptoms are not increasing. A good milk supply is essential, in order to avoid the necessity of pasteurization, which is contraindicated in scurvy, unless a good milk supply cannot be obtained. In such a case, a wet-nurse should be engaged if possible.

With a diet of raw milk, or human milk, complete recovery will occur without further treatment. The process of recovery will, however, take considerable time, and can be much hastened by the addition of fresh fruit juice to the diet. The juice of any ripe fruit is effective, but *orange juice* is to be preferred, as it can be easily obtained, and babies usually like it. It rarely disturbs digestion, and is less likely to do so when given in a single daily dose than when given in divided doses before each feeding, as is usually recommended. The daily dose should be one ounce, given an hour before the morning feeding. It may be diluted or sweetened, if taken better in this way. Orange juice is all that is necessary to effect a complete and rapid cure of scurvy. No drug has any influence upon the disease.

In older children, whose diet contains other things than milk, potato is a valuable antiscorbutic, if it can be digested. All fresh vegetables which can be given, are also antiscorbutic. These things should only be given, however, when the patient has reached the age which permits their introduction into the diet.

In the prophylaxis of scorbutus, the principal thing is the avoidance of the patent foods, and all foods prepared by heat. If babies are breast-fed, they will almost never develop scorbutus. The ques-

tion of the pasteurization of the cow's milk used in the artificial feeding of infants, is often raised in connection with the prophylaxis of scorbutus. Is it undesirable to pasteurize milk on account of the danger of the development of scurvy? It has been shown that scurvy is more likely to develop from the boiling or prolonged heating of milk, than from simple pasteurization. Still, it is probable that even pasteurization partially at least, destroys the antiscorbutic substances in milk, as scurvy develops more often in infants fed on pasteurized milk than in those fed on raw milk. The question therefore depends on the danger to be apprehended, from scorbutus with pasteurized milk, and from bacterial infection with raw milk. This has been discussed in the division on Feeding. In general, it depends on the character of the milk supply. With certified milk, or milk produced under properly inspected conditions, raw milk is preferable. With any other milk supply, the dangers from infection are greater than the dangers from scorbutus, and the milk should be pasteurized.

PROBLEMS AND RESEARCH.—Investigations on the subject of scurvy have been made by animal experiments, by metabolism experiments on infants, and by blood coagulation experiments.

Holst and Fröhlich first produced experimental scorbutus in animals in 1907. They, with Furst, have since continued the work, and have published numerous papers. Their work conclusively proved that scurvy is not due to starvation or inanition, but to the lack of some essential food element. Scurvy has been produced by various diets, and cured by various articles of diet. The antiscorbutic properties of various vegetables have been investigated, and it has been found that while they are weakened, they are not entirely destroyed, by cooking. Variations in the antiscorbutic power of various vegetables and fruit juices to withstand heat and drying have been demonstrated. Other observers have confirmed and extended these results None of these experiments has thrown any light on the nature of the antiscorbutic bodies, although efforts have been made to isolate them by extraction, dialysis, and other methods.

A few studies have been made on the metabolism of scurvy. While the results are interesting, they have as yet thrown no light on the essential nature of the disturbance seen in the disease.

Hess and Fish have recently published an interesting study bearing on the question of the cause of the hemorrhages in scurvy. They studied the coagulation factors in infantile scurvy, and were unable to demonstrate any marked decrease in the coagulation time of the blood. There was no evidence of calcium deficiency or of antithrombin excess, and the platelet count was normal. They then studied

the blood vessels by means of what they term the " capillary resistance test," which consists of binding a limb in such a way as to increase the blood pressure in the vessels, and observing the occurrence of hemorrhage. They found that there was a weakness of the vessel walls in scurvy; this is, however, not pathognomonic of scurvy, but is found in other hemorrhagic diseases. This work supports the theory that the hemorrhages of scurvy are not due to changes in the blood secondary to disease of the bone marrow, but are due to a condition of the blood vessels which is a primary manifestation of the disease.

THE VITAMINS.—The subject of scorbutus cannot be concluded without reference to the interesting and suggestive theories of Funk, published in 1914. He believes that there exists in food certain substances which play an important rôle in physiology and pathology, and which he calls vitamins. He believes that these substances are essential to proper metabolism and nutrition, and that their absence causes certain diseases, such as scorbutus, beriberi, pellagra, and rickets. He calls these diseases the "avitaminoses." He shows from his own work and that of others that milk contains substances which favor the growth of young animals, and attributes the development of scurvy in infants to the taking of food not containing milk, or containing an insufficiency of milk, or consisting of heated milk. He believes that the vitamins are the substances which are sensitive to heat; also that at times, raw milk or even breast-milk contains an insufficiency of vitamins. He advances evidence to show that the amount of vitamins in cow's milk varies with the food of the cows; cow's milk contains less of the vitamins in winter when the cows are eating dry fodder than in the summer when their fodder is green. He also calls attention to a diminution of the vitamins in the milk of women who are underfed.

The premises of Funk's conclusions are not yet established facts. Nevertheless the theory fully covers and explains all the clinical and experimental facts which have been established as to scurvy. It is possible that the subject of the vitamins, and the etiological relationship of scurvy to such diseases as beriberi and pellagra, will be the subject of much study in the next few years.

PURPURA

The term purpura is retained here chiefly on account of its frequent appearance in pediatric literature. Purpura is not a disease, but a symptom-complex, characterized by a tendency to spontaneous hemorrhage. Many writers, especially in Europe, recognizing the great variety of conditions in which hemorrhages occur, call this syndrome characterized by a tendency to hemorrhage the " hemor-

rhagic diathesis." This term is objectionable for several reasons. In the first place the word " diathesis " implies that there is some constitutional factor underlying this tendency toward spontaneous bleeding; this has not been proven, and is supported by little evidence. In the second place it groups together cases in which the hemorrhage is the primary essential feature of the disease, and cases in which the hermorrhage is secondary to proven causes. Among the conditions characterized by hemorrhage, three represent fairly definite disease entities, namely, hemorrhagic disease of the newborn, scorbutus, and hemophilia. These diseases are consequently considered separately. There remains only a symptom-complex, and it is to this symptom-complex that the term purpura is here applied.

Spontaneous hemorrhages are often seen in pathological conditions of which the nature is definitely known. In such cases, the hemorrhage is obviously a symptom of the known disease, and the hemorrhagic symptom-complex may be classified as *symptomatic purpura*. In a number of other cases, the spontaneous hemorrhages are apparently the primary pathological condition. It is probable that in these cases also the hemorrhagic symptom-complex is symptomatic of some other primary pathological condition, but as the underlying cause is unknown, this group may be conveniently described as *primary purpura*, the term primary being equivalent to "of unknown cause." In view of the fact that in many cases belonging to this group, abnormal changes in the composition of the blood have been demonstrated, it might seem that the primary purpuras should be classified with the diseases of the blood. The changes in the blood, however, do not appear to affect the cellular elements of the blood in such a way as to form a definite lesion, as is the case in such diseases as leukemia and pernicious anemia Moreover, the blood changes are strongly suggestive of either nutritional disturbance, or of the action of toxin. It is for this reason that the primary purpuras are classified among the disturbances of nutrition and metabolism.

SYMPTOMATIC PURPURA

ETIOLOGY.—The various known diseased conditions in which symptomatic purpura is seen at times, suggest the following factors in etiology:—Infection, toxemia, nutritional disturbance, and disease of the blood. Mechanical hemorrhages, such as are seen in pertussis and epilepsy, are also sometimes classed as purpura. At times after protracted illness, when patients first stand or walk, hemorrhagic spots appear on the lower extremities. It is probable that in such cases the cause is partly nutritional and partly mechanical.

INFECTION.—Purpura is often seen as a symptom of various known acute infections. It is most constant as a symptom in malignant endocarditis, but is sometimes seen in measles, scarlet fever, small-

pox, vaccinia, typhus fever, epidemic meningitis, diphtheria, septicemia, and pyemia. The frequency of its presence in infectious disease of the newborn has already been described. In these acute infections, the hemorrhagic symptom-complex is probably due to an altered condition of the blood, which in turn is caused by the bacterial toxins of the primary disease.

TOXEMIA.—Certain drugs, and certain known chemical poisons, can produce spontaneous hemorrhages. The effect on the blood of benzol poisoning is well known. Such drugs as potassium chlorate, quinine, and phosphorus have produced cutaneous hemorrhage when given in excessive dosage, or in long-continued administration. The pronounced tendency to hemorrhage seen in cases of jaundice is certainly of toxic origin. This group is distinguished from the first only in that the toxins are not of bacterial origin.

NUTRITIONAL DISTURBANCE.—Purpura often occurs as a late symptom of various known diseases in which marked disturbance of nutrition is a prominent feature. While most of these diseases are of infectious origin, the occurrence of hemorrhages late in the course of the disease, points toward a nutritional injury rather than a toxic injury. Also, purpura is seen in cachectic conditions which are not of infectious origin, such as starvation, functional gastro-intestinal disease, and malignant disease. Cachectic purpura is seen particularly often in the wasting diseases of infancy, and is met with in tuberculosis, meningitis, bronchopneumonia, and empyema. There is little doubt that the symptom is due to a disturbance of nutrition, either of the blood, or of the blood vessels. An example of a hemorrhage of nutritional origin is seen in scurvy.

DISEASES OF THE BLOOD.—Purpura is a fairly common symptom in primary diseases of the blood and blood-making apparatus, such as pernicious anemia, leukemia, and pseudo-leukemia.

SYMPTOMS.—The hemorrhages in symptomatic purpura are usually limited to the skin, and appear as hemorrhagic spots of various sizes and varying distribution. Rarely hemorrhage from the mucous membrane is seen.

DIAGNOSIS.—The hemorrhagic character of the spots is recognized by the fact that the color cannot be caused to fade by pressure. The diagnosis of the symptomatic character of the purpura depends upon the finding of an adequate cause.

PROGNOSIS.—The prognosis is that of the disease to which the purpura is secondary. In most of these conditions the appearance of purpura is a bad prognostic sign.

TREATMENT.—The treatment of symptomatic purpura is that of the cause.

PRIMARY PURPURA

ETIOLOGY.—The purpura is called primary when there is no recognizable cause. There has been much research devoted recently to the hemorrhagic diseases of early life, as a result of which a certain amoung of knowledge has been gained, which can form a basis for a theoretical conception of the nature of the purpura, and of the factors probably concerned in its etiology.

The symptom-complex called purpura is characterized by hemorrhage, and the tendency to spontaneous bleeding must be due to one or both of two immediate causes. The first is an abnormal composition of the blood, which lessens its coagulability. The second is an abnormal condition of the blood vessels which increases their permeability.

The coagulation of the blood has been extensively studied in recent years. While the full mechanism is still not perfectly understood, considerable advances have been made. In most of the cases of primary purpura which have been studied, a defective power of coagulation has been found in the blood. This is shown partly by a delay in the coagulation time, and partly by the formation of a clot of less firmness and contractility. Most of the cases studied have been typical cases of the clinical type called "purpura hemorrhagica," and the most usual finding suggests that the diminished coagulability is due to a deficiency in the prothrombin, the substance derived from the blood platelets. A marked diminution in the blood platelet count has been found repeatedly in purpura hemorrhagica. While this may not apply to all cases, and while some cases of purpura may show some other abnormality in the blood, this finding suggests at least that *there is often if not always in purpura an abnormal condition of the blood itself*, which manifests itself in defective coagulation.

The second possible factor, an abnormal condition of the walls of the blood vessels, has not yet been extensively studied. It cannot, however, be excluded in a condition which is not a single disease entity, but a symptom-complex due possibly to a variety of causes.

If the spontaneous hemorrhages of purpura are due to an abnormal condition of the blood or blood vessels, what causes this abnormal condition? It is not, as in hemophilia, an inherited constitutional anomaly, for the occurrence of purpura suddenly, at any age, without obvious cause, suggests that the cause is not an inherent tissue defect, but that it is acquired. In reaching an idea as to the probable cause of primary purpura, the varied etiology of the secondary, symptomatic purpuras is most suggestive. These secondary purpuras are associated with a number of known conditions, and *infection*, *toxemia*, and *nutritional disturbances* are the most probable causes of the hemorrhage. We know that all of these factors can exist with-

out definite recognition. It is probable that there are numerous types of bacterial infection which have not been recognized, and many writers believe that the chief cause of primary purpura is such unrecognized infection. Furthermore, we know comparatively little about the function of nutrition and metabolism, particularly about the so-called intermediary metabolism. There is much evidence that suggests that toxic products may be formed through a disturbance of metabolism of unknown nature. Again, emaciation is not a necessary manifestation of all nutritional disturbances, as is evident by such conditions as rickets and scurvy, in which loss of weight is not an essential feature. We must therefore admit that it is very probable that there are disturbances of metabolism of which we know nothing, but which may easily lead to the formation of toxic products, and to disturbed nutrition.

The blood is the result of the very complex work of all the organs and tissues of the body. Its composition probably depends on the activity of many organs, and of many cells, acting both as individual entities and in relation with each other. It is therefore easy to conceive how infection, disturbance of metabolism, toxin formation, and impaired nutrition, could impair the composition of the blood itself, and cause a defective condition of the walls of the blood vessels. I believe that these are the probable causes concerned in the production of purpura, and that unrecognized infection is probably the most frequent factor. The symptoms of the various clinical types of purpura are strongly suggestive of infection and toxemia, and also of a varied etiology. We shall be obliged to speak of primary purpura until the nature of the disturbances, which are the real primary conditions of which spontaneous hemorrhage is the symptom, is more fully understood.

Primary purpura is more common in children than in adults. It is met with at all ages, although more often in older children than in infants. It is commonest between the ages of two and ten years. The sexes are about equally affected. There is no constant association with any particular predisposing cause.

PATHOLOGICAL ANATOMY.—The pathological anatomy requires no detailed description, the essential lesion being the hemorrhage. The variation in the extent and location of the hemorrhages is so great, that enumeration is cumbersome and unnecessary. The changes in the blood are confined to its finer composition, and nothing abnormal is noted on ordinary examination, other than the characteristics of a secondary anemia, the severity of which is in proportion to the amount of the hemorrhage.

SYMPTOMS.—Purpura has been divided upon a purely clinical basis into various types. This division bears no definite relation to

the probable etiology of the disease, although it is possible that some such relation may be found in the future, when the various etiological factors are more fully understood. The division into clinical types is retained for purposes of clinical description only, the various types not representing definite disease entities.

PURPURA SIMPLEX.—This is the commonest form of primary purpura in children, and in it the hemorrhage is confined to the skin. There may be moderate constitutional disturbance, which may accompany the hemorrhages, or may precede them for one or two days. The general symptoms are malaise, loss of appetite, and slight fever; there may be a disturbance of digestion with vomiting or diarrhea. The hemorrhages appear suddenly, and consist of small petechiae. The spots are small, varying in size from a pin-head to a pea, and are of a bright red color. As the spots fade, they become brownish-purple, and disappear in a few days. The distribution of the spots is very variable; certain parts of the body only may be involved, or the whole body may be covered. The petechiae usually appear first on the lower extremities. The spots in the involved areas are usually thickly scattered, but there may be only a few spots widely scattered on various parts of the body. The spots are likely to appear in successive crops, coming sometimes in new areas, and sometimes in areas where a former crop is fading or has faded. Often when the child gets out of bed, a fresh crop will appear on the legs.

The course of the disease is usually from one to three weeks, but relapses are common, and attacks may continue to come at short intervals for a considerable period. I have seen cases in which the child was unable to get out of bed for several months without the appearance of fresh spots. The prognosis is good, although it is best not to give an unqualifiedly favorable prognosis, because occasionally a mild simple purpura has suddenly developed into one of the severe and fatal types.

PURPURA RHEUMATICA (Peliosis Rheumatica; Schönlein's Disease). This type resembles simple purpura in every way, except that there is an additional symptom, namely, arthritis. The joints are painful on motion, and show some swelling. This type may be considered a primary purpura with an associated arthritis, or a symptomatic purpura secondary to an infectious arthritis. The association with rheumatic fever is, however, not very definite. The arthritic symptoms are less severe, and the heart is not involved. There is a more frequent association with certain other cutaneous lesions, particularly erythema exudativum, urticaria, erythema nodosum, and angioneurotic edema. This type is strongly suggestive of a toxic origin, but whether the toxin is derived from bacteria or from a disturbance of metabolism is unknown.

The course of rheumatic purpura is from one to three weeks. Relapses may occur. The outcome is usually favorable.

PURPURA ABDOMINALIS. (Henoch's Purpura.)—This type closely resembles rheumatic purpura, except that it is more severe, and gastro-intestinal symptoms are prominent. The symptoms are more or less malaise, and pains not especially localized, but chiefly occurring in the extremities and back, sometimes accompanied by slight edema of the part affected. These early symptoms of pain occur in one or more joints, usually on the outer sides, and sometimes there are swelling and redness simulating rheumatic fever. In this stage there may be a sudden rise of temperature. Accompanying these symptoms there may be a few purpuric spots, but, as a rule, there is a period of several days between the appearance of the pains in the joints and the purpuric appearance on the skin. The purpuric spots may coalesce, and thus form ecchymoses of various sizes and colors. They are very apt to begin on the lower leg and spread up to the thighs, genitals and body. Somewhat later, intestinal symptoms develop. While the purpura is spreading there is severe colic, and the pain is very intractable to treatment. The abdomen is retracted and tender. There is obstinate vomiting. The pulse is weak, and the face has an anxious expression. There is more or less diarrhea, which usually occurs at the end of an attack of colic. The colic and vomiting sometimes last for one or two days. There may be a little blood in the vomitus and in the movements. The vomiting then diminishes, the colic ceases, and later the diarrhea stops, the pain in the joints passes away, the purpuric spots gradually fade and disappear, and the child, although left in an exhausted condition, is otherwise well.

There are very apt to be relapses, which may appear within a few days or not for several days.

These are the symptoms of a typical case; but there are many variations. As a rule, the younger the child the more typical is the case. Sometimes the purpuric spots closely simulate urticaria. They may occur, although rarely, in the mouth. They sometimes simulate the lesions of erythema nodosum. The attacks of colic have a paroxysmal character. There may be swelling of the joints.

This disease is rarely fatal unless it is complicated by some such disease as nephritis or endocarditis.

PURPURA HEMORRHAGICA. (*Morbus Maculosus Werlhofii.*)—This is a very severe form of primary purpura, characterized by hemorrhages from the mucous membranes as well as cutaneous hemorrhages. Bleeding from the mucous membranes is present from the start, and may even precede the cutaneous ecchymoses. There may be bleeding from the nose, mouth, pharynx, stomach, or intestines.

The most frequent symptoms are the vomiting of blood, and the passage of bloody discharges from the bowels. The urine may contain enough blood to be bright red in color. There may more rarely be bleeding from the ear, conjunctiva, or female genitals. Ecchymoses may sometimes be seen on the visible mucous membranes, and hemorrhages may occur into the retina or choroid of the eye.

Purpuric spots on the skin are always present. They are most often large ecchymoses, which appear suddenly in various parts of the body.

Constitutional symptoms, such as fever, and malaise, are often present, but not constant. All the abdominal symptoms described under Henoch's purpura may be present. Anemia and prostration finally become marked, and the loss of blood may be sufficient to cause death.

The course of the disease is very variable, the symptoms usually lasting from one to six weeks. Death may occur from loss of blood, or the patient may pass into a condition suggesting general sepsis, with high fever, delirium, great prostration, and irregular failing pulse, which finally ends fatally. A certain number of cases recover, but the prognosis in general is unfavorable in early life, depending on the age of the patient and the extent of the hemorrhage.

PURPURA FULMINANS.—A very malignant purpura hemorrhagica may occur, sometimes proving fatal within twenty-four hours. This form of purpura is usually spoken of as purpura fulminans. It is most commonly met with in infants and in very young children, and is characterized by excessive cutaneous hemorrhages which develop with great rapidity, death sometimes taking place before there has been any hemorrhage from the mucous membranes. There is high fever, vomiting, marked prostration, cerebral symptoms, and the disease has all the characteristics of an acute general infection.

DIAGNOSIS.—The diagnosis of purpura depends first upon the recognition of the hemorrhagic spots on the skin. The eruption is recognized as hemorrhagic by the failure of the spots to disappear on pressure. The diagnosis of the " primary " nature of the purpura can only be made by a careful exclusion of all the conditions in which purpuric spots sometimes appear as a symptom. The diagnosis of the clinical type of purpura depends on the associated symptom-complex.

TREATMENT.—The etiology of purpura being unknown, and probably multiform, there is no specific treatment for primary purpura. No drug has any influence on the underlying causes of the disease. The treatment must therefore be symptomatic.

The only serious symptom requiring treatment is the hemorrhage.

The clinical types of purpura characterized only by cutaneous hemorrhages, are not especially dangerous, and the hemorrhage requires no treatment. Simple purpura, purpura rheumatica, and Henoch's purpura, without hemorrhage from the mucous membranes, all run a definite course, and tend toward recovery. Not enough is known about the etiology to afford a definite basis for prophylactic treatment directed at preventing relapses, but in cases with a marked tendency toward relapse, serum therapy may be tried. Horse serum, rabbit serum, and human serum all have a theoretical basis in the hemorrhagic diseases. Their relative value is not definitely determined, but the evidence points toward human serum as the most valuable, and horse serum as the least; just the reverse of the ease with which the various sera can be obtained.

In simple purpura, no other symptoms need be treated. Rheumatic purpura may be symptomatically treated like rheumatic fever, with salicylates. Henoch's purpura should be treated like an acute infection with gastro-intestinal symptoms.

Purpura hemorrhagica, or any form of purpura in which blood is lost from the mucous membranes, is a dangerous condition, and requires active treatment of the hemorrhage. Various methods of treatment have been recommended from time to time for the hemorrhagic diseases, each based on some particular theory of the pathology of the condition. The evidence at present is against the value in purpura of injections of gelatin, of injections of adrenalin, and of the administration of calcium salts. There remains, serum therapy. Horse serum, rabbit serum, and human serum or human blood, all have theoretical evidence in their support, because any of them may supply the element which causes diminished coagulability of the blood. There is clinical evidence in favor of all three sera. Human serum is usually free from the possible danger of anaphylaxis, and experiment suggests that it is more potent than rabbit serum, while rabbit serum is more potent than horse serum.

In comparatively mild cases, horse serum may be tried first, given in doses of 30 c. c. or more, according to the severity of the hemorrhage. Rabbit serum may be tried first, or after the failure of horse serum.

In all severe cases, human serum should be preferred. The donor having been obtained, about 60 c. c. of blood should be withdrawn with a sterile syringe from the vein at the bend of the elbow. Of this, 30 c. c. should be injected subcutaneously at once, and the rest should be set aside to furnish serum for further treatment. In the subsequent injections, this serum may be used, and if possible, from 10 to 30 c. c. should be given every four to eight hours, according to the course of the case.

In any case in which the patient is so severely ill from loss of blood

that life is threatened, or if the injections of whole human blood or human serum are not followed by definitely recognizable improvement, the safest treatment is by transfusion. Whenever a surgeon can be obtained who is familiar with the rather formidable technique of this operation, it is the most promising method of treatment.

Serum therapy, and transfusion are the only available methods of checking the bleeding in purpura hemorrhagica, and are often strikingly successful. They do not always succeed, however. I have recently seen a case which died of hemorrhage in spite of repeated transfusions. This only emphasizes the probable multiplicity of the factors which are concerned in the pathology of purpura.

After convalescence from an attack of purpura, the anemia is to be treated with iron in the ordinary manner.

PROBLEMS AND RESEARCH.—The principal problem awaiting further research in connection with purpura, is that of etiology. The most promising field for investigation lies in the pathology of the blood. It is probable that the symptom-complex known as purpura, represents not only a variety of underlying causes, but also a variety of abnormalities in the blood and blood-vessels. The first step will be the recognition of all the various abnormalities in the blood and bloodvessels which are seen in purpura, and their classification. This problem has already been attacked. For a description of the known facts concerning the coagulation of the blood, the reader is referred to the Problems and Research in connection with hemorrhagic diseases of the newborn in Division III.

It is probable that there are various types of purpura representing deficiencies in different elements of the group of factors concerned in blood coagulation. Deficient coagulability of the blood may be due to a deficiency of prothrombin (blood platelets), of calcium salts, of kinase, or of fibrinogen. It may be due to an excess of antithrombin. Finally, in some cases, the tendency to hemorrhage may be due to an abnormality of the vessel walls.

The problem of purpura has already been attacked along these lines. The principal abnormality of the blood found in purpura hemorrhagica appears to be a deficiency of prothrombin. Duke found in typical cases an enormous reduction of the blood platelets. This has been confirmed by other observers. A low platelet count has also been found in certain cases of symptomatic purpura. Furthermore, purpura has been produced experimentally in animals by injections of the serum of an animal immunized with blood platelets. It has also been produced in rabbits by injections of diphtheria toxin, which reduced the platelet count. All this work, however, by no means proves that this is the only blood deficiency present in purpura.

Some evidence has been brought forward that purpura is not due

to a deficiency of calcium. It will, however, be necessary to investigate many cases of purpura by many different methods before a final solution of the problem is reached. The methods of investigation now in use are the following: Measurements of the coagulation time; studies on the firmness of the clot; studies on the effect of adding calcium salts; studies on the effect of adding defibrinated human blood; studying the effect of the purpuric blood on normal blood; artificial extraction of the thrombin in pure form; measurement studies on the condition of the vessels by capillary resistance tests. It is possible also that increasing knowledge of the obscure manifestations of disturbed metabolism may throw some light on the etiology of purpura.

HEMOPHILIA

Hemophilia is characterized by excessive and prolonged bleeding following an injury, and is a clear-cut clinical entity. It is an inherited, constitutional anomaly.

ETIOLOGY.—The inherited tendency of the disease is one of the factors which sharply differentiates hemophilia from other hemorrhagic conditions. The disease nearly always affects males, and is usually transmitted through healthy females. The tendency to hemorrhage usually persists unchanged throughout the life of the patient. There is no evidence of any connection with purpura, scurvy, infection, toxemia, or any other diseased condition.

The immediate cause of the hemorrhage is usually a mechanical injury to the tissues. From a wound which would ordinarily produce slight bleeding, the hemorrhage is excessive and prolonged. At times hemorrhage is produced by an injury so slight that bleeding would not follow in normal persons. Occasionally the hemorrhage appears to be spontaneous, but this is probably due to the fact that in hemophilia, slight mechanical shocks such as are incidental to ordinary daily life, may cause bleeding.

The nature of the constitutional abnormality in hemophilia is still an open question. Many theories have been advanced to explain the hemorrhages, such as an abnormal thinness of the walls of the bloodvessels, high blood pressure, disproportion between the total amount of blood and the capacity of the vascular system, and various abnormalities in the composition of the blood. There is no evidence in favor of any of these theories except the last, and the weight of modern opinion inclines toward the opinion that the inherited anomaly in hemophilia involves the blood.

As to the nature of the blood deficiency, various investigators claim various abnormal conditions, such as a diminution in the amount of fibrinogen, calcium salts, prothrombin, or thrombokinase, or an excessive amount of antithrombin. Lack of calcium has been

shown not to be the cause, and there is no convincing evidence of a deficiency of blood platelets, or of thrombokinase. The present state of the matter lies between a deficiency of fibrinogen, and an excess of antithrombin.

Cases are occasionally met with having all the clinical features of true hemophilia, but with no evidence of heredity. These cases occur later in life, affect both sexes, and probably represent an accidental sporadic type of unknown cause. Etiologically they probably belong with the purpuras.

SYMPTOMS.—In hemophilia the first manifestation of the disease does not often appear until the second year, or even later. This is probably due to the fact that wounds and traumatic injuries are uncommon until the child begins to run about. Occasionally there is excessive bleeding from the umbilicus at birth, but this is rare in hemophilia. The first hemorrhage may be either traumatic, or apparently spontaneous. In traumatic hemorrhage, a very slight wound, or the drawing of a tooth, is followed by severe and prolonged bleeding. A slight contusion may cause an extensive hematoma between the muscles. The commonest site of spontaneous hemorrhage is the nose, but bleeding is occasionally seen from the mouth, intestines, stomach, urethra, and lungs.

Hemorrhage into the joints is a fairly common form of apparently spontaneous hemorrhage in typical cases. The joints normally are constantly subjected to mechanical shocks, and in hemophilia the ordinary activities of life are sufficient to produce hemorrhage. The blood in articular hemorrhage is not all absorbed, but becomes partly organized, and this leads to symptoms resembling arthritis. The articular symptoms of hemophilia have been described as a coördinate manifestation, but they are really the result of a former hemorrhage which has been overlooked. Repeated hemorrhage may finally lead to marked articular deformity

The essential feature of the hemorrhages of hemophilia is their prolonged and uncontrollable character. Sometimes over a wound a crust may form and bleeding may cease externally, but continue into the subcutaneous tissue. Later, the accumulated blood breaks through, and then the hemorrhage is still more difficult to control.

There is a sporadic, accidental type, in which the hemorrhage resembles the bleeding of true hemophilia. This type is not inherited, usually appears later in life than childhood, and affects the sexes equally.

DIAGNOSIS.—The first hemorrhage of hemophilia so rarely occurs in the first week of life, that there is little difficulty in distinguishing from it the hemorrhagic diseases of the newborn. The only condition for which hemophilia might be mistaken is purpura hemor-

rhagica. In the latter the hemorrhage is always spontaneous, and there is almost always hemorrhage into the skin. The essential feature of hemophilia is that wounds and injuries tend to produce excessive hemorrhage. Even when the hemorrhage is apparently spontaneous, there is usually a history of previous excessive bleeding from a wound. The hereditary nature of hemophilia, and the absence of cutaneous petechiae, are further distinguishing points.

PROGNOSIS.—The first attack is rarely fatal. Many attacks may occur, without a fatal result, and the children may grow up and live indefinitely. Sometimes the tendency toward hemorrhage is outgrown in later life, but in typical cases it persists.

On the other hand, any attack, even from a slight cause, may lead to marked exsanguination, or even to a fatal ending. The chances are a little more than even that a patient showing signs of hemophilia in childhood will not live to enter adult life.

TREATMENT.—The hemorrhage from wounds is treated by the usual surgical measures for the control of bleeding, particularly compression. The treatment of severe cases is with serum, as described for purpura rheumatica. Transfusion should always be prepared for in every case, and used whenever the bleeding appears to be becoming dangerous.

PROBLEMS AND RESEARCH.—Hemophilia has been, and is being, investigated by the same methods as are used in the other hemorrhagic diseases, consisting chiefly of laboratory study of the factors concerned in the coagulation of the blood Among the recent investigations is one of Whipple, showing increased antithrombin in hemophilia. Several investigators have found a normal platelet count in hemophilia, in contrast to the diminished count in purpura hemorrhagica. Hess has recently devised a test for antithrombin, and has found that in animals it can be increased considerably without danger. His recent work showed, by a functional test, evidence of fibrinogen deficiency in hemophilia, but not in purpura. This did not seem to be the sole deficiency, as the addition of fibrinogen is often unable to bring the clotting of the blood to normal. Lack of calcium has been shown not to be the cause of the bleeding in hemophilia, but Hess found one sporadic case due to calcium deficiency, which he called hemophilia calcipriva. The evidence is rather against abnormality of the capillaries as a factor.

Schloessmann found that a tissue extract, made from hyperplastic thyroid material, was effective as a local application in hemophilia. Hess found a tissue extract, made of ground-up tissue, which he calls thromboplastin, similarly effective. Several European investigators have advocated the subcutaneous injection of propeptone (peptone de Witte), believing it to be more effective than serum therapy.

ACIDOSIS

In the broadest sense, the term acidosis refers to a diminished alkalinity of the blood. The physicochemical reaction of the blood, which depends upon the balance between the hydroxyl and hydrogen ion concentration, is fairly constant, even in acidosis. In acidosis, it is the titrable alkalinity of the blood which is diminished. Acidosis may be relative or positive. Relative acidosis is due to a deficiency of the bases which cause the normal titrable alkalinity of the blood; an example of relative acidosis is seen in severe diarrhea, and in indigestion from excess of fat. Positive acidosis is due to the production of acids in abnormal quantity. It is only positive acidosis which is to be considered here.

The most satisfactory definition of acidosis under this limitation is that of Cautley, who called it "an abnormal metabolism of carbon leading to the appearance of organic acids in the blood and urine, and to the formation of ammonia to neutralize these acids."

The only definite clinical evidence of acidosis obtainable is the finding in the urine of the so-called "acetone bodies" consisting of acetone, diacetic acid, and beta-oxybutyric acid. These acetone bodies are found in the urine in a great number of known pathological conditions. The most perfect example of a positive acidosis is seen in diabetic coma. The acetone bodies, however, appear very frequently in the urine of children, in conditions of known etiology. They are found in starvation and cachexia, in several known forms of drug poisoning, and in the so-called delayed anesthetic poisoning. They are found in hepatic disease of known type, such as acute yellow atrophy. They have been found in eclampsia, and in nervous disturbances due to fright. They are often present in the acute infections accompanied by high fever, such as the exanthemata, diphtheria, pneumonia, influenza, appendicitis. They are rarely found in the gastro-intestinal disorders of infancy, but are very common in those of later childhood. Frew, however, in his investigations at the Great Ormond Street Hospital, found transient acetonuria in most babies who were changed from the breast to the bottle. It persisted for about three days, and was ascribed by Frew to carbohydrate starvation due to a temporary failure of digestion. In most of these cases, the presence of the acetone bodies is not accompanied by toxic symptoms. From this point of view, acidosis as manifested by the appearance of the acetone bodies in the urine, must be regarded simply as a symptom of disturbed metabolism, with no more clinical significance than has albuminuria in similar conditions. There would consequently be no need to discuss acidosis under a separate disease heading.

There are, however, certain cases in which there is a fairly con-

stant and definite symptom-complex of disease, which cannot be readily attributed to known pathological conditions. The symptoms which have particularly attracted attention are attacks of severe vomiting without obvious or discoverable cause, but with a definite tendency toward recurrence. In other cases the prominent symptoms are vomiting accompanied by marked symptoms of nervous-system intoxication, again without obvious or discoverable cause, but without a definite tendency toward recurrence. To these symptom groups the names *Recurrent Vomiting*, *Periodic Vomiting*, *Cyclic Vomiting*, *Acid Intoxication*, and *Acidosis*, have been applied.

In 1901, Marfan noted the appearance of acetonuria in recurrent vomiting, and in 1903 Edsall called attention to the large quantity of the acetone bodies found in the urine in the same disease. This led to a general discussion of whether the formation of the acetone bodies is the cause of the symptoms, or whether it is only a coordinate manifestation of the disturbance of metabolism.

ETIOLOGY.—The relation of the acidosis to the symptoms in recurrent vomiting and the so-called acid intoxication is not definitely known. The cause of the disturbance of metabolism which produces the clinical symptoms and the acetonuria is not definitely known. The literature of the subject is filled with conflicting theories, but it is necessary for a student of the subject to have some knowledge of the views most widely held.

HISTORY OF THEORIES.—Langmead in 1906, doubted that the acetone bodies were the cause of the symptoms and ascribed the production of acid poisons to deficient oxidation of the fats in the liver, in consequence of an impairment of hepatic function caused by either alimentary toxins, or bacterial toxins from other parts of the body. Ewing also considered the condition due to a disturbance of fat metabolism, caused by deficient hepatic function, associated with the absorption of poisonous putrefactive products from the intestine. Rachford considers the condition an autointoxication, functional hepatic derangement causing an accumulation of purin bodies in the blood.

Another group of writers believe that it is the glycogenic function of the liver which is primarily disturbed. The analogy of the acidosis of diabetes is suggestive here, and it is known that carbohydrate starvation can produce acetonuria. Janeway and Mosenthal thought carbohydrate starvation important in acidosis, but more recent work appears to show that the symptoms are not due to a deficiency of carbohydrate in the diet. It is fairly well established that the normal metabolism both of fat and of protein is largely dependent upon proper carbohydrate metabolism, and is greatly disturbed by a deficiency of carbohydrate. Sedgwick called atten-

tion to increased creatin and creatinin excretion during attacks of recurrent vomiting; this is evidence of a disturbance of the intermediary protein metabolism. Mellanby also found creatinuria in recurrent vomiting. He considers acidosis secondary to derangement of the glycogenic function of the liver. The disturbance leads to imperfect Ratabolism of the fats, w th the formation of diacetic and beta-oxybutyric acids as intermediary products, and to imperfect protein metabolism and creatin formation due to carbohydrate insufficiency. The disturbed glycogenic function is due to absorption of alimentary toxins. He does not believe that the acidosis is ever the cause of the symptoms, because the quantity of beta-oxybutyric acid (the only toxic member of the acetone group) is never large, the quantity of the acetone bodies being always small compared with that seen in diabetes. The acidosis is only a symptom of the disturbance of metabolism. Mellanby's view is strengthened by the recent work of Underhill and Steele, who found in the urine lactic acid as well as creatin and creatinin—a definite evidence of disturbed carbohydrate metabolism.

Another group of writers lay stress on the unstable character of the nervous system in children as a cause of disturbance of metabolism. Ely in 1903, asserted that cyclic vomiting is a neurosis, and many writers have held this opinion. The most careful work on recurrent vomiting has been done by Howland and Richards, who ascribe the attacks to shock or excitement depending on the unstable state of the nervous system. Such a cause, they believe, leads to disturbed metabolism with diminished oxidation, and the circulation in the blood of unoxidized toxic substances. More recently Zade has published a similar theory. He believes recurrent vomiting to be a result of a sudden psychogenic upset of carbohydrate metabolism, and believes that the psychogenic origin explains many of the peculiar features of the attack.

Few writers have observed any marked connection between the diet and the occurrence of symptoms of acidosis. Kerley, however, believes that "the chief error in most cases rests in a defective oxidation, or in the giving of food substances of high carbon content in excess beyond the powers of normal oxidation." He believes diet important in prophylaxis and treatment.

Finally, a large group of writers have called attention to the frequency of acidosis in children exhibiting catarrhal conditions of the naso-pharynx, especially with adenoids and enlarged tonsils. This suggests a possible bacterial origin of the disturbance of metabolism seen in acidosis.

THE MOST PROBABLE THEORY OF ETIOLOGY.—I believe that the symptoms in recurrent vomiting and "acid intoxication" are due

to toxic products of disturbed metabolism. The products which actually cause the symptoms are unknown, the accumulation of the acetone bodies being only a coördinate symptom of the disturbance of metabolism.

I believe that the essential disturbance involves the carbohydrate metabolism, and that the frequency and severity of the attacks in childhood are partly due to the fact that the reserve supply of glycogen in the liver is not so great proportionately in childhood as in adult life. A disturbance of the glycogen-storing or glycolytic function of the liver would cause an insufficient supply of sugar, and this would induce an immediate disturbance of fat and protein metabolism. The result of the latter disturbance is the formation of the acetone bodies, and of other more toxic products.

The cause of the disturbance of carbohydrate metabolism which precipitates an attack, must next be considered. The key to this question lies in the known pathological conditions which are at times accompanied by acidosis. We find prominent among these conditions, *bacterial toxemia*, *alimentary toxemia*, and *disturbance of the nervous system* (eclampsia, etc.). We know that similar conditions frequently exist unrecognized, and it is highly probable that they are the cause of attacks of recurrent vomiting and acid intoxication which are apparently primary. Many children carry a chronic focus of infection, such as adenoids, diseased tonsils, or a chronically inflamed appendix. In many children there is a constant absorption of putrefactive products from the intestine, without recognizable symptoms. All these toxins act primarily on the liver, and cause an impairment of the function of carbohydrate metabolism, which is probably compensated elsewhere until an acute disturbance occurs. Such an acute disturbance may be caused by the cumulative effect of toxins, or by a sudden increase in toxic absorption, such as could appear in a *fresh infection*, or in an increased activity of the already existing infection, or after a dietary indiscretion. Perhaps more important in the production of such an acute disturbance, especially in recurrent types, is *nervous disturbance* such as is seen in *excitement or fatigue*. From these causes, there results a failure of the liver properly to perform its functions, and the symptoms of acute derangement of metabolism follow.

OCCURRENCE.—Our knowledge of the occurrence of acidosis is based chiefly on a study of the clinical type characterized by recurrent attacks of vomiting. The disease may occur in infancy, and in children approaching puberty, but it is commonest in the period of middle childhood, the first attack appearing most frequently between the ages of two and four years. The condition is about equally common in girls and boys. The disease is distinctly commoner in

private practice than in hospital practice, and frequently occurs in children with the best surroundings. The family history frequently shows neurotic antecedents, as well as rheumatism and a tendency to sick headaches and bilious attacks. The children are often of the highly-strung, excitable type, and subject to sea-sickness or car-sickness. They are not notably subject to digestive disturbance, but often have a tendency toward constipation. The attacks are apparently not precipitated by dietary indiscretion, but there is often a history of prolonged overeating without symptoms.

In many cases, no precipitating cause for the attack can be found. In many other cases, such a cause can be found, and is usually a nervous one. Unusual excitement or fatigue are the most common exciting causes, and an attack may be precipitated by fright, grief, anger, or a railroad journey, a children's party, anxiety or over-work at school, exposure to cold, or anesthesia with chloroform or ether. Sometimes a minor illness, such as tonsilitis, bronchitis, or an ordinary cold, seems to act as the precipitating cause.

Several epidemics of acidosis have been reported. In one epidemic in Birmingham, Ala., reported by Park, gastro-intestinal symptoms were prominent, and it is probable that in these cases the exciting cause was an intestinal infection. Another epidemic was reported by Metcalf, occurring in Concord, N. H., and vicinity. No definite evidence of an exciting cause was obtained in this epidemic, and it is probable that some unrecognized infection was responsible for the epidemic occurrence of the disease. Acidosis was unusually common around Boston in the winter of 1915 and 1916, but the type was apparently only symptomatic of a prevalent grippe-like infection of the upper air passages. In these epidemics the clinical type is not that of recurrent vomiting, but rather of a general intoxication.

SYMPTOMS.—There are two distinct clinical types seen in cases of acidosis. In the first type, vomiting is the principal symptom throughout the seizure, and the attacks of vomiting have a pronounced tendency to recur at intervals. In the second type, while vomiting is usually an early and marked symptom, it is not so prominent throughout the attack, the most important symptom being a pronounced intoxication affecting the nervous system. This type does not have a definite tendency toward recurrence. The distinction between these two types is purely clinical, and does not necessarily imply any difference in etiology. There may be a corresponding difference in etiology, but if so, it is not known. The distinction between these two types is not generally clearly drawn in pediatric literature, because both are characterized by the appearance of acetone bodies in the urine, and the second type is considered only as

a varying manifestation of acidosis. Nevertheless, the distinction is valuable, not only for purposes of clinical description, but also because the two types show a marked difference in prognosis, and in the indications for symptomatic treatment. The first type has usually been spoken of as recurrent, cyclic, or periodic vomiting, and will be described under that heading. The second type will be described as "acid intoxication," with the proviso that this term does not imply that the symptoms are due to the accumulated acids, but is only used for convenience of description.

RECURRENT VOMITING

(Cyclic Vomiting.). (Periodic Vomiting.)

The attack is very apt to come on suddenly, the child having been previously in good health, and not having shown any digestive disturbance. At times, prodromata have been observed, the most common being 'anguor, pallor, irritability, easy fatigue, restless sleep, coated tongue, anorexia, and constipation with stools that are either very light colored, or spongy and sour.

Whether or not there are premonitory symptoms, vomiting begins suddenly. The vomiting may be almost continuous, or may occur at intervals, usually of about fifteen minutes, but sometimes longer. The vomiting may be violent and projectile. After about twelve to twenty-four hours, it may become somewhat less frequent, but is still the principal symptom of the disease. The vomitus at first consists simply of the food which has been eaten, and after the stomach is empty, of mucus and serum. It may or may not have a sour odor, or the odor of acetone, and free hydrochloric acid may or may not be present. Later the vomitus often becomes streaked with blood, and occasionally, in untreated or in badly treated cases, considerable blood is vomited. Toward the end of the attack the vomitus is often bile stained. The vomiting is entirely uncontrollable, the stomach retaining nothing. Not only food, but all water, and any medicine given, are immediately vomited. The straining of the vomiting causes muscular lameness and tenderness of the abdomen, but there is no marked abdominal pain.

Thirst is always a prominent symptom, the child crying continually for water. Anorexia is usually very marked. Restlessness is usually great in the early stages of the attack, but later the patients appear to be afraid to move or to talk lest vomiting be provoked. The bowels are usually constipated, the stools being light colored and offensive.

The temperature in this type is rarely much elevated. It is ordinarily under 100.5° F. even at the onset, and later it is usually normal or subnormal. The pulse is often slow at the onset, but later becomes

rapid, weak, and sometimes irregular. The respiration is usually little if at all disturbed in this type; it is occasionally somewhat shallow and irregular. The tongue is ordinarily dry and coated, sometimes cracked, and the lips are dry and parched. The breath often has the odor of acetone strongly marked, but this cannot always be detected. The abdomen is usually retracted, and the skin may be dry and harsh, or cold and clammy.

During the first twenty-four hours the appearance of the child is not markedly altered. After this time emaciation is rapid, the child has a sunken-eyed appearance, looks severely ill, and prostration may become marked. Nevertheless, in this type, delirium, stupor and coma do not occur. The patient may be somewhat apathetic, but is always acutely conscious of his surroundings, and usually answers questions promptly and intelligently. In fact, the mental condition seems clear in comparison with the severity of the vomiting, and with the degree of prostration.

The urine is scanty and concentrated, but with proper treatment it may be more profuse. Toward the crisis of the attack its secretion becomes abundant, and this may be the first sign of improvement. It is strongly acid. The test for acetone usually gives a strongly positive result from the very start of the attack, and I have seen the sudden appearance of acetone precede the attack by twelve hours. The acetone test remains strongly positive until the crisis of the attack, when it becomes gradually less marked, disappearing in a few days. The test for diacetic acid is usually, but not always, positive from the start of the attack, and persists till the crisis is approached. Diacetic acid usually disappears from the urine before the acetone. The urine also contains an excess of indican, uric acid, creatin, the xanthin bodies, and sometimes beta-oxybutyric acid. During the height of the attack it may contain a trace of albumin, a few casts, and blood cells. The blood usually shows a moderate leukocytosis.

The duration of an attack is absolutely variable. It may be only a few hours, or it may be a week or even more. The most common duration is from two to four days. There is, however, a gradual tendency toward an eventual shortening of the duration of the successive recurrent attacks. The severity of the attacks is just as variable. This marked variation in the duration and severity of the attacks makes any opinion as to the value of any method of treatment in shortening the attacks, absolutely inconclusive and worthless.

The attacks recur at intervals, without any definite or obvious exciting cause. The intervals between the attacks are absolutely variable in length. The seizures may occur every week or two, or only after intervals of a number of months. In general, the

intervals tend to become longer as the patient grows older, but there are many irregularities, and several attacks may occur at short intervals after a long period of freedom. The length of time during which the tendency toward recurrence persists is also absolutely variable. The recurrences may continue throughout childhood, or may cease at any time. This variation in the intervals between attacks, and in the general duration of the disease, makes any opinion as to the value of any prophylactic treatment, or treatment in the interval, absolutely inconclusive and worthless.

The attacks in recurrent vomiting usually terminate by crisis. Within a period of twelve hours, usually, the paroxysms of vomiting become less frequent with longer intervals of sleep, and then cease for good. The other symptoms disappear rapidly, and appetite and normal digestion return almost at once. Convalescence is remarkably rapid, and within a few days the child is as well as ever.

"ACID INTOXICATION"

(Acidosis.)

This type is often spoken of as acidosis in distinction to recurrent vomiting, but many writers make no distinction. It is more frequent in infants than in later childhood, the commonest period being between one and three years. It is the type associated with the reported epidemics of acidosis. It is much more dangerous than recurrent vomiting, and does not tend to recur at intervals, although in the Concord epidemic some cases were seen in children who had had recurrent attacks.

The onset in this type is in every way similar to that of recurrent vomiting. It may be sudden, or preceded by the same prodromata. The first symptom is severe and uncontrollable vomiting. The acetone odor in the breath and the early appearance of the acetone bodies in the urine, are both present. In the first twelve or twenty-four hours, there is little to distinguish the case from the more common recurrent type. The temperature, however, is apt to be more elevated, often reaching 102° F., 103° F., or even more. There is also sometimes evidence of intestinal disturbance, as shown by diarrhea.

After this initial period of twelve to twenty-four hours, the vomiting becomes less frequent and severe. Everything taken by mouth is not immediately vomited, and while vomiting takes place at intervals, water and food are usually retained for a considerable period.

The essential clinical feature of this type is, that with the improvement in the vomiting, the general condition of the patient does not improve. Apathy becomes more marked, although wasting is often not so rapid. The patients finally become drowsy, and react

very sluggishly to their surroundings, and may become increasingly more stuporous. Delirium may be present, and occasionally convulsions.

With the increasing clouding of the mentality, there is usually a notable disturbance of respiration. In typical cases, the special dyspnea of air hunger is present.

The patient lies with the eyes half closed, with the eyeballs partly rolled up, and the conjunctivae injected. The lips are often of a peculiar cherry-red color. In severe cases prostration becomes very marked, and the patient sinks into coma, while the vomiting often ceases altogether In fatal cases the pulse becomes gradually more weak and rapid, the respiration more rapid and shallow, the temperature rises to 105°, 106°, or even higher, and death occurs in coma.

In milder cases the drowsiness gradually passes off, and the vomiting ceases, but recovery is less abrupt than in the recurrent type, and the acetone bodies often do not disappear from the urine for a number of days. After recovery, a second attack is very exceptional.

DIAGNOSIS.—The problems presented in diagnosis in the first attack of recurrent vomiting, and in the early stage of the toxic type, are the same. The conditions to be particularly considered are, acute indigestion, the onset of an acute infection such as scarlet fever or meningitis, appendicitis, intussusception, and tuberculous meningitis.

ACUTE INDIGESTION.—It is often difficult to distinguish acidosis from acute indigestion in the first twenty-four hours, vomiting being often the only symptom of both conditions. If the urine can be examined early in the attack, there will be a very strong acetone reaction in acidosis, and a slight or absent reaction in indigestion. Later, acetone may be present in indigestion with vomiting, but a very marked reaction points toward acidosis. Moreover, in indigestion, after the stomach is emptied of food, the vomiting usually occurs only after the giving of food. Everything taken is vomited, but there is not the continual retching characteristic of cyclic vomiting. Also, in indigestion, water is not often vomited, while it is almost always vomited in acidosis. These distinctions, however, are not always clearly marked, and the final test is the response to treatment. The vomiting of indigestion usually ceases with proper treatment, while that of acidosis continues irrespective of treatment.

ACUTE INFECTIONS.—A number of acute infections in childhood begin with vomiting at times. In particular, vomiting is often a prominent symptom of scarlet fever and meningitis. The diagnosis depends mainly on the results of a careful physical examination, which will often disclose the characteristic signs of the infection of

which the vomiting is a symptom. The presence of high fever is in favor of some acute infection; it is strongly against the recurrent type of acidosis, but does not rule out the other type, although the fever in acute infection is higher as a rule than in any form of acidosis. The examination of the urine for the acetone bodies is of help, if made early in the attack. The early presence of a marked acetone reaction points toward acidosis. It must be remembered, however, that the appearance of the acetone bodies in the urine is a frequent symptom in many acute infections, and acetone is likely to be present in any case in which vomiting has continued a number of hours. It is therefore very important not to conclude too hastily from the presence of acetone that the case is one of the so-called primary types of acidosis. Such a conclusion may lead to carelessness in physical examination, and to the overlooking of any signs of a recognizable acute infection which may develop in the course of the disease.

In general, when uncontrollable vomiting is the principal symptom, if the temperature is not much elevated, and if the urine shows a strong acetone reaction, it is safest to consider and treat the case as one of acidosis, while watching carefully for signs of some other disease. If the temperature is high from the start, it is safer to consider the case one of acute infection until characteristic signs of the infection fail to develop, or until signs of the toxic type of acidosis begin to appear. If, however, the acetone bodies are present in the urine, it is safe to use the treatment for acidosis, as this can do no harm if the acidosis be secondary and symptomatic rather than one of the primary types.

APPENDICITIS.—I have frequently seen appendicitis mistaken for acidosis, and have myself mistaken acidosis for appendicitis. The first mistake is due to the fact that with the increase in the use of accurate laboratory methods of diagnosis, physicians are apt to overemphasize the results of a laboratory examination, and to underestimate the importance of careful and thorough physical examination. In appendicitis, severe vomiting and acetonuria are common symptoms, and if the physician finds that the examination of the urine gives a positive test for acetone, and jumps at the conclusion that the case is one of acidosis, there is danger that he may neglect the careful physical examination necessary to reveal the presence of appendicitis in early life. In acidosis, while tenderness of the abdomen is usually absent, it may be present from the soreness of the muscles of the abdominal wall which often follows excessive vomiting. Appendicitis can only be ruled out when its characteristic signs, such as paroxysmal pain, abdominal tenderness and spasm, are all absent. When any of these signs are present, the differential

diagnosis may be quite difficult. The physician must take everything into consideration. Fever, localized tenderness, paroxysmal pain, a marked leukocytosis, and the retention by the stomach of some food or water, favor appendicitis. The absence of these signs, with a strong acetone reaction in the urine, favors acidosis.

INTUSSUSCEPTION.—This is another condition of which vomiting is a prominent symptom, and in which acetone may sometimes be found in the urine. It is distinguished from a primary acidosis by the appearance of the characteristic bloody discharges, and by the finding of a tumor on careful physical examination.

TUBERCULOUS MENINGITIS.—This condition is the one which presents the greatest difficulties in the differential diagnosis from acidosis. Severe vomiting is a common symptom in the early stages of tuberculous meningitis. If at this time the examination of the urine for acetone be negative, acidosis may be ruled out, and some other cause must be sought for the vomiting. While acetonuria is less common in tuberculous meningitis than in other acute infections, it may appear in any child who has been vomiting for some time. Consequently even in the early stages of either type of acidosis, tuberculous meningitis cannot be excluded with certainty. With the presence of a marked acetonuria, however, it is best to treat such cases as acidosis, because if the disease later proves to be tuberculous meningitis, no harm has been done.

In the further course of the two conditions, certain differences often appear. In the recurrent type of acidosis, vomiting continues to be the principal symptom, and continues to be uncontrollable, the stomach rejecting everything taken. In tuberculous meningitis, the vomiting is apt to be less severe, and often some food or water is retained. The abrupt cessation of the vomiting in the recurrent type of acidosis is accompanied by a favorable change in the condition of the child, and this distinguishes this condition from tuberculous meningitis, in which improvement in the vomiting is not attended by improvement in the general condition of the child. In the later stages it is very difficult to distinguish the second type of acidosis from tuberculous meningitis, because both diseases are characterized by clouding of the mentality. In this stage of tuberculous meningitis, however, when the vomiting remits, the acetone reaction is likely to disappear from the urine, while in acidosis, it continues to be marked. It is nevertheless often difficult to make a differential diagnosis in all stages of the disease, except by lumbar puncture. I believe that in any case in which the diagnosis lies between acidosis and tuberculous meningitis, lumbar puncture should be performed. It is true that if the case be one of tuberculous meningitis, the lumbar puncture does no good; but it is also true that if

the case be one of acidosis, lumbar puncture will do no harm. The advantage to be gained for purposes of prognosis and treatment in thus settling the diagnosis is very great. In acidosis as in tuberculous meningitis, the cerebro-spinal fluid is clear, and it may even be under increased tension. The cell count of the cerebro-spinal fluid, however, is usually normal in acidosis, while in tuberculous meningitis it is invariably increased. Rarely cases of the toxic type of acidosis will show some increase in the cell count of the cerebro-sp na flu'd, but this is usually moderate and much less than the usual cell count of tuberculous meningitis. The finding of the tubercle bacillus in the cerebro-spinal fluid is always positive evidence.

PROGNOSIS.—In recurrent vomiting the prognosis is in general favorable. A fatal ending is rare in this type, Cases have been reported in which death occurred as a result of extreme prostration in recurrent vomiting, but such cases are certainly rare. I have only seen two fatal cases in this type of acidosis. Both had been improperly treated and were characterized by the vomiting of large amounts of blood. I believe that this is the chief danger in recurrent vomiting. The continual retching seems to cause a mechanical injury to the mucous membrane of the stomach, leading to hemorrhage. The toxemia of the disease is certainly very rarely a cause of death. In the first attack, the prognosis is somewhat more doubtful, because the physician cannot tell at the start whether he has to do with the recurrent type, or the "acid intoxication" type. So long as no clouding of the mentality develops, the outlook is good. In subsequent attacks a favorable prognosis can be given in any case in which the vomiting of considerable blood has not already developed.

In the type of acidosis characterized by clouded mentality, stupor, or coma, the prognosis is much more unfavorable. Such cases are often fatal, particularly in infants, and the younger the child the worse the prognosis. The prognosis varies with the severity of the case, and this is measured by the degree of stupor which develops. If the infant's symptoms do not advance beyond apathy and slight clouding of the mentality, the prognosis is good, for the disease is a self-limited one. When marked stupor or coma deve'op w th disturbance of the respiration, the prognosis is very bad. In a child under two years, the chances are rather against recovery in any case of acidosis of this type.

TREATMENT. RECURRENT VOMITING.—The treatment of recurrent vomiting may be subdivided into that of the acute attack, and that of the interval.

In an acute attack of recurrent vomiting, the chief danger comes from the vomiting itself. Excessive vomiting may produce hemor-

rhage, or may cause severe exhaustion and prostration, which is increased by the deprivation of food and fluid. The chief indications therefore, are to diminish the frequency and severity of the vomiting, and to supply as much fluid and fuel as is possible.

The essential measure to be used for the excessive vomiting is to *give no food, fluid or medicine by mouth* until the attack ceases. This has more effect in diminishing the frequency and severity of the vomiting than any drug, or any other measure. Once that the diagnosis of recurrent vomiting is made, allow nothing to pass the child's throat. Do not give bicarbonate of soda, do not give aerated water, iced brandy or champagne, do not give cracked ice to be melted in the mouth. Every drop of fluid which enters the stomach will provoke vomiting, but if nothing enters, the vomiting will certainly decrease in frequency or severity. No drug is of any marked value in controlling the vomiting. The value of bicarbonate of soda as a specific weapon against acidosis will be considered later, but it is certainly not great enough to warrant its being given by mouth in the recurrent type of acidosis.

From the beginning of the attack, *the child should be put on rectal feeding*. Children can usually be satisfactorily nourished in this way during the comparatively short duration of an attack of cyclic vomiting. It is often surprising to see the favorable change which takes place in a child with whom feeding by mouth has previously been tried, when rectal feeding is instituted. Much of the prostration disappears, the sunken-eyed appearance improves, emaciation becomes less rapid, and the pulse becomes of better quality. These changes come from the much needed supply of food and fuel, which cannot be retained by the stomach, but which are usually readily retained and absorbed by the rectum.

The best food for use in the nutrient enemata is dextrose. It is the only food substance which can be given in the same form in which it is absorbed into the blood and utilized by the tissues. It is fortunately a carbohydrate, and as acidosis most probably involves a disturbance of the glycogenic function of the liver, with a consequent insufficient supply of sugar, this form of fuel is the one needed. Pure dextrose is expensive, and cannot always be readily obtained. Glucose is the next best substitute. If neither can be obtained at once, peptonized milk may be used during the interim, but is theoretically greatly inferior to dextrose.

The dextrose should be given in a 5% solution. More dextrose could be given in a stronger solution, but there is danger of irritation of the mucous membrane, and of intolerance of the rectum, which should be avoided at all costs. The amount to be given at each injection depends on the age of the child, and the tolerance of the rectum. It is safer to begin with a smaller amount, and later to

increase the quantity. To an infant, four ounces every four hours should be given (1.2 oz. dextrose in 24 hours). To an older child, six ounces every four hours should be given (1.8 oz. dextrose in 24 hours). If the rectum seems tolerant, and these enemata are well retained, the quantities may be increased to six and eight ounces respectively. If they are not well retained, the quantities must be somewhat reduced. A cleansing enema should be given every twenty-four hours.

If, in spite of the nutrient enemata, the child complains of excessive thirst, normal saline solution should be given by rectum during the intervals between the nutrient enemata. The amount at each injection should be from two to four ounces, and the frequency should depend on the thirst of the patient, enough being given to relieve this symptom.

The majority of writers advocate the use of bicarbonate of soda in recurrent vomiting. This is based on the theory that the disease being apparently an acidosis, an alkali is indicated to counteract the excessive acidity of the blood. As I have shown in considering the etiology of acidosis, there is considerable evidence against the theory that the toxic symptoms are directly due to the accumulation of the acetone bodies in the blood. The vomiting may be due to other toxic products of disturbed metabolism, the acidosis being only an accompanying symptom, or, even if the symptoms are due to toxic acids, there is no proof that the accumulation of acids can be effectively combatted by the administration of bicarbonate of soda. There would be no objection to the use of bicarbonate of soda, if its administration could do no harm. In as far as the giving of bicarbonate of soda by mouth is concerned, I believe that it certainly does do harm by increasing the vomiting, and that the possibility of harm is greater than the possibility of good. Bicarbonate of soda, however, can be given in the rectal enemata. Here also I believe that it can have a harmful effect, in rendering the rectum so intolerant that the nutrient injections are not retained. In recurrent vomiting, the chief danger comes from the vomiting itself, and our most effective weapon against this symptom is rectal feeding. If the rectum becomes intolerant, the proper treatment of the case becomes very difficult. The question arises whether the benefit to be expected from the counteracting effect of bicarbonate of soda is sufficiently great, to balance the possibility of harm. In my opinion the evidence of benefit is not sufficiently great. In view of the variability of the clinical course in recurrent vomiting, and the tendency of the vomiting to *cease abruptly at any stage without reference to treatment*, reports on the apparent favorable action of bicarbonate of soda cannot be accepted as conclusive Even if the urine becomes alkaline with bicarbonate of soda treatment *before* the vomiting

ceases, it is not conclusive evidence, because the urine often becomes alkaline as the crisis approaches without bicarbonate of soda treatment. I formerly used the bicarbonate of soda treatment, first by mouth, in various quantities including the very frequent administration of a saturated solution, and later by rectum. I have never seen any clear evidence of its value. The disease is self-limited, and the attack will run a favorable course, if the vomiting is not excessive, and if the patient can be nourished and supplied with sufficient fluid. I do not, therefore, advocate the use of bicarbonate of soda in recurrent vomiting. In a very severe case, in which it seemed necessary to try every available resource for saving the life of the patient, bicarbonate of soda would be a possible measure, but in such a case, I would give it intravenously.

Under the routine treatment of no food by mouth, and dextrose and fluid by rectum, most cases will run a course without severe prostration, hemorrhage, or other unfavorable symptoms. Some cases, however, are more sévere, and require additional treatment. Unfavorable signs are continued frequent and violent retching, with increasing prostration, or increasing blood in the vomitus. In such a case, ten grains of sodium bromide should be given by rectum, and if this does not lessen the vomiting, it may be repeated in two hours, with one and one-half to two grains of chloral hydrate. Subsequently the bromide may be given by rectum every four hours, if the vomiting continues to be severe. Morphine should not be used, except in the most desperate cases which resist the bromide. It seems to increase the toxemia of the disease, but should be tried if all other resources fail.

With very severe prostration, and a weak and rapid or intermittent pulse, the usual circulatory stimulants, caffeine, strychnine, and digitalis, should be used, given subcutaneously.

If the rectum becomes intolerant, the quantity of fluid given by rectum must be reduced, and only the dextrose should be given. Under these circumstances, physiological salt solution should be given subcutaneously, in doses of four ounces repeated as often as is necessary to control thirst. Dextrose solution, or a weak solution of sodium bicarbonate, may also be tried by mouth, under these circumstances.

When the vomiting has stopped for six or more hours, it is usua'ly a signal that the attack is over, and that feeding by mouth may be begun. It is best to begin with a five per cent dextrose solution in barley water, beginning with teaspoonful doses, and doubling the amount every two hours. The nutrient enemata should be continued for twelve hours, or until eight ounces of the dextrose-barley solution are taken, when they can be omitted. Zwieback, or dry bread crust can now be added to the diet, or even before the expira-

tion of twelve hours, if the child's appetite returns rapidly. Cereals are added during the next twelve hours, and then milk may be given. After this the child can rapidly return to its normal diet.

Next must be considered the treatment of the child subject to recurrent vomiting, in the intervals between the attacks. According to the theories outlined above, prophylactic treatment should aim at the prevention of the absorption of the products of intestinal putrefaction, the removal of chronic foci of infection, the prevention of nervous excitement or fatigue, and the avoidance of dietary indiscretions.

Many writers claim remarkably good results in recurrent vomiting from dietetic treatment. Unfortunately, clinical observation cannot be accepted as a satisfactory criterion of the efficacy of any dietary regimen, on account of the variability of clinical course which is an essential feature of the disease. Dietary treatment is usually advocated on a basis of some exclusive theory of etiology, while the facts suggest a diverse etiology. For instance, if the theory be accepted that the absorption of putrefactive products from the intestine damages the metabolic functions of the liver, the protein in the diet should be reduced. On the other hand, if the theory be accepted that an excess of food beyond the oxidizing power of the child is the cause of the disturbance, it is the fats and carbohydrate, with their high carbon content, which should be reduced. In view of the obscurity which still surrounds the etiology of the disease, it does not seem that any exclusive theory can be logically adopted as a basis for dietary treatment.

I believe that *the principal thing to be avoided is overfeeding*. The diet should be arranged according to the ordinary principles which govern the selection of the food for the normal child of the same age. A well-balanced diet, with a proper ratio between fat, carbohydrate, and protein, should be insisted upon. In one respect, I agree with those who favor reducing the carbon content of the food, and that is in the advisability of omitting sugar from the diet. Sugar is not a necessary ingredient of the diet of children, and indeed before the last three centuries, was only used as a condiment, not as a food. Cane sugar is a common source of a form of intestinal fermentation, which is very insidious and difficult of clinical recognition. All the carbohydrate needed can be supplied from the starchy foods which make up an ordinary, well-balanced diet. I believe that an excessive amount of milk should be avoided, and that cream had better not be given. All eating between meals should be strictly forbidden.

With a normal, well-regulated diet, the stools should be frequently examined for evidences of fermentation, or for indigestion of any particular food element. If any such indications are found, the diet

should be modified accordingly. If the stools suggest fermentation, lactic acid milk should be tried.

It is essential that a satisfactory daily evacuation of the bowels be insured by suitable measures.

Adenoids or diseased tonsils, if present, should be removed, and if the child be subject to attacks of infection of the upper respiratory passages, a change of climate is advisable. Every possible focus of chronic infection should be diligently sought.

The treatment of the nervous system must be carefully attended to. All such possible precipitating causes as emotional excitement and nervous fatigue should be prevented by careful attention to detail. Some children must be taken out of school. Sometimes a change of nurse or other attendant, or even the temporary absence of the mother, is of benefit. Rest in bed in the morning or afternoon, or both, is sometimes advisable.

The evidence in favor of the value of any form of medication in the intervals between the attacks of recurrent vomiting can only be of the inconclusive clinical variety. The physician may freely indulge his fancy, provided that nothing be given that can do harm. The disease is of such a character that whatever he uses, he is likely to be convinced that he is obtaining "good results." Personally, I have no suggestions to offer.

The question of whether in a child showing recognizable prodromata, the attack can be aborted by medication, is an open one. I cannot declare that it cannot be so aborted. Many writers have claimed that the giving of bicarbonate of soda upon the appearance of prodromata will sometimes abort the attack. I have seen cases in which I believed that I recognized prodromata, and have given bicarbonate of soda, and no attack occurred. On the other hand, I have seen cases in which I believed that I recognized prodromata, and have *not* given bicarbonate of soda, also proceed without an attack. Prodromata in recurrent vomiting are indefinite and uncertain. Certainly, when given at this stage, before vomiting has begun, the giving of bicarbonate of soda by mouth is not open to the objections which prohibit its use during the attack. It may be given in doses of ten to thirty grains every two hours until the urine becomes strongly alkaline and then enough should be given to keep the urine alkaline.

It is also advisable, upon the recognition of prodromata, to induce free catharsis, and to reduce the amount of food.

ACID INTOXICATION.—The treatment of the type of acidosis in which clouding of the mentality, stupor, or coma becomes the prominent symptom, is at first exactly like that of recurrent vomiting. Food or water by mouth should be interdicted, and the rectal ad-

ministration of dextrose and fluid should be instituted, and should be continued as long as vomiting is severe and uncontrollable.

When the development of clouded sensorium or stupor shows that the case is of the second type of acidosis, the indications for treatment undergo a marked change. We have now to do with a disease in which the danger comes, not from the vomiting, but from a pronounced intoxication affecting the nervous system, and tending toward coma and death. The only specific measure for combatting this intoxication which possesses any theoretical basis, is the administration of bicarbonate of soda. Whether or nor it is effective is an open question. At all events, it is the only thing which can be done, and should therefore be employed. Usually in this type, after the symptoms have developed the vomiting is not so severe. A considerable amount of bicarbonate of soda can usually be retained by mouth, and if any be retained, occasional vomiting is not a contraindication. As much bicarbonate of soda as possible should be given, usually from ten to thirty grains every two hours to an infant, and even more to older children. Often children take and retain it better when given in dry form instead of in solution. If the soda can be given by mouth, it is best not to give it by rectum, in order not to injure the tolerance of the rectum for the necessary food and fluid. If the soda is not well retained by mouth, it may be tried by rectum, and the giving of small amounts of dextrose solution in barley water by mouth should be tried. Either the mouth or the rectum should be used for the soda, but not both at once.

If under this treatment, the case continues to run an unfavorable course, the bicarbonate of soda may be given intravenously. From sixty to ninety grains (4-6 grammes) should be given in four to eight ounces of water according to the age of the child. In infants, the technique of intravenous injections is very simple, owing to the easy access to the longitudinal sinus through the open fontanelle In older children, some other vein must be chosen.

My personal experience w'th the bicarbonate of soda treatment has not been very encouraging. I have given it intravenously in large doses without effect on either the symptoms or the acidity of the urine, and then, two or three days later, the symptoms have improved and the urine has become alkaline spontaneously.

The other measures of treatment, such as stimulation, are the same in this type as in an attack of recurrent vomiting. Physiological salt solution subcutaneously is a valuable stimulant. In convalescence, the feeding is conducted along the same lines as recurrent vomiting, the rectal feeding not being omitted until sufficient food and fluid can be taken by mouth.

PROBLEMS AND RESEARCH.—The problems of acidosis are chiefly connected with the cause of the condition, and have been indicated in the discussion of the etiology. The fundamental problem is whether there is a primary acidosis with a specific cause, or whether the disturbance of metabolism which results in acidosis is always a secondary symptom-complex of some other pathological condition. It is probable that the increasing study of some of the more obscure infections will relegate to the secondary class many cases of acidosis now considered primary. It is probable also that the progress of research on the subject of the metabolism in early life will result in the subject of acidosis in children being attacked with the same thoroughness as has diabetes mellitus in adults. This is likely to throw much additional light on the subject, and may also result in revealing the secondary character of acidosis. It is very probable that the types of acidosis which are not recurrent, and wh ch sometimes occur in epidemics, will eventually be classified as secondary symptom-complexes, when the causes are more fully understood. As for the recurrent vomiting type, while this also may prove to be a secondary manifestation, the definiteness of the clinical picture is strongly suggestive of some specific form of metabolic disturbance.

Other problems are the relation of the acetone bodies to the symptoms, the nature of any other toxins which may be the cause of the symptoms, and the exact character of the disturbance of metabolism which results in the excessive formation of the acetone bodies and other toxins. For a solution of these problems we must await further progress in the complicated subject of the chemistry of metabolism.

The problems are being attacked by modern methods. Improved methods of studying the acidity and chemistry of the blood are being published. The method of studying acidosis by the estimation of the carbon dioxide content of the alveolar air has thrown much light on the relative form of acidosis, but has not yet been extensively applied to positive acidosis. Howland is at present doing much work on acidosis.

As an example of the methods now being employed, I note a recent publication of Marriott, describing an improved method of estimating quantitatively the acetone bodies in the blood. He found no fixed relation between the amount in the blood and the amount in the urine. There is also a recent work by Fischler and Kossow on the origin of the acetone bodies. This was an experimental study on dogs by means of Eck's fistula on the partially excluded liver, and showed that acetone, d acetic acid, and beta-oxybutyric acid, are mainly formed in the liver. This method of investigation seems very promising.

DIABETES MELLITUS

Diabetes mellitus is a disorder of metabolism characte
accumulation of grape-sugar in the blood, and by its
the urine over long periods. It is a rare disease in
compared with adult life, but is not so rare as was forme
An increased number of cases of diabetes mellitus have b
in the last ten years, and this is probably due, not to
frequency of occurrence, but to better recognition of
According to most statistics, only from .5 to 1 per cent
of diabetes mellitus occur in the first ten years of life. Va
statistics show the incidence for the first decade to be
and it is probable that the average incidence is about 2

ETIOLOGY.—Diabetes mellitus can occur at any
probably extremely rare in infants. Knox found but
in infants under one year reported in the forty-four ye
1852 and 1896, while it has been described in at least
in the fifteen years between 1896 and 1913. The freq
disease in childhood apparently increases directly with th
frequency of occurrence of diabetes mellitus in early lif
accurately estimated, because many cases have undou
overlooked, owing to the difficulty of obtaining urine
children, while on the other hand, diabetes mellitus is
diagnosed, owing to the tendency of young children to e
sugar and other reducing bodies in the urine, and to exl
sitory glycosuria.

The preponderance of male diabetics seen in adult st
not hold for childhood. The two sexes appear to be a
about equal frequency in the diabetes of early life. T
of race is also not so apparent in childhood as in adult life
race not showing so marked a predisposition towards diab
in childhood.

Heredity is an important factor in the etiology of diabe
There are many recorded instances of diabetes in chil
a diabetic family history, but the influence of heredit
evident in childhood as in adult life. Close investiga
family history has often shown that the parents of diab
were blood-relatives, or that intermarriage occurred in fo
tions. This is strongly suggestive that diabetes in ch
endogenous degenerative disease. Syphilis in the fathe
times been recorded, suggesting the possibility of a func
ness of the pancreas from the syphilitic virus.

The immediately exciting cause of diabetes mellitus
been most frequently described, is an injury to the cen
system. Such injuries have come from trauma, as in

or as the result of difficult labor, or from disease or deformity of the brain. The relation of these injuries to the disease is a subject of present day discussion, some authorities believing that diabetes can have a neurogenous origin independently of the pancreas, while others believe that an injury to the nervous system acts by affecting the pancreas. (Von Noorden.)

Diabetes mellitus in children sometimes appears to follow the consumption over a long period of an excessive quantity of sugar.

The general etiology, the pathogenesis, and the abnormalities of metabolism in diabetes mellitus have aroused great interest in recent years, and have been the subject of much experimental investigation. Owing to the rarity of the disease in early life, this is not the place for a lengthy consideration of these subjects, and the reader is referred to the works which consider the disease in adults, and to the voluminous special literature of the subject. A brief summary of the newer teaching concerning the disease may, however, be useful and interesting to those who have not followed the recent literature. Such a summary will be found under the heading of problems and research.

PATHOLOGICAL ANATOMY.—Diabetes mellitus shows no constant characteristic lesions. In a certain number of cases, some lesion of the central nervous system, either developmental, or the result of trauma, or from acquired disease, has been found. In many cases there is no lesion of the nervous system.

In the last two decades, increasing attention has been paid to the pancreas. In the older autopsies the condition of this organ was usually not recorded, while in more recent records there is frequent mention of a small size and shrunken condition of the organ. A diminution in the number of the islands of Langerhans has been often recorded of late, and some authorities believe that a careful count of the number of islets in a given area will often if not always show such a diminution in diabetes.

SYMPTOMS.—The chief symptoms of diabetes mellitus are the same in children as in adults, namely increase in the secretion of urine, excessive thirst and hunger. The onset of the disease has generally been described as much more rapid and sudden in children than in adults. This is probably not markedly the case, but the disease has appeared to come more suddenly, because it has not been recognized in its early stages. The urine of children is not examined as often as that of adults, and symptoms in children in the early stage are not pronounced, and consequently the disease is not recognized until marked symptoms have betrayed the existence of the disease which appears to be already in a severe stage. Nevertheless, it is probable that acute types are met with in childhood more often than in adult life.

The first symptom in children is usually *enuresis*, and no case of enuresis should be allowed to pass without examination of the urine for sugar. In some cases the *excessive thirst* is the first symptom which attracts attention; in other cases it is *flecks of sugar* on the child's underclothes. *Irritation of the external genitals* is a fairly common symptom in childhood, and should always lead to a urinary examination. Excessive hunger is a rare symptom, but is rather more common in children than in adults. The various complications, such as disorders of the skin, eyes, and nervous system, which so frequently cause the first suspicion in adults, are almost unknown in childhood. Sometimes, however, a marked change in the child's disposition is observed.

When the disease is recognized in an early stage, before the tolerance for carbohydrate fails, regulation of the diet will usually bring about a disappearance of all symptoms, including the glycosuria. Whenever, during this period, an increased quantity of carbohydrate is given, sugar will reappear in the urine. During this mild stage, the physical and mental development of the child is normal. The duration of this stage is very variable; it may be weeks, months, or even years.

Sooner or later the tolerance for carbohydrate fails. The failure may be precipitated by some intercurrent febrile disease or by a dietary indiscretion, but even in the absence of such causes, it is only postponed, and inevitably occurs in the course of time. When once the carbohydrate tolerance begins to diminish, the development of complete failure, with a severe type of glycosuria unmodified by carbohydrate withdrawal, is very rapid. With the loss of tolerance, symptoms develop. The patients lose physical and mental activity, tire easily, and complain of pain in the joints after exertion. Polyuria, excessive thirst, and sometimes excessive hunger become prominent, and emaciation is very rapid.

Even at this stage a carefully arranged dietary may cause some improvement in the symptoms, but the improvement is only temporary. With the rapid emaciation, the acetone bodies appear in the urine, the breath has the odor of acetone, and the patient presents the same picture of acid intoxication as is seen in adults.

Unless some intercurrent infectious disease occurs, the termination of diabetes mellitus in children is death in coma. The approach of diabetic coma is often indicated by such symptoms as loss of appetite, nausea, vomiting, gastric pain, nervous irritability, sleeplessness, and muscular weakness.

The urine in children shows the same abnormalities as in adults. During the mild stage, sugar (dextrose) is constantly present on a normal diet, or when the carbohydrate in the food is increased, and the quantity and specific gravity are proportionately raised. Under

dietary regulation, the sugar disappears. In the severe stage, the quantity of urine excreted in twenty-four hours is increased to from two to four times the normal, and the specific gravity is increased up to 1040 or even more. Sugar is constantly present, varying in amount up to 60 grammes or even more in twenty-four hours. The urine contains varying amounts, first of acetone, later of diacetic and oxybutyric acids, and large amounts of ammonia. There is usually a trace of albumin, and as coma approaches, an increasing number of the small, so-called "coma casts," are seen in the sediment.

The complicating organic diseases so common in the diabetes of adult life are almost never seen in the diabetes of childhood.

DIAGNOSIS.—The diagnosis of diabetes mellitus in children, as in adults, depends upon the finding in the urine of the constant presence of dextrose, when the patient is on a normal diet. This is positive proof of the existence of the disease.

Diabetes in children is often overlooked, especially in the early stages, on account of the common neglect of urinary examinations. In all cases, such symptoms as enuresis, thirst, abnormal appetite, irritation of the external genitals, and wasting, should lead to urinary examination.

Diabetes mellitus is sometimes wrongly diagnosed, especially in infants. In a number of conditions in infancy, lactose, or the split product galactose, enters the circulation through the intestinal wall, and is excreted in the urine. With the ordinary test with Fehling's solution, lactose will give a reduction, and such a reduction has led to many errors in diagnosis. For the tests which distinguish dextrose from the other sugars, such as the phenylhydrazin test, the reader is referred to works on chemistry. A simple test for ordinary clinical use is the following: Boil the urine with an equal amount of a 10% solution of sodium hydrate for three minutes. Then add Fehling's solution and boil again. If the Fehling's solution is reduced, the sugar is lactose, not dextrose.

Another difficulty in diagnosis comes from transitory glycosurias, which are fairly common in children. Some children have a definite intolerance toward carbohydrate, which is not progressive in character. These cases are recognized by the fact that glycosuria does not occur with a normally balanced diet, but appears temporarily after the eating of an excessive quantity of carbohydrate The finding of sugar in a single specimen of urine should never be considered evidence enough to warrant a diagnosis of diabetes. Only if the sugar is constantly present with a normal diet, or if intolerance as shown by glycosuria toward an excess of carbohydrate is increasing, should diabetes mellitus be diagnosed. Rare cases have also been reported of glycosuria in children, probably dependent upon

some transitory disorder of the pancreas, which disappears in a few weeks. Diabetes mellitus should be strongly suspected in such cases, but a positive diagnosis should not be made unless the glycosuria persists for a considerable time, or unless other symptoms of diabetes mellitus are present.

PROGNOSIS.—If the diagnosis of true diabetes be certain, the prognosis in children is almost without exception unfavorable. Practically the only hope for the child is that some form of transitory glycosuria is present. The lapse of time, or the progressive appearance of symptoms finally settles the point. After complete failure of carbohydrate tolerance occurs, progress toward a fatal ending is very rapid in children. Before complete failure occurs, the course of the disease is very variable in length.

TREATMENT.—In spite of the hopelessness of the prognosis, it is the physician's duty to prolong life as much as possible. The treatment is mainly dietary, and is conducted in the same way as in the diabetes of adults. Carbohydrate should first be gradually withdrawn from the diet until the urine becomes sugar-free, and then, by the gradual addition of a measured quantity of bread, or some other carbohydrate, the tolerance can be definitely determined. In mild cases, in which there is a definite carbohydrate tolerance, the diet should be carefully arranged in such a way that the carbohydrate given is within the limit of tolerance, with an occasional day of strict carbohydrate-free diet. The details of the arrangement of the diet depend upon the age, appetite, and powers of digestion of the individual child, and cannot be illustrated by definite schemata. The quantity of the protein should also be reduced if possible, but should not be below 2 grammes per kilogram of body weight. Usually the protein cannot be reduced as low as this, as the caloric value of the diet cannot be maintained by fat without indigestion, or by carbohydrate without overstepping the tolerance. The experience of many observers suggests that oatmeal is the form of carbohydrate best tolerated in the diabetes of children, although this has not been finally proven.

In infants, in whom the development of the digestive apparatus does not permit a varied diet, and whose food consists of milk, it is often difficult to determine the limits of carbohydrate tolerance on account of the quantity of lactose contained in all milk modifications. Carbohydrate can be greatly reduced, if not completely excluded, by the use of precipitated casein, either alone in a suspension, or in the form of albumin milk. With precipitated casein and top milk (16% cream) a formula can be arranged containing—Fat 2%,—Lactose .40%,—Protein 2.5%. (See Division on Feeding.) If with such a food glycosuria is present, the carbohydrate tolerance

may be considered to be practically zero. It may be accurately determined by gradually increasing the percentage of lactose in the food, and when this is determined, the formula may easily be arranged to meet the requirements.

When the tolerance for carbohydrate is zero, as shown by the presence of glycosuria when any carbohydrate is given, or when none is given, it is useless to continue strict dietary rules. A certain amount of carbohydrate, except cane-sugar, may now be given, either in the form of milk, or cereals. The so-called "oatmeal cure" of von Noorden may be tried. This consists in putting the patient for one or two weeks on a diet consisting of 150 grammes of oatmeal, 150 to 200 grammes of butter, and four or five eggs daily, and in addition, some wine. This sometimes shows a marvelous result in increasing the carbohydrate tolerance, but unfortunately, the improvement does not last.

No drug has any influence on the disease. When the acetone bodies are present in the urine, bicarbonate of soda should be given in doses of two to four drachms daily, to neutralize the acid products of the impaired metabolism. Some recent writers have advocated the use of the lactic acid bacillus (bacillus bulgaricus) in diabetes, on the theory that it diminishes intestinal putrefaction and the resulting autointoxication which disturbs the function of the pancreas, adrenals, thyroid, nervous system, and hypophysis. As lactic acid milk is low in carbohydrate content, this treatment might well be tried.

When diabetic coma occurs, the only measure which can even temporarily relieve the symptoms, is the intravenous injection of bicarbonate of soda. This has given good results in certain cases, but can only postpone the fatal termination.

PROBLEMS AND RESEARCH.—The following is a summary of the newer teaching as to diabetes mellitus:

Diabetes mellitus is a condition in which the liver shows an abnormal tendency to discharge dextrose into the blood in excess of the capacity of the tissues to utilize it. The hepatic function of dextrose formation from the stored glycogen is normally regulated by two factors, one stimulating, the other inhibitory. The stimulating factor lies in the chromaffin system of the adrenals, and this in turn may be stimulated by influences coming from the central nervous system. The inhibitory factor lies in the internal secretion of the pancreas, and this in turn is normally stimulated by the secretion of the parathyroids. The inhibitory action of the pancreatic secretion may also be *lessened* by disease of the pancreas, or by overactivity of the thyroid or hypophysis. Any influence which overstimulates discharge of sugar, or lessens the inhibition to dis-

charge of sugar, may theoretically produce diabetes m explains the experimental production of diabetes throu the nervous system, and its occasional clinical associatic injury. Practically, however, von Noorden and his follo that the most important causes in human diabetes are lessen the inhibitory action of the pancreas, such as dis pancreas, and disorders of the organs which influence the function. Whatever the ultimate cause, there is in all c betes an abnormal irritability of the dextrose-forming me the liver. The rational treatment of diabetes consequent in efforts to calm this excessive irritability by the avoidan ulation. The taking of sugar acts as a stimulus to the function of the liver, and it is for this reason that the carb are to be so limited as to lie within the tolerance of the i as shown by absence of glycosuria. There is evidence th certain circumstances the proteins may act as stimulant liver cells, but the fats are not excitants. The protein, cannot be kept low too long. Acidosis and diabetic coma result of an insufficient supply of carbohydrate. The success cases of the "oatmeal cures" suggests that some forms of c drate stimulate the discharge of dextrose less than others. I so, the reason is unknown. Some recent writers believe tha is no special value in oatmeal, having obtained equally good with wheat flour. This is explained on the theory that th results come from the use of a single variety of carbohydrate than from a special variety, the use of a single variety allow increased tolerance to take place in the same way that toleran a drug is increased.

DIABETES INSIPIDUS

Diabetes Insipidus was described by Johann Peter Frank in as "a long continued, abnormally increased, secretion of non-sa rine urine which is not caused by a diseased condition of the kidn This original definition still holds good. The disease is much common than diabetes mellitus, its occurrence being about 14 100,000 dispensary cases. It is less common than diabetes mell even in childhood, although more common in proportion to total number of cases at all ages. About 10 to 15 per cent of total number of cases occur in the first ten years of life, the majo being in the latter half of the decade.

ETIOLOGY.—Diabetes insipidus has no constant pathologi anatomy. The cases which have come to necropsy have sho various pathological conditions, mainly involving the brain. T most common anatomical lesion has been tumor, either of neopla or inflammatory origin. The most frequent situation for these tum

has been in or near the floor of the fourth ventricle, or in the hypophysis. On the other hand, tumors in these situations often do not cause the symptom-complex characteristic of diabetes insipidus.

Syphilis is frequently mentioned in the literature as an etiological factor in connection with the lesions found in diabetes insipidus. In such cases the lesion is either a gumma in the floor of the fourth ventricle or hypophysis, a syphilitic basilar meningitis, or a syphilitic endarteritis.

A certain number of cases have been reported in which no demonstrable anatomical lesion was found. The facts have led to the classification of diabetes insipidus under two types: (1) primary or idiopathic, and (2) secondary or symptomatic.

The real etiology and pathogenesis of diabetes insipidus remains unknown. There are three theories in vogue at the present time: (1) that the symptom-complex is due to a faulty power of concentration on the part of the kidney; (2) that it is due to a primary polydipsia of neurogenous origin, with normal kidney function; (3) that it is due to a symptomatic polyuria caused by stimulation of the kidney from a variety of sources, including stimulation through the nervous system, and from hypersecretion of the hypophysis. In my opinion the first theory is improbable, and the weight of evidence is about equally balanced between the last two.

SYMPTOMS.—The symptoms are directed especially to the urine, which is passed in very large quantities; in one case of Trousseau's, in an adult, amounting to twenty-eight quarts a day. There is a very low specific gravity, varying from 1001 to 1005 or 1007. Glucose is never present, but inosite (muscle sugar) has at times been noted. Albumin and casts are of only rare occurrence. The total solids are normal or in many cases greatly increased.

Intense thirst, a dry skin, disturbance in the surface circulation, and general nervous symptoms are often present. Emaciation is not so marked as in diabetes mellitus, but usually takes place gradually. The children are often stunted in growth, the development of bone and muscle being particularly deficient. The appetite is usually good. Often the general health is not interfered with, and the disease may last for a period of years.

DIAGNOSIS.—This condition is to be distinguished from nervous or hysterical polyuria, which is of temporary duration and is associated with other hysterical symptoms.

It is differentiated from diabetes mellitus by the absence in the urine of dextrose or grape sugar, and by the low specific gravity of the urine.

Chronic nephritis may be excluded from the diagnosis by the presence of albumin and casts and the low percentage of solids elim-

inated, together with the clinical symptoms of chronic nephritis, and the abnormal response to tests for renal function.

PROGNOSIS.—The prognosis and course of diabetes insipidus cannot be predicted with any certainty. In the symptomatic cases, the prognosis is that of the disease of the brain which is responsible for the symptoms. Spontaneous recovery has been reported in a few cases, but there is doubt as to whether these were true cases of diabetes insipidus, or only some temporary form of symptomatic polyuria. In the idiopathic cases, the usual course is either gradual exhaustion with a fatal termination, or else death occurs from some intercurrent infection. Some cases, however, may run a chronic course of many years without much affection of the general health, and well-defined polyuria and polydipsia have continued through life, regarded as an inconvenience rather than as a disease.

TREATMENT.—The treatment of diabetes insipidus is very unsatisfactory. A certain number of cases of syphilitic origin have been reported as improving under anti-syphilitic treatment, and this should be tried in all cases in which there is the remotest suspicion of syphilis. Fully as many cases have shown no improvement under anti-syphilitic treatment.

A gradual restriction of fluid intake has been advocated by some writers. Although favorable cases have been reported under this treatment, the benefit is too doubtful, and the discomfort of the patient under restriction of fluid too great, to warrant its general use.

A few cases have been reported of apparent recovery under treatment with hypophysis extract. Herrick has reported a case in which the symptoms appeared to be strikingly relieved by lumbar puncture. There is no conclusive evidence in favor of these forms of treatment, and they have been tried in other cases without apparent result. There is, however, no contraindication for trying them in a chronic case in which the general health is growing progressively worse.

There is considerable evidence that the symptoms can be influenced by changes in the diet. No particular form of feeding has appeared preeminently successful, but the most rational is to use a bland and sufficiently nourishing diet which will not excite excessive polyuria. Such a diet should contain a relatively small amount of sodium chloride, and of nitrogen.

A great variety of drugs have been tried in diabetes insipidus, but no one of them has given any evidence of favorable action.

PROBLEMS AND RESEARCH.—The principal problems awaiting research in connection with diabetes insipidus have to do with the pathogenesis of the symptom-complex. Even if we assume that the unknown cause of the idiopathic cases probably lies in the brain

or hypophysis, as in the symptomatic cases, the most important thing is to determine whether the polydipsia or the polyuria is the primary condition. In favor of the theory of a disturbed function of the kidney being the primary factor, is the experimental production of polyuria by injury to the nervous system, and various studies on the diuretic action of hypophysis extract. A number of studies on the action of the kidneys in diabetes insipidus have been made, with varying interpretations. The weight of evidence appears to point against the theory of loss of concentrative power of the kidneys, and in favor of the fact that the polyuria is produced by pathological conditions of renal stimulation. The principal argument in favor of the theory that the disease is a primary polydipsia, with a resultant polyuria, is that if the kidneys were primarily at fault, the specific gravity would not increase when the fluid intake was restricted, whereas many observations have recorded such an increase of concentration.

R. Fitz has recently reported a case studied by means of the modern methods of testing renal function. In this case the tests showed that the kidneys were apparently normal but gave findings suggesting a vascular hyposthenuria in which the glomerular vessels appeared to be oversensitive to the stimulation of sodium chloride, while other diuretics were inert. These results support the theory of primary polyuria from some pathological stimulus.

Goodall has recently reported metabolism experiments on a case of diabetes insipidus. He believes that the polydipsia is probably primary, as with restricted fluid intake the kidneys secreted a more concentrated urine. His results suggested that the increased katabolism of the body was the result of under-nourishment rather than of the ingestion of large amounts of fluid.

SPASMOPHILIA

(Tetany.)

Spasmophilia is a condition peculiar to the early years of childhood, characterized by hyperirritability of the peripheral nerves to mechanical and electrical stimulation, and by a tendency toward tonic and clonic spasm.

The disease is very common in infancy, and is responsible for much of the tendency toward convulsions which is characteristic of early life.

ETIOLOGY.—Researches on the nervous system have not disclosed any tissue changes characteristic of spasmophilia. There is, however, little doubt that the hyperirritability of the peripheral nerves is due to a *disturbance of metabolism*. No evidence of any

toxin caused by disturbed metabolism has been found. Considerable evidence has been found, however, that the condition is caused or accompanied by an *anomaly of the metabolism of the mineral salts*. It has been shown that the brain of spasmophilic children is poorer in calcium content than that of normal children (Quest), and metabolism experiments have shown that there is a negative calcium balance in spasmophilia. Other investigators have found abnormalities of the metabolism of the alkaline salts, potassium in particular. It is not yet definitely established whether these changes in the mineral metabolism are the actual cause of the nervous hyperirritability, or whether, as in rachitis, they are simply accompanying manifestations of a disturbance of unknown nature which causes both the symptoms and the negative salt balance. The fact that in both rickets and spasmophilia a disturbance of salt metabolism exists, is strongly suggestive of an etiological similarity in the two diseases, which suggestion is strengthened by certain other peculiarities in their occurrence, such as the rarity of spasmophilia in countries where rickets is rare (Japan).

Whether the nervous hyperirritability is caused by disturbance of salt metabolism, or whether both are caused by a metabolic disturbance of unknown nature, the ultimate cause or causes which produce disturbance are not definitely known. We do, however, know a number of facts as to the occurrence of the disease, which are strongly suggestive of an etiological rôle.

In the first place there is evidence that the factor of *heredity* often plays an important part. The tendency toward laryngeal spasm and convulsions often runs in families Furthermore, spasmophilia is frequently observed in infants with a *neuropathic family history*. The frequency of occurrence of spasmophilia varies with *the time of year*, cases being more common in the winter and spring, as is the case with rickets, especially in March, April, and May. This suggests the influence of lack of fresh air, and bad hygienic surroundings. The age of the child also plays a part; while the disease has been observed in rare cases even in the newborn, it is uncommon before the fourth month, and the most frequent period of occurrence is that *between six and eighteen months*. There can be little doubt that *disturbances of digestion and assimilation* often form the immediately exciting cause for the appearance of spasmophilic symptoms, and the evidence in favor of this connection is so great, that spasmophilia was formerly supposed to be exclusively due to such disturbance, either a reflex phenomenon, or a manifestation of autointoxication ("gastric tetany"). There is evidence that *acute infectious diseases* can increase the severity of spasmophilic symptoms, or cause the appearance of symptoms in latent or cured cases. Finally, *the diet* undoubtedly plays an important rôle in the etiology of the disease.

Spasmophilia is almost never seen in breast-fed infants. Moreover, when spasmophilic infants previously fed on cow's milk are put on human milk, there is usually a rapid disappearance of the symptoms. Finkelstein believes, as a result of feeding experiments, that it is the whey of cow's milk which is responsible for the disorder, and other investigators have reached the same conclusion. The evidence here is very contradictory, another group of investigators concluding that there is no direct connection between spasmophilia and cow's milk (von Pirquet, Thiemich). Under the last hypothesis, the frequent occurrence of spasmophilia in artificially-fed infants is explained by the frequency of disturbances of digestion and metabolism in infants fed on cow's milk, the spasmophilia being secondary to such disturbance rather than due to any specific element of cow's milk.

Finally must be mentioned the recent work on the influence of the *parathyroids* on calcium metabolism, and the experimental production of spasmophilia in animals. This has led to the parathyroid theory of Escherich, which attributes the entire symptom-complex of the disease to anatomical lesions or functional disturbance of the parathyroids.

It thus appears that spasmophilia is probably a disease of complex etiology. The disturbance of metabolism, whether it produces the symptoms *through* an anomaly of salt metabolism, or whether it produces *both* the symptoms *and* the disturbance of salt metabolism, is probably due to numerous etiological factors, such as faulty diet, digestive disturbance, poor hygienic surroundings, lack of fresh air, infections, and so forth. Whether these causes produce disturbances of metabolism directly, or whether they produce it through disturbance of the parathyroids, or in both ways, is yet to be determined.

SYMPTOMS.—Symptomatically, cases of spasmophilia may be divided into four principal clinical types, as follows:

1. Cases in which laryngeal spasm is the principal manifestation.
2. Cases in which eclamptic attacks form the principal manifestation.
3. Cases in which the carpopedal spasm described as *tetany* is the principal manifestation.
4. Cases of a latent character, which do not attract attention and are recognized only by examination.

In all types the principal diagnostic signs characteristic of hyperirritability of the peripheral nerves are found. The clinical types cannot be regarded as different stages, or different degrees of severity. Many children show only the manifestations characteristic of one clinical type. Other children show a combination of laryngeal spasm with either convulsions, or tetany. We do not know why spasmo-

philia is manifested in one child by one clinical type, in another child by another.

The disease shows few general symptoms, and usually the first thing to attract attention is either a convulsion, or a tonic spasm of the extremities, more frequently the former. Laryngeal spasm, although the commonest manifestation of spasmophilia, is rarely recognized as abnormal by the parents unless severe, and is usually recognized only by the physician when the infant comes under his care for some other ailment. In very pronounced cases, the patients are often restless, irritable, fussy, easily frightened, cry a great deal, and allow only certain persons to care for them.

LARYNGEAL SPASM.—The milder forms of laryngeal spasm are the most common manifestation of spasmophilia. The only noticeable symptom is *attacks of crowing inspiration*. The sound of the inspiration much resembles that of common croup, or the whoop of pertussis. The attacks usually come on when the child is disturbed in some way, particularly when it cries or laughs; often every disturbance produces an attack. To the trained ear the inspiratory stridor of spasmophilia is very characteristic, but is usually unnoticed by parents, or by persons not familiar with its significance. Whenever in a crying infant each inspiration is audible as a distinct crowing sound, the physician should suspect spasmophilia and should seek the characteristic diagnostic signs of the disease. It is important to recognize these mild attacks, and to establish the diagnosis, because they sometimes pass over suddenly into the more dangerous, severe form.

The severe form of laryngeal spasm caused by spasmophilia is that often described under the name *laryngospasmus*, or *laryngismus stridulus*. These attacks consist in a sudden complete arrest of respiration through spasm of the larynx. The child's head drops back, and he makes violent efforts to draw air into the lungs, but without result. His eyes start forward, his lips turn blue, his skin becomes covered with a cold sweat, and takes on a bluish-gray color. His consciousness leaves him, his limbs become relaxed, urine and feces are passed involuntarily, and in a few minutes he takes on the appearance of death, except for slight twitching of the eyes and corners of the mouth. Suddenly the spasm relaxes, and a prolonged crowing stridor accompanies the drawing in of air between the closed vocal cords. Deeper inspiratory efforts follow, the stridor diminishing with each, and in a few minutes normal respiration is reestablished, and the child appears somewhat exhausted, but otherwise as well as ever. This is the usual course of the attack, which may be repeated at varying intervals. Sometimes, however, the end is not so favorable, as an attack of laryngospasmus may be fatal. In such

cases, death occurs not from suffocation, but from a stopping of the heart, and consequently artificial respiration is of no avail. One attack may precipitate another, and twenty or more seizures have been observed in twenty-four hours, some severe, some of the mild type. The attacks are much commoner by day than by night, are usually precipitated by some psychic excitation, and do not often come on during sleep, except at the moment of sudden waking. Sometimes an attack of laryngospasmus may be followed by general convulsions. Occasionally the spasm occurs after inspiration when the lungs are filled; this is a dangerous form, and its character is often overlooked because there is no crowing inspiration at the end of the seizure.

Laryngeal spasm is the spasmophilic manifestation which is particularly associated with rachitic children; it is a rare manifestation after the second year.

CONVULSIONS.—The eclamptic attacks of spasmophilia consist in localized or general clonic twitching of the muscles with loss of consciousness. They in every way resemble attacks of true epilepsy. In the milder cases the twitching is confined to the muscles of the cheeks, eyelids, eyeballs, and often the neck. More commonly the entire face and all the extremities are involved, and the convulsion may occasionally be bilateral at the start. Consciousness is lost from the beginning of the attack, the pupils do not react, and the cutaneous reflexes are absent. Only exceptionally is there a stage of tonic spasm. In severe cases the picture is very characteristic, with violent twitching of the face, staring, wide-open eyes, either twitching or turned upward, froth at the mouth, and the entire body shaken by rythmic jerks. The fontanelle in an attack is tense, the pulse is rapid and irregular, and urine and stool may be passed involuntarily.

The duration of an attack is usually from one-half to two minutes; only very rarely does it last longer than three minutes. One attack may, however, be closely followed by another, causing a "status eclampticus." At the end of the attack the motor irritability usually gradually diminishes, and the child sinks into a sleep, from which he awakes apparently normal. An attack may be followed by a brief febrile reaction, and fever is usually seen when the seizures are frequent. Apart from these attacks, fever is not seen in spasmophilia.

The convulsions of spasmophilia may appear only once, but this is uncommon. Usually the attacks are repeated, but the intervals show the greatest extremes of irregularity. The seizures may occur daily, twice a day, twenty times a day, or at intervals of weeks or months. There is absolutely no rule as to the time and frequency

of the recurrences. Attacks may continue daily fo weeks, and then cease abruptly without treatment; tl spontaneously at any time. Relatively, these eclampt much less dangerous than the severe form of larynge

The attacks often show evidence of being precipit special cause, such as acute digestive disturbance, abd tion, or the onset of an acute infection. It is probal of the convulsions of early life which are considered reflex, such as those associated with teething, worms and foreign bodies, would show a background of spasr cases were thoroughly examined. Many cases ori epilepsy, which later recover, are probably also spasm

TETANY.—This type is characterized by a peculia of the hands and feet. The hands tend to assume "obstetrical position." The wrist is sharply flexed ar

FIG. 101

Showing the characteristic spasm of tetany in an infant of 6 mon breast fed and showed only slight degrees of malnutrition an

ward; the thumb in the position of extension is dra palm of the hand beneath the fingers, which are flexe carpo-phalangeal joints, but otherwise are in the positio The feet are rigidly extended and may be in the posi equinus or of equino-varus. The phalanges of the t and extended in the same way as those of the fingers

These tonic contractions are paroxysmal, and vary g duration. They usually last about an hour, but may tion of only a few minutes, or may last for hours or ev contractions are painful at times, especially if effort overcome the spasm. In prolonged spasm, edema or feet may appear. The position of the hands is oft in very young infants, the phalanges not being so rig but the abnormal character of the position can usu recognized. Tetany of the feet is unmistakable.

Other groups of muscles may be involved in tonic s cases, especially the muscles of the face, causing a p

sion; in severe cases the lips are pushed forward. Occasionally there is a spastic strabismus. The spasm sometimes involves the muscles of the neck, causing a stiffness of the neck and retraction of the head suggesting meningitis. Tonic contraction of the muscles of the back or extremities is seen at times. In rare cases even the musculature of the bladder may be involved, with retention of urine. Occasionally persistent cases of tetany are seen, in whch the tonic spasm of the limbs persists for days or weeks; these persistent forms are seen most often in infants emaciated from gastro-intestinal disease.

LATENT CASES.—A certain number of babies do not show any of the three foregoing types of spasmophilia, namely, laryngeal spasm, convulsions, and tetany, but nevertheless show the signs of the disease on physical examination. Such cases may be considered as showing a latent type of spasmophilia.

PHYSICAL SIGNS.—All the clinical types described above are recognized as belonging to the general symptom-complex of spasmophilia, because they show certain signs on physical examination which are characteristic, and diagnostic of the disease. There are three signs characteristic of spasmophilia, and the presence of any one of them is sufficient to establish the diagnosis. They are, (1) increased irritability to galvanic stimulation; (2) Chvostek's sign; (3) Trousseau's sign.

GALVANIC HYPERIRRITABILITY. (*Erb's Phenomenon.*)—This was described by Escherich, and studied by Mann and Thiemich. It depends upon the fact that in the normal child, when the Stintzing electrode is applied over the peroneal nerve, no cathodal or anodal opening contraction is produced with less than five milliamperes of galvanic current. In the hyperexcitability characteristic of spasmophilia, cathodal and anodal opening contractions are produced by much weaker current, down to one milliampere. For ordinary clinical testing of the electrical reactions, *the presence of a cathodal opening contraction with less than five milliamperes of galvanic current, may be considered to establish the diagnosis of spasmophilia*, while the absence of such a contraction excludes it.

The testing of the electrical reactions is a difficult procedure, requiring special training, and cannot be generally used for diagnostic purposes. In very young children, or during convulsions, or with very marked tetanic contraction, a satisfactory test is sometimes impossible. The indifferent electrode is applied to the breast, while the differentiating electrode is applied to the part of the nerve nearest the surface. The median nerve at the bend of the elbow may be used, or the peroneal nerve, or the ulnar nerve. Voluntary movements are easily to be distinguished from the contractions caused by the galvanic current.

CHVOSTEK'S SIGN.—While the testing of the elect is the most thorough method of investigation in susp philia, the testing for mechanical hyperirritability is more easily carried out in ordinary clinical examinatic sists in percussion over the nerve trunk, which causes a tion just as if the nerve were electrically stimulated easiest to carry out on the facial nerve. The physi series of sharp taps with the tip of the finger on variou outer part of the cheek. When he strikes a nerve tr be a rapid contraction of the muscles supplied by the is called *Chvostek's sign*. Sometimes all the muscle the facial nerve are involved in the contraction, bu either the lower branches are struck, producing a cont muscles about the mouth, or the upper branches, pro traction of the muscles about the eye (Weiss' phenom

It must be remembered that Chvostek's sign is not as constant in spasmophilia as is the increased galvani Its chief value is in infancy; it is usually present in infants, and when present is diagnostic. It can, howe even in the spasmophilia of infancy, and is more likely in older children. Sometimes when the facial phenome contraction in the hand or foot may be obtained by tap appropriate points of the radial or peroneal nerves. T tried in such cases, particularly if the physician is nc test the electrical reactions.

The presence of Chvostek's sign is sufficient to estal nosis. The absence of the sign, however, does not mophilia.

TROUSSEAU'S SIGN.—This depends on the fact tha philia, compression of the nerve trunks of the upper a the hand to assume the contraction characteristic of elastic constriction band is put about the arm in the bicipital fork, and is left in place for several minutes cases, the hand assumes the characteristic "obstetri from which it can only be moved by the use of force.

When present, Trousseau's phenomenon is diagnosti ever, often absent in spasmophilia. An objection to i is that it is somewhat painful. Furthermore, in chil the type of spasmophilia characterized by laryngea testing of Trousseau's phenomenon may precipitate an its attendant dangers. This sign should only be testec for testing the electrical reactions is not at hand, and C is absent.

COURSE AND COMPLICATIONS.—Spasmophilia

rather a long course, but the progress of a case is very irregular. When untreated, it usually tends to persist for months, but at any time, without any particular treatment, there may be a sudden improvement, or even a disappearance, of all symptoms and signs, including the electric hyperirritability. This irregularity in the course of the disease makes it very difficult to draw any positive conclusions as to the result of any particular method of treatment. When the instituting of treatment is followed by prompt disappearance of symptoms, and the electric reactions become normal, one is tempted to conclude that the treatment brought about the improvement. It may be so, but one should remember that similar improvement may occur suddenly without treatment.

Even when, with or without treatment, improvement occurs, it does not necessarily mean that the disease is cured. The manifest symptoms of the disease—laryngeal spasm, convulsions, or tetany—usually improve rapidly under treatment. The increased electrical excitability may be more obstinate. This too, however, may disappear with or without treatment. Later, with a change of diet, change of surroundings, or without obvious cause, the symptoms are very apt to recur. In cases treated by feeding with human milk, the symptoms are particularly apt to recur when cow's milk is given again. Patients may be free from signs of spasmophilia in summer, with recurrence in winter. The course of every case is different. The symptoms may be confined to one clinical type, or may pass over into another.

One complication, or rather manifestation, of the disease remains to be mentioned. In all forms of spasmophilia, sudden death occasionally occurs. The cause is sudden cessation of the heart, and this may occur not only during an attack of laryngeal spasm, or less often during a convulsion, but occasionally without any paroxysmal manifestation. A large meal appears to be the usual precipitating cause in such cases. These sudden deaths in spasmophilia have been particularly described by European writers, but I have seen them in this country. The cause of death is supposedly a tetany of the heart-muscle. It is probable that many deaths attributed to "status lymphaticus" for lack of other discoverable cause, were really cases of spasmophilia.

DIAGNOSIS.—The diagnosis depends on the finding of electrical or mechanical overexcitability of the peripheral nerves. Chvostek's sign, or Trousseau's sign, or increased electrical reactions, when positive, are diagnostic.

In a well-marked case of the tetany type, or in a case with typical severe laryngospasmus, the diagnosis is unmistakable. The common mild type of laryngeal spasm is, however, not necessarily of spas-

mophilic origin, as the same symptom is sometimes seen in other diseased conditions. In an infant, however, it should always suggest spasmophilia, unless some other obvious cause be present. The diagnosis is confirmed by the finding of the characteristic physical signs of the disease.

The type characterized by convulsions must be differentiated from the various other conditions in which convulsions are seen. Those principally to be considered are epilepsy, convulsions of reflex or toxic origin, and convulsions symptomatic of organic disease of the brain. The spasmophilic origin of the convulsions is recognized by the signs of electrical and mechanical hyperirritability of the nerves. If the presence of the characteristic signs shows the child to be spasmophilic, it makes little difference whether or not the immediately exciting cause be reflex or toxic. It must be remembered, however, that the spasmophilic child may have meningitis, encephalitis, or other organic disease of the brain, and the presence of spasmophilia should not lead to the overlooking of the signs of these other diseases. Epilepsy is distinguished from spasmophilia by absence of Chvostek's sign, and normal electrical reactions.

PROGNOSIS.—The majority of cases of spasmophilia eventually recover. The only immediate danger is the sudden death which occurs in a few cases. A study of the later history of spasmophilic children has shown, however, that quite a number of them show some signs of neuropathy or of defective intelligence, and in only about a third is development wholly normal. There is no evidence that spasmophilia ever passes over into epilepsy.

TREATMENT. PROPHYLACTIC.—The important factors in the prophylaxis of spasmophilia are *maternal nursing*, *sunlight*, and *fresh air*. If a baby is breast-fed, with good hygienic surroundings and plenty of fresh air, spasmophilia will not develop.

HYGIENIC.—Hygienic treatment is fully as important in spasmophilia as in rickets. The ideal treatment is the same as that described for rickets, the essentials being fresh air, sunlight, and exercise. All the hygienic measures described for rickets should be employed in cases of spasmophilia.

DIETETIC.—The most valuable of all known curative agents in spasmophilia is *human milk*. Whether or not it can be employed in an individual case depends upon various circumstances, such as the ease with which a wet-nurse can be obtained, and the severity of the spasmophilic manifestations. It must be remembered, however, that the most certain and effective cure for spasmophilia is human milk.

When in spasmophilia it is necessary to use artificial feeding, the basis which can serve as a guide in the modification of cow's milk

is somewhat uncertain, because it has never been finally proven whether some particular element of cow's milk is responsible for the symptoms, or whether they are caused by the general difficulty of digestion with cow's milk. With our present knowledge it is best not to use a whey-mixture, and to substitute as far as possible some other form of carbohydrate, such as dextri-maltose, for lactose. It is also advisable to begin to add starch to milk-mixtures as early as the digestive powers of the child will permit. The whey salts may be further reduced by using precipitated casein. Some such formula as fat 3%, dextri-maltose 6%, protein 1.5%, starch .75%, may be chosen to begin with, and if precipitated casein can be made or obtained, the protein may be given in that form. Further modification must be guided by the digestive peculiarities of the individual child, according to the general principles of infant feeding. It is important to eliminate all forms of indigestion.

With older infants, able to take a more varied diet, cow's milk can be excluded, or reduced to a minimum. Bread, cereals, butter, beef juice, meat, and vegetables can take the place of milk at this age. Overeating should be avoided, and the child should never be allowed to eat a very large meal.

When the symptoms of spasmophilia have disappeared, cow's milk should be added gradually to the diet. It is often necessary to exclude cow's milk for weeks or months, if a recurrence of symptoms is to be prevented.

MEDICINAL.—Three methods of specific medicinal treatment have been recommended in spasmophilia, as follows: (1) The giving of calcium salts; (2) the giving of phosphorus and cod liver oil; (3) the giving of parathyroid extract.

Calcium was first given in the form of calcium lactate. Some observers claimed good results, others failed to get them. The original dose was 3 to 5 grains three times a day. Later larger doses were recommended. At the present day *calcium chloride* is preferred to calcium lactate. Several observers have reported good results as measured by the electrical reactions, from the giving of calcium chloride, if the drug is given in sufficiently large doses. Bluhdorn gives from 2 or 3 to 4 or 8 grammes daily. Sedgwick, in this country, has published very good results from calcium chloride given in doses of at least 3 grammes (75 grains) daily. The salt is given in divided doses in the feedings, and infants do not seem to object to the taste. The question of the value of calcium therapy must be regarded as still *sub judice*. Most observers believe that the effect is only to diminish the symptoms, and that it is only temporary. It is difficult to conclude as to the value of any therapeutic procedure in a disease of indeterminate course like spasmophilia. It is certain

that in effectiveness calcium chloride cannot be compared with human milk. I believe, however, that it should be tried in severe or obstinate cases for which human milk cannot be obtained, or in which human milk fails to effect a cure It may be given in doses of 75 grains daily, in divided doses in the feedings.

The combination of phosphorus with cod liver oil has been widely advocated in Germany as a method of treating spasmophilia as well as rickets. The basis for its use is certain metabolism experiments which show that the combination favors the retention of calcium. There is, of course, no positive proof of its clinical value in spasmophilia. Its advocates believe that its good effect is more permanent than that of the calcium salts. Rohmer has recently published a paper in which he claims the best results from a combination of calcium therapy and phosphorlebertran. The combination of phosphorus and cod liver oil may be given in the same doses as described under rachitis.

Parathyroid extract has been recommended in spasmophilia, on account of recent research on the rõle of the parathyroids in the etiology of the disease. There is at present little evidence that it has any value.

SYMPTOMATIC.—The clinical manifestations of spasmophilia may be aided by various temporary measures. At the beginning of an attack, whether the manifestation be larnygeal spasm, a convulsion, or an attack of tetany, a dose of two teaspoonfuls of castor oil should be given, and feeding with cow's milk should be stopped at once. If the abdomen be distended, a high irrigation of the colon should be given, and this may be repeated if necessary. For twenty-four hours, only boiled water or barley water should be given, and then feeding with human milk should be begun, and continued as long as possible. If human milk be unobtainable, the cow's milk modification given after twenty-four hours should be weak at first.

When the patient is seen in a convulsion, a warm bath should be given, followed by castor oil and irrigation of the colon. If the convulsions continue or are frequently repeated, sodium bromide, 5 to 15 grains with chloral hydrate 1 to 5 grains in 1 ounce of water should be given by rectum, and repeated if necessary every two hours for three or four doses. An infant of one month can be safely given by rectum 1 grain of chloral; of six months, 2 grains ;of twelve months or over, 5 grains. If this is not effective, or while waiting for it to take effect, the inhalation of ether in small amounts may be used for severe convulsions. At times, a tendency to repeated convulsions can be checked by lumbar puncture.

The milder attacks of laryngeal spasm require no symptomatic treatment. It is important, however, to prevent all possible causes

of sudden psychic disturbance. Sudden noises or disturbance, inspection of the throat, unnecessary physical examination, the entrance of strangers into the room—all these should be avoided. If there has ever been an attack of severe laryngospasm, the child should be watched day and night, and a basin of cold water, together with a hypodermic syringe filled with camphor in oil should stand ready beside the bed. When a severe attack occurs, if the child does not quickly breathe, it should be sprinkled with cold water. If apnea continues, the finger should be pressed under the tongue, and if this is not followed by an inspiration, an injection of camphor in oil should be given, followed by efforts at resuscitation by artificial respiration. The moment the crowing inspiration occurs, all therapeutic endeavors should cease, in order not to precipitate a second attack.

When the only manifestation of spasmophilia is carpopedal spasm, symptomatic treatment is not usually indicated, reliance being placed on the effect of the general hygienic, dietetic, and medicinal treatment. When the tonic spasms are particularly severe, sodium bromide may be given in doses of 5 grains every four hours.

PROBLEMS AND RESEARCH.—An enormous amount of research work has been devoted to the subject of spasmophilia in the last decade. The principal problems confronting the investigator have been indicated in the discussion of the etiology of the disease.

The first feature of spasmophilia to attract the attention of investigators was the apparent relation to the diet. Feeding experiments with the various elements of cow's milk gave no positive evidence in support of Finkelstein's theory of a whey injury. Since there is five times as much calcium in cow's milk as in human milk, the theory was advanced that spasmophilia represented a calcium intoxication. Stoelzner explained this by assuming that the excessive calcium in cow's milk is absorbed, but must be re-excreted by the intestinal mucosa. On account of the extraordinary demands thus made upon the excretory functions, they become insufficient, with a resulting calcium retention and intoxication. While certain experiments in feeding with excessive calcium have apparently given positive results in support of this theory, it has been attacked by many writers, and the weight of evidence is decidedly against it, most experiments proving that there is no increased calcium retention in spasmophilia.

Certain researches tending to prove that the action of calcium on nerve tissue was depressant, and that its withdrawal was excitant, led to the experiments of Quest, who found a diminished calcium content in the brains of spasmophilic infants. These results turned the attention of investigators to the theory that the symptoms in spasmophilia are due to calcium deficiency. The results of a great

mass of research work in connection with this theory may be summarized as follows: (1) There is in spasmophilia evidence of a negative calcium balance; (2) there is evidence of a disturbance of the metabolism of the mineral salts in general; (3) there is evidence that the metabolism of the mineral salts has an influence on the functioning of the nervous system, some salts acting as excitants, others as depressants.

If these conclusions be accepted, we are next confronted by three further problems:

1. Are the symptoms produced directly by the disturbance of salt metabolism, or is this only a coordinate manifestation, the symptoms being produced by other substances, possibly of a toxic character?

2. If the symptoms are produced by a disturbance of salt metabolism, how can the relation of spasmophilia to the diet be explained?

3. If the symptoms are produced by a disturbance of either the salt metabolism, or the intermediary tissue metabolism, what is the ultimate cause of the disturbance?

In the investigation of the first question, no evidence has been found of any toxic substances causing nervous hyperirritability. There is considerable evidence that the symptoms are produced by an actual disturbance of the mineral metabolism. For example, in a recent research, MacCallum, Lambert and Vogel found that when calcium was removed from blood by dialysis, and the blood perfused through an isolated extremity, extreme hyperexcitability of the nerves was produced. There is some evidence that it is the relative proportions of calcium and other salts which is concerned in producing the symptoms, rather than the actual calcium content.

In connection with the second question, the fact remains that spasmophilia is usually seen in artificially-fed infants, and that human milk is its most effective curative agent. This is strongly suggestive of some special exciting substance in cow's milk, and this does not fit well with the theory that a disturbance of salt metabolism is the cause of the disease. There are still many authorities who believe that the whey of cow's milk has a particular rôle as an etiological factor. Pediatricians are especially prone to attribute pathological processes to diet. The weight of evidence certainly suggests that cow's milk is not the only factor in spasmophilia. If it has no specific rôle, the relation of spasmophilia to artificial feeding can only be explained on the ground that disturbances of metabolism in general are more common in artificially-fed infants.

The third problem, that of the ultimate cause of the disturbance of metabolism, is the one which is stimulating the greatest activity in research at the present day. It is obviously closely connected

with the question of the rôle of diet. There are two principal theories for the cause of the disturbance of metabolism. These are the following:

1. That the disturbance is due to a variety of causes, among which are unsuitable food, indigestion, lack of fresh air, faulty hygienic surroundings, acute infections, and so forth.

2. That the disturbance is due to anatomical or functional insufficiency of the parathyroid glands.

These two theories are not mutually exclusive. It is very probable that the various causes enumerated in the first theory may all play an etiological rôle. The question is whether the influence of the parathyroids is a necessary feature in the production of the disease. The literature of the last few years is full of investigations upon this point

The parathyroid theory of tetany and spasmophilia depends on the fact that tetany may be produced experimentally in dogs by extirpation of the parathyroids. A great number of writers have attempted to show that this experimental tetany is identical with postoperative and spontaneous human tetany. The chief argument in favor of this is the identity of the clinical symptoms, and of the disturbance of calcium metabolism.

The theory has been attacked by a number of writers, chiefly those who believe strongly in the dietetic origin of spasmophilia. The arguments against the parathyroid theory are that it is a mere hypothesis, unsupported by any pathological or anatomical findings. Also, since milk diet seems to be favorable in the experimental tetany of dogs, it has been argued that spasmophilia and experimental tetany are entirely different conditions. The reply to this has been that pediatricians are too anxious to find things to prove the dependency of tetany on diet.

A number of statistical post-mortem researches have been published on the parathyroids. Erdheim first described hemorrhages into the parathyroids, occurring at birth, and a number of investigators have found hemorrhage, or the residuals of hemorrhage, in the parathyroids in numerous post-mortems of infants. On this ground, a number of authorities believe that spasmophilia is caused by disturbed or lowered parathyroid function, due to the effect of these hemorrhages.

Other investigators have failed to find very much evidence of an anatomical lesion of the parathyroids. Still others have found such traces, without evidence of spasmophilia.

Among recent investigators are a number supporting the hypothesis that spasmophilia is caused, not by traumatism of the parathyroids during birth, but rather by subsequent disturbance of their

function. This theory is consistent with the theory that diet, hygienic conditions, and so forth, play an etiological rôle.

There will probably be much research on the metabolism and the chemistry of the blood, both of human and experimental spasmophilia in the next few years

In therapeutics the principal question awaiting final solution is the value of calcium chloride medication. Also, a recent contribution by Berend is based on the theory that there is disturbance both of calcium and magnesium metabolism in spasmophilia, and claims good therapeutic results from the subcutaneous administration of magnesium sulphate. Parathyroid extract will probably be more extensively tried before it is finally condemned.

PELLAGRA

Pellagra is a disease characterized by certain definite pathological changes involving the skin, the nervous system, and the gastro-intestinal tract, and other organs. The disease was first described by Gaspar Casal of Spain in 1735. The name pellagra was first used by Frapolli of Italy in 1771, who adopted the term in general use among the Italian peasants, meaning rough skin. Since then the disease has been described under many different names.

The disease is very common in certain localities. It is apparently very much on the increase in the United States. It is only comparatively recently that its occurrence among children has been adequately recognized. It has been known that in Italy pellagra is of comparatively frequent occurrence in children, and even in infants. Recently its occurrence among children has been recognized in this country, but there are few statistical studies showing its frequency. Lavinder found in a study of 15,870 cases of pellagra, 412 under five years of age, and 1,719 between the ages of five and fifteen years. The consensus of opinion among the physicians of the South, so far as can be determined from a number of recent publications, appear to be that the frequency of the disease in childhood is about 10 per cent.

From the time of its first recognition the disease has been associated with the eating of Indian corn. Until recently the prevalent theory of etiology has been that pellagra is due to the eating of spoiled maize. The most recent investigations have brought forward evidence which renders the spoiled maize theory very improbable. I have classified pellagra among the disturbances of nutrition and metabolism, not because I am convinced that the preponderance of evidence is in favor of the dietary theory of etiology, but simply for convenience of reference until the weight of evidence shall, by future research, be brought to incline toward one view or the other.

ETIOLOGY.—The cause of pellagra is unknown. The available evidence bearing on etiology, has given rise to two distinct theories as to the cause of the disease.

1. The first theory is that pellagra is due to an improper diet. The most recent evidence, while against the spoiled maize theory, points toward the association of pellagra with a vegetarian diet, containing a preponderance of cereal. This has been explained on the theory of the vitamins, advanced by Funk. A cereal diet is supposed to be deficient in certain substances essential to proper nutrition and metabolism. This theory would bring pellagra into the same etiological group as beriberi and scurvy. Other suggestions in connection with the dietetic origin of pellagra, are that it may be caused by the toxic action of substances like aluminum, which are present in large amounts in cereals, or to silica, which is present in excess in certain drinking waters. A deficiency of amino acids has also been suggested as a possible explanation of a dietetic etiology.

2. The second theory is that pellagra is an infection, caused by a parasite. The brilliant researches of Sambon constitute the chief foundation for the parasite theory. Sambon believes that the disease is an insect-borne infection caused most probably by a protozoal parasite, and transmitted by a fly of the genus Simulium The Simulium reptans, incriminated by Sambon, is not found in this country, but the Simulium vittitatum, or buffalo gnat, is frequently encountered. Some writers are inclined to suspect this insect, while others believe that in this country the evidence should eliminate the Simulia, but believe that the disease is transmitted by some other variety of biting insect.

The evidence for and against these two theories appears to be fairly evenly balanced. It will be further discussed under Problems and Research. There are certain known facts of importance in connection with the etiology of the disease.

AGE.—No age is exempt. The disease is comparatively rare in infants under two years of age, and very rare in infants under three or four months. In children, pellagra occurs most often between the ages of two and ten years. It becomes less common again in the first years after puberty.

SEX.—The preponderance of victims among the female sex is a notable characteristic of the disease in adults. In children this does not hold, and the disease appears to be about equally distributed between the sexes.

CLIMATE AND DISTRIBUTION.—Pellagra appears to be very generally distributed, and no climate or country is entirely exempt. There are, however, very marked variations in the frequency of

pellagra in different localities. In the United States, the disease is commonest in the Southern States. Even in the South there are interesting variations in the distribution of the disease, which contribute additional interest to the problem of etiology. In the North the disease has generally been regarded as rare or non-existent. In recent years this has been shown to be untrue, and an increasing number of cases are being reported from Northern communities. Physicians in every part of the United States should be on the watch for cases of pellagra. The disease appears to be notably on the increase in this country.

SEASON.—There is a well-marked seasoned incidence in pellagra. Most of the cases occur in the spring, early summer, or autumn. It is very unusual for cases to appear during cold weather. Recurrent attacks are seen at times, as in malaria, occurring each year at a certain season.

HEAT.—There is no definite evidence that it is the heat of our Southern climate which is responsible for the greater frequency of pellagra in the South. There is some evidence, however, that the rays of the sun aggravate the cutaneous eruption, and that they may act as a contributory cause in the acquirement of the disease.

SURROUNDINGS.—There is a notable relation between the occurrence of pellagra and the surroundings of the patients. It has not been established that pellagra is more common in the rural districts, or among field workers. The disease is found most often under conditions of unsanitary surroundings, whether in rural districts, or in cities. In certain parts of the South it is particularly common in the mill villages.

DIET.—In spite of the fact that the maize theory of pellagra is improbable, nevertheless there is some evidence of a relation to the diet. Pellagra seems to be associated with a vegetarian diet, and many cases have improved when more animal protein was given. The diet of the poorer classes in the South is largely cereal. No more definite relation between pellagra and the diet has been established.

IN FAMILIES.—Sambon states that in pellagra districts he has often found the parents and all of the children attacked. Siler and Garrison report that in one-half of the cases observed, only one case occurred in a family, in a quarter of the cases there were two in a family, and in the remaining quarter there were family groups of three, four, and five. There is, however, no evidence of direct contagion.

PATHOLOGICAL ANATOMY.—The lesions of pellagra in children show no essential differences from those of adults. For a de-

tailed account of the pathological anatom
is referred to the special literature of the su
skin is an angioneurotic process similar to
forme. In the internal organs the essentia
infiltration with mononeuclear cells. The
lesions are found in the nervous system an
tract. In the nervous system various d
found, which apparently represent, not a
process, suggesting a toxic degeneration, a
cells and nerve fibres. In the spinal cord
marked in the columns of Goll and Burdac
gastro-intestinal tract appear to be an esser
rather than secondary manifestations. T
chronic catarrhal inflammation, with exfolia
and infiltration of the stroma.

SYMPTOMS.—The principal features of
of pellagra, are *symmetrical* skin lesions, *gastro*
and *certain nervous manifestations*.

CUTANEOUS MANIFESTATIONS.—The symm
pellagra are considered pathognomonic. The
in intensity, which causes corresponding variat
The general character of the rash is that of
mildest form, the eruption presents the appea
reddish erythema. In more severe cases, there
ing of the skin resembling sunburn. In a late
present a grey scaling appearance. After the
some time, there is often marked thickening of
numerous fissures are formed. In some cases th
tion of bullae, this being called the wet type, in
the dry type characterized only by erythema an
ing of the skin.

Both the wet and the dry types occur in chil
is, however, very much more common in chil
type, and was the only form of rash observed by
ages of the South.

The distribution of the rash is peculiar. It
monly on the exposed portions of the body. T
of the rash is the backs of the hands, and the d
feet. In some cases only the hands are affected,
backs of the hands were lacking in only 5 per c
cases in children. In a certain number of case
appearing on the backs of the hands and feet, ex
side of the arms to the elbows, and up the calves
knees.

The rash may less commonly invade other portions of
The neck and face are affected next in frequency to the
The extent of the invasion is variable. Casal's "necklac
ing completely around the neck, and the "mask," in
entire face, are rare. Weston has observed the rash
occasionally on the palms and soles, on the front of th
on the abdomen.

FIG. 102

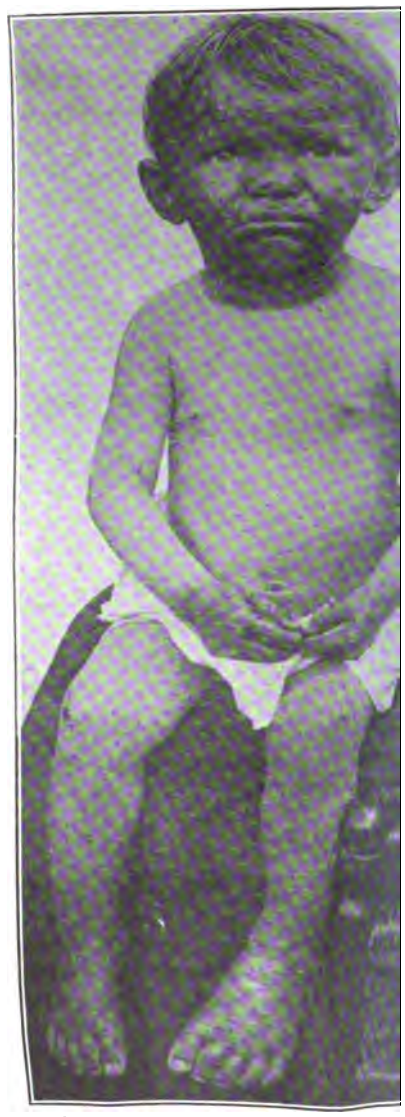
Annie V., white, aged 6; fifth attack of pellagra; first attack occurr
the age of 2

The duration of the rash is very variable. It ma
rapidly, leaving the skin somewhat smooth and glistenin
however, it persists for an indefinite period, and finally
by desquamation, leaving the skin smooth, and somewh
color than the adjoining skin. Itching and burning may
especially during the stage of desquamation.

The essential feature of the rash of pellagra is its
character. Although it appears chiefly on the exposed
cannot be prevented from appearing by protection from

THE MOUTH.—The tongue in pellagra
and is often fissured. These appearance
of epithelium, and to a glossitis. The r

FIG. 103



Josiah P., aged 18 months; wet variety of
(By courtesy of Dr. J. J. Watson, Columbi

able at the sides and tip. The entire mucous membrane of the mouth is sometimes redder than normal, but does not often in children have the fiery redness seen in severe cases in adults. The lips are also often fissured.

GASTRO-INTESTINAL SYMPTOMS.—These are more pronounced, according to Weston, in very young children than in those over four

FIG. 104

Pellagra in Italy. Photograph from Dr. Eugenio Bravetta, Mombello, Italy

years old. Young children show a tendency to indigestion, and often suffer from severe diarrhea. Constipation is seen at times.

NERVOUS SYMPTOMS.—In general, the manifestations of disturbance of the nervous system in children are less common and less marked than in adults. Rice found few nervous manifestations in his series, except in the patellar reflexes which were most often exaggerated, but were sometimes decreased or absent, and only

infrequently normal. Weston states that i
children, insomnia is often marked, and d
cases.

BLOOD AND URINE.—The blood changes
to be especially characteristic. An increas
of small lymphocytes has been observed in
canuria is very common in pellagra, but is

DIAGNOSIS.—The only pathognomonic
sis of pellagra can rest with any certainty, i

FIG. 105

Pellagra (By courtesy of Dr. William Weston, C

metrical dermatitis. It is on this that the diag
majority of cases. Prompt recognition of the d
success in treatment, and the presence of pellagr
country, makes it within the range of possibilit
be encountered at any time by any physician.
are likely to be overlooked chiefly because this
to be forgotten, and in districts where the disea
may not be recognized only because it is unfan
little meaning to the physicians of that locality.
of the occurrence of pellagra be remembered, and i

infrequently normal. Weston states that paresthesia is common in children, insomnia is often marked, and depression occurs in many cases.

BLOOD AND URINE.—The blood changes in pellagra do not appear to be especially characteristic. An increase in the relative number of small lymphocytes has been observed in a number of cases. Indicanuria is very common in pellagra, but is not constant.

DIAGNOSIS.—The only pathognomonic sign on which the diagnosis of pellagra can rest with any certainty, is the characteristic sym-

FIG. 105

Pellagra (By courtesy of Dr. William Weston, Columbia, S. C.)

metrical dermatitis. It is on this that the diagnosis depends in the majority of cases. Prompt recognition of the disease is essential to success in treatment, and the presence of pellagra in all parts of the country, makes it within the range of possibility that a case may be encountered at any time by any physician. Cases of pellagra are likely to be overlooked chiefly because this possibility is likely to be forgotten, and in districts where the disease is uncommon, it may not be recognized only because it is unfamiliar, and has but little meaning to the physicians of that locality. If the possibility of the occurrence of pellagra be remembered, and if the characteristic

cutaneous manifestations be present, the diagnosis of the disease will present but little difficulty.

The question is important whether or not the diagnosis of pellagra in children can be made without the presence of the rash. In a locality in which pellagra is rare or has been unknown, such a diagnosis must always be doubtful; in a region where pellagra is endemic, it will be less hazardous. When in a child are found such symptoms as a red tongue, fissured lips, diarrhea, insomnia, restlessness, paresthesia, exaggerated or absent knee-jerks, or other nervous symptoms not to be explained by any other recognizable disease, the possibility of pellagra should be considered. Often under such circumstances, the

FIG. 106

Pellegra in Italy (By courtesy of Dr. William Weston, Columbia, S. C.)

appearance of an erythema, however slight or transient on the characteristic parts of the skin, will confirm such a tentative diagnosis.

There is no positive laboratory method of making the diagnosis. The changes in the blood, and the indicanuria are not sufficiently marked and constant, to be of much diagnostic value. Whenever the classical triad of symptoms, dermatitis, intestinal disturbance, and nervous disturbance is present, the diagnosis is certain.

PROGNOSIS.—The prognosis is probably very uncertain in young infants. In older children, the disease is milder, and with proper treatment, recovery usually occurs. The period during which re-

lapses are likely to occur is very indeterminate, and consequently cases which have apparently recovered should be kept under observation for a number of years.

TREATMENT.—Hygienic measures are of the first importance in the treatment of pellagra in children. Whenever possible, a complete change of environment should be carried out. The patients should, if it can be arranged, be taken to a locality in which pellagra is unknown. If such a change cannot be managed, the hygienic conditions under which the patients live should be improved as much as possible.

The uncertainty as to the etiology of the disease gives rise to a lack of definite therapeutic indications. The only suggestion comes from the dietary theory of the disease. With our present knowledge, a change of diet is advisable in all cases. Certainly, with infants nursing pellagrous mothers, the infant should be weaned. In artificially-fed infants, the diet should be as liberal as the digestive powers of the child will permit. Cereals should be kept at a minimum, and such articles as beef juice, broths, meats, and eggs, given as soon as possible.

In older children, corn products should be prohibited, and the diet should be so arranged as to contain plenty of animal protein. Foods leaving a large undigested residue should be avoided.

Among other hygienic measures, hydrotherapy has always ranked high in pellagra. The affected areas of the skin should be protected from the direct rays of the sun. Soothing lotions, such as that containing calamine, should be applied to the dermatitis. Internally tonics, such as arsenic and iron, are valuable.

PROBLEMS AND RESEARCH.—At the present time, pellagra presents a most fascinating problem in etiology, in which many methods of investigation are available. It is quite probable that the continued statistical study of the peculiarities of distribution of the disease, such as is being carried out by various commissions, will throw much light on the pathogenesis of the condition. There is also a wide field for experimental study, by means both of dietary methods, and by animal inoculations.

The principal evidence in favor of the nutritional theory is of a negative character, its advocates regarding the burden of proof as resting upon those who believe the disease to be of infectious origin. Lavinder and Francis carried out a very thorough series of inoculation experiments on monkeys, failing to obtain any positive results. Willets and Waring, in their epidemiological studies in the Georgia Sanitoria, failed to find evidence of transmissibility, but believed that their results supported the dietary theory, the disease being most prevalent among those whose diet contained little meat and

animal protein. The influence of diet in promoting recovery is believed by many to be sufficiently obvious to constitute evidence as to etiology.

The arguments in favor of the parasitic theory are many. In the first place, the peculiarities of distribution and of seasonal incidence are strongly suggestive of an insect-borne contagion, especially the seasonal recurrences which have been observed. There is no definite evidence in favor of the maize theory or the dietary theory. The recent report of the Illinois Pellagra Commission found no connection with occupation, but evidence of definite spreading from certain centres. There is no evidence, however, incriminating the Simulium.

DIVISION VI

SPECIFIC INFECTIOUS DISEASES

An infectious disease is one caused by a parasitic microörganism. A *specific* infectious disease is one in which the pathological changes in the body are caused by one variety of microörganism only, and are peculiar to infection with that one organism. There are certain microörganisms which produce pathological changes which are in no way specific, sometimes in one part of the body, sometimes in another. The lesions produced are not peculiar to the particular microörganism, but the same tissue changes, and the same disturbances of function, may be produced by other organisms. Examples of such infecting parasites are the common pyogenic organisms, such as the staphylococcus pyogenes, and the streptococcus pyogenes. Other examples are seen in the gonococcus, the bacillus of dysentery, and the colon bacillus. The clinical picture presented by infection with this group of organisms depends not so much upon the nature of the infecting organism, as upon the organ or part of the body which happens to be attacked. For this reason these diseases are described under the various organs or regions of the body which may be involved.

The border line between this group and the *specific* infections, however, is not very sharp, and in certain cases the distinction must be somewhat arbitrary, dependent upon convenience of description. For example, in lobar pneumonia the pathological anatomy of the lung, and the clinical picture produced, are peculiar to infection with the pneumococcus, and from this point of view the disease should be described among the specific infections. The pneumococcus, however, does not produce lobar pneumonia only, but may attack a great variety of organs, producing pathological changes which are in no way specific. Furthermore, the clinical description of lobar pneumonia is much more conveniently placed under the Diseases of the Lungs, where it can be brought into telling contrast with other forms of pneumonia.

Again, it is a question whether the lesions or the symptoms of infection with the bacillus of influenza are sufficiently peculiar to that organism to warrant the classing of influenza as a specific infection. Its chief title to a place in this division is its tendency to epidemic occurrence.

Neither the lesion nor the clinical picture of epidemic cerebrospinal meningitis is in any way characteristic, as practically the same changes in the central nervous system can be produced by the streptococcus, the pneumococcus, the bacillus of influenza, and other organisms. The diplococcus of epidemic meningitis, however, is concerned in the production of no other disease, and the occurrence of this form of meningeal infection in epidemics, together with the fact that it has a specific serum therapy, are the reasons for describing it among the specific infections.

The organisms causing rheumatic fever and erysipelas have not been shown to be specific, but belong in the general streptococcus group. On account of the peculiarities of the lesions and symptomatic manifestations, they are for convenience described in this Division.

There are a certain number of diseases classified among the specific infections, in which the cause has not been discovered. The evidence of an infectious origin for these diseases lies in the fact that they are highly *contagious*, that is, easily transmitted from one individual to another by contact, the same peculiar lesions and symptoms being reproduced after transmission. There can be no doubt that each of these diseases is a specific infection, of which the cause is yet to be discovered. In this group belong scarlet fever, measles, rubella, varicella, variola, vaccinia, and mumps.

The contagiousness of the infectious diseases is a matter of degree. It is probable that most if not all of the specific infections are transmitted from one individual to another. The degree of contagiousness depends upon the manner, ease and frequency, with which such transmission occurs. With each infection, modern medicine must concern itself largely with the mode of transmission.

In former times, descriptions of the specific infections were concerned almost exclusively with the symptoms, differential diagnosis, and symptomatic treatment of the diseases belonging in this group. Under the most modern views, we must consider two other aspects of a specific infection as of particular importance. The first of these aspects is the *manner of transmission and invasion*, that is, how the parasite is brought to the host, and how it gains access to the tissues of the host. The second of these aspects is *the defensive or "immune" reaction of the host against the invading parasite*. Upon these two aspects of the specific infections depend most of our prospects of progress in knowledge and in ability to combat these disease processes successfully.

Classification of the specific infections upon any definite scientific basis is impossible. For convenience of description, the following grouping will be employed:

1. The Exanthemata	Scarlet Fever, Measles, Rubella, Varicella, Variola, Vaccinia
2. Highly contagious diseases without exanthem	Diphtheria, Pertussis, Mumps, Influenza
3. Diseases with constant infection of the blood	Typhoid Fever, Malaria
4. Diseases tending to occur in epidemics	Epidemic Cerebrospinal Meningitis, Polio-myelo-encephalitis
5. Diseases of doubtfully specific cause	Rheumatic Fever, Erysipelas
6. Diseases essentially chronic	Tuberculosis, Syphilis

I. THE EXANTHEMATA

In contradistinction to the various diseases of the skin which dermatologists are accustomed to designate as exanthems of local origin are certain acute diseases characterized by exanthems symptomatic of the specific infection, which are called the exanthemata. This group of infectious diseases is of especial interest in connection with children, as it is among children that they most frequently occur. They can, however, attack individuals of any age. Although none of these diseases is entirely self-protective, yet the instances in which they develop in an individual more than once are rare. Each of these diseases is characterized by certain conditions common to all. Besides being highly contagious, each disease runs a definite course and is self-limited, facts which should be remembered in the study of its diagnosis and treatment.

The course of these exanthemata from the time when the infection takes place up to the appearance of their later manifestations may be divided into distinct stages. In the first of these a more or less definite period elapses between the acquirement of the infection by contact, and the appearance of any disease manifestations. During this period the infecting organisms apparently remain dormant for a time, constituting what is called the *stage of incubation*. The stage of incubation is followed by certain general symptoms resulting from the supposed development of the special organisms and constituting the *prodromal stage*, or stage of invasion. These prodromal symptoms are, after intervals varying according to the special disease, followed by an efflorescence on the skin, which marks the third stage of the disease, called the *stage of efflorescence*. The efflorescence in its turn is followed by what is called the *stage of desquamation*, this desquamation being more or less pronounced in proportion to the intensity of the lesions of the skin which have occurred during the stage of efflorescence.

SCARLET FEVER

(Scarlatina)

Scarlet fever is a specific infectious disease, characterized by angina, exanthem, and a tendency to certain special complications. It is contagious, self-limited, and one attack confers an immunity which usually protects the individual throughout life.

ETIOLOGY. THE MICROÖRGANISM. — The organism which causes scarlet fever is unknown. In the necrotic tissue and inflammatory

exudate of the throat is found almost always a streptococcus. The same type of organism has been found in the blood, not only in severe and fatal cases, but also in blood cultures from mild cases. These findings have led some writers to regard this streptococcus as the cause of the disease. Favorable reports on the effect of both streptococcus vaccine and antistreptococcic serum in the treatment of scarlet fever have lent additional weight to this theory, and some investigators have even reported biological differences between the streptococcus found in scarlet fever and other strains of streptococci. Nevertheless, although scarlet fever has been successfully transmitted to the anthropoid apes, all attempts to produce the disease experimentally with streptococcus cultures have failed. There is much other evidence against the streptoccocus origin of scarlet fever, such as the peculiar contagiousness of the disease, the immunity conferred by one attack, and the fact that the streptococci are usually not found in the earliest stage of the disease. Most authorities believe that these streptococci represent a secondary infection, which, however, may have an important influence on the course and complications of the disease.

Within the last few months, Mallory has reported the finding of an organism, which he believes may prove to be the specific cause of scarlet fever. This organism is a bacillus which in appearance somewhat resembles the bacillus of diphtheria. The evidence in favor of its etiological importance is its constant presence in the deeper layers of the tonsillar tissue in scarlet fever, and its absence in other cases. At the present writing, inoculation experiments are being conducted with cultures of this organism, the outcome of which will probably decide the question of its relation to scarlet fever.

TRANSMISSION.—Every case of scarlet fever comes from some other case, the disease being transmitted from a sick to a healthy individual. The virus cannot, however, as many persons suppose, fly through the air across a considerable space. Under ordinary circumstances the mere coming into a room occupied by a scarlet fever patient, provided that nothing in the room be touched, would not be a sufficient exposure to cause infection. The usual means of infection in scarlet fever is *actual contact with infected material.* Infectious material from the patient, such as secretions from the mouth and nose, reach the outside world where it can contaminate any object with which it comes in contact. It most often reaches the healthy individual through *direct contact*, such as occurs in kissing, or handling, or sleeping together. In addition to direct contact, *indirect contact* plays an important rôle in scarlet fever. In this mode of transmission, healthy persons, and various objects, such as handkerchiefs, food, toys, carpets, and so forth, become contaminated by the patient, and constitute the carriers of the disease. This in-

direct contact, while much less common than direct contact, is nevertheless important in scarlet fever, because the virus is able to retain its infecting power for a considerable time outside the human body.

There is a possibility of transmission through the air without actual contact, the virus being carried in the fine, moist particles expelled in sneezing and coughing. In scarlet fever, however, this plays a very slight part, and is not likely to occur unless the patient cough directly in the face of the exposed child.

THE INFECTIOUS PERIOD.—Scarlet fever is contagious from the very beginning, possibly even a day or two before the appearance of symptoms. It was formerly supposed that it was not contagious in the prodromal stage, but it is now regarded as *certainly contagious from the appearance of the first symptom.* The spread of the disease is largely due to mild ambulant, or unrecognized cases. The duration of the contagious period is long, and probably very variable. It has long been known that scarlet fever is usually contagious throughout the stage of desquamation, the average duration of which is about six weeks. It has been wrongly considered that the contagious period necessarily ends with the completion of desquamation. Under this view, the scales of desquamation were considered a source of infection. There is, however, no definite evidence that the scales contain the virus, and those instances in which the disease has apparently been transmitted by the desquamating particles of epidermis can be explained on the ground that the scales became contaminated by secretion from the mouth or nose. We cannot say with absolute certainty that the scales are not contagious, but the weight of evidence is decidedly against such a theory. Certain it is that there are cases on record in which the patient was discharged well after desquamation had been completed, even as long as ten weeks after the onset of the disease, and still caused the infection of others.

Altogether too much attention has been paid to the scales of desquamation. The chief danger comes from the secretions and discharges of the patient, which have been proven to be infectious. Early infection probably occurs simply from the secretions of the mouth and nose, while the later infection in the stage of desquamation may come from the discharges from otitis, rhinitis, suppurating glands and so forth.

The infectious period of scarlet fever being variable, and there being no means of determining whether the patient is still a possible source of infection, some definite limit must be assigned, for the purpose of isolation and the protection of others. The contagious period of scarlet fever has been arbitrarily placed at six weeks by most writers. While the majority of children are not capable of infecting others after six weeks, I believe this period to be too short for the purpose of complete protection. Certainly no patient who has any visible discharge from the mucous membranes should be

released from isolation, even if six weeks have elapsed and desquamation has been completed. I would not allow my own patients to receive in their homes, or to play with any child who has just been discharged from scarlet fever isolation at the end of six weeks, but in order to take every possible precaution, would require the lapse of ten weeks.

THE PORTAL OF ENTRY.—The route by which the virus of scarlet fever enters the body has not been established by certain proof. The weight of evidence however, is strongly in favor of the throat as the first localization of the infection, which points to the mouth as the probable portal of entry.

THE INCUBATION PERIOD.—The period of incubation of scarlet fever is variable, but is short in comparison with the other exanthemata. In the great majority of cases it is less than seven days. The commonest period is *from two to four days.*

PREDISPOSITION.—Scarlet fever may occur at any age. It is very much commoner in childhood than in adult life. The commonest period of occurrence is from three to six years of age Susceptibility continues to be marked up to about the age of thirty years, after which it diminishes. The disease is rare in the first year of life, especially in the early months.

The following table was compiled by McCollom. It represents the age and the number of deaths in one thousand cases of scarlet fever treated in the contagious wards of the Boston City Hospital:

TABLE 41

One Thousand Cases of Scarlet Fever, by Ages, With the Deaths

YEARS	CASES	DEATHS	YEARS	CASES	DEATHS
Under 1 year	9	2	19 years	10	1
1 year	24	8	20 years	7	0
2 years	66	20	21 years	8	3
3 years	115	21	22 years	8	0
4 years	99	7	23 years	15	0
5 years	124	9	24 years	9	0
6 years	106	7	25 years	7	0
7 years	64	6	26 years	5	0
8 years	62	5	27 years	7	1
9 years	58	1	28 years	6	1
10 years	53	3	29 years	3	0
11 years	21	1	30 years	9	0
12 years	20	0	31 years	3	0
13 years	23	1	32 years	3	0
14 years	10	0	33 years	2	0
15 years	12	0	34 years	3	0
16 years	10	0	35 years	1	0
17 years	7	0	41 years	0	0
18 years	9	1	50 years	1	0
				1000	98

The predisposition of children toward scarlet fever is much less than toward measles and whooping-cough. In the most severe epidemics, not more than twenty per cent of the exposed who had not had a previous attack, including adults, contracted the disease, while about fifty per cent of the children escaped. In families with numerous children, there are often only one or two cases, whereas with measles or whooping-cough all are usually affected.

This peculiarity of the predisposition of children to scarlet fever has usually been explained on the ground of a varying individual susceptibility to the infection, some children having a natural immunity. I do not believe this explanation to be correct, but believe that the apparently varying susceptibility depends on the method by which scarlet fever is transmitted. Measles and whooping-cough are not contact infections, but what the German writers call "droplet infections" (tröpfcheninfektion), in which the virus is carried through the air by the moist particles expelled in coughing, and these particles can traverse a very considerable air space. In a contact infection such as scarlet fever or diphtheria, the acquirement of the disease depends on whether or not the virus is properly carried to the exposed child, and all sorts of irregularities in the carrying over of the virus into the required contact are possible, which irregularities might easily cause the apparent variations in susceptibility. In other words, I believe that the exposed children who escape do not come in contact with the virus, which consequently never reaches the portal of entry. This theory is fully adequate to explain the known facts, and I believe it to be more in harmony with them than the theory of natural immunity. The latter theory could only be proved by the impracticable method of actually inoculating a series of healthy children.

Similarly, it is generally supposed that the greater frequency of scarlet fever in childhood is due to the fact that children are more susceptible than are adults. Even when those adults are excluded who are protected by a previous attack, scarlet fever is much more likely to attack children than unprotected adults, but I do not believe that this is due to a greater susceptibility in childhood, or that adults acquire a relative immunity. I believe that the frequency of scarlet fever among children is also to be explained by the mode of transmission of the disease. *Close contact* is a condition much commoner in childhood. Children live close together, often sleep together, play together, and are often assembled in groups, as in school and kindergarten. Children are often habitually uncleanly with their secretions, which soil their faces, hands, beds and clothes. All the conditions favoring the spreading of a contact infection such as scarlet fever are multiplied in childhood, and this I believe to be the true explanation of the greater frequency of the

disease at this age. Whether or not the rarity of the disease in the first year is due to less opportunity for effective contact, or to a natural immunity, it is impossible to say.

IMMUNITY.—An attack of scarlet fever confers a pronounced and lasting immunity. Nevertheless, second attacks, though rare, are seen at times, and are commoner than in measles.

PATHOLOGICAL ANATOMY.—The only constant and characteristic lesions of scarlet fever are found in the skin, in the mouth and throat, and in the lymphnodes.

THE SKIN.—The lesion of the skin in scarlet fever is an acute dermatitis, of variable depth and intensity. The process goes through the stages usual in acute inflammation, first hyperemia, then exudation of serum and cells into the corium, then death of the epidermis, which is cast off in flakes.

The changes in the mucous membrane of the mouth are similar to those in the skin, the inflammation taking a catarrhal form. It frequently extends to the nose and middle ear, from which places in turn it may invade the mastoid cells or the accessory sinuses. It rarely invades the larynx.

THE THROAT.—Superficially the mucous membrane of the throat may show nothing more than an intense redness, suggesting only a catarrhal inflammation. In reality, however, the inflammatory process in the scarlet fever angina is much deeper, particularly in the tonsils and adjacent mucous membrane. It is always really a phlegmonous inflammatory process which tends toward a certain amount of purulent softening, and which extends deeper into the tissues than does the inflammation of diphtheria, although it often does not present such characteristic appearances on the surface. In the more severe cases the inflammation may produce a coagulation necrosis, which gives the appearance of a gangrenous or membranous angina which superficially resembles the lesion of diphtheria. The false membrane in scarlet fever is usually found filled with streptococci. It is in the deeper layers that Mallory has found his bacillus. The necrosis may be more or less extensive over the mucous membrane of the throat, and the inflammation may extend to the cervical lymphnodes, causing purulent softening. It is the streptococci which are concerned in this latter process.

THE LYMPHNODES.—The most constant change throughout the body in scarlet fever is a hyperplasia of all the lymphoid tissue.

TOXIC LESIONS.—The poison of scarlet fever may produce a variety of lesions in the various parenchymatous organs of the body, which do not differ from the lesions associated with other varieties of toxemia. The one particularly associated with scarlet fever is an acute toxic nephritis. Among the toxic lesions seen in fatal cases

are fatty degeneration of the myocardium, focal necrosis of the liver, and proliferation of the cells of the Malpighian bodies of the spleen.

SECONDARY LESIONS —The streptococcus infection may extend to various parts of the body, causing secondary lesions. Beside otitis media and purulent cervical adenitis, secondary lesions which may occur at times are bronchopneumonia, pleurisy, abscess of the lung, endocarditis, pericarditis, arthritis, and abscesses in the cellular tissue.

THE TYPICAL FORM OF SCARLET FEVER

PRODROMAL STAGE OR STAGE OF INVASION.—The invasion of the disease is usually sudden, and, as a rule, active. The child feels very sick, looks dull, and in a large number of cases vomits continuously. The vomiting usually ceases in the stage of efflorescence and often before the prodromal stage has ended. The pulse is rapid. The temperature is high, 103°, 104°, 105° F. In infants and very young children if the temperature rises to 104° or 106° F., convulsions are very likely to occur. Older children complain of sore throat, and often of headache. Young children show marked apathy or restlessness, and often are delirious

At this stage the physician finds nothing on physical examination which can account for the high fever and toxic symptoms, except in the throat. The appearance of the mucous membrane of the throat, although perhaps not characteristic, as at times a simple non-specific pharyngitis may simulate it quite closely, is, in connection with the general symptoms, at least suggestive. The mucous membrane of the soft palate and of the pharynx is much congested. The redness covers the soft palate only, being sharply bounded at the hard palate.

In this stage, a positive diagnosis of scarlet fever cannot be made, but the symptoms and appearance of the throat should always arouse suspicion.

EFFLORESCENCE —The prodromal stage in scarlet fever is comparatively brief, and the efflorescence usually begins to appear in from twelve to twenty-four hours after the onset. It appears first on the neck, chest, or back, and next extends over the entire trunk, and to the upper arms and thighs. Finally it reaches the forearms, legs, hands, and feet. The full extension of the eruption occupies about two days, at the end of which time the entire body is involved with the exception of the face. The cheeks may be reddened, but the redness is a simple congestion, not the punctate rash of scarlet fever. The nose, upper lip, and chin remain entirely free, even from reddening, and this pale area shows a marked contrast to the redness of the skin elsewhere, and is a characteristic symptom of scarlet fever.

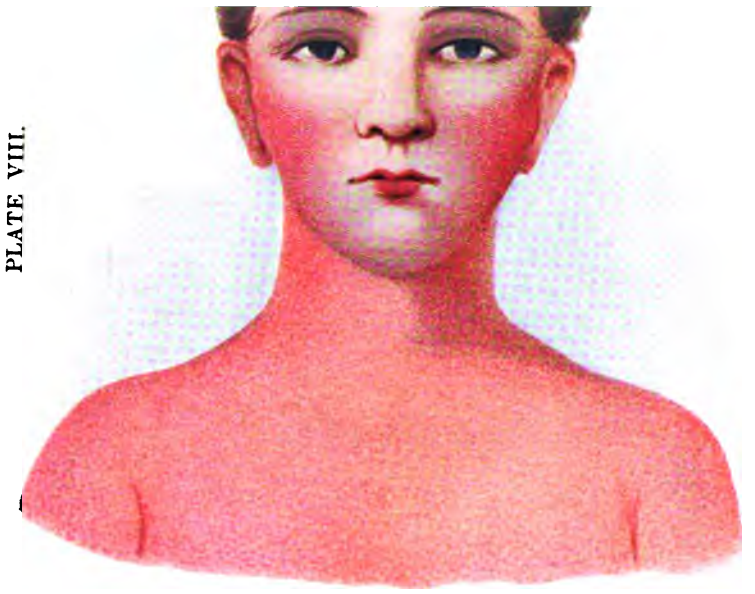
The eruption is a punctate erythema, which becomes confluent. The first appearance is that of scattered, very small, light red spots, between which the normal skin is clearly visible. Between the punctate spots new ones keep appearing, until after one or two days, they become confluent. In the meantime the eruption has been constantly becoming a brighter red, until at the time of full development, the skin presents a brilliant erythematous blush, which does not appear to be made up of single punctate spots, except in certain situations such as the inner surface of the thighs, and the backs of the hands. At all times pressure with the fingers will cause disappearance of the exanthem, and when the pressure is released, even if the rash is in the confluent stage, the discrete punctate spots will reappear first, and then rapidly become confluent.

In the early stage of the efflorescence, the punctate spots are flat. With full development they become slightly raised, so that on stroking the skin with the finger, a very fine unevenness is detected. The swelling of the follicles in the regions of the forearms, lower legs, and backs of the hands and feet, sometimes causes the formation of larger papules. Sometimes the papules become slightly vesicular. These variations of the rash have no relation to the severity of the case. Very small hemorrhages may sometimes be seen in those regions where the clothing binds or chafes, such as the elbows or the axillae. The eruption sometimes itches.

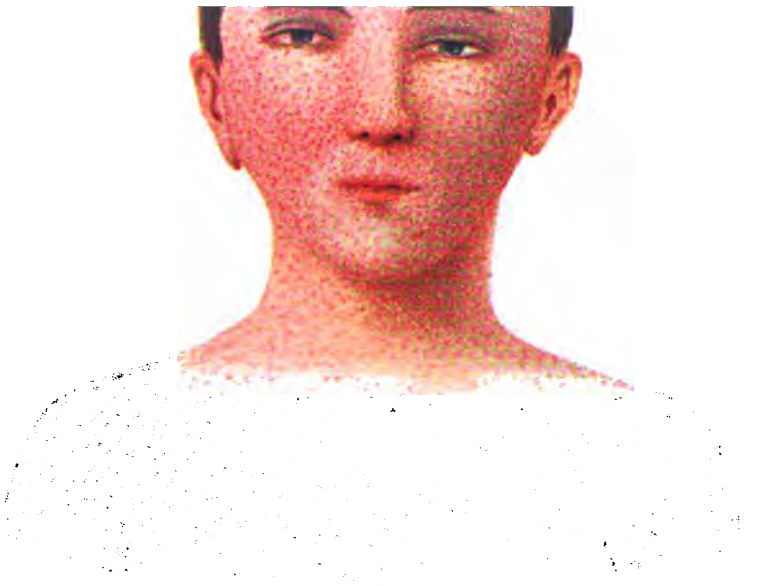
The efflorescence reaches its height in from three to five days, and then gradually disappears in the order of its appearance. It is usually entirely gone by the beginning of the second week. A certain amount of pigmentation of the skin may persist.

DESQUAMATION.—The stage of desquamation begins at about the seventh day from the time when the efflorescence first appeared and on the parts of the skin first attacked. The desquamation, however, is not always proportionate to the intensity of the efflorescence. It is at first composed of small particles of cutis, but these soon become larger, and early in the third week from the beginning of the disease they fall from the body in large flakes, and are most characteristic and pronounced on the fingers and toes. This form of desquamation is called *lamellar*. Here, again, we have an important means of distinguishing scarlet fever from measles, for in measles the desquamation is almost universally of a furfuraceous character throughout the whole course of the disease, while the characteristic desquamation of scarlet fever is lamellar. This lamellar form of desquamation may, at times, in certain individuals, and following more intense inflammation of the skin, be represented by large and extensive pieces of skin. This is well shown in a specimen in the Warren Museum, where large strips of skin have come from the hand of a patient with scarlet fever so as almost to form a glove.

PLATE VIII.



Scarlet Fever.



Measles.

Sometimes the desquamation lasts only ten days, but it usually continues for two or three weeks, and may not be complete for six or eight weeks. It is especially slow in disappearing from the hands and feet, and it may remain between the fingers and toes for a number of weeks. Sometimes after the desquamation has apparently ceased, and the skin has been smooth and normal for several days, it may begin again, and thus prolong the period of convalescence.

THE MOUTH AND THROAT.—The redness and swelling of the tonsils, soft palate and pharynx which is present during the stage of invasion, increase for the next three or four days during the stage of efflorescence. Older children complain of sore throat, while in younger children pain on swallowing is often evident. The color of the throat is a fiery red, of an intensity seldom seen in ordinary pharyngitis or tonsillitis. The swollen tonsils often show yellowish exudate in the crypts. The submaxillary lymphnodes are enlarged, and usually tender to pressure. In typical cases the scarlet fever angina reaches its height in from three to five days, after which it gradually improves, but a membranous type may develop even after this time.

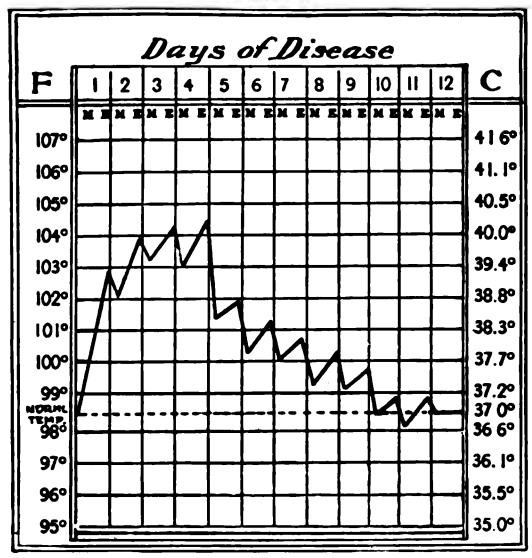
The tongue in the stage of invasion is heavily coated, but clears rapidly during the stage of efflorescence, and by the third or fourth day shows a bright red surface on which the enlarged papillae stand out prominently. This is the characteristic "strawberry tongue" of scarlet fever, which has been considered diagnostic by some writers. It is an aid in diagnosis, but is sometimes seen in other conditions, and is occasionally lacking in scarlet fever.

THE TEMPERATURE.—The fever continues high, 103° F., to 105° F., for the first three to five days of the disease, and often mounts steadily higher until the full development of the exanthem. The height of the temperature depends more upon the severity of the angina than upon the intensity of the efflorescence. After the full development of the rash, the temperature begins to fall by a step-like lysis, reaching the normal from the seventh to the tenth day. Occasionally in cases otherwise typical, the fever may persist as long as two weeks, without any discoverable cause or complication to account for its persistence.

THE PULSE.—In scarlet fever the pulse is very rapid, the acceleration in children being greater than would ordinarily be accounted for by the temperature. Younger children often have a pulse rate of from 160 to 180 in typical mild cases. Increase in pulse rate persists after the temperature has fallen to normal, and disappears gradually.

THE GENERAL CONDITION.—Apathy, restlessness, sleeplessness, weakness, and anorexia are common symptoms accompanying the fever, and disappear as the temperature falls. Vomiting usually ceases during the stage of efflorescence. The nose and conjunctivae are not notably affected, and the ears are usually not involved during the stages of invasion and efflorescence. All the palpable lymph-nodes are noticeably enlarged, and the tenderness of the cervical lymphnodes may make movement of the head painful.

CHART 6



Benign and regular form of scarlet fever

THE BLOOD.—The blood shows regularly a leucocytosis, the white count being between 18,000 and 40,000. The increase is entirely in the polymorphonuclear neutrophiles. The leucocytosis gradually disappears in the second or third week. There is often, but not always, a diminution of the eosinophiles, beginning at the end of the first week. Certain inclusion bodies, first described by Döhle, are regularly found in the leucocytes during the febrile stage. They were at one time considered diagnostic, but have been shown to be present in other acute infections.

THE URINE.—The urine is lessened in amount during the febrile period, returns to normal amount after defervescence, increases during the stage of desquamation, amounting at times to polyuria, and returns again to the normal amount at the end of this stage. During the stage of efflorescence, especially if the temperature is considerably heightened, there may appear in the urine a small amount of albumin, and a few casts and red blood cells, but these disappear as

the temperature subsides, are probably only the result of the fever, as in many other diseases accompanied by a high temperature, and their appearance is not to be confounded with the albuminuria of the nephritis which in some cases complicates the stage of desquamation. Acetonuria is often seen in scarlet fever, especially in younger children.

CONVALESCENCE.—All the symptoms subside with the fall of the temperature. The patient appears fully convalescent by the middle or end of the second week, often before desquamation has begun.

ATYPICAL FORMS OF SCARLET FEVER

The form of scarlet fever described above as typical, is the commonest. It represents a medium or average severity of the disease. Deviations from this typical form are, however, extremely common, both on the mild side and on the severe side. In fact they are so frequent that the term atypical is hardly suitable.

THE MILD FORM.—Very mild cases of scarlet fever are quite common, and at certain times the majority of cases in any locality may be of the mild type. Their recognition is important, as they are often largely responsible for the spread of the disease.

In the mild type the sore throat, fever, and constitutional disturbances are very slight, often not enough to attract attention. In such cases the only symptom noted by the child's parents may be the efflorescence. The temperature may not go above 101° F., there may be no sore throat, and the child does not feel sick enough to want to go to bed. Even the efflorescence itself may be very slight, appearing only on part of the body at a time, and never becoming confluent. Sometimes even the rash is overlooked, and the disease runs its course unsuspected, until the appearance of desquamation, or of a nephritis, arouses suspicion that scarlet fever has gone before. Even the desquamation, especially in children who are bathed daily, may be overlooked, unless special note be taken of the appearance of the fingers and toes.

THE "RUDIMENTARY" TYPE.—In this type the angina, fever, and constitutional disturbance are typical, but the efflorescence is very slight, or even absent. The rash may not appear till the third, fourth, or fifth day, and is slight in intensity, and limited in distribution. It is usually most pronounced on the back. Cases without any rash are seen occasionally in older children and adults. These rudimentary forms resemble ordinary tonsilitis or pharyngitis, and are known to be scarlet fever only through simultaneous occurrence with other cases in the family.

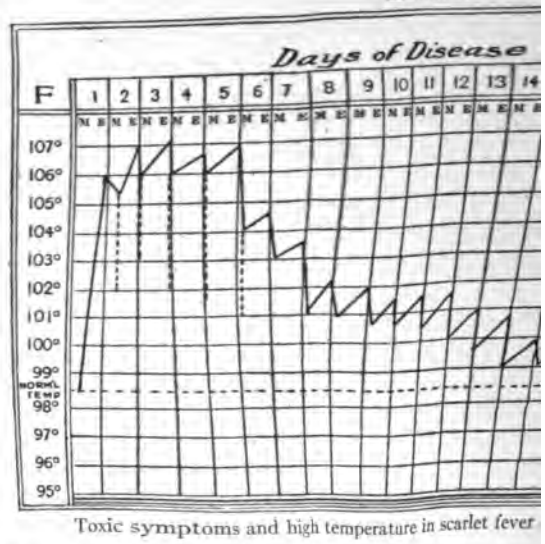
SEVERE FORMS.—The most severe type, sometimes called *malignant scarlet fever*, is comparatively rare. The child is suddenly

most commonly seen in cases which h
angina from the start, or which have d

THE LYMPHNODES.—The acute infl
and surrounding cellulitis, may go on
tion may extend to the mediastinal lym
mediastinitis, and eventually leading to
tatic abscesses in various parts of the bo

THE EAR.—The middle ear is so clos
the Eustachian tubes with the naso-pha
tions are exceedingly common. The sym
a secondary infection of the ear is taking
as they may differ much in their manifes

CHART 8



Toxic symptoms and high temperature in scarlet fever

fore watch with the greatest solicitude and exa
the greatest care during the course of scarlet feve
may be active and represented by aural pain and
On the other hand, there may be no apparent p
infants and young children, who are often unable
location of the pains by which they are affected.
the symptoms may be merely a somnolent conditio
attacks of fretfulness.

Cases of scarlet fever occurring in the fall and w
more likely to develop middle ear complication tha
sons. When it does occur, both ears are usually a
the second week. If the constitutional symptoms

The severe symptoms are not, as in the malignant f scarlet fever contagion, but to the *associated second infection*. The onset is severe, with high fever, m tional disturbances, and severe angina. The efflore ordinary course. About the third or fourth day, how instead of showing the usual appearance of catarrha or follicular tonsillitis, takes on the appearance angina. Part or all of the tonsils becomes covere or greyish-white false membrane, which at first cl the membrane of diphtheria. After a time, the me more adherent, and more deeply implanted in the mu than does that of diphtheria. The membrane frequ the soft palate and pillars, and occasionally to the p geal wall. In the meantime the enlargement of the nodes becomes very marked, and the inflammation the adjacent connective tissue, making movement of ful, and sometimes forming a hard swelling on each s This phlegmonous inflammation may be so marked, lings on the two sides meet under the chin. The flammation often extends into the nasopharynx, and This causes obstructed nasal breathing, and a purul

In favorable cases the membrane begins to dis five to eight days after the onset, and the fall of lysis occurs. In bad cases the tissue necrosis becon the tissue of the tonsils, and often of the adjacent part forming a brownish ulceration, covered with a bloo charge. The process occasionally extends to the epi the larynx, causing stenosis.

Continued fever and marked constitutional dist the development and spreading of the membranou course of these severe cases depends upon the varie of the complications.

COMPLICATIONS.—The complications of scarl occur in the early stages of the disease are due to t the associated secondary streptococcus infection to the body. All these complications may occasionall course of a typical mild case of scarlet fever. Tl run what is apparently a normal course for a time. T may begin to fall, and then suddenly rise again, w immediately clear what is the cause. In such cases c tion will eventually disclose that the patient has de membranous angina, or some other complication.

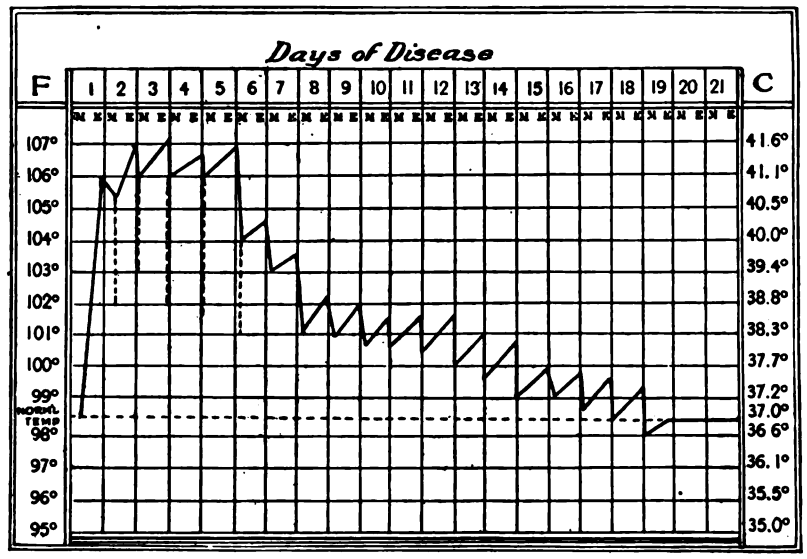
While the various complications due to secondar infection may occur in cases otherwise apparently t

most commonly seen in cases which have either shown a membranous angina from the start, or which have developed a membranous angina.

THE LYMPHNODES.—The acute inflammation of the lymphnodes and surrounding cellulitis, may go on to suppuration. The infection may extend to the mediastinal lymphnodes, causing a purulent mediastinitis, and eventually leading to general sepsis, with metastatic abscesses in various parts of the body.

THE EAR.—The middle ear is so closely connected by means of the Eustachian tubes with the naso-pharynx, that aural complications are exceedingly common. The symptoms which indicate that a secondary infection of the ear is taking place are not always clear, as they may differ much in their manifestations. We should there-

CHART 8



Toxic symptoms and high temperature in scarlet fever treated by baths

fore watch with the greatest solicitude and examine the ear with the greatest care during the course of scarlet fever. The symptoms may be active and represented by aural pain and great restlessness. On the other hand, there may be no apparent pain, especially in infants and young children, who are often unable to indicate the location of the pains by which they are affected. In these cases the symptoms may be merely a somnolent condition and occasional attacks of fretfulness.

Cases of scarlet fever occurring in the fall and winter are much more likely to develop middle ear complication than at other seasons. When it does occur, both ears are usually affected early in the second week. If the constitutional symptoms of the primary

disease are severe, no new symptoms show themselves, so that the ear complications may be easily overlooked until much damage has been brought about. The otitis is purulent, and much of the membranum tympani is usually destroyed. Mastoiditis and septic sinus thrombosis are not very rare. The scarlet fever otitis has the peculiarity that it occasionally produces necrosis of the ossicles, and sometimes more widespread necrosis. This results in impaired hearing or deafness. May has shown by statistics that of 5613 cases of deaf-mutism, 572 were due to scarlet fever. The absence of symptoms pointing to the ear does not, therefore, justify us in overlooking an otitis media. Routine examinations of the ears should be made in all cases.

THE JOINTS.—An acute inflammation of the joints, usually the larger ones, is not infrequently met with during the course of scarlet fever. It is more frequent in adults than in children, and usually occurs at the end of the first week. As a rule, these cases are not of long duration, and if effusion takes place in the joints it is serous, does not become purulent, and does not give an especially serious prognosis.

A more severe form of arthritis, apparently caused by secondary infection with streptococci, may also occur during the course of scarlet fever. The effusion into the joints in these cases becomes purulent and leads to serious and permanent disorganization of the tissues and often to death from general septic infection.

THE HEART.—The rapid pulse which is seen as a rule in scarlet fever shows that the heart is particularly susceptible to the toxin of the disease. In severe cases this rapid pulse persists after the end of the febrile period, and is sometimes accompanied by other signs of cardiac weakness.

In addition to this toxic manifestation, a cardiac lesion sometimes complicates scarlet fever. It usually appears at the end of the first or in the second week of the disease. The commonest manifestations are a systolic murmur at the apex, enlargement of the cardiac dulness to the left, and sometimes bradycardia. These symptoms can last several weeks, but finally disappear entirely. They are due not so much to a true endocarditis, as to an atony of the heart muscle with slight dilatation and relative mitral insufficiency.

A true endocarditis, however, can also complicate scarlet fever, and cannot be distinguished from the condition just described. Occasionally it is ushered in by a reappearance of fever. In such a case the mitral murmur is permanent.

Pericarditis is rare.

THE LUNGS.—Scarlet fever may be complicated by purulent bron-

chitis, bronchopneumonia, and empyema. These complications are, however, comparatively rare.

SEQUELAE.—Most of the complications described above occur comparatively early in the course of the disease. Occasionally, however, between the end of the second week and the sixth week, a reappearance of fever is observed, which is found to be due to the late development of fresh lymphnode infection, otitis, arthritis, or endocarditis. Rarely is seen an actual scarlet fever relapse, with a fresh appearance of angina and efflorescence.

NEPHRITIS.—This is the most common and important of the sequelae of scarlet fever. It usually appears in the third week of the disease, almost never before the twelfth day, and often not until the fourth, fifth, or even the sixth week. It is a toxic nephritis, the glomeruli chiefly being affected. The advent of nephritis is most commonly recognized by the appearance of edema and bloody urine, although if the physician examines the urine daily, he can sometimes discover an approaching nephritis before the appearance of edema. Occasionally the development of nephritis is heralded by fever, vomiting and headache, but more often edema is the only symptom noted. The urine is much diminished in quantity, and sometimes, in severe cases, anuria is seen. The urine usually contains considerable blood, and in color is red or "smoky." There is usually a rather large quantity of albumin, while the sediment shows numerous red blood corpuscles and casts. The edema begins about the eyes, and usually involves the skin of the whole body. Fluid is often found in the peritoneal cavity, the pleural cavities, and the pericardial cavity. Enlargement of the area of cardiac dullness, bradycardia, and raised blood pressure appear in a short time. In cases with anuria, or marked diminution in the amount of urine, headache and vomiting may appear, followed by slow pulse, convulsions and coma.

In most cases the scarlet fever nephritis is completely healed after five or six weeks, or somewhat longer. In some cases chronic nephritis may persist. In still less common cases, death may occur either from uremia, or from increasing edema and dilatation of the heart.

The frequency of nephritis is very variable at different times, and in different epidemics, the incidence being from 2 per cent. to 30 per cent. The development of nephritis also appears to bear no relation to the severity of the scarlet fever.

DIAGNOSIS.—In typical cases of scarlet fever the combination of fever, characteristic angina, efflorescence and strawberry tongue, makes the diagnosis easy. The diagnosis cannot be made with certainty before the appearance of the rash. Every case of sore

throat in a child should at the start be regarded a of scarlet fever, and the patient should be isolated. of the throat may or may not be sufficiently chara the suspicion of scarlet fever very strong. Fort dromal stage is so short, that the appearance of t firm the suspicion within twenty-four or thirty-si fever be present.

It is the frequent occurrence of atypical cases mild and rudimentary types, which makes the di fever difficult. In many cases the physician canno he is confronted by an atypical case of scarlet f other condition. Cases of scarlet fever without e and can only be recognized by their occurrence in other cases to which the patient has been exposed. and rudimentary types, the efflorescence may be aty and the diagnosis may remain doubtful. In this ever, it is well to remember that *a scarlatiniform ra for only twenty-four hours or less, is usually not sca*

The usual problem in diagnosis which presents it cian, is whether a certain rash or efflorescence on t scarlet fever or not. Many diseased conditions ma by a cutaneous rash which resembles more or less c cence of scarlet fever. When a definite angina is with fever and constitutional disturbance, the evide scarlet fever. When, however, fever and angina a larly if the efflorescence be not general, the diagnc difficult.

The following are conditions which are with entiated from scarlet fever:

TOXIC ERYTHEMA.—This is seen in a number childhood, particularly in cases of disturbed nutrit ism. The rashes which occur after the administ drugs, and the serum rashes, belong in the same pared with scarlet fever, these rashes are more fl show a changing character, so that cutaneous l urticaria or measles are seen alongside of the scarla Furthermore, angina is absent in these erythemas, t ing normal. Fever and constitutional disturban absent. The leucocytosis characteristic of scarlet and, especially in serum rashes, there is apt to be relative diminution of the polynuclear forms.

INFECTIOUS ERYTHEMA.—An erythematous rash symptom of a variety of acute infections. It is up of the punctate areas characteristic of scarlet

absent. The recognition of the underlying infectious process is of aid in the diagnosis. The most common infections which sometimes produce a rash in children, are lobar pneumonia, typhoid fever, and influenza.

MEASLES.—Scarlet fever and measles should easily be differentiated. The long prodromal stage, the catarrhal symptoms and conjunctivitis, the Koplik's spots, the absence of characteristic angina, and the different appearance of the efflorescence, differentiate measles so completely that one rarely has to think of it in diagnosis. The rash of measles is characterized by larger macules; if larger macules are seen in scarlet fever, they are never general, but are usually confined to the extremities. The measles rash involves the cheeks, nose and chin. If it is so confluent in places as to resemble the appearance of scarlet fever, there are always other parts of the body where the macules of measles are discrete and typical.

RUBELLA AND "DUKES' DISEASE."—Typical German measles, with the larger macules which never become confluent, should never be confused with scarlet fever. It is a question whether the condition described by the German writers as "Dukes' Disease" or "Fourth Disease," represents a distinct specific infection, or whether it is a type of German measles with scarlatiniform rash. The efflorescence resembles scarlet fever, but angina is absent, and fever and constitutional disturbance are slight. Mild cases of scarlet fever have the same characteristics, and Dukes' Disease is difficult to distinguish from such cases. The "strawberry tongue" is absent in "Dukes' Disease," and desquamation does not occur. The disease is rare.

MILIARIA.—In infants and young children who are too warmly dressed, and who sweat freely, there occurs a punctate rash, popularly called "prickly heat," which superficially bears a close resemblance to the scarlet fever exanthem. When it is localized, the distinction is not difficult, but miliaria may involve the entire body. It is recognized by the prominent raised character of the rash, which gives it a peculiar rough or prickly feeling. The absence of angina and the other symptoms of scarlet fever strengthens the diagnosis of prickly heat.

DIPHTHERIA — The membranous angina of scarlet fever may suggest diphtheria, but the latter disease is recognized by the abscence of a cutaneous efflorescence, and by the presence of the diphtheria bacillus in culture from the throat.

PROGNOSIS.—In mild and typical types of scarlet fever, the prognosis is favorable. In the severer types with membranous angina the prognosis is more doubtful, depending on the severity of the case, and on the number and character of the complications. The

violence of the invasion and severity of the constitutional disturbance at the onset is some indication as to the severity of the case, but even in cases with a comparatively mild invasion, the physician should always give a guarded prognosis, because in no disease is the course to be reckoned with less certainty than in scarlet fever. Mild cases may suddenly become severe, and unforeseen complications may develop at any time.

Every variation from the typical temperature curve is an unfavorable sign, and every rise in temperature after the fall by lysis has begun or after the temperature has reached the normal, should be regarded with suspicion, as it may mean a troublesome or dangerous complication or sequela.

The severity of the disease varies greatly in different epidemics, and at different times, so that the mortality varies between 1 per cent and 50 per cent. The average mortality is about 15 per cent. Children between the ages of two and four years are most in danger. Mild and typical cases, in which no membranous angina develops, are usually soon out of danger, without any treatment. In severe and toxic cases, the physician is practically powerless.

PROPHYLAXIS.—We must remember the fact that when the child has passed its tenth year the chances of its ever contracting the disease are very much lessened. We should, therefore under all circumstances discountenance the opinion so often expressed by the laity, and sometimes even by physicians, that it is well for children to have scarlet fever while they are young, on the ground that otherwise they will probably contract it at a later period of life, when the type of the disease may be more severe. The assertion that the type of the disease is more severe in adults than in children is not corroborated by my experience. Scarlet fever is so uncertain in its course, that it constitutes a very real danger, and every effort should be made to prevent exposure.

Isolation is the only effective prophylactic measure. As soon as there is any suspicion of scarlet fever, and throughout the course of the disease, the child should be strictly isolated with its nurse. Whenever conditions in the home prevent strict isolation, the child should be sent to a hospital.

An upper room should preferably be chosen, having its own bathroom. If possible a second room communicating with the sick room should be set apart for the nurse to change her clothes, and the physician to put on his gown. A disease which renders confinement to the room necessary for weeks demands a room with good ventilation and plentiful sunlight. Therefore, a room on the sunny side of the house, having an open fireplace, should be selected. The room should be free from all cotton or woolen materials except such as

can be destroyed by fire at the end of the disease. The blankets, sheets, towels, and clothes can, of course, be disinfected, but it will save much ultimate trouble to remove the carpet and the curtains and replace them with pieces of old carpet and sheets. The pictures, and, in fact, everything worth preserving, should be removed. The room can be made sufficiently cheerful by means of cheap colored prints and destructible toys to amuse the child.

During the whole course of the disease, the greatest care must be taken to disinfect the linen of both the patient and the nurse. This should be done by soaking it for twenty-four hours in a five per cent solution of carbolic acid, then boiling it for half an hour in water, and finally washing it with soft soap solution. All dishes and utensils which enter the sick room should be thoroughly washed before leaving it, and should be boiled as soon as they are taken down stairs.

Isolation should be continued until at least six weeks have elapsed. At the end of this time, if desquamation has entirely ceased, and if there is no discharge from the nose or throat, the patient may be released. The child is to be thoroughly washed: first in a solution of corrosive sublimate 1 to 10,000, then in water; it is then to be taken to another room to be wiped and put into fresh clothes, which have not been in the scarlet fever room. The mattress is to be tied up in canvas wet with a corrosive sublimate solution 1 to 500, and sent out of the house to be disinfected, if possible by steam. I usually advise the family never to have it brought back again. In place of the mattress it is far better to use old blankets, which, if in sufficient number, are comfortable, and at the end of the sickness can be thoroughly boiled and thus disinfected. The useless articles which have been in the room during the sickness should be burned in the open fireplace.

The room must next be disinfected. If there be paper on the walls, it should be scraped off and immediately burned. The floor should then be washed with a solution of corrosive sublimate 1 to 500, followed by soap water (a mop should be used so as to avoid irritation of the hands). The ceilings, the walls, all the woodwork, and the furniture are to be thoroughly rubbed with bread and then wiped with the corrosive sublimate solution 1 to 500. The room should then be thoroughly vaporized for eight or ten hours with formaldehyde. For every 3500 cubic feet of space, 250 grammes of formaldehyde should be used. This can be done with a vaporizing-lamp, the room first having been tightly sealed.

The room should then be aired for several days. If there are other children in the house, it is well to have the whole room painted, including the ceiling and the floor.

The physician should also bear in mind that the hair, beard, and

clothes are possible means of transmitting the contagium from one patient to another, and it is his manifest duty to the public to protect himself and others from the danger of carrying the contagium. It is best to wear a gown which extends from the neck to the floor, a cap with a flap to protect the hair, rubber over-shoes and rubber gloves. These articles are put on outside the sick-room and are discarded as one leaves the room. They are then left with the nurse and disinfected with corrosive sublimate and are kept outside the sick-room.

TREATMENT.—Scarlet fever is a self-limited disease, and there is no drug or therapeutic measure which can shorten the course of uncomplicated cases.

The scarlet fever patient should be put to bed at once. It is customary to keep even mild uncomplicated cases in bed for three or four weeks. This is done with the idea of avoiding nephritis, which often develops late. I do not believe, however, that nephritis can be avoided by keeping the patient in bed. In cases which run a mild or typical course, when the temperature has been normal for a week, and when the child seems fully convalescent, showing no evidence of complication, the patient may be allowed to get up. All cases in which any fever, rapid pulse, or any signs of complications persist, should be kept in bed till they have been thoroughly convalescent for at least a week, and show no more evidence of any infectious process.

The diet should consist of milk and farinaceous foods. Plenty of water should be given. After three weeks eggs, broths and bread can be added to the diet, and after four weeks, a little meat, and from this time on the child can gradually return to its normal diet. The strict milk diet formerly prescribed is unnecessary, and it has been shown that nephritis is just as apt to develop as when the diet is more liberal. The cereal gruels can do no harm. When there is extreme anorexia, and it is difficult to get the child to take sufficient nourishment, it may be tempted by a more liberal diet.

Isolation, proper nursing, and proper diet being established, it is the duty of the physician to watch carefully for the various symptoms and complications which may require treatment. I have known physicians who actually contented themselves with watching the progress of the efflorescence, which is of no importance whatever in the care of scarlet fever. The important things, which should be examined and noted at each daily visit, are the *temperature*, *the throat*, *the ears*, *the cervical lymphnodes*, and *the heart*. Any rise of temperature, or departure from the normal temperature curve, which is not explained by the condition of the throat, ears, or lymphnodes, should be the signal for a thorough, complete physical examination.

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4. The value of antistreptococcic serum t
5. Streptococcus vaccine therapy in the
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acute nephritis, and the treatment is described in the Division on Diseases of the Kidney.

Stimulation is required in scarlet fever, as in other acute infections, whenever there are any signs of cardiac or vaso-motor weakness, as shown by a rapid pulse. The best stimulant in such cases is caffein-sodium-benzoate. Camphor in oil given subcutaneously may be used in sudden threat of cardiac failure.

During the stage of *desquamation* the application of a simple ointment to the whole body is desirable both for the purpose of softening the disintegrated epithelium and lessening the duration of this stage, and also to prevent the spread of the contagium by means of the loosened scales.

SERUM THERAPY.—The treatment of scarlet fever with antistreptococcic serum has been highly recommended by many authorities, and its value has been the subject of much discussion. It is impossible from the evidence at hand to draw any positive conclusions. This treatment was based on the theory that scarlet fever is caused by a streptococcus, which theory is now considered highly improbable. It is admitted, however, that the severe types and complications are probably caused by a secondary streptococcus infection, and this would explain the reported good results from serum therapy. Antistreptococcic serum should not be used as a routine measure. In mild and typical cases, the disease runs a favorable course anyway, and it is not advisable to run the risk of uncomfortable serum sickness. In severe cases, with membranous angina, running an unfavorable course, or in cases with severe complications, the antistreptococcic serum should be tried.

TABLE 42

Percentage of Mortality of Scarlet Fever in the Annakinderspital in Vienna from January 1, 1893, to July 1, 1904

YEAR	PER CENT	YEAR	PER CENT
1893	25.2	1899	16.4
1894	21.0	1900	12.4
1895	20.0	1901	8.9 serum
1896	17.6	1902	6.7
1897	11.1	1903	8.5
1898	12.3	1904 to July, 1904	7.6

PROBLEMS AND RESEARCH.—The one great problem in scarlet fever is that of the specific etiology. The diphtheroid bacillus of Mallory is promising, and we are now awaiting the results of its further investigation.

The following subjects in connection with scarlet fever have recently figured largely in the literature of medical research:

1. The diagnostic significance of the leucocytic inclusion bodies found in scarlet fever.

2. The streptococcus in connection with etiology.
3. The experimental transmission of scarlet fever to monkeys.
4. The value of antistreptococcic serum therapy.
5. Streptococcus vaccine therapy in the production of artificial immunity.

The inclusion bodies have been finally shown not to be characteristic.

There is still considerable difference of opinion as to the rôle of the streptococcus. A number of European authorities still believe it to be the cause of the disease. Kretschmer advances the ingenious hypothesis that scarlet fever represents an anaphylactic phenomenon due to a hypersusceptibility to the streptococcus. The arguments in favor of the streptococcus theory, and against a specific etiology, are, the varying incubation period, the occurrence of sporadic cases apparently without exposure, the good results of serum therapy, and the fact that certain observers have claimed to have found specific antibodies for the scarlet fever streptococcus, which they think differs in its biological reactions from other streptococci. Other observers, however, have failed to confirm the experimental evidence in favor of the streptococcus, and the weight of the evidence inclines toward the view that the streptococcus is a secondary invader.

There has been much difference of opinion as to whether scarlet fever has been successfully transmitted to monkeys. There have been many failures, and the reported successes have been questioned, on the ground that monkeys frequently have transient erythematous eruptions, and bran-like desquamation. It seems probable from the evidence, however, that true scarlet fever has actually been transmitted to the higher anthropoid apes, although the transmission is difficult. Lower monkeys appear to be insusceptible. This question of experimental scarlet fever is of extreme importance, as every organism which shall in the future appear as a claimant to the title of the specific cause of scarlet fever, will stand or fall by the results of animal experiment. The organism must be isolated in pure culture, and scarlet fever must be produced experimentally by inoculation with the culture, before it can be accepted as the proven cause.

The question of serum therapy has already been discussed. In a disease of which the severity, course, and fatality is so varying as in scarlet fever, it will always be extremely difficult to prove the value of any specific treatment. Serum therapy has proved unable to change or interrupt the course of typical cases.

Vaccine therapy has been employed in scarlet fever, and the usual "good results," based on utterly inconclusive evidence, have been reported in scarlet fever just as they have in every disease in which vaccine therapy has been used. There is no evidence of any value in favor of vaccines as a means of treatment. The question of vac-

cines in prophylaxis has been discussed. The e favor is mainly of the character of percentage figur hospital wards, one with vaccine prophylaxis, th Owing to the irregularities of the transmission of s evidence is of no value whatever, and it will be im the value of vaccine prophylaxis without experim of children, or more certain experimental productio in animals.

MEASLES

Measles is a specific infectious disease, characteri them, and by a characteristic affection of the muc the mouth and respiratory tract, and of the conjur commonest of all the specific infections. Measles is limited, and one attack usually confers an immunit the individual throughout life.

ETIOLOGY. THE MICROÖRGANISM.—The organi measles is, in spite of much research, still unknow demonstrated that the virus is contained in the s from the nose and mouth, and also in the blood. It demonstrated that the cause of measles belongs in t able virus, as it can pass through a Berkefeld filter teristics of the virus have been brought to light by production of measles in monkeys.

TRANSMISSION.—The virus of measles is an air-b It possesses the power of floating in the air, and cons is transmitted from one individual to another witho It is probable that the virus is contained in the moist in coughing and sneezing. A susceptible child can t the disease by simply approaching a measles patien a room in which there is a measles patient. It is cons that the transmission of measles is much easier tha fever, which requires actual contact, or an infecte as a carrier.

On the other hand, the virus of measles is extra lived outside the body. It must be transmitted d patient to the exposed child. If objects are contam quickly dies. Consequently indirect transmission, t of the virus on contaminated objects, or by health extremely rare, that it is practically negligible. A leaving the same bed with a measles patient, and go bed with another person, can carry the disease. circumstances as this, indirect transmission does n are no recorded instances of a physician carrying tagium from one house to another.

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rather increase, during this *remission of t*
an important point to remember, as the ch
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physician, who, because the child appears s
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ment of the larynx. The nose begins to run
red and swollen, as is the caruncle at the in
The discharge from the eyes is usually watery,

The whole picture during the prodromal sta
a severe catarrhal cold, or an attack of *grippe*,
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ance of Koplik's spots, or of the measles *efflores*
membrane of the mouth. These signs are often
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KOPLIK'S SPOTS.—If the mucous membrane of t
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days before the appearance of the efflorescence. *Th*
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appear on the mucous membrane lining *the chee*
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white points, no larger than the finest grains of sa
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of the mouth, that the light red spots do not stand
else, especially in anemic children, there is no redde
only the minute white points are seen.

The number of Koplik's spots is very variable. So
are only two or three, and the white points are so sm
are found with the greatest difficulty. They can onl

due to the failure to distinguish between measles and rubella. Second attacks are commoner in scarlet fever than in measles.

EPIDEMIOLOGY.—As a result of the predisposition of the human race toward measles, the disease tends to occur in epidemics, which spread rapidly, soon reach their height, and quickly end, there being no more susceptible children within reach. In small and comparatively isolated localities, there may be no measles for a number of years, until one case introduces the disease, and causes a widespread epidemic. In the large towns, epidemics occur every three or four years, with freedom from measles in between. In the big cities there are always cases of measles, but widespread epidemics are not seen. There are little epidemics at times in various parts of the city.

Measles is more common in the autumn, winter, and spring than in the summer. This is probably because the schools are open at this time.

PATHOLOGICAL ANATOMY.—Beyond the morbid conditions which appear on the *skin* and on the mucous membrane of the throat, there is no especial characteristic pathology of measles. The chief lesions found post-mortem in fatal cases are those of the complications which caused death. The most common fatal complication is bronchopneumonia. Otitis media is frequently found.

In cases coming to autopsy during the stage of efflorescence, the pathological changes in the skin are clearly marked. The capillaries of the papillae are dilated and there is round cell infiltration about the walls of the vessels, about the sweat and sebaceous glands, and about the hair follicles.

The mucous membrane of the respiratory tract shows a catarrhal inflammation, which has no peculiarities characteristic of measles.

SYMPTOMS. PRODROMAL STAGE.—This is the period between the beginning of the symptoms and the first appearance of the efflorescence on the skin. The length of the prodromal stage varies from one to five days. In the great majority of cases it is two or three days, sometimes four days, rarely one or five days.

The invasion is characterized by severe catarrhal conditions affecting the nose (coryza), the eyes (lachrymation), and the throat and upper-air passages (cough). In the first twenty-four hours the temperature rises to 101° or 102° F., and often to 104° F. The height of the temperature on the first evening is a fair indication as to the severity of the coming disease. Thus a temperature of 105° F. indicates a severe case. An important point to be noticed regarding the prodromal symptoms is that after the first twenty-four hours there is in a large number of cases a remission in the temperature, which goes down, perhaps to 99.5° or 98.6° F. and remains down for about twenty-four hours, when it again rises. The *cough*, *coryza*, and *lachry-*

mation, which appear early in the prodromal stage, do not abate, but rather increase, during this remission of the temperature. This is an important point to remember, as the child who seems quite sick and loses its appetite while the temperature is high during the invasion of the disease, seems brighter and has a return of appetite on the second day when the temperature is lower. This peculiarity of the prodromal stage is often misleading both to the parents and to the physician, who, because the child appears so much better, are led to believe that one of the infectious diseases is not developing. In infants and young children, the prodromal stage may begin with a convulsion, but this is unusual, and if it occurs it is not, as a rule, particularly severe, and does not necessarily make the prognosis more grave. Headache in the prodromal stage is quite frequent; vomiting is rather rare. The tongue is usually furred, and the mucous membrane of the throat shows at first only the appearance of an ordinary catarrhal inflammation. The cough is dry, and often severe and racking, and sometimes hoarse and ringing, showing the involvement of the larynx. The nose begins to run. The conjunctiva is red and swollen, as is the caruncle at the inner angle of the eye. The discharge from the eyes is usually watery, sometimes purulent.

The whole picture during the prodromal stage resembles that of a severe catarrhal cold, or an attack of grippe, and there is nothing to show that it represents the beginning of measles, until the appearance of Koplik's spots, or of the measles efflorescence on the mucous membrane of the mouth. These signs are often overlooked, so that the true diagnosis is not suspected until the appearance of the efflorescence upon the skin.

KOPLIK'S SPOTS.—If the mucous membrane of the mouth be carefully and systematically examined during the prodromal stage, it is often possible to make a positive diagnosis of measles two or three days before the appearance of the efflorescence. This is based on the finding of Koplik's spots, which are pathognomonic of measles. They appear on the mucous membrane lining the cheeks, usually near the molar teeth, and consist of light red spots about the size of the head of a pin, in the center of which are minute white or bluish-white points, no larger than the finest grains of sand. The white points consist of fatty degenerated epithelium and detritus. In many cases there is so much catarrhal reddening of the mucous membrane of the mouth, that the light red spots do not stand out clearly, or else, especially in anemic children, there is no reddened base, and only the minute white points are seen.

The number of Koplik's spots is very variable. Sometimes there are only two or three, and the white points are so small, that they are found with the greatest difficulty. They can only be satisfac-

PLATE IX.

Fig. 1.

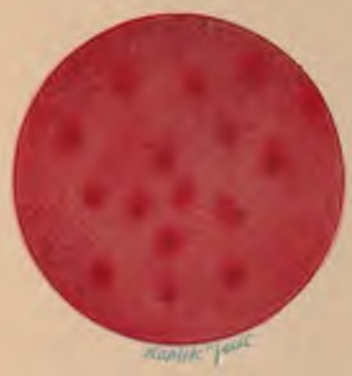

Fig. 2.

Fig. 3.

Fig. 4.

The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in line. Minute *yellow points* are surrounded by a red area. Always discrete.—*Med. News*, June 3, 1899.

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discomfort and constitutional disturb
and the catarrhal symptoms, remain
On the day before the appearance of t
toms reach their maximum severity.

THE EFFLORESCENCE.—The physician
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and finally to the forearms and legs, so tha
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above the level of the surrounding normal skin

The color of the spots is at first light red, b
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In the beginning, when the spots are small
scattered, but as they grow larger, new spots keep
In certain parts of the body, particularly on the
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The fading of the rash is rapid, lasting about
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DESQUAMATION.—A light desquamation begins w
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is thus distinguished from the large lamellar flakes oc

torily seen by daylight, and require a strong light, good eyes, and close observation. The child should be held close to a window, in the position used for the examination of the throat, and a spatula or handle of a teaspoon should be inserted at the corner of the mouth, and the cheek retracted from the teeth. At other times the spots are very clear and numerous, covering thickly the lining of the cheek, and often extending onto the mucous membrane of the lower lip. Their number increases up to the time of the appearance of the skin eruption, and they rapidly disappear on the first or second day of the stage of the efflorescence.

Koplik's spots appear at a variable time before the efflorescence on the skin, *most commonly two days before.* They may in rare cases appear as long as four days before the rash, while sometimes they do not appear until simultaneously with the rash. They may not be seen at all in very young or atrophied infants.

Koplik's spots are a sign of extreme importance in the diagnosis of measles. When present, they are diagnostic, as they are seen in no other disease. The difficulty lies in the diagnosis of the Koplik's spots themselves. They are easily distinguished from thrush, and are much smaller than the lesions of aphthous stomatitis.

THE ERUPTION IN THE MOUTH.—The rash of measles begins on the mucous membrane of the mouth toward the end of the prodromal stage. This is to be distinguished from the Koplik's spots. The eruption in the mouth is in every way similar to the later eruption on the skin. It appears one or two days before the efflorescence on the skin, usually later than the first appearance of the Koplik's spots. These lesions, which are especially pronounced on the soft and the hard palate, are represented by papules or macules of a dark red and later purplish-red color, of different sizes, and considerably larger than the punctate macules which were described in connection with the throat in scarlet fever. These papules in certain cases are arranged crescentically, and may sometimes be found to have coalesced in some parts of the fauces. The mucous membrane between the lesions is comparatively normal in color, although there may be a slight hyperemia of the entire throat. This hyperemia, however, is not nearly so intense as is seen in the throat in scarlet fever. The exanthem in the throat is at its maximum with the beginning of the cutaneous efflorescence, and may take as long as three or four days to disappear. It is often not clear and characteristic, and is not so important in the early diagnosis of measles as are the Koplik's spots.

END OF THE PRODROMAL STAGE.—After the peculiar remission of the fever, which most commonly occurs on the second day of the disease, the temperature rises again, and remains high, or continues

to creep higher, during the remainder of the prodromal stage. The discomfort and constitutional disturbance accompanying the fever, and the catarrhal symptoms, remain severe, or increase in severity. On the day before the appearance of the efflorescence, all the symptoms reach their maximum severity.

THE EFFLORESCENCE.—The physician who is watching the development of a case of measles, now looks for the first appearance of the rash, which begins as little red spots, which usually first appear behind and in front of the ears, on the face, or on the neck. From these situations the efflorescence spreads rapidly, first to the back, then over the entire trunk, then to the upper arms, then to the thighs. and finally to the forearms and legs, so that the entire body becomes covered. It usually takes two days for the entire body to become involved, and another day for the rash to reach its greatest intensity. At this time, however, the rash has already begun to fade on the parts of the body where it first appeared, so that for the rash to reach its greatest intensity in any part, and then begin to fade, takes about two days. The rash fades in the order of its appearance.

The single spot of measles begins as a tiny macule, which increases in size. As the spot grows, a prominence forms in the middle, which becomes palpable as a slightly raised papule, and corresponds to a gland or hair follicle. The spot quickly becomes of a size varying from a pin-head to a pea, and as it grows it becomes irregular in outline. The fully developed spot is slightly but entirely raised above the level of the surrounding normal skin.

The color of the spots is at first light red, but as the papules develop, the color becomes a bright, fiery red.

In the beginning, when the spots are. small, they are sparsely scattered, but as they grow larger, new spots keep coming in between. In certain parts of the body, particularly on the face and back, the rash becomes confluent, forming large red areas, with only small islands of normal skin.

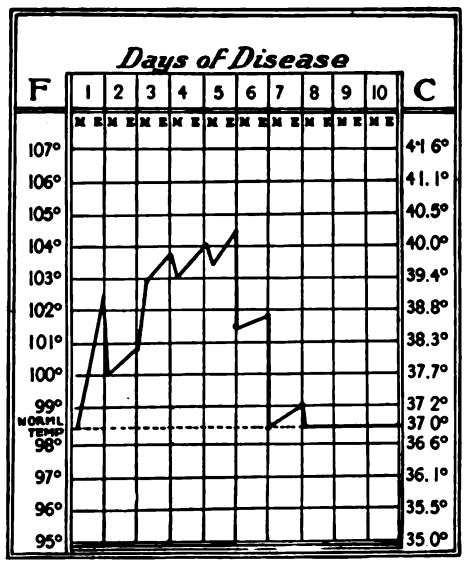
The fading of the rash is rapid, lasting about two days, so that the entire period of the exanthem is from four to five days. Until the rash begins to fade, the color disappears entirely on pressure, but after fading has begun, a certain amount of color remains on pressure, as some blood pigment has escaped from the vessels. This brownish pigmentation becomes more conspicuous after the rash fades, especially in full-blooded children, and may remain visible for from ten to twenty days.

DESQUAMATION.—A light desquamation begins with the disappearance of the efflorescence. The desquamation is usually furfuraceous in character, that is, the epithelium is cast off in fine flakes, and is thus distinguished from the large lamellar flakes occurring during

the period of desquamation in scarlet fever. The desquamation begins in the order in which the efflorescence came out, namely, first on the face and later on the chest. The furfuraceous character of the desquamation is especially noticeable on the sides of the nose. The desquamation on the body is so slight and fine as to be hardly noticeable. Older children sometimes complain of itching, especially of the legs, during the stage of desquamation.

THE TEMPERATURE CURVE.—In typical cases of measles, the temperature curve is often quite characteristic. The peculiar fall of temperature on the second day of the prodromal stage, one or two days before the appearance of the efflorescence, has already been

CHART 9



Typical measles

mentioned. The temperature reaches its maximum on the second day of the efflorescence, remains high another day until the full development of the eruption, and then falls abruptly by a sharp or broken crisis. If the temperature fails to fall before the fourth or fifth day after the appearance of the exanthem, it is strongly suspicious of a complication. The highest point reached by the fever is usually at least 103° F., and may be as high as 105° F. Deviations from the typical temperature curve are common in cases otherwise typical, especially a fall of temperature by lysis beginning at the height of the exanthem.

ACCOMPANYING SYMPTOMS.—The general condition is much affected both in the stages of invasion and of efflorescence. Severe cough, headache, and photophobia are the most troublesome symptoms.

Delirium, especially at night, is often present. The appetite is almost entirely lost.

The conjunctivitis increases in the stage of efflorescence. The secretion may become purulent, and the eyelids are often stuck together in the morning. The rhinitis also increases, and the swollen mucous membrane and purulent secretion often render breathing difficult.

The tongue is dry, and heavily coated. The throat is somewhat reddened, and the exanthem may still be visible on the mucous membrane after its appearance on the skin.

The cough often improves with the beginning of the eruption, as does the laryngitis.

The lungs in the majority of typical cases are normal to auscultation and percussion. Often, however, medium and coarse moist râles are heard.

The heart and circulatory apparatus show little evidence of special disturbance. The pulse is rapid, corresponding to the fever, and in young children may reach 160 to 180 without becoming poor in quality.

The urine in mild cases is concentrated, and slightly diminished in quantity. With high fever there is usually a small amount of albumin, and a few casts and red blood corpuscles. A true nephritis is very rare. During the stage of efflorescence the diazo reaction is present.

During the period of efflorescence, when the temperature is still raised and the eruption is at its maximum, it is usual to have, in addition to the symptoms of cough, coryza, and lachrymation, a slight disturbance of the intestines, represented by small, frequent, loose discharges, apparently arising from irritation of the rectum and descending colon. This condition is seldom a serious one, and no especial attention need be paid to it unless it should continue for some days, or after the maximum of the temperature and efflorescence has been passed for a day or two.

The lymphnodes are slightly enlarged. The spleen is not palpable.

THE BLOOD.—The blood picture in measles is very characteristic, and recent researches have shown that the changes begin during the stage of incubation, so that a change in the blood finding is really the first symptom of the disease. In the blood of normal children the lymphocytes predominate over the neutrophiles. During the stage of incubation there begins a relative decrease in lymphocytes, and increase in neutrophiles, which begins on the average six or seven days before the eruption. This is caused by a diminution in the absolute number of lymphocytes, which becomes marked enough to cause an actual *leukopenia*, which may appear anywhere from eight

days before the eruption to simultaneously with the rash, but is seen on the average four and one-half days before. The neutrophiles come to predominate in the differential count, and this displacement of the blood picture appears on the average four days before the beginning of the rash. The eosinophiles diminish during the incubation period, and are absent during the stage of efflorescence. The white count reaches its lowest point at the height of the exanthem. With the fall of temperature, the lymphocytes increase, the eosinophiles reappear, and the polynuclear neutrophiles may, after defervescence, be relatively below the normal.

These changes are considered by many writers of the highest importance in the early recognition of measles, as they usually occur several days before the appearance of Koplik's spots. If they were as constant and typical as described, it would be possible by a daily examination of the blood of a child who had been exposed to measles, to recognize the approach of the disease during the stage of incubation, and this would be very useful in prophylaxis, especially in hospitals. It is often possible to make an early diagnosis of measles in this way, but I have not found the changes as constant and typical as described.

The most useful feature of the blood examination in measles is the leukopenia. A leukocytosis always points toward a complication.

CONVALESCENCE.—With the fall in the temperature, all the symptoms disappear rapidly. In one or two days the child feels well. The cough lessens, and ceases in a few days. The conjunctivitis and rhinitis clear up more slowly, but usually a week after the appearance of the efflorescence the child is fully convalescent, and by another week or ten days is well, the only trace of the disease being the slight pigmentation of the skin.

VARIATIONS IN TYPE OF MEASLES.—Measles during epidemics and in sporadic cases varies much in its type, and presents great differences in its prodromal stage, in its dermal lesions, in its desquamation, and in its entire course. Through a lack of appreciation of this fact, the diagnosis of other diseases, such as rubella and various forms of erythema, is continually being made, when, in fact, the disease represents one of the more unusual forms of measles. If these variations in measles were better understood, we should not find the disease rubella so often diagnosed.

At times, the duration of the stage of incubation of measles varies considerably. Instead of the usual prodromal stage, certain cases during epidemics of undoubted measles show few, if any, prodromal symptoms.

In addition to the usual catarrhal symptoms, in some cases there are vomiting and sore throat. Again, instead of a considerable ele-

vation, the temperature may be scarcely above the normal degree. Epistaxis of a mild form is sometimes met with.

The efflorescence, which in typical cases usually consists of papules, may vary so as to simulate closely a common erythema, constituting the form called *laevis*, or may closely simulate a papular erythema. Again, the efflorescence may in certain cases be represented by minute vesicles or milia, characterizing the form called *miliarius*. Any of these forms may be confluent, but, as a rule, only upon the face.

There is another form of efflorescence which occurs in measles, which is rare, and of a more serious nature than the common benign forms which are met with ordinarily. This is called the *hemorrhagic* or *malignant* form, and is represented on the skin by small capillary hemorrhages. It is often rapidly fatal, and at times appears to be part of a general hemorrhagic diathesis or symptomatic purpura, characterized by epistaxis, hematuria, and hemorrhages from other localities. The temperature in these cases is not typical, as it does not remit in the prodromal stage, thus depriving us of an important means of diagnosis; but a doubt as to the nature of the disease does not last long, as the other symptoms soon become prominent. The more prolonged the course of this form, the better the prognosis, for if fatal it is usually quickly so. It may be complicated by a malignant bronchopneumonia.

The efflorescence, besides differing in its form, may vary to a great degree in its intensity. Thus, we may have every grade of papule or macule, from the smallest to the largest, and varying from a dark purplish to a light pink color. In like manner, although the arrangement of the efflorescence, especially on the chest, is somewhat crescentic, yet during epidemics of undoubted measles this crescentic shape is often absent. Instead of the efflorescence first appearing on the face and then extending to the thorax and extremities, we may find in undoubted measles that it begins first on the chest or some other part of the body; or the efflorescence may appear on the face and thorax simultaneously. We may also find that in certain cases the efflorescence appears first on the abdomen, or on the thighs, and yet the presence of other typical and undoubted cases of measles in the vicinity or in the same house assures us that we are dealing with the same disease. The efflorescence, instead of lasting for a number of days, may be evanescent, and may subside within twenty-four hours.

The desquamation of measles is of so light a grade that it is not surprising that in some cases no desquamation whatever is detected. Cases in which desquamation occurs without efflorescence are highly improbable, although such have been reported.

During certain epidemics of undoubted measles, cases have not

infrequently been noted in which the post-aural a
were markedly enlarged.

There is a form of measles, called the *recurrent*
relapsing fever. The main characteristic of this
fever. The temperature will sometimes be raised f
will then become normal for seven or eight days,
again with a recurrence of the symptoms. This
form, and one which needs merely to be menti
accompanied by the general symptoms connected w
and bronchi which are met with in the typical for

Relapses occur in measles, but they are uncom

In reviewing the pictures which have been giv
tions, it must be evident that, although in the la
cases measles runs so typical a course that the
easily made, yet such great variations in type a
to occur that we should be extremely careful not to
of certain other diseases, such as rubella, except u
cumstances. This is important, because we know
demics of well-marked measles all these great variat
tion, prodromata, efflorescence, desquamation, and
not infrequently arise.

COMPLICATIONS AND SEQUELAE.—There
ber of complications and sequelae which may occu
measles. The most common of the serious ones a
tuberculosis.

BRONCHOPNEUMONIA.—The bronchitis which i
accompaniment of measles sometimes appears in a
attacking the smaller bronchi as well as those of
may result in a bronchopneumonia, which is mu
as a complication of measles than is lobar pneumon
pneumonia does not, however, appear to be mo
arises as a complication of measles than when it
from that disease. Bronchopneumonia as a compl
may occur very early in the course of the disease
stage of invasion; but it occurs most commonly t
the second week.

When, therefore, after the efflorescence has fad
has subsided, the temperature again rises without
inflammation in the throat, ears, or glands, we sho
bronchopneumonia is developing. The addition
quickened respiration and movement of the alae
still more probable the supposition that this comp
even though no consolidation is detected in the l

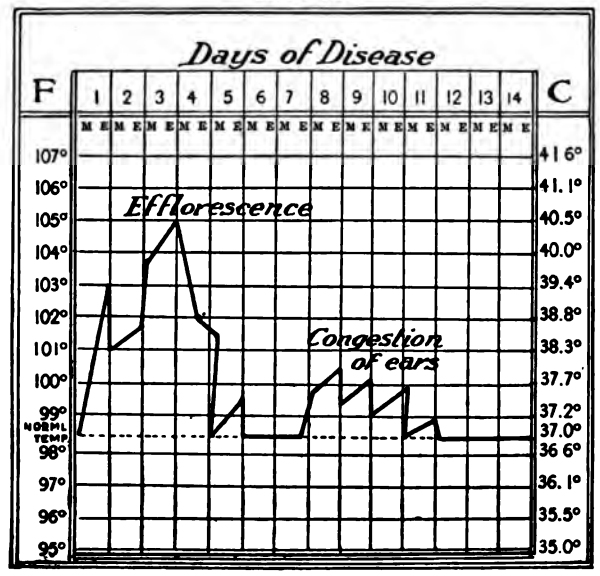
The congestion of the larger bronchi, which app

a part of the measles, may become subacute and chronic, instead of, as is usually the case, passing off soon after the maximum of the temperature and efflorescence.

Pleuritis may occur in the course of measles, but is not so common as pneumonia.

OTITIS MEDIA.—This is a common complication of measles. It shows no special characteristics differing from the otitis media occurring as a complication of other acute infectious diseases.

CHART 10



Measles with congestion of membranae tympanorum during stage of efflorescence

EYE.—In addition to the conjunctivitis which is a common accompaniment of measles, and which, as a rule, requires no treatment beyond the protection of the eyes from light, the inflammatory process may extend to the deeper tissues of the eye and cause other grave lesions, such as *blennorrhagic conjunctivitis*, *keratitis*, and *iritis*. These complications should be treated at once by a skilled ophthalmologist.

THYROID GLAND.—In a very few cases an acute swelling of the thyroid gland may take place during the course of measles. This swelling of the thyroid gland may even cause marked dyspnea by pressure, but it usually disappears in two or three days. In some cases, however, a formation of pus has taken place, followed by destruction of a part of the gland. In intractable cases of this kind it has been found that the external application of iodine is useful.

LYMPHNODES.—Enlarged cervical lymphnodes are not so common

in measles as in scarlet fever, but they may occur, a
prove serious from the occurrence of suppuration.

TUBERCULOSIS.—The most common sequela of mea
culosis. This means that in a child already infected
culosis in the primary stage, measles markedly predis
the development of secondary manifestations. These r
form of general miliary tuberculosis, or secondary tu
some organ or part of the body. Tubercular disease
seems to show a special liability to follow attacks of
is noticeable that when a patient with a tubercular j
attack of measles, the process in the joint is apt to bec
rarily more active, and the prognosis is comparatively
The organ which in measles is most commonly affected
losis is the lung, and the most common form of tubercu
lung is a tubercular bronchopneumonia.

PARALYSIS.—Another sequela, although a rare one, i
Cases thus complicated have shown mostly a paraplegia, a
ing to Osler, frequently can be classified as post-febrile p
although it is possible that some of them may be due t
ascending myelitis.

NOMA.—A very rare sequela of measles is the disease n
crum oris).

Among the rarer complications of measles are *empy*
carditis, *pericarditis*, and *membranous laryngitis*. *Catarrha*
and *tracheitis* are not infrequent accompaniments of
stage of measles. Edema of the glottis is rare, but has b
to occur.

The irritation of the intestine, occurring commonly
height of the efflorescence and temperature, sometime
much more severe from the development of *colitis* as a co

DIAGNOSIS.—In order to understand how difficult it
is to diagnose measles, we must recognize that it is one o
variable diseases with which we have to deal. During
of undoubted measles, cases arise which differ materiall
disease as it appears in its typical form, yet these cases, by
the typical form in other individuals, prove that they are
by the same contagium. In like manner, certain epidem
characterized by irregular forms of the disease, and, as tr
can rarely occur more than once in the same individual,
nition of a sporadic case is often difficult. As in other
the skin, we should recognize measles not by any particu
lesion, but by the peculiarities of the prodromal symp
general course and location of the efflorescence, the time o
mum of the efflorescence, and the character of the des

Thus, a prodromal stage of three or four days, characterized by catarrhal symptoms of the eyes, nose, and upper air-passages, by the presence of Koplik's spots in a large majority of cases, and a papular efflorescence appearing first on the face, differentiates the disease from the various conditions for which it might be mistaken.

During the prodromal stage, a positive diagnosis depends usually upon the finding of Koplik's spots. Until these appear, the diagnosis must always be in doubt. The next diagnostic sign to develop is the eruption in the mouth, and ultimately the diagnosis rests on the recognition of the character of the eruption on the skin.

When the exanthem of measles is markedly papular, one might think of the possibility of *variola*. After one or two days, however, the typical vesicular character of the smallpox eruption develops. Furthermore, in variola the temperature falls with the appearance of the eruption, while in measles it rises.

A very markedly confluent rash in measles might suggest *scarlet fever*, but there are always parts of the body where the measles rash is discrete, and these bear no resemblance to scarlet fever. In scarlet fever the absence of catarrhal symptoms, of Koplik's spots, and of a long prodromal stage, and the presence of angina, strawberry tongue and leukocytosis, makes the differentiation usually easy.

The efflorescence of *rubella* is similar to that of measles, but is usually paler, more sparsely scattered, and the spots are smaller. The absence of prodromal symptoms, Koplik's spots, high fever, and marked constitutional disturbance, is the characteristic feature of rubella.

In various infections, and also in certain toxemias not due to infection, such as serum sickness, or after the taking of certain drugs, a *toxic erythema* may appear on the skin, which may resemble the efflorescence of measles. In most of these cases, however, the rash is polymorphous or changing in character, there are areas where it does not resemble measles, and it is usually not distributed all over the body. The catarrhal symptoms, Koplik's spots, and blood findings of measles are absent.

In rare cases the *syphilitic roseola* has been mistaken for measles, in spite of the absence of the other characteristic symptoms. I have even seen a case of *typhoid fever* with a very profuse crop of rose spots, diagnosed as measles.

PROGNOSIS.—The prognosis of measles is good. The disease lacks the uncertainties of scarlet fever, and most children recover completely. The only dangers are from the development of bronchopneumonia, or of one of the fatal secondary forms of tuberculosis. Both of these dangerous complications appear most often in infants or very young children. In children over three years of age, they

are comparatively rare. Bronchopneumonia is seen most often in feeble or rachitic infants. In such cases, when the disease begins with a severe bronchitis, and when the eruption is not well marked, the prognosis is very uncertain.

PROPHYLAXIS.—It is often said that as every individual must have measles, it is useless to attempt to protect healthy children, and that it is best to allow them to become exposed while well, in order to protect them from acquiring the disease at some future time, when they may be in poor health, or when measles may be more inconvenient. I do not at all agree as to the desirability of such a plan. One can never be sure, no matter how well a child seems, that the disease will run a mild course, and if a complication does develop, the physician who is responsible for the health of the child will have much to reproach himself for. It must also be remembered that measles is mainly dangerous in young children. It is consequently especially important to protect the very young, or the weak and sickly. It is not true that measles runs a more severe course in adults.

Whenever a child becomes sick with any symptoms compatible with measles, it should be rigidly isolated. If this is done, there is a possibility that the other children in the household may escape, especially the younger ones. If the diagnosis of measles is confirmed, the isolation should be continued, the patient and its nurse being treated in the same way as in scarlet fever, except that rigid isolation of the nurse is not necessary. She may go in and out, but it is better for her to be kept away from the other children.

The rigid disinfection of all clothing and other articles used in the sick room, which is practiced in scarlet fever and diphtheria, is not necessary in measles. On the other hand, other children should not be allowed near the sick room. Whenever possible, in private practice, I send them out of the house.

When measles occurs in kindergartens, day nurseries, or any place where *young* children congregate, the institutions should be closed. It is not, however, necessary to close the common day schools on account of measles, as epidemics cannot be checked in this way. In children's hospitals, the ward in which measles occurs should admit no new patient until fourteen days after the appearance of the last case.

The measles patient should be kept in isolation until fully convalescent, usually two weeks at least. After release, disinfection of the sick room is unnecessary. It should simply be given a good airing.

TREATMENT.—The treatment of measles is essentially symptomatic. There is no known means of producing immunity to the

disease or of shortening its course. It is a self-limited disease, and the treatment should be directed towards the protection of the organs which are most likely to be attacked by complications. Bearing in mind that the eyes, the nose, and the throat are affected in the prodromal stage, that later the skin is in a very sensitive condition, and that the lung is frequently the seat of some complication, we should direct our treatment especially to the care of these organs.

The child should be placed in a room kept at an equable temperature, 68° to 70° F., and well ventilated. The room should be darkened, and the eyes should be protected from light during the whole course of the disease. Unless this precaution is taken, the eyes are often seriously affected for many months after the measles itself has disappeared. The child should be kept in bed until the temperature has been normal for a few days, the efflorescence has faded entirely, and the desquamation has almost ceased.

The diet during the period of the high temperature should be soup, milk, and bread. Later, when the temperature is normal and desquamation has begun, the child can gradually have its diet increased, until by the third week from the beginning of the attack it is having its usual food.

The cough, though very troublesome at times, does not, as a rule, require any special treatment, as it will in most cases pass off of itself in a few days. While it continues, it can be treated with some simple medication, such as camphorated tincture of opium in cold water in doses of 5 to 10 minims.

For the irritation of the nose, some simple refined oil, such as oleum petrolatum album dropped into the nose with a medicine-dropper is useful. Atomization should be avoided because of the danger of starting an acute inflammation of the middle ear through the Eustachian tubes. During the invasion of the disease, however, these catarrhal symptoms are exceedingly difficult to control by any treatment whatever.

As at times there is great irritation of the skin during the period of efflorescence, a powder should be applied thickly to the entire body and limbs. In place of the powder, some simple ointment such as petrolatum, may prove to be more soothing.

As a rule, the child should be kept in an equable temperature for at least three weeks, and at the end of that time, if the desquamation has ceased, it may be allowed to go out of its room, and out of the house a few days later in pleasant weather. For several months, however, it should be carefully protected from sudden changes of atmosphere, as the catarrh of the air-passages is so likely to leave them in an extremely sensitive condition that a very slight irritation may cause a recurrence.

PROBLEMS AND RESEARCH.—The central probl
is that of the etiology. Every effort to discover the s
örganism which undoubtedly causes the disease, has
results of blood cultures have been uniformly negative.

The most significant step in advance taken by res
on measles, has been the successful transmission of t
monkeys, by inoculating their noses and throats with
of a measles patient. Through this means it has be
that the virus is present in these secretions as well as
It has further been demonstrated that the virus can
a Berkefeld filter, and that the infectivity of the vir
as defervescence is approached, and disappears in conv
has further been demonstrated that the products of
are not infectious.

This production of experimental measles opens the
further study of many characteristics of the virus.
that these researches will eventually be crowned by t
liant result as in poliomyelitis, and that a way will
cultivate the virus artificially.

The order of appearance of the rash bears a connec
tain characteristics of the blood supply. It has been s
the virus may agglutinate in the capillaries.

Herrman, taking advantage of the immunity of infa
protective inoculations in infants under five months. T
promising but not conclusive.

RUBELLA

(German Measles; Rötheln)

Rubella, or German measles, is a specific infectious
acterized by an efflorescence resembling measles but of
and by the absence of catarrhal symptoms, high fever
constitutional disturbance. The disease is contagiou
chiefly in epidemics.

There has been in the past considerable doubt as t
clinical picture of rubella represents a disease *sui ge*
authorities believe it to represent a mild and atypical fo
This doubt as to the separate identity of rubella coul
exist in the mind of anyone who had ever seen an ep
evidence in favor of the separate existence of rubella li
that in epidemics, the clinical picture in children who
disease by known exposure always retains its particu
istics, and never resembles typical measles. Furthermo
tical evidence of most epidemics has shown that rubel

seen more often in children who have already had measles, than in children who have not.

ETIOLOGY. THE MICROÖRGANISM.—The organism causing rubella has not been discovered. Practically nothing is known of the characteristics of the virus.

TRANSMISSION.—Rubella is transmitted from one individual to another in much the same way as measles. The contagium is only exceptionally carried by a healthy person, or on objects. The virus is short-lived outside the body.

Sporadic cases are rare. The disease occurs chiefly in local epidemics, which do not affect a large percentage of the population. Epidemics occur at rather long irregular intervals, which may amount to many years. They are seen most often in the spring, and usually last several months.

The disease is contagious from the time of the first symptom, the infectivity reaching its height at the time of the exanthem, and ceasing with its disappearance. The portal of entry is unknown.

PREDISPOSITION.—The susceptibility to rubella is much less than to measles. When an epidemic occurs in a children's institution so that all are exposed, about half contract the disease. Outside of such institutions the incidence is much less.

The most susceptible are children from three to twelve years of age, especially school children. Whether this is due to a special susceptibility in childhood, or to the fact that the conditions of life in childhood favor the spreading of any contagious disease, is not known. Older infants are frequently infected, and the disease is fairly common in young adults. Immunity to measles gained by a previous attack does not protect against rubella. More than one attack is rare.

INCUBATION.—The stage of incubation in German measles is long, from *seventeen to twenty-one days*. Rarely is it as short as fourteen days.

SYMPTOMS. PRODROMAL STAGE.—In the majority of cases there are no prodromal symptoms in rubella, the first thing to attract attention being the efflorescence. Sometimes for from *half to one day* before the appearance of the rash there is malaise, fretfulness, slight sore throat, slight fever, and the symptoms of a mild cold in the head. These symptoms are not severe enough to be especially noted, and do not suggest a severe process of any kind, and the physician usually first learns of them through his inquiries made after the appearance of the rash.

THE EFFLORESCENCE.—The exanthem of rubella appears first on the face, or about the ears, and spreads very rapidly, so that usually

in half a day the skin of the entire body is involved. The spots at first are about the size of the head of a small pin, and grow somewhat larger, but do not reach the size of the measles papules. As compared with measles the spots are not only somewhat smaller, but also paler, not so much raised above the normal surface, and more regular in outline. Their shape is round or oval. In further contradistinction to the rash of measles, that of rubella is more uniform in the size of the papules, more sparsely distributed, more evenly distributed, and does not become confluent, except sometimes on the cheeks. In the beginning the entire face sometimes seems reddened.

The rash is often so much paler than that of measles, that it may be overlooked entirely. The color disappears on pressure, except that when fading it leaves behind a slight pigmentation which is less than that of measles.

The efflorescence of rubella is usually most marked on the face, back, and extensor surfaces of the extremities, or on parts of the body where the clothing presses. It is seldom seen at its fullest development all over the body at the same time, so that on the face it is already fading when the body is involved, and is fading on the body when it reaches the limbs. Often large areas of the body remain uninvolved.

The rash remains visible in any one place from one to two days, and the whole period of efflorescence is usually from two to three days.

At times the efflorescence is composed of such small papules, that it resembles scarlet fever (scarlatiniform type). According to some observers this type is never seen on all parts of the body in epidemics of rubella, but only on certain parts such as the chest and thighs. According to other observers a general scarlatiniform type has been seen in rubella epidemics. The former authorities believe a general scarlatiniform rash means not rubella, but Dukes' disease ("fourth disease"). The latter authorities do not believe in the separate identity of Dukes' disease. Occasionally the rash of rubella shows urticaria-like lesions.

No desquamation is observed in the majority of cases, but at times there is a very fine, powdery desquamation.

OTHER SYMPTOMS.—The most important symptom of rubella beside the efflorescence, is an ***enlargement of the peripheral lymphnodes***. This is a constant symptom of German measles, and may occur from two to four days before the appearance of the exanthem. The lymphnodes most markedly involved are those of the neck, especially the post-auricular group. The glands reach a size varying from that of a bean to that of a hazel nut, and are often tender to pressure. The swelling does not disappear for one or two

weeks. The axillary and inguinal lymphnodes are also involved. Cases have been described of rubella without exanthem, but with lymphnode enlargement, occurring during epidemics.

The mucous membranes are slightly if at all affected. At most are seen slight redness of the conjunctivae, slight rhinitis, slight reddening of the tonsils and pharynx, and slight enlargement of the lymph follicles. Forchheimer has described an eruption in the mouth as an enanthem which he believes is characteristic of German measles, and states that it is very short-lived, fading away within the first twenty-four hours, and is localized upon the velum of the palate and on the uvula, but rarely invades the hard palate or any other part of the mouth. The efflorescence consists of macular, distinctly pink-red spots resembling the roseola of typhoid fever, arranged irregularly, not crescentically, about the size of large pin-heads, very little elevated above the level of the mucous membrane, and with very little infiltration. Koplik's spots are never seen in rubella.

There is often a brief and moderate elevation of temperature in the prodromal stage. The fever may go to 101° F., 102° F., and rarely to 103° F., on the first day of the efflorescence. It quickly falls, even while the eruption is still spreading. Many cases run their entire course without fever.

The general condition is usually little disturbed. Often the children do not feel sick at all. The first case in a family is seen by the physician usually only through fear of measles or scarlet fever, and the parents often do not seek medical care for later cases.

Only very exceptionally are there any severe symptoms, such as severe angina, or bronchitis. Complications are practically never seen.

The blood shows no characteristic changes.

DIAGNOSIS.—In sporadic cases the diagnosis of rubella is often difficult; in epidemics it is comparatively easy. The diagnosis, as in all the exanthemata, cannot be based on the efflorescence alone, but the entire clinical picture must be taken into consideration.

Measles presents the most difficulty. If the case is observed daily from the beginning of the symptoms, Koplik's spots should be observed at some time in measles. Their presence excludes rubella; their absence is in favor of rubella, but is not positive, as they may be very few, and are easily overlooked. The second point of most importance in the differential diagnosis, is whether or not the child has already had measles. A foregoing measles speaks strongly for rubella. The rash in measles is more marked, redder, more raised, more diffuse, and presents a much more imposing picture. It is in mild cases of measles with atypical efflorescence that the most difficulty in diagnosis is encountered. Notable conjunctivitis, catarrhal

affection of the upper air-passages, and high fever, fa
The absence of a prodromal stage and the presence of
of the cervical lymphnodes favor rubella. The lon
period of rubella, at least fourteen days, is sometimes a
nosis. The diazo reaction in the urine is said to be abse
while it is present in measles. The blood examination is
assistance, as neither disease is accompanied by a leuk

Scarlet fever rarely comes into consideration in diffe
nosis. The cases of rubella with a scarlatiniform rash
some areas where the appearance of the rash is incom
scarlet fever. The mildness of the constitutional distu
the absence of marked angina and strawberry tongue, ex
fever.

Some of the *toxic erythemata* with a measles-like r
considerable difficulty at times in differential diagnosis
is usually less uniform, but the diagnosis can only be mac
all the circumstances into consideration.

PROGNOSIS.—The prognosis is good. Rubella is
and most favorable of all the specific infectious diseases

PROPHYLAXIS.—Special measures of prophylaxis a
sary. Isolation should be enforced only when there is
of the diagnosis, while the physician is waiting to deci
measles and rubella.

TREATMENT.—No special treatment is required.
any fever or constitutional disturbance, the child should
bed, with a light diet, the treatment being that of a
measles. Children should be kept out of school for a
days, but when the temperature is normal and there a
toms, may go out doors.

PROBLEMS AND RESEARCH.—The chief proble
is that of the etiology. Very little research work has
devoted to the disease. Physicians have been engaged
exact description of the clinical features.

DUKES' DISEASE

("Fourth Disease")

In 1900 the English physician Dukes described as a
gious disease a condition which resembled mild cases of
but which did not appear to be either scarlet fever, m
bella, and which he consequently designated as "the fou

Dukes observed several epidemics, and described th
follows: There are usually no prodromal symptoms, a

malaise and sore throat. In a few hours the body becomes thickly covered with a pale, punctiform efflorescence. There is some reddening of the conjunctiva and throat, and moderate enlargement of the cervical lymphnodes, but no strawberry tongue. The eruption disappears rapidly, and is followed by a very slight furfuraceous desquamation, lasting one or two weeks. There is little or no fever and constitutional disturbance. Full recovery without complications occurs rapidly. The contagious period appears to last two or three weeks.

This description is that of a disease with an efflorescence resembling that of scarlet fever, but in which the mildness or absence of other symptoms is suggestive of rubella. A very similar clinical picture is seen in certain mild cases of scarlet fever, occurring in certain epidemics, in which only the connection with other more typical cases proves them to be scarlet fever. Dukes, however, distinguishes his disease from scarlet fever on the ground that in the epidemics observed by him the clinical picture was frequently seen in children who had had scarlet fever, or in whom true scarlet fever occurred later. Furthermore, he found that all the cases ran a mild course without complications, and the incubation period appeared to be longer than in scarlet fever, namely, from nine to twenty-one days.

Filatow in 1886 had described the same clinical picture under the name of *rubeola scarlatinosa*, but he did not mean by this rubella with a scarlatiniform rash, but regarded it as a distinct disease. Many authorities have regarded the "fourth disease" as representing rubella with a scarlatiniform rash, and the long incubation period would well fit rubella. Dukes, however, was unwilling to admit the identity of the disease with rubella, on the ground that it frequently occurred in children who had had rubella.

Opinions differ as to the separate identity of Dukes' disease. The majority of authorities deny it, and regard the clinical picture as representing either a very mild abortive form of scarlet fever, or an atypical rubella with scarlatiniform rash. Others believe that Dukes' disease has a separate existence. In epidemics of rubella scarlatiniform rashes are not uncommon, but they usually involve only parts of the body, and a general scarlatiniform rash has hardly ever been described. In an epidemic in Philadelphia, described by Ostheimer, scarlatiniform rashes were seen, but were limited in extent. Also, the mildest scarlet fever cases usually show some features more characteristic of scarlet fever, than does the clinical picture described by Dukes. I have recently seen in the Infants' Hospital a case exactly fitting Dukes' description, but in which a recent exposure to scarlet fever could not be excluded. No further case occurred.

The question of whether Dukes' disease represents a separate

disease entity must be regarded as still open. It can only be solved either by the discovery of the causative organism, or by more extended epidemiological studies, or by hematological and serological research. For the safety of the community, cases presenting the clinical picture described by Dukes, should be treated as mild scarlet fever.

VARICELLA

(Chicken-pox)

Varicella is a contagious disease characterized by a vesicular exanthem, and mild constitutional disturbance.

ETIOLOGY. THE MICROÖRGANISM.—The cause of varicella, like that of most of the exanthemata, is as yet undiscovered. The virus does not appear to be contained in the vesicles, as, in contradistinction to variola, inoculation of children from the vesicles of varicella has not been followed by uniformly positive results.

TRANSMISSION.—Varicella is highly contagious. The contagium is usually air-borne, and the virus is extraordinarily light and easily carried in currents of air, so that it can cross a considerable air space. It is for this reason that the Germans call the disease "Windpocken," and the French "petite vérole volante." Transmission through the medium of healthy carriers or contaminated objects is rare, but has been known to occur.

The manner in which the virus leaves the patient, and the portal of entry are still unknown. It seems probable that it enters with the air in respiration.

The contagious period possibly begins shortly before the appearance of the exanthem. The infectivity is at its height at the beginning of the stage of efflorescence. By the time the vesicles appear, transmission to the other children in the family has already taken place. How long the contagious period lasts is unknown, but the infectivity appears to be slight at the time of the drying of the last vesicles. The longevity of the virus outside the human body appears to be short.

PREDISPOSITION.—The susceptibility to chicken-pox is very great. Usually when the infection enters a family for the first time, all the children are attacked. The disease confers a lasting immunity and second attacks are rare.

Varicella almost always occurs in childhood. The disease is very common up to the tenth year. Infants under three months are less often attacked than older children, but the disease can occur in the newborn. After the tenth year the number of cases becomes much less, and the disease is very rare in adults. The infrequency of chicken-pox in adults is, however, probably not due to a lessened

susceptibility, but to the fact that almost all persons acquire immunity through having the disease in childhood.

In general the disease is commoner in the colder months, probably because the schools are open and because housing conditions involve more intimate contact among children. The disease is prevalent at all times. Recognizable widespread epidemics are not seen.

INCUBATION.—The period of incubation in the majority of cases is fourteen days. It is almost never less than thirteen days, and may be as long as nineteen days.

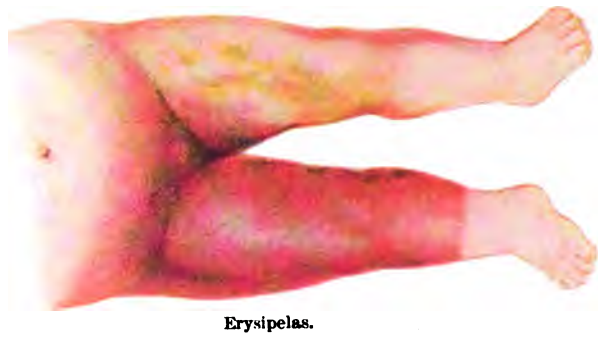
PATHOLOGICAL ANATOMY.—Deaths from varicella are so extremely rare that our knowledge of the pathology of the disease is necessarily limited. It is evident, however, that the efflorescence of vesicles, which represents the principal morbid lesion of the disease, is of a somewhat different type from that which occurs in variola. The vesicle is much nearer the surface than in the latter disease, being formed mostly by the upper layers of the epithelium. The vesicle itself is seldom multilocular, a condition which is frequently present in variola. The contents of the vesicles are usually a clear serum, the progression to a pustule being rare in comparison with the lesion of variola. The lesion so rarely involves the deeper layers of the skin, and the process is usually so very mild, that it is only in a few widely scattered lesions that sufficient destruction of the tissue takes place to produce a scar.

The lesions may appear on the mucous membranes as well as on the skin. At times the lesions assume a much more serious form and may become gangrenous. This occurs most often in poorly nourished, atrophic infants. In gangrenous varicella, the vesicles, instead of drying up in the ordinary way, become black and larger, so that a number of rounded, black crusts are scattered over the surface of the body. If a crust be removed, it is found to cover an ulcer more or less deep. Around it the skin is of a dusky red color. All the vesicles do not become gangrenous, so that we find crusts of the ordinary appearance mixing with the blackened crusts. The gangrenous process often penetrates deeply through the skin to the muscles. The lesions at times are so extensive as to form ulcers which may invade and destroy large areas of tissue.

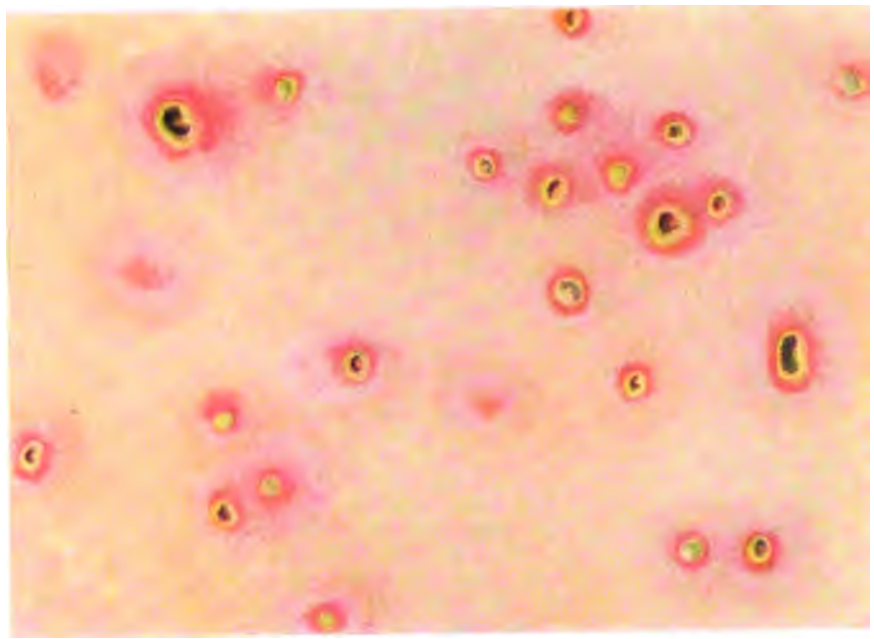
SYMPTOMS. PRODROMAL STAGE.—There are rarely any prodromata in varicella, beyond a slight malaise for a few hours. At times, however, especially in young infants, the onset of the disease may be severe; it may be characterized by vomiting, and, when the temperature is high, even by convulsions. In rare cases the prodromal stage is of considerable length and the prodromata resemble somewhat those of the other exanthemata.

EFFLORESCENCE.—The first symptom usually noted by the child's

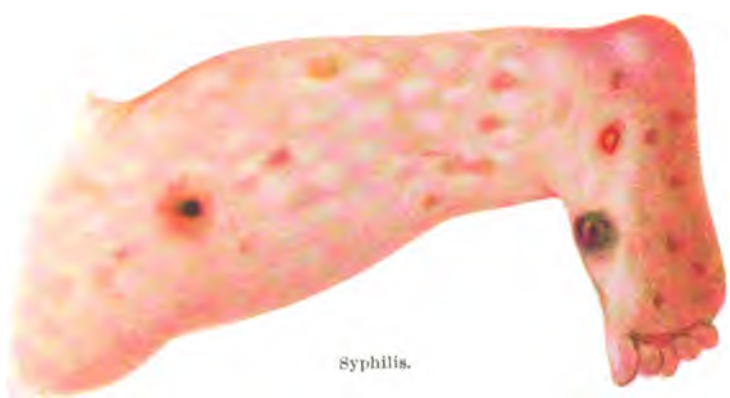
PLATE XI.



Erysipelas.



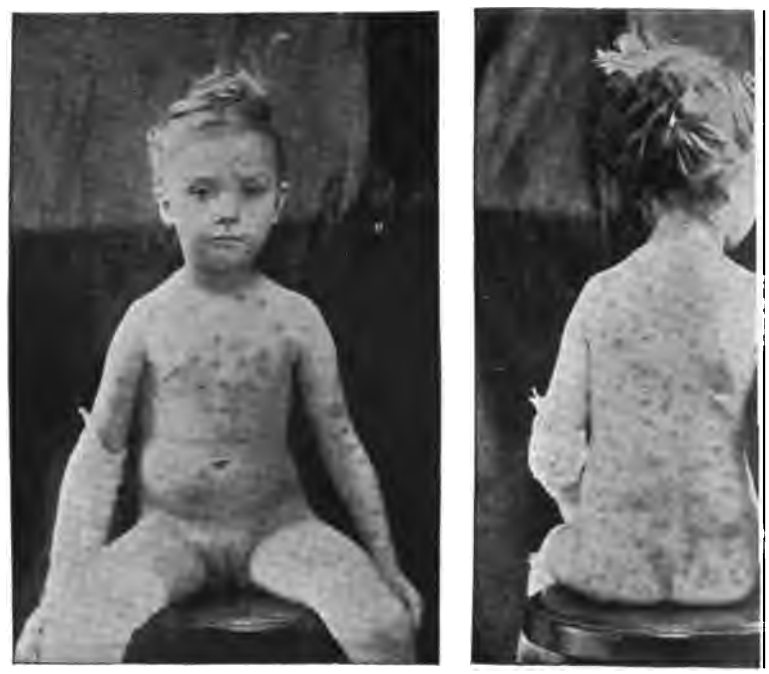
Varicella.



Syphilis.

parents is the rash. This ordinarily appears first on the scalp, and soon appears on the body and extremities. It at first of small red spots about the size of the head of a pi rapidly increase in size, becoming papular, and about as small pea. In the middle of a certain number of these p pears a small vesicle, which quickly spreads, often co entire papule. The margin of some of the vesicles borde upon normal skin, while others do not involve the who and are surrounded by a reddened areola. The vesicles v developed are about the size of a small pea. Their conte

FIG 107



Varicella. Stage of efflorescence, third day

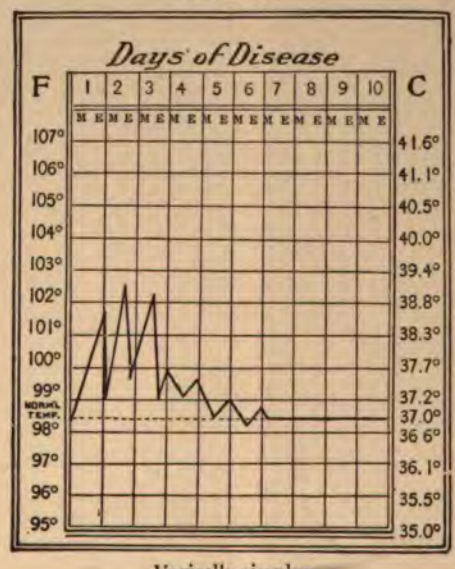
first clear, sometimes slightly cloudy, and later they become but only occasionally become actually purulent. The vesic umbilicated, and is unilocular. After one or two days it l dry up and flatten out, and in three to six days after its ap it becomes a dry yellowish or brownish crust. These scale at the end of the first or in the beginning of the second week leaving no scar, unless the scab has been picked off premat the patient.

It is characteristic of the disease that not all the lesion appear go through all these stages. A certain number reach

macular or papular stage before fading, and never form the characteristic vesicles. Also new spots keep appearing for a number of days, or a week, many of which go through all the stages. Consequently at any time in the stage of efflorescence it is usually possible to see the lesions in all stages, macules, papules, vesicles, and crusts.

The number of lesions varies widely. Sometimes there are only a few, less than a dozen on the entire body, while at other times there are several hundred. On the face they can even become confluent. There is often a peculiar odor coming from the body, especially when the contents of the vesicles are very cloudy or purulent. During the crusting stage there is often itching, especially on the scalp, and the lesions may be infected by scratching, showing then more marked suppuration or the lesions of impetigo contagiosa.

CHART 11



Varicella simplex

The eruption is frequently seen on the mucous membrane of the mouth and throat, involving most often the soft palate, but occasionally being seen on the tongue, pharynx and lining of the cheeks. The lesion on the mucous membrane is a rather large vesicle, but is rarely seen in this vesicular stage, as its covering becomes broken almost as soon as the vesicle is formed, and the usual appearance is that of a shallow greyish-yellow ulcer resembling the lesion of stomatitis herpetica.

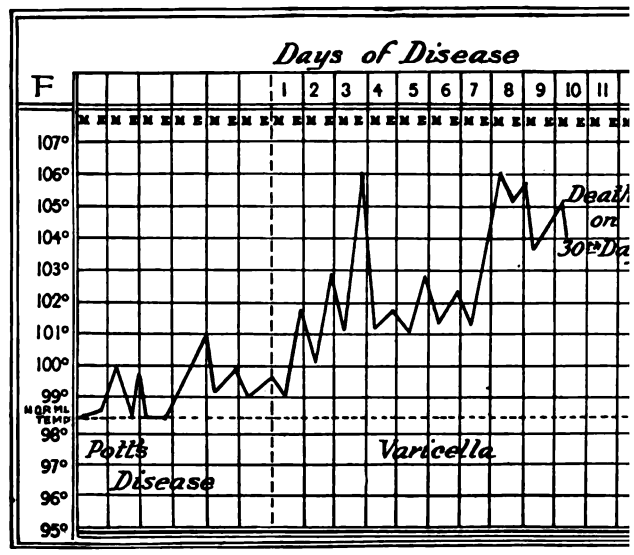
The eruption occasionally involves the nasal cavity, conjunctiva, and vulva. In the last situation itching is often severe, and scratching may cause troublesome secondary infection. In rare cases there

is involvement of the vocal cords, with hoarseness, cr
even symptoms of laryngeal stenosis.

OTHER SYMPTOMS.—The temperature in varicella
not high, and is very irregular. It usually rises when
of any considerable number develops, and falls again
There is little or no constitutional disturbance in
cases. There may be restlessness, disturbed sleep, a
tite. In a few cases there is high fever, headache,

ATYPICAL FORMS.—In some very severe cases, the
the first be papular and hard like that of variola, the
vesicles may become notably purulent, and they ma
by a marked inflammatory areola. With such sever

CHART 12



Varicella gangrenosa

fever is higher, and the constitutional disturbance
severe. A markedly purulent eruption may lea
clothing children tends to produce a more severe typ

Occasionally single vesicles may reach an abnor
resembling the lesion of pemphigus. The contents
rare cases is hemorrhagic. The vesicles are part
become pustules in poorly nourished infants, in sub
and in the uncleanly.

A special type is sometimes seen, chiefly in ill-no
which is called the gangrenous form of varicella.
secondary infection of the vesicles causes large p
inflammatory process extends into the deeper layers

subcutaneous tissue, and may even reach the muscles. The process causes gangrene of the skin. The lesions become covered with black crusts, which fall off, leaving deep, crater-like ulcers, often with a peculiar punched-out appearance. The process may lead to pyemia, metastatic suppuration, general sepsis, and death.

COURSE AND COMPLICATIONS.—The course of varicella is rapid. It is characterized by a sudden onset of mild constitutional symptoms, with the almost immediate appearance of the efflorescence. The efflorescence runs a rapid course, appearing quickly on different parts of the skin, and disappearing almost as quickly as it appears. The disease lasts about a week or ten days, and, as a rule, has no serious sequelae. It is rarely complicated by any other disease.

During the course of certain epidemics, however, it has been noticed that the *kidneys* are affected. This complication usually occurs after the efflorescence has almost disappeared, and in the second week from the time of the beginning of the attack. In these cases albuminuria is present, and in all probability is caused by some form of nephritis, although nothing definite is known about this class of cases.

DIAGNOSIS.—The diagnosis of varicella is not difficult if we bear in mind the characteristics of the diseases which it is most apt to simulate.

VARIOLA.—In differentiating it from variola we must consider the great difference in the rapidity of the development of the efflorescence in the two diseases. In variola it is essentially slow, in varicella it is characteristically quick. The papules of variola are hard to the touch, those of varicella are soft. The vesicle of variola, as a rule, is umbilicated and soon becomes a pustule; these characteristics are absent in varicella. The whole course of variola occupies a period of from two to three weeks; the course of varicella is much shorter, and is often limited to one week. Finally, the severe constitutional symptoms and the long prodromal stage in variola differ essentially from the lack of prodromata and the mild constitutional symptoms in varicella.

The following table gives the chief points of difference between varicella and variola:

TABLE 43

	VARICELLA	VARIOLA
Incubation.....	Two to three weeks.	One to two weeks.
Prodromata.....	None or slight.	Three to four days in length. Active. Severe.
Efflorescence.....	Rapidly becomes vesicular. Not umbilicated. Unilocular. Irregular. Lesions numerous. Universally distributed in successive crops. Vesicles differ	A slow progressive development from a macule to a papule, from a papule to an umbilicated vesicle, then to a pustule. Multilocular. Regular.

TABLE 43—*Continued*

	greatly in size. On pricking, collapses entirely.	Lesions not numerous. Defined in its localization. Lesions, as a rule, of uniform size. On pricking, collapses partially.
Desquamation.....	Slight crust formation.	Pronounced crust formation.
Duration.....	Short, one week to ten days.	Long, three to four weeks.
Type.....	Mild.	Severe.
Temperature	Irregular, not high.	Rises suddenly. Remains high until papules are developed, when it falls considerably. Rises again during the development of the pustules.

VACCINIA.—In vaccinia the slow progression of the lesions from papules to pustules, and the rather limited areas affected, serve to distinguish it from the successive crops of vesicles, with the rapid development and extensive distribution which are met with in varicella.

HERPES ZOSTER.—The differential diagnosis of varicella from herpes zoster is not difficult, if we consider that the vesicular efflorescence in herpes zoster follows the course of some set of nerves, while that of varicella is perfectly irregular and is in no way connected with the distribution of the nerves.

PROGNOSIS.—In the majority of cases, the prognosis of varicella is good. Severe cases are very rare. The only danger is in wretched atrophic children, in whom gangrenous varicella, and general sepsis sometimes develop.

PROPHYLAXIS.—When a case of varicella occurs in a family, it is well to isolate the patient. Although the disease is a mild one, there is always a possibility of a severe course, and I do not believe in ever intentionally allowing children to become exposed to a contagious disease. It is especially important to protect if possible young infants and sickly or poorly nourished children. In nine instances out of ten, however, isolation will be too late to prevent the spread of the disease to the other children. In children's hospitals, epidemics can only be prevented by closing the ward to new patients for three weeks after the last case.

TREATMENT.—The treatment of varicella is merely symptomatic. The child should stay in the house, and its room should be kept at an even temperature. The diet should be light. The child should be carefully watched to prevent it from scratching, as lesions deep enough to produce scars may often be obviated in this way. This treatment should be continued until all the constitutional symptoms have passed away and the efflorescence has disappeared. The skin should be kept clean. Itching may be relieved by sponging the skin with a solution of bicarbonate of soda, or a wash containing 1 per cent carbolic acid in lime water.

PROBLEMS AND RESEARCH. The only important problem in connection with varicella is that of the microörganism which causes the disease. The portals of entry and exit for the virus are also still in doubt. Some investigators have failed to produce any lesion in human beings by inoculation with the contents of the vesicle. Others have apparently obtained a local lesion. Kling has reported the attainment of a local lesion by inoculation which protected the children exposed from acquiring the disease, whereas 73 per cent of those exposed, but not inoculated, contracted chicken-pox.

VARIOLA

(Smallpox)

Smallpox is a specific infectious disease of unknown etiology. It is extremely contagious, and is characterized by a typical fever curve and a typical exanthem.

ETIOLOGY. THE MICROÖRGANISM.—The organism which causes smallpox has not been discovered. The most prevalent theory is that it belongs to the group of protozoa. It is known that the virus is contained in the vesicles, pustules and crusts which are formed upon the skin and mucous membranes. The virus is also present in any of the secretions and excretions which can become contaminated with the products of the exanthem or enanthem. There is evidence that the virus is sometimes contained in the blood.

The virus can pass through ordinary filters, but not through colloid filters. It is highly resistant to such injurious influences as light and drying, and is also resistant to disinfectants.

TRANSMISSION.—Both contaminated objects and droplet infection play a part in the transmission of smallpox. Not only can such objects as linen, clothing, and utensils which have been in contact with the patient, transmit the disease, but the contagium can also be carried on the skin, hair, or clothing of third persons who have been in contact with a patient. The contagium is also air-borne, dust and droplet infection playing a part, and there is evidence that the infection may be carried for a considerable distance through the air. It is possible that insects may help in the distribution of the virus.

PORTAL OF ENTRY.—The principal portal of entry is the mucous membrane of the respiratory tract, as the virus usually enters with the inspired air. The possibility of infection through the digestive tract cannot be excluded. When the contents of a smallpox lesion are inoculated into the skin, the resulting disease produced is never true smallpox, but consists in a local lesion, which was described by Brinckerhoff as a *variola inoculata*.

THE CONTAGIOUS PERIOD.—The disease is contagious at all stages, from the beginning of symptoms until after the completion of desquamation. The disease is most contagious during the stage of efflorescence.

PREDISPOSITION.—Mankind seems to be generally susceptible to smallpox. At all ages, however, some individuals appear to be insusceptible. The disease is commoner among children than among adults for the same reasons that explain the greater frequency of all contagious diseases in early life.

IMMUNITY.—One attack of smallpox confers an immunity which usually persists throughout life. There have, however, been rare recorded instances of second attacks. The immunity conferred by variola inoculata or vaccinia, is not so persistent.

PATHOLOGICAL ANATOMY.—The pathological conditions found in variola are chiefly those of the skin and the mucous membranes. The lesion begins as a round, somewhat raised macule, which develops into a hard papule, and later a small vesicle arises on its summit. This vesicle enlarges very rapidly and changes to a tensely filled pustule with a central depression. The size of this pustule corresponds to that of the original macule. Microscopically the macule consists of a circumscribed spot of hyperemia in the capillary layer of the skin. The papule is formed by a sharply defined necrobiotic degeneration of the under layers of the rete mucosum, by which process the nuclei of the epithelial cells are destroyed. By the transudation of fluid into these areas the cells are pushed apart and the epithelial layer is filled up as a whole, covering the area affected, and forming a vesicle, the inner part of which is composed of a mesh-work filled with lymph. In the vicinity of the necrobiotic focus, an inflammation is set up, causing an increased growth of the cells of the rete which surround the cells of the focus on all sides. The developed pustule extends through the whole thickness of the cutis to the subcutaneous tissue. A net-work inside the pustule, which is most tense in the central part, connects the roof and the floor of the pustule, and, in conjunction with the above-mentioned growth of the cells of the rete around the focus, causes the central depression. If the vesicle is pricked, only a part of the lymph flows out of the mesh-work within. The lymph contains some white and red blood-corpuscles, streptococci and staphylococci, fibrin-flocculi, and molecular granules. The contents of the pustule are purulent, and those of the hemorrhagic variety contain blood. Clumps of bacteria with corresponding localized degeneration and its associated changes are found in the neighborhood of the pustules, also in the parenchyma of the internal organs and lymph-glands, as well as in the skin. These secondary infections are largely responsible for the

stage of pustules and for the toxic symptoms. The severe hemorrhagic and malignant forms are probably dependent more upon the activity of the secondary streptococcus and staphylococcus infections than upon the primary infecting agent. When the lesion of variola has reached its height, the central depression in the pustule disappears, because the increased tension in the contents tears away the meshwork. The vesiculation begins in the upper central part and spreads downward towards the periphery. The pustule then collapses and changes to a crust, which after a certain number of days falls off, leaving a more or less deep scar covered with young epithelium. If the suppuration extends into the deeper layer, scarring invariably results; it does not necessarily follow if the suppuration is confined to the upper layer. A distinct difference in the anatomy of a pustule of variola vera and one of varioloid does not exist.

On the mucous membrane of the mouth, nose, conjunctivae, bronchi, esophagus, rectum, sometimes of the vagina, and also on the tonsils and the tongue, the same pustular efflorescence may be found, and is either superficial or extends more deeply. At times also a pseudomembrane is found on the ulcers.

In the intestines, swelling of Peyer's follicles is not uncommon. In the larynx, the efflorescence may be associated with a fibrin exudate, and sometimes with edema sufficient to cause death. Occasionally the inflammation extends deeper and involves the cartilages. In the trachea and bronchi there may be ulcerative erosions, but the characteristic lesions seen on the skin do not occur. There are no special lesions of the lungs, but congestion or broncho-pneumonia is very common.

Conjunctivitis, keratitis, and inflammation of other parts of the eye may occur in the course of the disease or afterwards.

Acute otitis media, with or without suppuration, is of common occurrence.

The pathological changes in the other organs consist of enlargement of the spleen and fatty degeneration of the liver, kidneys, and heart. Metastatic processes in the various organs and in the joints sometimes occur. In the hemorrhagic form, hemorrhages in the various cavities, in the different organs, and, according to Golgi, in the medullary cavities of the bones, may occur, also on the serous and mucous surfaces and in the muscles. The blood shows an active leukocytosis during the pustulous stage.

INCUBATION.—The incubation of the disease varies from twelve to fourteen days, the latter being the most frequent period.

SYMPTOMS.—According as the symptoms of variola are mild or severe, the disease has been divided into a number of forms, designated as follows: (1) *Discrete*, (2) *confluent*, (3) *hemorrhagic*, and

(4) *modified*. In all these forms the initial fever, convulsions, and general symptoms may be severe, and do not necessarily indicate which type of the disease is about to follow.

The mildest and most typical form of the disease is that which is called *discrete*.

PRODROMATA.—In this form, the invasion, although sometimes less severe than in the confluent and hemorrhagic forms, is in infants and young children almost always of a grave type. In infancy and early childhood the disease commonly begins with convulsions. There may be vomiting, great restlessness, rapid pulse, high temperature, and in a number of cases the children quickly succumb to the disease from the virulence of the toxemia. If they survive this early stage of the disease they usually present the same sequence of symptoms as in cases occurring in later life, but may eventually die from the exhaustion which often rises from a prolonged suppurative fever. In the prodromal stage the pulse is much accelerated and the temperature may be as high as 104°, 105°, or 106° F. In this stage we at times, especially among children, meet with an evanescent erythematous efflorescence. It has a peculiar distribution and generally a limited extent, usually affecting the lower abdominal areas, the inner surface of the thighs, the sides of the thorax, and the axillae; sometimes, however, it involves the whole surface. This efflorescence is distinct from the typical lesions of variola which occur later.

EFFLORESCENCE.—On the third or fourth day of the prodromal symptoms an efflorescence appears on the skin, and at this time the frequency of the pulse lessens, the temperature usually falls considerably, and the more severe symptoms improve, so that the patient appears much more comfortable. The efflorescence is at first represented by small red macules or papules, which, as a rule first appear on the forehead, or on the face and mucous membranes, and later on the trunk and limbs. The papules are rather scattered in their distribution, and have the feeling of shot under the skin. The macules when present soon become papules. On the third day by means of a good light a small vesicle can be seen at the apex of the papule, and by the fifth or sixth day the vesicular stage is well established and the vesicle becomes distinctly umbilicated. This appearance on careful examination can also be seen in the lesions of the mucous membranes. At about the eighth day the vesicles become pustules, the tops soon flatten, and the umbilication disappears, leaving an areola of injection and the intervening skin in a swollen condition.

TEMPERATURE.—The temperature at this time rises, from the suppuration which is taking place in the pustules. This rise of tem-

perature is called the secondary fever, or fever of suppuration. The temperature remains high for from twenty-four to forty-eight hours, and then gradually falls until by the twelfth or thirteenth day it usually becomes normal. The contents of the pustules dry up, and crusts are formed. On the palms and soles small hard disks form, which may of themselves fall off in infants, but in children as old as ten years would remain for a long time unless removed with the point of a knife.

BLOOD.—On examining the blood in cases of variola, Arnheim found the hemoglobin diminished at the beginning of the disease. After the formation of pustules and during their exsiccation, he found an increase of the hemoglobin with diminution of the erythrocytes. When complicating suppuration occurred, both the erythrocytes and the hemoglobin remained for a long time abnormally diminished.

Pick, in forty-two cases found no leukocytosis except in the stage of suppuration or in some complication like pneumonia. This was the case even when the temperature was high, the disease severe, and the termination fatal.

DESQUAMATION.—By the fourteenth or fifteenth day the stage of desquamation is established. In some cases extensive scars are left on the skin where the crusts have fallen off. This is more apt to occur in severe cases.

CONFLUENT FORM.—In contradistinction to the mild or discrete form of variola is the more severe form, called *confluent*, on account of the tendency of the lesions to coalesce. In the confluent form of variola the efflorescence usually appears at the same time as in the discrete form. At about the fourth day the lesions become confluent, the skin becomes reddened and swollen, and the face may be much distorted by the severity of the eruption. In this form the initial temperature does not fall to the same degree as it does in the discrete form, and diarrhea is likely to occur, particularly in children. The pharynx and larynx are especially apt to be involved, and the cervical lymphnodes to be enlarged. The crusts adhere longer in the stage of desquamation than they do in the same stage of the discrete form.

HEMORRHAGIC FORM.—The third or *hemorrhagic* is the most virulent form of variola, and may occur in children as it does in adults, although not so frequently in the former as in the latter. Its symptoms in children are so severe that in almost every case it very quickly proves fatal. It is characterized by punctiform hemorrhages in the skin, appearing from the first to the fourth day of the prodromal stage, ecchymoses in the conjunctivae, and hemorrhages from the mucous membranes. According to Osler, hematuria is the most common form of hemorrhage, hematemesis the next.

MODIFIED FORM.—The fourth or modified form of variola occurs when the disease attacks individuals who have been successfully vaccinated. This form is called *varioloid*, but would be better termed *modified smallpox*. Modified smallpox is usually much milder in its symptoms than any of the other forms of variola, although the initial fever may be as high as in a severe case. The lesions are fewer in number, the temperature becomes normal sooner, and the child seems comfortable in a shorter period of time, since there is usually no secondary fever from suppuration. The nearer the attack comes to the time when the child was vaccinated, the less severe will be the symptoms.

In any of these forms of variola the prodromal symptoms may be of a very severe nervous type, and this is especially characteristic of the disease as it occurs in children. For this reason variola may simulate other diseases in its prodromal stage, and may often cause death before the efflorescence has appeared. This is especially the case with the prodromal symptoms of the hemorrhagic form.

COMPLICATIONS.—The most common complications of variola are those of the larynx and the lungs. When the larynx is affected, edema of the glottis may suddenly arise and death take place from suffocation. When a lesion of the lung develops, it is usually in the form of a bronchopneumonia.

In the throat, the presence of the efflorescence occasions great irritation, and the accompanying secretions cause nausea and at times dyspnea, with a cough which in weak children is very exhausting.

When acute inflammation of the middle ear has taken place, the pain during the formation of the pus is very intense, but it subsides as soon as the sac bursts or is incised. This complication, therefore, requires early and careful treatment.

Although albumin is very frequently present in the course of the disease, nephritis is rare.

DIAGNOSIS.—There is no other acute infection accompanied by an efflorescence on the skin which in a typical case would be likely to be mistaken for variola. The severe constitutional symptoms, the slowly developing and rather scattered macules and papules, with the shotty feeling of the latter, the umbilicated vesicles gradually becoming pustules, the extensive crust formation, and the initial and suppurative fevers all render the diagnosis in most cases quite plain.

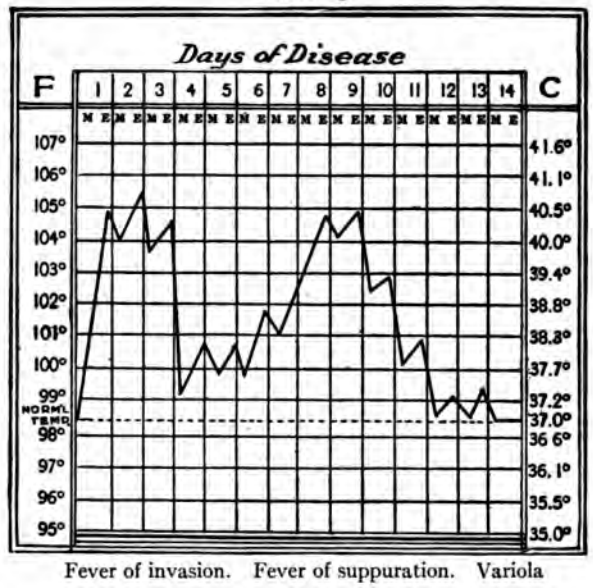
Extreme cases of varicella have been mistaken for variola. The differential diagnosis between the two diseases is given in the table under varicella.

Variola differs materially in its prodromal symptoms from measles, in which the pronounced catarrhal symptoms of the nose and eyes

make the differentiation comparatively easy. Although the prodromal symptoms of scarlet fever and of variola, such as the convulsions and vomiting, are often of equal severity and somewhat similar, yet the pronounced symptoms connected with the throat in scarlet fever, and the appearance of an erythematous efflorescence, instead of the scattered papules of variola, serve to differentiate clearly the two diseases. We must, however, be careful not to mistake the evanescent efflorescence which occurs in the prodromal stage of variola for the erythema of scarlet fever. The distinction can usually be made by remembering that the efflorescence in variola has the peculiar distribution described under prodromata, while the typical location of the efflorescence of scarlet fever is first on the neck and chest.

The following chart represents the usual temperature curve:

CHART 13



Fever of invasion. Fever of suppuration. Variola

In making the diagnosis of variola we must bear in mind the efflorescence which appears on the skin in the course of a general *vaccinia*. In vaccination the single lesion and the absence of severe constitutional symptoms make it hardly necessary to do more than refer to it in this connection. The differential diagnosis from general vaccinia is not difficult, and yet general vaccinia is so rarely met with that when it appears it almost always creates a suspicion that we may be dealing with variola. As a rule, in vaccinia the general symptoms are not severe, the disease being represented almost entirely by a slight malaise and loss of appetite, in conjunction with

the appearance on the third or fourth day of an efflorescence on the skin, represented by papules, vesicles and pustules, few in number and irregularly distributed, some on the face and nose, and a few on the body and extremities. As these manifestations almost invariably appear after vaccination, this fact is of great aid in the differential diagnosis from variola. The subsequent course of a case of vaccinia is so much milder and shorter than that of variola that in a few days the differential diagnosis can easily be made.

PROGNOSIS.—The prognosis of variola depends upon whether the child has been vaccinated, and also upon whether children who have been vaccinated when infants have been revaccinated. In the unvaccinated the prognosis is always bad, and is proportionately grave the younger the child. In the vaccinated the prognosis is good, and is favorable proportionately to the shortness of the interval between the invasion of the variola and the time of the previous vaccination.

TREATMENT.—There is no specific treatment for variola, but it is of the utmost importance that the best hygienic care should be employed. The air of the room should be perfectly fresh. The crusts should be kept softened with a mixture of glycerin, oil, and carbolic acid, and the odor arising from them should be modified by the application of a dilute solution of carbolic acid. The general principles of treatment are the same as in any severe acute infection.

In the initial stage of the disease, stimulants should be given freely if the symptoms are severe, and the high temperature should be controlled by sponging with water at a temperature corresponding to the power of the child's reaction.

The greatest care should be taken during the stage of convalescence, and when the child is considered well the most rigid measures for preventing the spread of the contagion should be enforced. The clothing and everything connected with the child and its attendants, and the room in which they have been kept during the sickness of the child, should be thoroughly disinfected, the same precautions being taken to prevent the spread of variola as in scarlet fever. The immediate transfer of a patient from its room to a smallpox hospital is in most communities considered the wisest method of dealing with the disease, and is usually enforced by law.

VACCINIA AND VACCINATION

(Cowpox)

Vaccinia is a specific eruptive disease occurring in cattle, especially in cows. It is a question whether cowpox represents a separate disease, or is smallpox modified by animal passage. Authorities differ

on this point, but the weight of modern opini
that vaccinia is modified smallpox. The dis
may be either a local affection spreading from
such as from vaccination, or may rarely be a
It is the latter condition which is usually mean
as applied to human beings. It has been su
may occur by the introduction of the vaccine
but it is now believed that the generalized er
auto-inoculation. The disease is characterized
papules, vesicles and pustules of different sizes
the body and limbs, as well as on the face, and
course. It may be said to be a rather rare disease
follow vaccination, they appear at about the fifth
lation. At the end of four days, however, minu
seen with the magnifying glass.

VACCINATION.—By vaccination is commonly
inoculation with the virus of vaccinia as a preventiv
During a period of fifteen years no death occurr
in Boston of a child who had been vaccinated before
old. During the same period the percentage of de
unvaccinated at the Boston Smallpox Hospital was 7
that of the vaccinated was 3 per cent. For the past
no person who has been successfully vaccinated w
has died of variola, and those who have been attac
have had the disease in a very mild form. In orde
relative frequency of deaths occurring in the vaccinat
unvaccinated, I have arranged the following table, bas
by Dr. Barry of an epidemic of variola at Sheffield, En
1887 and 1888. The table shows the percentage of thos
in houses invaded by variola, were attacked by the
also how many of these died. It also gives the percen
ages, and for those under ten years and over ten years.

TABLE 44

Individuals Living in Houses Invaded by Variola

		1 ALL AGES PER CENT	2 OVER TEN YEARS, PER CENT	T P
Vaccinated	Attacked	23.0	28.1	
	Died	1.1	1.4	
Unvaccinated	Attacked	75.0	68.0	
	Died	37.2	37.1	3

The low percentage of children as shown in column 3 is ver
ing in comparison with column 2, which represents older indiv

and emphasizes the importance of revaccination. It also impresses upon us the significance of the difference in the number of deaths between the vaccinated and the unvaccinated. When large numbers of cases of variola have been reported, figures show that among the vaccinated nineteen out of twenty recover, while of the unvaccinated fifty individuals out of one hundred die. It is therefore evident that vaccination is highly protective against variola, and physicians should insist on the vaccination of every individual in the community. One vaccination, however, does not protect for a lifetime. On the contrary, revaccination is just as important as the primary operation.

Revaccination should be performed at intervals of eight or ten years, *and in a shorter time when cases of variola appear in the community*. The danger of serious results arising from vaccination is extremely small.

The time at which vaccination should be done is of considerable importance. The infant should be vaccinated early, before it begins to be exposed to the danger of contagion from sources outside of its home. We must, however, remember how low is its vitality at birth, and how readily this vitality is affected by what would be considered trifling conditions in the older child or in the adult. A time should be chosen when the infant is not subject to the other disturbing conditions which naturally arise in the first two years of life, such as weaning and the irritation of the dental periods. If it is found necessary to vaccinate the infant after the sixth or seventh month, or before the twentieth, it should be done in an interdental rather than in a dental period, and not at the time when its food is being changed, or when it is suffering from either slight catarrhal conditions or some definite disease. I prefer to vaccinate the infant when it is four or five months old, that is, just before the period when the first tooth appears. At this age it has usually become accustomed to its food, its digestion is in equilibrium, and its vitality is much above what it was in the early weeks of its life, By the fifth month also, it will usually have developed the outward symptoms of syphilis if it has inherited that disease from its parents.

The vaccine virus can be introduced into any part of the body through the skin according to the fancy of the physician or parents. Girl infants can be vaccinated just below the knee on the outer side of the leg, so as to avoid having on the arm a scar to which women usually object. We are accustomed to vaccinate boys on the outer side of the upper arm. Whether the vaccination is performed upon the leg or the arm, we should first inquire if the person who is to take care of the infant is right-handed or left-handed. If the nurse, for instance, is right-handed, she will naturally hold the infant on her left arm. and in this case, the infant's right arm being towards the nurse, it is better for the vaccination

to be on the left arm. The process should be reversed when the nurse is left-handed, and in this case, for the same reason, it is better to vaccinate on the right arm or leg. The form of virus which I have been accustomed to use is lymph obtained from cows, put up by the State Board of Health. It is essential to use vaccine known to be good, and put up under the best conditions. It should be very carefully prepared by those who have made a scientific study of the subject, and, if possible, on farms which are under state supervision. A very small surface is amply sufficient for the proper introduction of the virus. A pointed ivory quill charged with glycerinated lymph (such as is shown in the plate), was formerly used for removing the epithelium, for exposing the smaller blood-vessels, and for introducing the virus. The vaccine is now, however, generally put up in sterilized glass capillary tubes. An ordinary glover's needle is preferable to the quill for removing the epithelium. It can be sterilized by being held for a moment in the flame of a match or candle, and then wiped on sterile gauze.

The utmost precaution should be taken to insure against infection from other microörganisms. The part of the skin chosen for the vaccination should be thoroughly washed with soap and water and with alcohol. The hands of the operator should be clean and aseptic. A series of short scratches should be made about one-half centimeter (about one-fourth inch) long, until the epithelium is sufficiently removed to show that the blood vessels are exposed, but not to a degree sufficient to cause bleeding, for in the latter case the virus may be prevented from gaining an introduction to the tissue. The capillary tube is then broken, and the lymph allowed to run on the abrasion, and rubbed in with the side of the needle. The skin should be protected for four or five minutes from contact with anything and then covered with a sterile gauze pad held in place by adhesive plaster. The plate shows the different stages of a vaccination as they occurred in one case carefully observed by the artist every day.

Every case of vaccination does not present exactly the same appearances. The lesions may differ in shape and size, and one individual may be affected more intensely by the virus than another; one may have accompanying severe constitutional symptoms and another may have none. The chain of lymphatics may be affected as far as the axilla or the groin.

The following description represents pretty well the usual course of the disease. After the vaccination, the skin shows nothing new until the third, fourth, or even the fifth day, when a red point appears. This soon becomes a papule; by the next day a vesicle has developed; about the sixth day this vesicle usually becomes umbilicated, and is surrounded by a faint red zone. By the eighth

PLATE X.

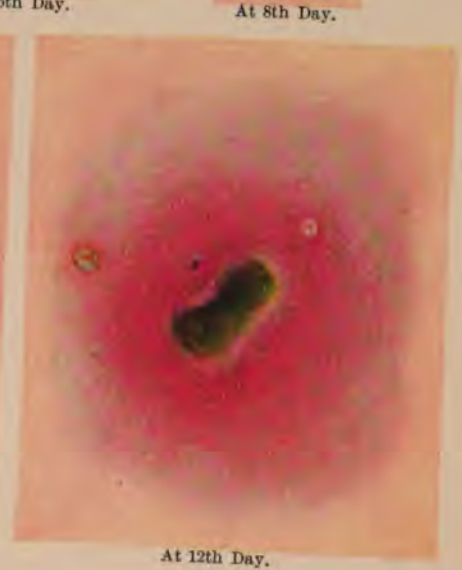
Vaccine Quill.

Vaccination Scratch.

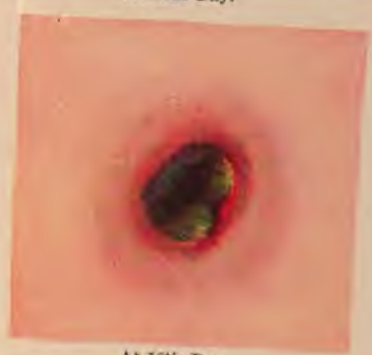
At 5th Day.

At 8th Day.

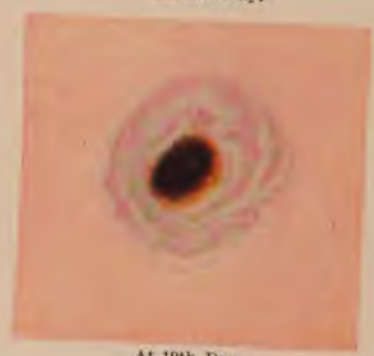
At 10th Day.



At 12th Day.



At 16th Day.



At 19th Day.

Scar at 1 Year.

Scar at 21 Years.

day the vesicle is fully developed, and by the ninth day the red zone increases rapidly and the vesicle soon becomes a pustule. By the eleventh or twelfth day a crust is formed, and this crust falls off from about the fourteenth to the twenty-first day. In some cases an ulcer is left which heals by the formation of another crust, in others the skin remains intact. From the eighth to the twelfth day there may be a slight amount of fever and a coated tongue, with some loss of appetite, and the glands of the axilla or groin may become enlarged and tender. The scar, although perhaps not typical, can usually be recognized by its small depressions (pits) and its location.

In some cases, instead of the healing of the scratch in a few days, or the formation of the vesicle of a successful vaccination, irregular excresences of a fungus-like character may appear. These in all probability have no connection with the vaccine virus, and are not protective. In addition to the rather rare cases of vaccinia, various efflorescences at times appear on the skin, not only in the neighborhood of the vaccination lesion, but also in other parts of the body. They may be present on the fourth or fifth day, or even later, in the second week, and are probably caused by some toxic substance connected with the vaccination lesion. They vary considerably in form, but are usually represented by a multiple or papular erythema or an urticaria.

II. HIGHLY CONTAGIOUS DISEASES WITHOUT EXANTHEM

This group includes infections which resemble the exanthemata in that they are transmitted very easily from one individual to another by contact, but do not show a characteristic cutaneous efflorescence. The diseases of this character are diphtheria, pertussis, mumps and influenza.

DIPHTHERIA

Diphtheria is a contagious specific infection, caused by a known microörganism, and characterized by a membranous inflammation of the mucous membranes, particularly of the throat and upper air-passages, and by certain more remote effects of the action of a soluble toxin produced by the organism.

ETIOLOGY. THE MICROÖRGANISM.—Diphtheria is caused by a bacillus, which was first described by Klebs in 1883, and was found to be the specific cause of diphtheria by Löffler in 1884. It is usually called the Klebs-Löffler bacillus. It has been isolated in pure culture, inoculations with which reproduced the essential lesions of the disease in animals. The bacillus secretes a soluble toxin, which has been isolated from cultures, and with which the general toxic lesions of diphtheria have been reproduced in animals.

The Klebs-Löffler bacillus shows great variations in morphology, growth, and virulence. The typical bacillus is a straight rod about the length of the tubercle bacillus, but decidedly thicker. It is easily stained by the aniline dyes, particularly by methylene blue. It is apt to stain irregularly, presenting an appearance suggesting spores. The stain may also give it a bipolar appearance. Long involution forms are frequently seen.

The organism grows rapidly on Löffler's blood serum, moist grey colonies appearing in sixteen to twenty-four hours. It resists cold, but dies when heated at a temperature over 56° C. It may remain alive outside the body for a considerable time when conditions are favorable, namely, moisture and darkness. It dies in sunlight.

The organism is found chiefly in the necrotic or inflamed tissue of the membranous lesion. In certain cases it has been found in the cervical lymphnodes, lungs, cerebrospinal fluid, blood and urine.

The organism remains upon the mucous membrane after the disappearance of the local lesion for a varying time which may reach many weeks. During this time it may retain its virulence. The organism may reach the mucous membrane without producing a

lesion or clinical diphtheria, but nevertheless retaining its virulence. Many healthy persons, especially those who have been in contact with a case of diphtheria, have virulent Klebs-Löffler bacilli in their noses or throats. Such persons are called "*diphtheria carriers*"

There is also frequently found in the noses and throats of healthy persons a bacillus which morphologically bears a close resemblance to the Klebs-Löffler bacillus, and which is called the *pseudo diphtheria bacillus*. This form is distinguished with certainty from the true organism only by the effect of animal inoculation. It does not produce diphtheria, and represents an avirulent type of the Klebs-Löffler bacillus.

TRANSMISSION.—Diphtheria is transmitted from a patient having the disease to a healthy person. The connection is not always close, however, owing to the fact that both convalescent and healthy persons may be diphtheria carriers. A healthy carrier may receive the organism from a diphtheria patient, may carry it indefinitely, and then may transmit it to a third person in whom it produces the disease. Diphtheria is, however, most commonly transmitted directly without the intervention of a healthy carrier.

Diphtheria is a contact infection. The individual who contracts the disease must in the majority of cases come into actual contact with the diphtheria patient or carrier, or with an object contaminated by the secretions of the patient. The contagium is not usually airborne, as the big, heavy diphtheria bacillus is not very volatile. Only when the patient coughs or sneezes directly in the face of the susceptible individual is the contagium carried through the air on the moist drops expelled from the patient. Usually close contact is required. As the bacilli live in the mouth, and come in and go out through the mouth, kissing is a common form of contact by which the disease is transmitted. The hands, bed and clothing of the patient are easily contaminated by secretions, and from these the hands of the second individual may be contaminated by contact, and then convey the contagium to the mouth. The bacilli are also frequently carried over by contaminated objects such as handkerchiefs, playthings, articles of food and the like.

The mode of transmission of diphtheria does not resemble that of measles, rubella and varicella, but is in every way similar to that of scarlet fever. Crowded conditions of life and uncleanliness favor the spread of the disease, which is easier among the children of the proletariat than of the well-to-do.

The diphtheria bacilli, after transmission as described, usually enter the mouth with the fingers or food, and are most apt to develop upon the tonsils or soft palate.

TOXIN FORMATION AND IMMUNITY.—The lesions of diphtheria, both

local and general, are due to the action of a
by the bacilli. Whether or not all the *lesion*
single toxic substance, or by more than one,
There is evidence in favor of the theory of E
lesion and general poisoning are produced by t
theritic paralysis is produced by the toxone.
through the formation by the cells of the body
neutralizes the toxin.

This immune body is consequently an *antito*
diphtheria does not produce a permanent immu
body appears to become used up in some way,
third attacks of diphtheria are not at all uncomm
produced by serum treatment is neither permane

There is a *natural immunity* to diphtheria, whic
the immunity produced in a patient who under
It has long been known that a certain proporti
immune to diphtheria, and the majority of very
known to be immune. This immunity has been
to the presence in the blood of a supply of dip
Whether or not this antitoxin has resulted from
in the ancestors is not known. The amount of na
the blood may be estimated by an animal test (R
neous method on guinea-pigs), but this method ca
than 1.16 units of antitoxin in 1 c.c. of serum, an
amount has been proven sufficient to produce im
has described a method of determining natural imm
tine use.

The Schick test for susceptibility to diphtheria consi
into (not under) the skin a very small amount of di
the quantity used being one-fiftieth of the minimum
a guinea-pig weighing 300 grams. The toxin is so di
dose is contained in 0.1 c.c. of fluid. This is injected
of the forearm with a small needle. If properly made
should produce a small, raised, white spot. Within
hours, if the reaction is positive, the area becomes redd
edematous, and slightly cyanotic. This passes off i
hours, leaving a brownish pigmentation. The reactio
only in individuals without antitoxin in their blood, and
they are susceptible to diphtheria. A negative reaction i
the individual has one-thirtieth of a unit or more of antit
of blood, and is immune. Children sick with diphtheria
tive reaction before antitoxin is given.

The Schick test is of great value in determining whet
who have been exposed to diphtheria, or who are adm
open wards of a hospital, require an immunizing dose

Only those with positive reactions need be immunized. It has been determined that the artificial immunity produced by antitoxin, and by the disease itself, is very varying in length, but does not usually last more than three weeks. The value of the Schick test has been confirmed by Park and his associates, and by other observers.

PREDISPOSITION.—The general susceptibility to diphtheria is much less than toward measles, chicken-pox and whooping-cough, and probably less than toward scarlet fever. The Schick test has confirmed the clinical observation that a certain proportion of children are immune. According to Schick's observation 93 per cent of the newborn are protected by natural antitoxin, 57 per cent of children in the first year, 37 per cent of children between two and five years, and 20 per cent between five and fifteen. In two years at the Infants' Hospital in Boston, where the Schick test is performed as a routine on admission, about 60 per cent of children under two years were found immune, the majority being in the first year.

Adults show a much smaller proportion having natural immunity than children, but yet diphtheria is much commoner in childhood. The frequency of the disease in children is not due to greater susceptibility, but to the same reason which causes all of the contagious diseases to attack children more than adults, namely, that the conditions of life in childhood favor the spread of all diseases acquired by close contact.

Diphtheria is most common between the ages of two and five years. It is infrequent after ten years. Among individual predisposing causes are adenoids, enlarged tonsils, and a tendency to catarrhal affections of the mucous membranes. For this reason the disease is seen more often in autumn, winter and spring than in summer. There are other factors. Certain children seem to have an individual constitutional predisposition. The chances of infection and the severity of the case depend not only on the constitutional predisposition, but upon the virulence of the particular diphtheria bacilli causing the disease.

EPIDEMIOLOGY.—Sporadic cases of diphtheria are seen at all times. The frequency of the disease varies from year to year. At times the prevalence of the disease is such that it amounts to a more or less widespread epidemic. The contagiousness and severity of the disease tend to increase with the frequency of cases, and are greatest in epidemics.

INCUBATION.—Diphtheria, unlike the exanthemata, has no definite period of incubation. The disease may appear within twenty-four hours of exposure, or may not appear for two or three days.

PATHOLOGICAL ANATOMY.—The lesions of diphtheria are due to the local action of the Klebs-Löffler bacillus and associated

bacteria, and to the absorption of toxins produced by the bacteria. The most constant lesion is a pseudo-membrane, produced by the local action of the bacilli in the upper air-passages, and due to exudation and coagulation necrosis. The exudation coming from the vessels of the tissue beneath the membrane meets the necrotic tissue containing a fibrin ferment, and coagulation takes place, the fibrin of the coagulum being intimately associated with the necrotic epithelium. The bacilli can also produce other lesions, such as simple inflammation, necrosis without membrane formation, and even abscess. There is no pathological condition directly characteristic of the action of the Klebs-Löffler bacillus, and the same anatomical conditions may be caused by other bacteria, and even by irritants. The process may rarely be simply a catarrhal inflammation, which does not go on to the formation of a pseudo-membrane.

The primary infection in diphtheria is most commonly in the throat, from which the bacilli may extend into the adjacent mucous surfaces, producing in most cases, membrane formation, in some only catarrhal or purulent inflammation. They may also extend into the nose, producing nasal diphtheria, or along the Eustachian tubes into the middle ear, or from the nose into the accessory sinuses, downward into the trachea and air-passages, or into the esophagus and stomach. There are certain differences in the membrane formation due to the anatomical character of the tissue. Thus the membrane in the pharynx clings tightly to the surface, while in the trachea and lower passages it is easily removed.

The extension of the bacilli into the lungs may produce a membrane formation in the smaller bronchi, areas of bronchopneumonia or even abscesses. The pathological process may be further modified by the association with the Klebs-Löffler bacillus of the common pyogenic cocci, and it is difficult to determine exactly the part which these various organisms play in the production of the local lesions. It is certain, however, that the lung lesions which are ordinarily attributed to these associated organisms may be produced by the Klebs-Löffler bacillus alone.

The membrane in diphtheria is usually of a peculiar greyish-white color and, as a rule, cannot be easily detached; in some cases, on the contrary, it may be white and easily separated. It sometimes assumes, early in the disease, a gangrenous appearance, which shows that the case is serious. Nasal diphtheria is characterized by a profuse nasal discharge, and often by a membrane. Transmission of the germ of the disease from the nose to the eye is infrequent, and does not always result in the formation of a false membrane. Frequently the pharyngeal inflammation extends through the Eustachian tube, causing inflammation of the middle ear, and in these cases a membrane may be formed. The membrane may extend downward

to the larynx, causing marked dyspnea. Below the vocal cords the membrane is not very firmly attached, and is frequently coughed up. If death occurs late in the disease or in apparent convalescence no macroscopic lesions, as a rule, are found. Microscopic examination of the nerves, however, often shows marked degeneration of the nerve tissue.

The internal lesions of the disease are not due to the presence of the bacilli but to the action of the toxic substances which are absorbed from the places where the bacilli are growing. These lesions consist chiefly in diffuse degenerative changes, and in intense local processes, also of a degenerative character. The organs most commonly affected by these lesions are the kidneys and the lymphnodes. The adjacent lymphnodes are apt to be swollen, and on microscopical examination they often show small foci of cell-necrosis; similar smaller necrotic foci may be found in other parts of the body, as in the liver, and are due to absorption of toxins. There is also a general lymphatic hyperplasia, which is relatively greatest in the abdomen. The kidneys ordinarily show only parenchymatous degeneration, but in a few cases of recurrent infection they may present acute lesions. Hemorrhages into the serous membranes are often met with, and the organs in general show degenerative changes due to action of the toxins. Endocarditis is rarely seen. Catarrhal bronchitis and bronchopneumonia frequently complicate diphtheria, and are caused by the inspiration of pyogenic cocci as well as by the invasion of the Klebs-Löffler bacillus itself.

SYMPTOMS.—The symptoms of diphtheria vary much in severity, and also vary with the locality attacked. The following types will be described: (1) *The Typical form* or general disease picture. (2) *The Mild form.* (3) *The Severe form*, or malignant diphtheria. (4) *Nasal diphtheria.* (5) *Laryngeal diphtheria.* (6) *Special localizations.*

THE TYPICAL FORM

The prodromal symptoms of diphtheria are not especially typical. They may be acute in character, or very mild and of a subacute variety. In young children there may be at the onset of the disease a slight convulsion. In certain cases, a peculiar dark-red appearance of the mucous membrane of the mouth is seen, which is quite characteristic. There is apt to be a sensation of chilliness, some heightening of the temperature, and more or less pain in the back and limbs. There is nothing, however, to distinguish this stage from many other affections of children, such as simple tonsillitis. The child may often complain of discomfort on swallowing, and on examining the throat the fauces are found to be reddened.

In from twelve to twenty-four hours, however, a more typical appearance will be seen in the throat. A white or greyish-white

pseudo-membrane, commonly appearing first on the tonsils, develops, and on the second or third day extends to the soft palate and uvula. It may also extend backward to the pharynx. During this stage the throat becomes much swollen and the tonsils considerably enlarged, so as almost to meet at times in the median line. The diphtheritic membrane is usually firmly adherent to the mucous membrane, and, as the case progresses, assumes a brownish or yellowish-grey color, sometimes becoming gangrenous, with an extremely fetid odor. A profuse nasal discharge may appear at this stage. Listlessness is present, but delirium of an active type is not common. In addition to these lesions in the throat, the cervical glands are usually involved and become considerably swollen.

The child, as a rule, shows grave constitutional symptoms. The temperature is not characteristic. It is usually not especially high, and ranges from 101° to 102° F. It may, however, rise to 104° F. A subnormal temperature is more serious than a moderately elevated one. The pulse is increased in rapidity, and is weak in proportion to the severity of the disease, but does not always correspond to the temperature; sometimes it is very slow. The slow pulse is indicative of the action of the toxin on the nervous centers, as well as of the weakness of the myocardium. Diarrhea is a frequent but not a constant symptom. Loss of appetite, nausea, and vomiting frequently occur.

In typical cases the symptoms abate towards the end of the first week, the psuedo-membrane separates, leaving a raw surface behind, the neck becomes less swollen, and the child feels much better. It is, however, usually left in a prostrated condition for a number of weeks; and even in these comparatively mild cases the toxic effects of the disease may show themselves in the form of a neuritis with an accompanying paralysis many weeks after the diphtheria has run its course. There may also, even in mild cases, be a slight discharge from the nose, owing to the inflammation of the posterior nares. Slight albuminuria is not infrequent.

A very prominent symptom in all forms of diphtheria may be cardiac weakness. In some cases the child dies suddenly without any warning, or death may be preceded by attacks of semi-collapse. In other cases there may be a weak intermittent pulse, which continues throughout the disease and during convalescence. Under these circumstances the child should be considered to be in a critical condition, as death is likely to occur suddenly.

THE MILD FORM

A great number of cases, especially in older children, are of a very mild type. These cases are accompanied by very slight constitutional disturbance. The temperature is moderately raised, 100° F.

PLATE XII.

1

Thrush.

2

Varicella.

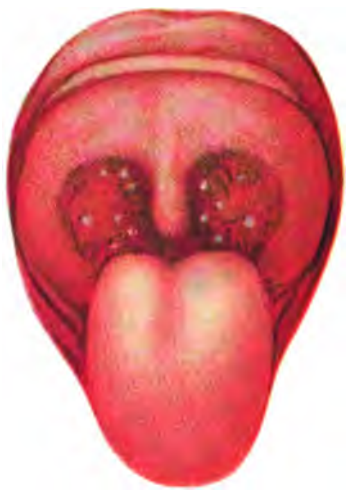
3

Stomatitis herpetica.
(Aphthosa.)

4

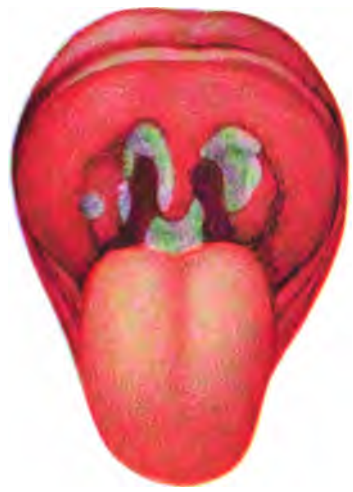
Stomatitis ulcerosa.
(Scorbutus.)

5



Follicular tonsillitis

6



Diphtheria.

to 101° F., and there are few other symptoms except slight apathy, loss of appetite, and slight sore throat. The membrane is confined to the tonsils, does not spread, and disappears in from three to five days. The enlargement of the cervical lymphnodes is only moderate.

Some cases are still milder. No typical membrane is seen. The tonsils are little inflamed, and in their crypts are seen greyish-white spots about the size of the head of a pin, or a little larger. The appearance closely resembles follicular tonsillitis, but the areas really consist of bits of membrane which are more adherent than the exudate in tonsillitis, and contain diphtheria bacilli. Sometimes these bits of membrane later coalesce into typical membrane, or the diagnosis may be clinically confirmed by the appearance of laryngeal stenosis, or by the later appearance of paralysis.

There is a still milder form, which is often called catarrhal diphtheria. In these cases no membrane or exudate appears, and the throat presents the appearance of an ordinary catarrhal angina, with moderate redness, swelling and increased secretion of mucus. These cases can transmit the bacillus to others, and in more susceptible individuals a severe form may be produced. A purely clinical diagnosis in such cases is impossible, and at most diphtheria can only be suspected from the presence of the disease in the family, house, or neighborhood. The diagnosis can only be made by culture and bacteriological examination.

THE SEVERE FORM—MALIGNANT DIPHTHERIA

In addition to these mild cases, the Klebs-Löffler bacillus at times produces a most severe form of diphtheria. In these cases the child either shows a fairly mild form of the disease for a few days and then suddenly develops the severe form, or it may be attacked at once by very severe symptoms. It becomes dull and markedly prostrated; the temperature is either slightly raised or may rise to 103° or 104° F. or higher; vomiting and headache may be present; the pseudo-membrane spreads rapidly. There may be a dusky efflorescence on the skin, simulating closely that which has been described in the malignant form of scarlet fever. There may also be a purpuric condition of the skin. The tonsils and throat are markedly swollen. The picture of these septic cases is very characteristic. There is a peculiar sweetish odor to the breath. There is cyanosis and a marked waxy pallor. There are hemorrhages from the throat and nose, with a profuse muco-purulent discharge from the latter. The cervical glands are often enormously enlarged. The breathing becomes difficult from the extension of the membrane in the pharynx, and from the swelling of the tonsils and pharyngeal tissue, and nasal breathing may be impossible. Swallowing is very difficult, and the speech is thick. The membrane has been known to extend in all directions, and some-

times even through the Eustachian tubes to the ears. All degrees of severity are met with between the mild and malignant types of diphtheria. The membrane, instead of extending upward to the nasopharynx, as occurs in the malignant cases just spoken of, may spread downward, attacking the epiglottis and the larynx, and cause serious obstruction.

The general condition is much affected. The children lie in apathy, with open, staring eyes. Occasionally they toss about in bed. There is complete loss of appetite. Although thirsty, it is difficult to get them to take any fluid. The fever may be high and remittent, or only slightly raised. The pulse is rapid, of small volume, and very easily compressible; it may be thready and hardly palpable. The extremities become cold and cyanotic.

The heart sounds are feeble. There may be a systolic murmur at the apex. Later, dilatation with enlargement of the liver, may occur. The spleen is somewhat enlarged, but may not be palpable. The urine contains albumin and casts.

In some severe forms the symptoms may be partly due to an associated streptococcus infection of the throat, which may spread as in scarlet fever. Such types have been described as "septic diphtheria." The streptococcus, however, may be present without playing much part, the severity of the symptoms being due to the diphtheria infection. The term "malignant diphtheria" is preferable.

The course of severe diphtheria has been much modified by serum therapy. Before the days of antitoxin, the majority of these severe cases died in the second half of the first week, either from intoxication and circulatory failure, or from bronchopneumonia. With early serum treatment, the severe progress of the case can often be arrested in time. In such cases the throat becomes clear of membrane by the end of the first week. Convalescence is, however, slow, taking a number of weeks, and there remains still the possibility of a sudden death from heart failure.

NASAL DIPHTHERIA

Diphtheria sometimes begins in the nose and spreads no farther. Primary nasal diphtheria may be either very mild or very severe, according to where the membrane is situated. If the membrane forms in the anterior nares, the probability of absorption is reduced to a minimum, and there is usually very little constitutional disturbance. The condition is especially liable to be overlooked, as the child for one or two days may show merely the symptoms of fever, malaise, loss of appetite, and a discharge from the nose. On examining the nose carefully, however, a pseudo-membrane will often be found. It is, therefore, very important in cases of this kind to have a bacteriological examination made, and to isolate the child until it is

determined that the Klebs-Löffler bacillus is not present. These cases are a prolific source of infection to the community at large, for even when antitoxin has been given it does not immediately kill the bacilli, although it may stop the nasal discharge.

The importance of recognizing nasal diphtheria of this type lies in preventing the spreading of the disease to others. The history of many serious outbreaks of diphtheria is due to the fact that some member of a family or some inmate of an institution has had a profuse discharge from the nose without any constitutional disturbance, and has been the focus from which has arisen many severe cases of diphtheria. This is not a theoretical statement, but is the result of experience. Every profuse discharge from the nose, particularly if there is any excoriation about the nostrils, should be looked upon with suspicion and cultures should be taken. A unilateral discharge is always suspicious, usually meaning either diphtheria or a foreign body. If adenoids are to be removed, cultures should always be taken before the operation is performed, for it very frequently happens that a child who has had a profuse discharge from the nose, has a severe attack of diphtheria, immediately after the removal of the adenoids or the excision of the tonsils. If the operation for cleft palate is to be performed, it is still more important to take cultures; because, if there are any bacilli of diphtheria in the nasal discharge, this organism is sure to grow upon the surfaces of the operative tract, and the patient not only has an attack of diphtheria, which may be mild or severe, but also the edges of the wound slough and render a second operation much more difficult.

When the disease is situated in the posterior nares and the nasopharynx is affected, either primarily or secondarily through the nares or the pharynx, the constitutional symptoms are, as a rule, marked. This is in all probability accounted for by the great mass of lymphatics in the nasopharynx, where absorption takes place so easily that general septic poisoning quickly follows, often with fatal result. In these cases the toxin generated is very readily absorbed and may cause death from its effects on the heart. Many patients die from unrecognized nasal diphtheria, because no membrane is seen, and the first indication of serious trouble is a rapid action of the heart known as the *bruit de galop*, or, in some instances, there may be a slow pulse, and the physician is often unable, if he does not have the idea of nasal diphtheria in mind, to explain the symptoms.

LARYNGEAL DIPHTHERIA

This form of diphtheria is what is commonly known as "*true croup*," or "*membranous croup*." It includes all cases in which the membrane extends into the larynx, trachea, and bronchi. It is the most dreaded of all the clinical forms of diphtheria. It is most

common in infants and younger children, its frequency bearing an inverse proportion to the age.

Extension of the diphtheritic process to the larynx can occur in the course of diphtheria of the nose, tonsils or pharynx. The involvment of the larynx may occur simultaneously with the process in the throat, or may develop later, in three to seven days. It may occur in the course of a mild catarrhal faucial or nasal diphtheria, in which no membrane has been observed. In these cases cultures from the nose and throat are positive for Klebs-Löffler bacilli.

It is important to remember, however, that in young children *the larynx may be the primary site of the diphtheritic process*, and may be severely involved, while the nose and throat remain entirely free. In such cases cultures from the nose and throat may be negative.

The first symptoms of laryngeal diphtheria are *hoarseness* and *cough*. The chief symptom is *increasing inspiratory dyspnea*.

The hoarseness increases until complete aphonia is present. The cough is at first of a harsh ringing character, but later becomes husky and painful.

Either at the beginning in very young children, or a little later in older children, inspiration becomes prolonged and noisy. It resembles the breathing in an attack of common or false croup, but becomes steadily worse, is uninfluenced by the ordinary treatment of common croup, and does not improve by day.

Steady progress, steadily increasing severity of symptoms, is characteristic of laryngeal diphtheria. There is, however, the greatest variation in the rate of progress. The full development of the picture may be reached in twenty-four hours, or may not be reached for four or five days. The inspiratory obstruction becomes dyspnea, and this becomes more and more marked. If the lungs are clear, the rate of respiration is not increased, but the noisy and prolonged inspirations cause marked effort, and are accompanied by retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and lower chest. These signs become more and more marked, and finally *increasing cyanosis develops*.

The temperature may or may not be raised. As the toxic absorption is slight, on account of the locality affected, the constitutional symptoms are correspondingly mild. The child is very restless, is forced to sit up in order to breathe, and, for the same reason, bends its body forward with its head back.

Inspection of the throat shows signs which vary according to whether the laryngeal process is primary, or secondary to a primary process in the throat. There may be membrane visible in the throat, or only redness and swelling of the tonsils and mucous membrane, or there may be nothing abnormal seen. If a tongue depressor be placed on the back of the tongue, and strong pressure made, the

reddened and swollen epiglottis can often be seen, and sometimes a bit of membrane. Laryngoscopic examination would of course show the membrane in the larynx, but this is rarely practicable in children.

The labored, obstructed breathing and cyanosis grow worse, if no treatment is instituted. The face and hands become bathed in cold sweat, and the child's restlessness gives place to somnolence. Sometimes spontaneous improvement occurs, and this may be brought about by the child expelling some membrane in coughing. The usual ending is death from asphyxia.

SPECIAL LOCALIZATIONS

The Mouth.—Diphtheria of the mouth is rare, and is almost always secondary to diphtheria of the throat. There is typical membrane formation, usually on the cheeks and lips rather than on the tongue.

The Conjunctiva.—Diphtheria of the conjunctiva is not extremely rare, and is often primary. In mild cases there is a thin membrane on the palpebral conjunctiva, with swelling and redness of the lids, and purulent secretion. This form may recover without further damage. In more severe cases the membrane is more adherent, and the swelling and inflammation is much greater. This may result in destruction of the cornea.

The Skin.—Diphtheria of the skin is seen at times, when there is a wound or lesion. It is usually secondary to a process in the throat, but may be primary. The membrane is often thin and hardly visible.

The Vulva.—Diphtheria of the vulva is rare. It is usually secondary, the infection being carried from the mouth by the fingers. Any inflammatory condition of the vulva favors infection. There is a marked phlegmonous inflammation with a thick membrane, and the affection runs a severe course, sometimes leading to gangrene.

COMPLICATIONS AND SEQUELAE.—The principal complications and sequelae of diphtheria involve *the lungs, the heart, the ears, the kidneys* and *the nervous system.*

THE LUNGS.—Severe bronchitis and bronchopneumonia are frequent complications of diphtheria. Bronchopneumonia is often seen not only in laryngeal diphtheria with membrane extending into the trachea and large bronchi, but also in the severer cases of faucial diphtheria. The clinical type shows the characteristics of a secondary bronchopneumonia, with nothing characteristic of, or peculiar to, diphtheria. The diphtheria bacilli may be found in the areas of consolidation, but the process is due to a mixed infection, the commonest bacteriological finding being numerous streptococci. It is probable that the diphtheria acts as a predisposing cause. Bronchopneumonia is a frequent cause of death in severe cases of diphtheria, particularly in cases of laryngeal diphtheria which die in spite of intubation or tracheotomy.

THE HEART.—The toxin of diphtheria always affects the heart, signs of cardiac weakness having already been mentioned as characteristic of diphtheria, so that they constitute a symptom rather than a complication. The severity of the cardiac weakness is in proportion to the severity of the case. The weak pulse, dilatation and murmur have already been described. Cardiac weakness is the commonest cause of death in the malignant type. The blood pressure observations are not very reliable in children. The circulatory disturbance is probably more vasomotor than cardiac.

There is sometimes seen in diphtheria a peculiar form of heart failure which is characteristic of the disease and which causes *sudden death,* the "*Herztod*" of the German writers. Sudden cardiac failure may occur during the height of the disease. It is often preceded by malaise, pallor, abdominal pain, and a weak irregular pulse, but these symptoms are not severe enough to suggest so sudden a fatal ending.

More characteristic of diphtheria is the so-called post-diphtheritic heart failure, which occurs after the disease has subsided, in the fourth, fifth, or occasionally even as late as the eighth week. It is most often seen after severe cases, but occasionally occurs after typical or even mild cases. The children usually show marked prostration after the disease has subsided, and remain weak, and pale. The pulse remains rapid and irregular, but is sometimes slow at times—a still more dangerous sign. Every slight exertion greatly increases the frequency of the pulse. Occasionally there are attacks of weakness, sudden pallor, dyspnea, vomiting, or abdominal pain. These premonitory symptoms can go no farther, and can be recovered from in the course of one or two months. At times, however, after the child is believed to be wholly out of danger, sudden death may occur. The cause is believed to be either a myocarditis of toxic origin, or more commonly, a toxic degeneration of the nervous mechanism governing the heart.

THE EAR.—Otitis media is a frequent complication of diphtheria. In most cases it is not due to the Klebs-Löffler bacillus, but to a mixed infection, as in any infectious disease involving the throat. Secondary diphtheria of the ear, caused by the excursion of the specific organism from the throat to the nasal passages, and thence to the ear, through the Eustachian tube, can occur. Improper methods of treatment, such as unwise irrigation, increase the danger of ear complications.

THE KIDNEYS.—In most cases of diphtheria albuminuria is present, with numerous casts. This represents a degeneration of the epithelium of the convoluted tubules. A severe nephritis involving the glomeruli, with diminished urine and edema, is rare.

THE NERVOUS SYSTEM; POST-DIPHTHERITIC PARALYSIS.—This is the most important sequela of diphtheria. The toxin of diphtheria has an especial affinity for the nervous system, resulting in peripheral nerve paralysis. This is a late manifestation of the disease, and appears at a time when the ordinary toxic manifestations of diphtheria are over, usually in the second, third, or fourth week. It is a question whether this manifestation is due to the same poison as produces the other symptoms, or whether there are not more than one poison, paralysis being due to the "toxone" as suggested by Ehrlich. The anatomical basis for the paralysis is a peripheral nerve degeneration. The fact that cases have been observed in which there was membrane on only one side of the throat, and in which a unilateral paralysis of the palate occurred, and that in diphtheria of the skin the nearest muscles are most apt to be paralyzed, suggests that the poison reaches the central nervous system along the course of the nerves rather than through the blood.

The commonest type of post-diphtheritic paralysis involves the muscles of the soft palate. The symptoms are a *nasal voice*, and the fact that the *taking of fluid causes cough* and the *regurgitation of part of the fluid through the nose*. Inspection shows that the soft palate and pillars hang limp, and do not move with gagging, phonation, or inspiration. The paralysis may be unilateral. The palatal reflex is absent. Sometimes the muscles of deglutition are involved, so that swallowing is difficult or impossible. In palatal paralysis recovery occurs gradually in from two to four weeks.

Next in frequency to paralysis of the palate is paralysis of accommodation of the eye. This is often overlooked in young children, but if careful observation is employed, it will be noted that the fixing of the eyes on a finger or object held near the face is either not so complete, or not so prompt as it should be. Older children complain of inability to read and write. At times, in addition to paralysis of accommodation, there is paralysis of other muscles of the eye, which causes strabismus or double vision.

Besides these two most common types of post-diphtheritic paralysis, in rarer cases any of the other groups of muscles all over the body may be involved. In the lower extremities weakness or ataxia is commoner than actual paralysis. The muscles of the neck are sometimes involved. Very rarely is there paralysis of the back, face or larynx. Paralysis of all the muscles of the body has been observed. Paralysis of the diaphragm, or of other muscles of respiration may be dangerous to life.

Disturbances of sensation are very uncommon. In severe cases there may be incontinence of urine and feces.

In most cases of diphtheritic paralysis, although there may be no involvement of the lower extremities, there is *disappearance of*

the patellar reflex, which lasts a number of weeks, and is sometimes permanent. The electrical reaction of the paralyzed muscles shows a mild reaction of degeneration.

DIAGNOSIS OF DIPHTHERIA. BACTERIOLOGICAL DIAGNOSIS.—The positive diagnosis of diphtheria depends upon the finding of the Klebs-Löffler bacillus in the lesions present. Bacteriological examination should be made in every suspicious case. Although faucial diphtheria can often be recognized clinically with a fair degree of certainty, there is always some doubt without bacteriological proof, and the most typical-looking membrane may not be diphtheritic. So important is the early recognition of diphtheria, that I believe that a bacteriological examination should be made in every case with sore throat, or with any lesion or acute inflammation involving the throat. It should also be made in cases with nasal discharge and constitutional symptoms, or with unilateral or bloody nasal discharge.

The bacteriological examination may be made both by means of *cultures*, and of *fresh cover-glass preparations*. The results of cultures are more certain, but they require more time. I believe that both methods should be employed.

The material both for cultures and for fresh cover-glass preparations is obtained in the same way, by means of a small sterile cotton swab. This should be preferably rubbed at the edges or under the edges of the membrane rather than upon the surface. If typical membrane be not present, the swab should be rubbed upon any spots which may appear in the crypts of the tonsils, or if there be no spots, upon any part of the mucous membrane which appears most inflamed. If there is a suspicion of laryngeal diphtheria, the swab should be applied to the posterior wall of the pharynx. Two swabbings should be taken, one of which is rubbed upon the surface of Löffler's blood-serum culture medium, and the other upon the surface of the cover-glass. The culture should be incubated for twelve hours, and then cover-glass preparations are made from the surface of the medium. The fresh cover-glass preparation may be examined at once. The cover-glasses are dried, fixed, and stained with Löffler's methylene-blue. The diphtheria bacilli are recognized by their comparatively large size, by their irregular staining, and by their tendency to occur in clumps. In cases with any clinical symptoms, it is better for purposes of treatment to regard all microörganisms having a characteristic appearance as true diphtheria bacilli, although the pseudo-diphtheria bacillus cannot be excluded with certainty. In cases where it is a question of a carrier, it is best if possible to differentiate the true from the pseudo type. This can only be done by means of animal inoculation.

DIFFERENTIAL DIAGNOSIS OF FAUCIAL DIPHTHERIA.—While the

diagnosis of faucial diphtheria depends mainly upon the bacteriological examination, it is necessary that the physician be familiar with those conditions which bear the closest clinical resemblance to diphtheria. Among these are *catarrhal pharyngitis*, *follicular tonsillitis*, *membranous angina*, *Vincent's angina*, and atypical appearances in various *other forms of angina and stomatitis*.

A simple *catarrhal sore throat*, showing no membrane or exudate, is not clinically suggestive of diphtheria, and most cases of this type are not diphtheritic. Nevertheless, infection with the diphtheria bacillus may produce only a simple catarrhal inflammation, and such cases can only be recognized by bacteriological examination. It is for this reason that I recommend the taking of cultures from all cases of sore throat.

Diphtheria may show only bits of membrane in the crypts of the tonsils, resembling the appearance of *follicular tonsillitis*. On the other hand, in follicular tonsillitis the exudate in the crypts may increase in amount, and the areas may coalesce, so that all or a large portion of the tonsil may be covered, resembling the appearance of diphtheria. In follicular tonsillitis high fever is much more constantly present than in diphtheria. Moreover the exudate in tonsillitis is usually yellowish, sticky, purulent, and foul-smelling, and is more easily wiped off than is the membrane of diphtheria, which is whiter, tougher, and more adherent. Most cases with discrete spots in the crypts of the tonsils are tonsillitis, but the differential diagnosis can only be made with certainty by bacteriological examination.

A membranous lesion of the tonsils is not always diphtheria. It occurs frequently in scarlet fever, and occasionally from other causes. The *scarlet fever angina* is recognized by the other features of the disease, but bacteriological examination is essential in every case of membranous angina.

Vincent's angina may bear a close resemblance to the lesion of diphtheria. It usually involves only one tonsil, and may often be recognized by the fact that destruction of tonsillar substance and ulceration begin early. In diphtheria the membrane appears to be plastered over the surface of the tonsil, while in Vincent's angina it often appears to fill a large ragged crater. The inflammatory reaction of the tissues around the lesion is much less in Vincent's angina than in diphtheria. The gangrenous odor from Vincent's angina is often marked enough to be characteristic. Constitutional disturbance is less marked in Vincent's angina than in diphtheria. While all these points may enable the physician to make a probable clinical diagnosis, certainty can only be attained by bacteriological examination. In Vincent's angina fresh cover-glass preparations are characteristic, showing the bacillus fusiformis with its associated spirochetae.

In certain cases atypical forms of thrush, stomatitis herpetica, syphilis of the mouth or throat, and Bednar's aphthae may be mistaken for diphtheria. Bacteriological examination will settle such doubtful questions.

DIFFERENTIAL DIAGNOSIS OF LARYNGEAL DIPHTHERIA.—The diagnosis of laryngeal diphtheria in cases which have already shown the evidences of faucial or nasal diphtheria, is not difficult. The signs of laryngeal obstruction, hoarseness, harsh cough, and increasing inspiratory dyspnea are easily recognized, and in these cases their connection with the diphtheria infection already present is obvious.

Difficulty is encountered in the cases, not uncommon, in which the laryngeal process is the primary manifestation of the disease. The clinical diagnosis mainly depends on the *steadily increasing* signs of laryngeal obstruction. Bacteriological examination of material taken from the pharynx often shows the presence of diphtheria bacilli, particularly in cultures. On the other hand, the results of cultures cannot be used in excluding laryngeal diphtheria, as the cultures are often negative. Consequently, the clinical diagnosis is more important than in faucial diphtheria.

The conditions to be considered in differential diagnosis are all which may cause obstructive dyspnea. The most important are *acute laryngitis* (*spasmodic croup*, *common or false croup*), *retropharyngeal abscess*, and *foreign body in the larynx*. Occasionally the dyspnea from thymic enlargement, or pressure from enlarged bronchial lymphnodes, has led to a suspicion of laryngeal diphtheria, but the diagnosis is usually clear from a careful history of the case. Papilloma of the larynx produces symptoms which come on so much more gradually, that it is rarely confused with laryngeal diphtheria.

In *spasmodic croup*, the attack is sudden, usually occurs at night, and is spasmodic in character. The voice is almost never lost. The attack usually remits, and obstructive dyspnea disappears the next morning. Rarely some dyspnea may persist on the day following the attack, but it is well to treat any such case as diphtheria. . The history of previous attacks points toward spasmodic croup.

In the severe form of *acute catarrhal laryngitis*, with dyspnea, but without spasm, the diagnosis may be more difficult, especially in the first twelve hours. Laryngoscopic examination will often clearly differentiate the two conditions, but it is neither practicable nor conclusive in infants. The dyspnea in catarrhal laryngitis is more likely to be paroxysmal and remittent, and is mainly inspiratory, while in laryngeal diphtheria it is both inspiratory and expiratory, and increases steadily. The voice is usually not lost in catarrhal laryngitis, and the temperature is usually higher than in diphtheria. In cases in which any doubt exists, it is best to give antitoxin at once.

In *retropharyngeal abscess* there is increasing obstructive dyspnea, and the extension of the inflammation of the mucous membrane to the larynx often causes hoarseness and cough. There is not usually loss of voice in retropharyngeal abscess, and the dyspnea is of a somewhat different character, often accompanied by a peculiar sound on expiration, and with inspiration proportionately less labored. The diagnosis depends on the results of careful examination of the pharynx, both by inspection and by exploration with the finger.

I have mistaken *foreign body* in the larynx for laryngeal diphtheria. Usually with foreign body the onset of obstructive symptoms is sudden, not preceded by hoarseness and cough. The history is often of aid. Occasionally, however, the foreign body does not at once produce marked obstruction, but causes an inflammation which leads to an increasing severity of symptoms. In such cases, with an unsatisfactory history as to the disappearance of any plaything or other object, the diagnosis is difficult, and can only be made by laryngoscopic examination.

DIAGNOSIS OF NASAL DIPHTHERIA.—The taking of cultures from every case with nasal discharge would be required in order to be sure of the recognition of every case of nasal diphtheria. This, while theoretically desirable, would involve so much labor, on account of the frequency of catarrhal rhinitis, that it will probably never be carried out. I would advise, however, that cultures be taken in all cases with *unilateral discharge, blood stained discharge, or unduly persistent discharge.*

DIAGNOSIS OF POST-DIPHTHERITIC PARALYSIS.—This condition is usually so characteristic, that even in cases with a defective previous history, or in which the diphtheria was of so mild a type as to have been overlooked, the recognition of the diphtheritic origin of the paralysis is usually easy.

PROGNOSIS.—Diphtheria is an extremely fatal disease, especially the septic and obstructive cases. The mortality varies decidedly in different epidemics and according to the age. It is greatest in children under two years of age, but has been lessened in cases in which the antitoxin treatment has been thoroughly used. In the acute stage, death may occur either from laryngeal stenosis, bronchopneumonia, or by the effect of toxin generated from the growth of the bacillus. In the later stages or during convalescence it may be due to the action of the toxin on the nervous centers. The symptoms which make the prognosis especially unfavorable are the extension of the membrane to the naso-pharynx or the larynx, profuse nasal discharge, marked septic odor, extensive glandular enlargement, hemorrhage from the nose or into the skin, a high grade of albuminuria, bronchopneumonia, and a weak heart. In cases of post-

diphtheritic paralysis the prognosis is good, as they almost invariably recover. The prognosis in all cases of diphtheria is uncertain and should be given with caution, and no case of diphtheria should be considered benign, for at times in certain mild cases serious symptoms may arise, and death from heart failure is liable to occur at any stage of the disease. A child who has had diphtheria is liable to suffer from the deleterious effects of the disease for months and even years. Before antitoxin was used, the mortality from diphtheria in the Boston City Hospital was 50 per cent. Since its use in a series of 4500 cases in the diphtheria wards of the Boston City Hospital the mortality has been reduced to 13 per cent., and if the moribund cases, which numbered 179, by which are meant those dying within twenty-four hours after admission to the hospital, are deducted the mortality was about 10 per cent.

PROPHYLAXIS.—As diphtheria is a disease having the same degree of contagiousness and the same mode of transmission as scarlet fever, the measures of isolation, quarantine, and disinfection employed in preventing the spread of the disease are much the same. The diphtheria patient should be isolated, with the nurse, and a most rigid quarantine should be established, the arrangement and management of the sick room being the same as for scarlet fever. Whenever such strict isolation and quarantine cannot be obtained in a private house, the patient should be sent to a diphtheria hospital. The quarantine must be very rigidly enforced in the case of the nurse who cares for a diphtheria patient, on account of the fact that a healthy person may become a carrier of virulent Klebs-Löffler bacilli. The nurse of a diphtheria patient should come in contact with no other person. Similarly, no member of the patient's family, and no other person, should be allowed within the quarantined quarters, unless such person is prepared to submit to permanent quarantine with the patient.

The only exception is the physician, who must visit the patient from time to time. He should take the most rigid precautions against a possible contamination of his person with infected material, wearing gown, cap and gloves as described for scarlet fever, and being most thorough in cleansing his hands and face after each visit. I further believe that every physician caring for a case of diphtheria should take frequent cultures from his own throat, in order to make sure that he has not become a carrier.

The other children in the family in which a case of diphtheria has occurred, should be kept out of school, and should not be allowed to play with other children. Cultures should be taken from the throats of every member of the household, and of every person who has been exposed. This is important in detecting mild incipient

cases of diphtheria as well as carriers. All persons in whom a positive culture is obtained should be placed under quarantine.

Not only should every undoubted case of diphtheria be immediately isolated, but also every suspected case. It is my practice to isolate every case of sore throat occurring among the children under my care, until the results of cultures for diphtheria, and the expiration of the prodromal period of scarlet fever, have demonstrated that neither of those diseases is present. In an epidemic of diphtheria every sore throat and nasal discharge is particularly suspicious, and the child should be isolated until the results of the cultures are known. While such extreme watchfulness is generally only recommended in institutions, I believe that it is equally important in private families. It should be remembered, however, that cases isolated on suspicion only of a contagious disease, should be kept by themselves and not exposed to possible infection from other cases, until it is certain that they actually have the disease. In cases in which the clinical manifestations are strongly suggestive of diphtheria, in times of epidemic, and in persons who have been actually exposed, the negative result of a single culture should not be accepted as conclusive, but several, at least three, should be taken before quarantine is relaxed.

The immunization by giving antitoxin of individuals who have been exposed to diphtheria will prevent the development of the disease. It does not, however, prevent an individual from being a carrier, and should not be allowed to take the place of the taking of cultures from the noses and throats of such persons. Until recently the immunization of every person exposed has been recommended. Since it has been shown that many children have a natural immunity, which can be demonstrated, it is not necessary to immunize a child who has a negative Schick reaction. *Every exposed child, however, who has a positive Schick reaction, or on whom the Schick reaction is not performed, should receive an immunization dose of antitoxin.* The quantity of antitoxin which has usually been recommended for immunizing purposes is 500 units for infants, and 1000 to 1500 units for older children. Recently numerous writers have been recommending the proportioning of both prophylactic and therapeutic doses of antitoxin to the body weight of the patient, which gives us a more definite procedure. For immunizing purposes Schick recommends 50 units per kilogram of body weight.

In hospitals devoted to the care of children, the practice of periodic immunization of all patients admitted to the hospital has proved highly successful. At the Children's Hospital, Boston, from May 1, 1897 to October 1, 1905, 10,575 patients were treated, and at the Infant's Hospital, from February 1, 1900, to October 1, 1905, 1276 cases were treated. During these years, under prophylactic immuniza-

tion, no case of diphtheria broke out in the wards. At present at the Infant's Hospital a routine Schick reaction is performed on every case, and only those not having natural immunity receive antitoxin.

The length of time during which quarantine should be continued depends on the results of bacteriological examination. The patient must not be released until the bacilli have disappeared from the mucous membrane. This time is very variable. In about half the cases the organisms disappear within three days after the membrane is gone, while in the great majority of the remaining cases they disappear within a week. In certain individual cases they may persist for a much longer, indefinite period, which may reach many weeks. The results of a single culture should not be taken as conclusive. The Boston Health Board requires three successive negative cultures, which I believe to be a wise rule. Cultures should be taken from both the throat and the nose.

Many cases in which the bacilli persist for a long time are instances of nasal diphtheria. It has been shown that in many of these cases the bacilli are non-virulent. In healthy persons in whom positive cultures are obtained, the bacilli may also be non-virulent. It has been questioned whether the isolation of healthy carriers, and of cases of nasal diphtheria with continued positive cultures, is desirable or necessary. It has also been shown that in many of these cases of nasal diphtheria or of healthy carriers the bacilli are very virulent. Therefore it is better to isolate all such cases unless the bacilli are shown to be non-virulent by animal experimentation.

The measures which are employed in diphtheria in the disinfection of clothing and utensils, are the same as those described for scarlet fever. Also the measures which are taken upon the release of a child from quarantine, and in the subsequent disinfection of the sick room, are in no way different from those used in scarlet fever, and have already been fully described.

TREATMENT. SERUM THERAPY.—It is in diphtheria that serum therapy has won its greatest triumph, and for this reason the antitoxin treatment of diphtheria must first be considered. The immune body through which recovery from diphtheria occurs is an antitoxin, which acts by neutralizing the toxin which causes the injurious effects of the disease. The serum of horses which have been treated with repeated injections of a filtrate from cultures of the Klebs-Löffler bacillus, contains a large quantity of this immune body. The antitoxic power of such serum is measurable, as the neutralizing action of the immune body is as definite as a chemical reaction. The unit of measure is the amount of antitoxin which will protect a guinea-pig weighing 250 to 300 grammes against one hundred times the fatal dose of diphtheria toxin. Behring's original serum contained this

amount in one c.c. of horse serum, but improvements in the method of preparation have permitted a much greater degree of concentration of the serum, so that many units are contained in one c.c. In using the serum, the physician must know the number of antitoxin units to the cubic centimeter in the particular serum employed. Only serum prepared by a reliable manufacturer should be used. When kept properly in a cold ,dark place, preferably upon ice, serum will retain its full antitoxic properties for a considerable time. If, however, the serum is more than six months old, it is better if possible to obtain a fresh preparation.

While the chief result of the injection of immune serum in diphtheria is the neutralizing of the toxin, there is also evidence of some injurious action upon the bacilli themselves, the nature of which is as yet imperfectly understood. On account of the nature of the immune body, the serum used in the treatment of diphtheria is genally called *antitoxin*. This is not strictly correct; the antitoxin is the principal immune body contained in the serum.

Indications for Serum Therapy.—While the beneficial results of the serum treatment of diphtheria are now generally recognized, there are some authorities who, on account of the untoward effects which sometimes follow the administration of antitoxin, advocate that it be not used in certain mild cases in older children. I do not agree with this opinion. One can never know beforehand that a case of diphtheria will continue to be mild throughout its course, as severe symptoms may appear unexpectedly at any time. Furthermore the advantages of an early administration of antitoxin are very great. I believe, therefore, that serum therapy should be employed in every case. The question does arise as to whether antitoxin should be given to suspected cases immediately, and before the bacteriological investigation has confirmed the diagnosis. This depends on the severity of the case, and the amount and character of the clinical evidence pointing toward diphtheria. I would summarize the indications for serum therapy as follows:

1. In all cases proved to be diphtheria by positive bacteriological investigation.

2. In cases in which, before the bacteriological examination is completed, membrane is seen.

3. In cases in which the patient, while not having typical membrane, shows appearances in the throat compatible with diphtheria, and marked constitutional disturbance, or any dangerous and threatening symptoms, while the physician is waiting for the results of the cultures.

4. In cases which have developed any signs of laryngeal obstruction which cannot definitely and certainly be attributed to some other cause.

The dose of diphtheria antitoxin.—This varies greatly with the age of the child, with the situation of the lesion, and with the severity of the case. The initial dosage may be summarized as follows:

1. In mild cases of diphtheria involving the throat or nose alone, 3,000 to 5,000 units.
2. In cases of average severity, involving the throat alone, 5,000 to 7,000 units.
3. In severe cases of faucial diphtheria, cases involving both the throat and the nose, and average cases of laryngeal diphtheria, 7,000 to 10,000 units.
4. In malignant diphtheria and severe cases of laryngeal diphtheria, 10,000 to 15,000 units.

In each group enumerated above, the lower figures apply to younger children, and the higher figures to older children.

Schick has recently done some experimental work on the dosage of diphtheria antitoxin, measuring the results by the effect upon his cutaneous reaction. He recommends the following dosage:

1. In mild cases, 100 units per kilogram of body weight.
2. In severe cases, 500 units per kilogram of body weight.

The injection of serum should produce a noticeable and continued beneficial effect. If after eight hours such an effect is not seen, or if at any time this improvement ceases, the injection of antitoxin should be repeated.

The method of administering antitoxin serum in diphtheria.—The serum was formerly injected into the subcutaneous cellular tissue, the seat of the injection being of no particular importance. It has been shown that absorption from muscular tissue takes place much more rapidly than from the subcutaneous tissue, and that intramuscular injections are less painful. Consequently, ***as a routine, intramuscular injections should be used.*** The preferable points for the injection are either the muscles of the outer side of the thigh or the muscles of the gluteal region. The skin should be thoroughly cleansed with water and alcohol, and the needle and syringe should be boiled.

Animal experimentation has shown that the intravenous injection of antitoxin produces a much more rapid effect than intramuscular injection. The intravenous route has been more and more used of late, and the most favorable results have been reported. I believe that intravenous injection should be employed in all very severe cases, in which the serious condition of the patient demands a rapid effect. The ease with which intravenous medication can be used in infants, in whom the open fontanelle gives ready access to the longitudinal sinus, has led me to adopt the intravenous method of injection in all severe cases in infants.

The beneficial effects of serum therapy.—In diphtheria treated with antitoxin, the signs of the favorable action of the serum are unmis-

takeable. The temperature and pulse rate begin to fall within the next eight hours, and the general condition improves. During this period the membrane ceases to spread. In laryngeal cases the obstruction may increase for the next twenty-four hours, owing to the necrosis of epithelium injured before the injection, but after this time it diminishes. The favorable effect is further shown by the formation around the membrane of a distinct red line of demarkation, by the fact that the membrane shrinks, softens, rolls up at the edges, and finally after two or three days, loosens, and is cast off. The effect on the process in the nose is seen in cessation of the discharge. The swelling of the cervical lymphnodes diminishes. The membrane in the larynx and trachea loosens and is cast off rapidly, and frequent removal of the tube is sometimes required when intubation has been performed, to clear the larynx of detached membrane.

Value of early treatment.—The results of serum therapy in diphtheria depend very largely on the time of administration of the serum. The antitoxin can only prevent further damage, but cannot remedy damage already done to the tissues. The cells of the heart and nervous system are particularly susceptible to the deleterious action of the diphtheria toxin, and death is likely to occur at the height of the disease from cardiac failure, or from cardiac or respiratory paralysis. Damage sufficient to cause a fatal ending may occur in a variable period which may be very short, and this damage cannot be undone by the subsequent administration of antitoxin. Many severe cases are fatally injured before antitoxin is given, and the importance of early diagnosis and treatment cannot be overemphasized.

It must also be remembered that antitoxin is powerless against secondary streptococcus infection, such as phlegmonous inflammation of the throat or cellular tissue, bronchopneumonia and suppuration of the lymphnodes. It also does not prevent the occurrence of post-diphtheritic paralysis, which is not, however, a serious sequela.

Results of serum therapy in diphtheria.—The antitoxin treatment has since 1895 been so thoroughly tested all over the world, that it is no longer necessary to cite figures and statistics in support of its value. The percentage mortality in hospitals has been reduced to about a third of the previous figures. The mortality in all cities and countries where antitoxin has been used has been very strikingly reduced. There has been a striking reduction in the percentage of cases requiring operation for laryngeal stenosis, and in the mortality after tracheotomy or intubation.

The importance of early administration is emphasized by some statistics recently collected by Deycke, based on 78,028 cases.

TABLE 45

Mortality in Serum-treated Cases of Diphtheria, According to the Time of Injection

TIME OF INJECTION	MORTALITY
1st day	4.3%
2nd day	7.6%
3rd day	14.7%
4th day	19.7%
5th day	31.6%
6th day	31.3%
After 6th day	31.6%

Untoward effects of serum therapy; serum sickness.—When one injects into an animal organism the serum of a foreign species, there may occur a reaction which causes pathological symptoms. These are in no way due to the specific antibodies contained in the serum, and are seen in any form of serum therapy, but as horse serum is used more in diphtheria than in any other disease, these manifestations of serum reaction are best described in this place. The symptoms are not seen in every patient, but only in about 10 to 20 per cent, and appear to depend upon some constitutional peculiarity. Some individuals will always show a serum reaction, even when serum from different horses is used. On the other hand, the serum from a particular horse will sometimes produce a reaction in every patient, while the same patients do not react to the serum from another horse.

In susceptible individuals, there appears in the days after the injection of serum a swelling and sensitiveness to pressure of the lymphnodes which drain the site of the injection. This may persist two or three weeks. The commonest clinical manifestation is the ***exanthem***, or ***serum rash***, which belongs in the class of toxic erythemata. The serum rash appears usually from the seventh to the twelfth day after the injection. It begins ordinarily at the site of the injection, and may remain limited to this area, but more commonly spreads with an irregular distribution over the entire body. The commonest appearance is an urticaria-like erythema, with very large irregular areas. There may be smaller areas of erythema in between, with spots resembling those of measles. Scarlatiniform eruptions are also seen at times, but are rare, as are hemorrhagic lesions. Itching is usually severe. Fever may accompany a serum rash, and may last several days. Malaise and pain in the joints are seen at times. During serum sickness there is usually a pronounced diminution in the white blood count, the leukopenia being due to a drop in the number of polymorphonuclear cells. At times precipitins for horse serum can be demonstrated in the serum of the patient.

The diagnosis of a serum rash depends mainly on the history that horse serum has been injected. The itching, the irregularities in the character and distribution of the eruption, and the fact that

it never involves the mucous membrane of the mouth, are important points in differential diagnosis.

These serum rashes usually last for two or three days, but are rarely severe for more than twenty-four hours.

A more severe serum reaction is sometimes seen, although instances are rare. Such a severe reaction may occur in a particularly susceptible individual after the first injection, but is seen more often after a second injection made during the period from about twelve days to three months after the first. In such cases the reaction appears immediately, from within a few minutes to within a few hours after the injection. There is marked malaise and muscular pain. Severe edema and erythema rapidly appear at the site of the injection, more rarely a marked edema of the face, a general urticaria, and occasionally marked dyspnea and threatening collapse, are seen. Usually these manifestations subside rapidly. In a very few instances sudden death has followed the injection of antitoxin, but the evidence has not always been clear that the injection was the cause of death. In a few cases, however, the death seemed to be due to the serum injection. In so many of these cases the patients have been asthmatic, that the association cannot be accidental, and some of them have been known to have the form of asthma which is undoubtedly an anaphylactic phenomenon, excited by contact with horses.

I have not space in this book to go into a full description of the phenomenon of anaphylaxis on which the untoward manifestations of serum therapy depend. The reaction depends upon the fact that the horse serum acts as a foreign body, against which the human organism forms antibodies, which are produced in from seven to twelve days after the injection, and which cause the toxic manifestations. In cases in which a rapid severe reaction is seen, the tissues are so sensitive that they form antibodies with great rapidity. Such sensitiveness may be a constitutional peculiarity, or may have been produced by a previous injection.

The question has been raised whether the possible dangers from anaphylaxis are sufficiently great to affect the indications for serum therapy. Certainly in a dangerous disease like diphtheria, the possibility of serum sickness should not deter the physician from using antitoxin as early as possible in every case. The only exception would be in a known asthmatic, to whom it is better not to give horse serum in mild cases of diphtheria. The possibility of anaphylaxis has led some authorities to advise against the use of *immunizing* doses of antitoxin, on the ground that in susceptible individuals serious symptoms have followed small doses, and that the risk should not be taken until clinical diphtheria is actually present. Furthermore, anaphylactic phenomena are seen most often after a repetition of the serum injection, and an immunizing dose would be a source of

danger if a subsequent therapeutic dose ever became necessary. In my opinion the danger from anaphylaxis is too slight to counterbalance the advantage to be derived from immunizing injections of antitoxin. The routine practice at both the Children's and Infant's Hospitals for years was to give an immunizing dose to every patient admitted, and to repeat the injection every three weeks as long as the child remained in the hospital. Instances of severe anaphylactic manifestations have been exceedingly rare. Immunizing injections, however, should never be given to asthmatics. Furthermore, in children the use of the Schick reaction has greatly reduced the number of cases in which immunizing antitoxin injections are indicated.

GENERAL MEASURES IN DIPHTHERIA.—The general measures of nursing and hygiene which are used in any severe acute illness, are applicable to diphtheria. Fresh air and sunlight are particularly important. Diphtheria patients should be kept in bed throughout the entire attack, and for a longer period during convalescence than is required after any other acute illness of the same comparatively short duration. As long as there are any signs of cardiac weakness, as long as the child has not regained its strength, absolute rest in bed should be continued, even though fever and local symptoms have been gone for weeks. The child must be got out of bed gradually, the heart being carefully watched.

The nutrition of diphtheria patients is often a matter of considerable difficulty. Anorexia is frequently extreme, both on account of the pain on swallowing, and on account of the septic condition of the child and the ease with which vomiting is produced. Milk is the main reliance; it should not be diluted unless distinct symptoms of indigestion are produced. Cereal gruels may be given to older children. Fresh fruit juices are often better taken than water, and plenty of fluid is required. In severe and protracted cases, beef juice should be added to the diet. As convalescence comes on, zwieback scraped beef, bread, eggs, and so forth, are gradually added, till the child returns to its normal diet. Breast-fed infants with diphtheria should not be put to the breast, but should be fed with breast-milk obtained with the breast-pump.

When anorexia is very severe, it is sometimes better to give nourishment in small amounts more frequently; but it is better not to disturb the child oftener than every two hours. In obstinate cases, it is necessary to resort to gavage.

In children who have been intubed, or who have post-diphtheritic paralysis, the swallowing of fluids is often more difficult than that of foods of more solid consistency. In such cases junket, milk toast and jellies are useful.

Stimulation.—Owing to the action of the diphtheria toxin upon

the circulatory system, stimulants are frequently required. The indi
cations for stimulation are a very rapid, feeble, or intermittent puls
a weak first heart sound, and marked prostration. The stimulan
which have formerly been most widely recommended in diphther
are alcohol and strychnine. As a result both of clinical experien
with these drugs, and of recent experimental work upon their acti
as circulatory stimulants, I have given up their use in the acu
stage of diphtheria. Much better are the drugs whose value as c
culatory stimulants has been adequately proven, namely, *caffei*
camphor and *digitalis*. In the acute stage, the best routine stin
lant is caffeine-sodium benzoate or salicylate, given in doses sui
to the age of the patient. (See Division II.) Digitalis is useful w
cardiac weakness persists after the acute stage. Camphor in oi
indicated in sudden threat of cardiac failure. In severe colla
with cold extremities and cyanosis, due to vaso-motor paral
adrenalin is useful. Alcohol is most useful in cases where nutri
is difficult and prostration prolonged. Strychnine in small reg
doses three times a day should be used in the treatment of p
diphtheritic paralysis.

There are few other symptoms beside circulatory weakne
diphtheria which require internal medication. The giving of c
is disturbing to the child, no drug influences the course of the dis
and all drugs should be avoided except necessary stimulants.

LOCAL TREATMENT.—Since the introduction of serum therap
local treatment of diphtheria has retired into the background.
painting and spraying of the throat, which was formerly r
mended, is bad practice, and can do harm. Local treatment s
not be entirely abandoned, but its benefits are not great enou
warrant its use when it encounters marked resistance on the p
the child, as the exhaustion caused by resistance is more h
than any condition which local treatment can remedy.

The mouth can usually be kept clean by alkaline washes, w
causing resistance. Older children can often gargle the throa
a solution of boric acid, or Dobell's, or Seiler's solution, or
one per cent solution of hydrogen peroxide. In younger c
the same solutions can be used for syringing the mouth and
unless this causes great resistance. I do not believe in sy
through the nose in diphtheria. Even in septic cases with a
fetid discharge, nasal syringing should only be used in exc
cases. In nasal diphtheria one should rely mainly on serum t
and should rest content with removing as much secretion as
with a cotton stick, and with protecting the upper lip with
or some other simple salve.

For the painful swelling of the cervical lymphnodes,

should never be employed. The application of cold b the ice bag is indicated in such cases.

CONVALESCENCE.—This is slow after a severe attack o No matter how rapid the apparent recovery, no patien allowed out of bed for at least a week after the disap the membrane. The heart should be carefully watche pulse remains abnormally rapid, slow, or weak, or is dicrot mittent, if there is any weakness of the first heart sound recumbency in bed must be continued. A slight irregular cardiac rhythm is not a contraindication to the child getting about, provided that no other signs of cardiac wea present. The appearance of the typical post-diphtheritic also does not require the child to be kept in bed. As lo child does not regain its full general strength, it shoul kept in bed.

Iron, and other tonics are sometimes indicated in conv for anemia or malnutrition.

The persistence of diphtheria bacilli in the throat is occ an annoying feature of the convalescence. Sometimes posi tures continue to be obtained after the child has become fu valescent, and this prevents release from quarantine. T means of getting rid of the bacilli is to syringe through the nos or four times daily, and in addition, children old enough should frequently. Simple salt solution, Dobell's solution, or bori solution should be used. It is doubtful if the dilute soluti bichloride of mercury (1 to 10,000) sometimes recommended, any real antiseptic value.

TREATMENT OF LARYNGEAL DIPHTHERIA.—In the early stag laryngeal diphtheria, before obstruction has become marked, physician, while waiting for serum therapy to produce its effect, try local measures before resorting to operative treatment. A w pack is sometimes useful. Hot fomentations should be applied the region of the larynx. The use of steam by means of the cr kettle and bed tent, as employed in common croup, may be tried. emetic dose of ipecac may be given, as for common croup. T opiates, however, should be avoided.

If in spite of these measures, the obstruction shows a tendenc to increase, the physician must be prepared to resort to operativ interference. No hard and fast rule can be laid down as to whe operation is indicated. More harm has been done by waiting to long than by operating too early. One should never wait for th appearance of cyanosis, as this sign is a precursor of death. I general when the obstruction is increasing, and has resulted in marke supraclavicular and epigastric inspiratory retraction, or if the ob

structed breathing appears to be exhausting to the patient, or if there is any tendency toward somnolence or toward suffocative attacks, interference should not be longer deferred. Some German writers state that the moment for interference can be definitely fixed at the time when the sterno-cleido-mastoid muscles are brought into action as auxiliary muscles of respiration, the contraction with inspiration being detected by palpation.

When operative interference becomes indicated, the question which formerly had to be decided was whether to use *intubation* or *tracheotomy*. It is now generally recognized that intubation is the best primary operation for the relief of membranous laryngitis, tracheotomy being reserved for those cases in which for some reason intubation fails. The drawbacks to intubation are annoyances rather than real evils; the most conspicuous occasional troubles being obstinate difficulty in swallowing, repeated coughing up of the tube and ulcerations from pressure. The drawbacks to tracheotomy are the necessity of narcosis and trained assistance, the danger from hemorrhage and wound infection, the long period before the canula can be removed, and the frequency of sequelae such as disturbance of voice, decubitus, and tracheal stenosis.

INTUBATION

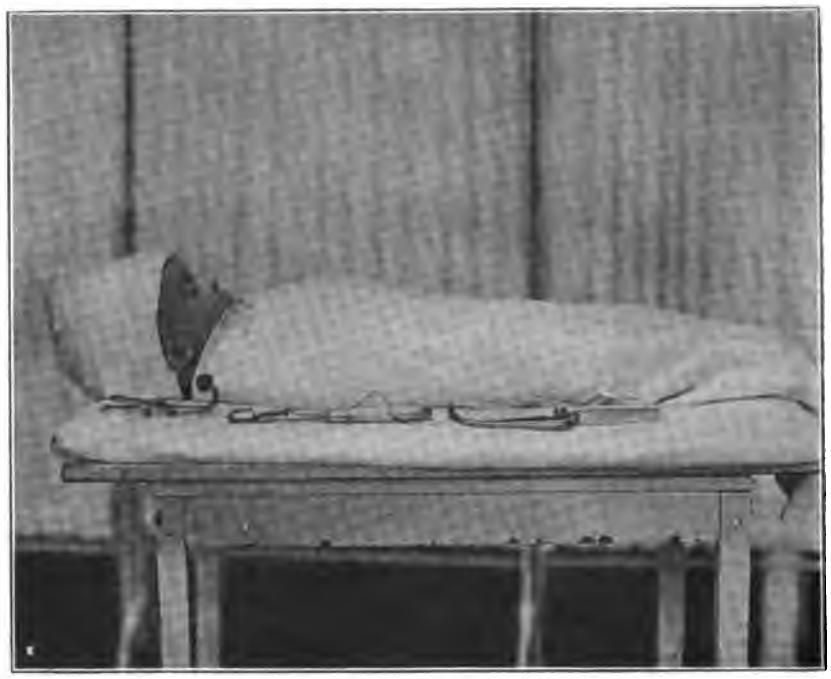
Intubation is the introduction through the mouth into the larynx of a tube of special construction. The instruments and technique were devised by Dr. Joseph O'Dwyer of New York, and were so perfected by him, that practically no improvements have since been added.

An intubation set consists of seven tubes of graded sizes, an introductor, an extractor, a mouth-gag and a gauge. The tube is selected according to the age of the patient, as indicated on the gauge. A large child often requires a larger tube than the one called for by its age.

TECHNIQUE OF INTUBATION.—The patient is wrapped firmly in a blanket, so that he cannot move his arms, and then placed in a horizontal position, with the head slightly raised The mouth is held open by the gag, with its jaws resting on the molar teeth. The gag should be on the left side. Care must be taken not to have the cheek injured by the gag, and special pains must be taken to prevent its slipping. The head should be steadied by the assistant who holds the gag. The operator takes the introductor carrying the tube in the right hand with the index-finger around the hook on the under surface of the handle, the loop of silk passing over his little finger, and his thumb resting on the button on the upper surface of the handle. The index-finger of the left hand is then passed down to the epiglottis, which is hooked forward. The finger on entering the

pharynx is brought forward until it encounters the upper border of the cricoid cartilage which is felt as a hard nodule, and is a useful landmark. The epiglottis lies immediately in front, and should be drawn strongly forward. The tube is now introduced into the mouth, the handle of the introductor being well down on the chest of the patient. The tube is kept vertical in the middle line, and its tip is passed down the radial side of the left index-finger which serves as a guide, the handle of the introductor being raised to permit the tube to follow the course of the finger. When the tip of the tube approaches the tip of the finger, the operator moves the finger slightly toward his left while still maintaining the forward pressure on the epiglottis

FIG. 108



Apparatus for intubation

and tongue, and raises the handle of the introductor to a vertical position, which allows the tip of the tube, still following the radial side of the finger, to enter the opening of the larynx. It is best to choose for this moment of introduction, an inspiratory effort on the part of the child, as at such a time the glottis opens most widely. When the tube has entered for about one-third of its length, the operator should feel around it rapidly with the left index-finger, in order to be sure that it is in the proper position. If it has entered properly, it will be closely surrounded by a ring of soft tissue. The

right thumb then presses forward the thumb-piece, disengaging the introductor, which is withdrawn. The left index-finger is placed on the head of the tube, and pushes it down into place. It is well before withdrawing the finger to make sure once more that the tube is tightly surrounded by a ring of soft tissue. Further signs that the tube is properly in place are a peculiar hissing breathing sound, a paroxysm of coughing, and relief of the dyspnea. While waiting for these signs the operator passes the silk loop about the ear of the patient, and removes the gag.

The silk loop is best left attached to the tube, whenever there is evidence of loose membrane below, or when the patient is to be left in the care of someone who would not be able to remove the tube with

FIG. 109

Intubation. Beginning the insertion of the tube

the extractor in case it should become obstructed. In such a case the silk passes out at the corner of the mouth, and is attached to the cheek with a piece of adhesive plaster. The child's hands must then be secured so that he cannot seize it. When anyone is to be constantly within reach who is competent to use the extractor, it is best to remove the silk loop from the tube. For this purpose one strand of the loop is cut, the gag is inserted a second time, the left index-finger is placed on the head of the tube, and the loop pulled through and free from the tube.

It cannot be overemphasized, that very little force is required for the passage of the tube into the larynx, no more than is used in the passage of a catheter through the urethra into the bladder. The operation must be performed quickly, as sometimes the holding of the mouth open with the gag will cause a cessation of respiration. The entire manipulation can be completed in a very few seconds

The most common mistake made in intubation is the passage of the tube into the esophagus instead of into the larynx. This is recognized by the following facts: The finger in the pharynx does not find the posterior wall of the larynx between it and the tube; the tube can be pushed down indefinitely, instead of coming to a point where it can be pushed no farther down without depression of the

FIG. 110

Intubation. Completing the insertion of the tube

whole larynx, and it may continue to pass downward as shown by shortening of the silk loop; the hissing breathing, paroxysm of coughing, and relief of dyspnea do not occur. When these signs show that the tube has been passed into the esophagus, it is withdrawn by means of the silk loop, and after a few minutes a second more careful attempt is made.

False passages are sometimes made, the tip of the tube going into one of the ventricles of the larynx. It can be pushed quite through into the cellular tissue, but this would only happen from the use of the most brutal force. The cause of the tube entering the ventricle

is that the operator works from the angle of the mouth, instead of keeping the tube in the median line. A false passage of this kind is recognized by the fact that although the tip of the tube is felt to enter the opening of the larynx, it cannot be pushed down into place without undue force, and the head of the tube projects above the epiglottis. The tube must be withdrawn, and another attempt made, with care to keep tube and introductor always in the median line.

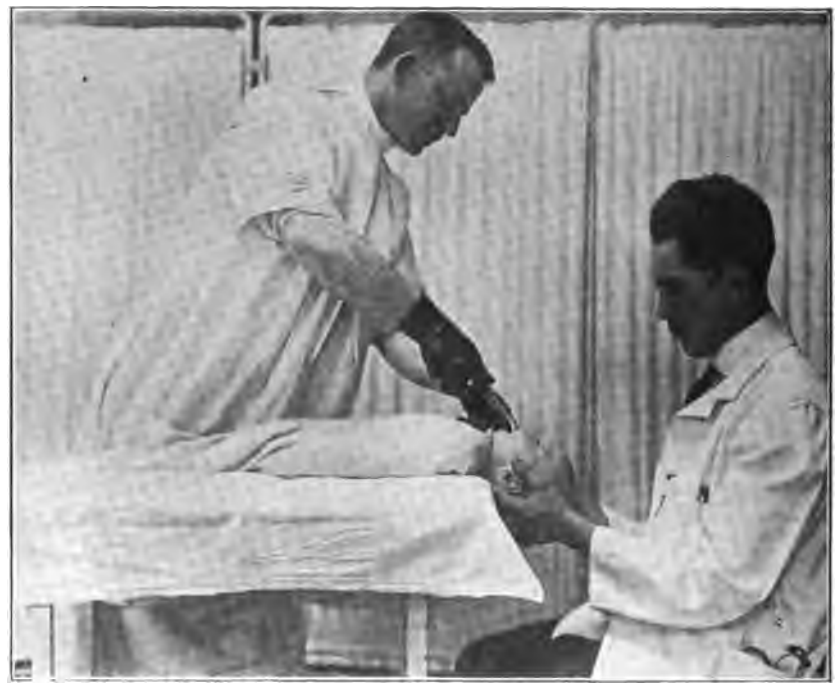
Rarely the operation of intubation shows all the signs of having been properly performed, except relief of dyspnea, and instead of relief, breathing appears to have become more or completely obstructed. This is serious, and usually means that loosened membrane has been pushed down ahead of the tube and is obstructing the larynx, though it may be due to an unskillful prolongation of the operation. The tube must be immediately withdrawn, and sometimes this is followed by a paroxysm of coughing in which the obstructing membrane is expelled. If this does not occur, give the patient half a teaspoonful of whiskey or brandy undiluted to excite cough. If there is still threatening asphyxia, try artificial respiration, and if no air enters, tracheotomy must be performed at once.

CARE OF INTUBED CASES.—The feeding of patients who have been intubed is sometimes difficult. Infants who take their food by sucking usually have no difficulty, but older children sometimes experience considerable difficulty in swallowing, especially in the taking of fluids. At other times the taking of fluids causes excessive coughing. In such cases the patient may be able to take semi-solid articles of food, such as cereal jellies, junket, ice cream, scrambled eggs and milk toast. The method of feeding suggested by Casselberry of giving fluids with the head lowered, has been widely recommended, but I have seen at autopsy an undoubted case of inhalation pneumonia after this method of feeding, and prefer other methods. Gavage is a preferable method, especially in the younger patients. Nutritive enemata may be an important adjunct in certain cases.

The tube is often coughed up prematurely. This may be due to the fact that the nurse has laid the child face downward, or held him over one shoulder; in both these positions the tube is very easily expelled. In some cases, frequent coughing up of the tube is due to the fact that too small a size has been used. When the tube is expelled, dyspnea may return at once, or may not return for several hours. When it appears, the tube must be replaced, a size larger being used if necessary. Occasionally the tube is coughed out of the larynx and swallowed. This need cause no alarm, as the tube is soon passed from the intestine without harm. When the tube is not coughed up until after two or three days, dyspnea often does not return, and the tube can be dispensed with.

The sudden blocking of the tube with membrane loosened from the trachea or bronchi may occur at any time after the introduction of the tube. This is ordinarily not serious, as the usual result is the immediate expulsion of the tube by coughing, followed by the discharge of the loose membrane. No definite rule can be given regarding the length of time that the patient should wear the tube. It is well to remove it at the end of the third or fourth day, but it is frequently necessary to reinsert it. In some instances three or four extractions and introductions may be required. The most favorable cases are those in which the child coughs up the tube at the end of the third day, and does not require reintubation.

FIG. 111



Intubation. Withdrawing the tube

REMOVAL OF THE TUBE.—The first steps of an extraction are similar to those of an introduction. The extractor is passed into the lumen of the tube and the lever on the handle pressed so as to open the jaws of the instrument, and the tube extracted by a reverse of the movements used in introduction. Sometimes there is considerable difficulty in extraction, but by patience and gentleness the end can be accomplished.

After a tentative removal of the tube the child must be carefully

watched, and if dyspnea recurs, the tube must be replaced. If after two or three hours dyspnea does not return, the tube can usually be dispensed with.

COMPLICATIONS OF INTUBATION.—There is always a certain amount of hoarseness, and sometimes aphonia, following intubation. This usually passes off within a week, sometimes persists for three or four weeks, but is never permanent.

There is also usually some superficial ulceration from pressure at the base of the epiglottis, and at the end of the tube in the trachea. This is not serious. Deep ulceration only occurs in poorly nourished children, usually in association with other serious complications of the disease.

In a very few cases after intubation, difficulty is encountered in dispensing with the tube. All attempts to remove the tube permanently are followed by recurrence of dyspnea. This does not occur nearly so often with intubation as after tracheotomy. In most cases, the patient can do without the tube in a few weeks, but occasionally a case is more obstinate. The cause is probably swelling and edema of the tissues, which have become the seat of chronic inflammation, and which swell when the pressure is removed. In a few cases cicatricial contraction from ulceration has been found. The condition is best treated by increasing the pressure by the insertion of larger and larger intubation tubes. Some cases may be so obstinate as to require tracheotomy.

The only serious accident which occurs with intubation is the sudden blocking of the tube with loosened membrane. When this cannot be relieved by removal of the tube and reinsertion, tracheotomy must be performed.

INDICATIONS FOR TRACHEOTOMY.—Tracheotomy is indicated only when for some reason intubation does not cause the necessary relief of symptoms.

PROBLEMS AND RESEARCH.—There are fewer unsolved problems of importance connected with diphtheria than with the majority of the other infectious diseases. Most of the important facts as to the organism, its transmission, its mode of entrance, its action in the body, and the mechanism of recovery and immunity are known. Much of the research in the past few years has been devoted to confirming the value of the Schick reaction as a measure of immunity and a means of studying natural immunity and the duration of the immunity produced naturally by infection, and artificially by antitoxin injection. The result of this work has been incorporated in the description of the disease.

The following additional points have been the subject of study in diphtheria in the last few years:

1. Active immunization. 2. The dosage and mode of administration of antitoxin. 3. The relation of the toxins to diphtheritic paralysis. 4. The mechanism of circulatory failure. 5. The treatment of diphtheria carriers.

ACTIVE IMMUNIZATION.—It has been known for many years that a vaccine composed of a mixture of toxin and antitoxin could be used to produce an active immunity in animals. Von Behring began experimenting with the use of toxin-antitoxin mixtures in immunizing children in 1912, publishing his report in 1913. Since then a number of studies have been made, the observations of the different observers not being uniform. The results of many of the investigations have been judged only by the percentage of exposed cases which acquired the disease, and this is a very unreliable method of estimating the results. Park, Zingher and Serota have reported their experiences in producing active immunity by this method, controlling their results by determining the antitoxin content of the blood before and after injections of vaccine. The antitoxin content was estimated both by Römer's intracutaneous method and by means of the Schick reaction. They found that in susceptible persons, active immunity may be produced after a period of from two to six weeks. Persons already immune through having a supply of natural antitoxin in the blood, showed a decided increase in the antitoxin content as a result of the injection in a relatively short time, but of course such patients do not require active immunization. Of the remainder, those not showing natural immunity, less than 25 per cent reacted to the diphtheria toxin and antitoxin mixtures to a sufficient degree to be thoroughly immunized. Of the 75 per cent which could not be perfectly immunized, only 20 per cent of the exposed contracted the disease. It would seem therefore from the results of these studies, that the use of toxin-antitoxin injections is not certain enough to supersede passive immunization. The matter stands at present about as it did before, namely, that the Schick reaction should be used to determine immunity, and that every exposed patient having a positive reaction should receive an immunizing dose of serum.

THE DOSAGE OF ANTITOXIN.—Most of the work on this subject has been devoted to demonstrating the superior value of massive doses. Most of the evidence used in support of this contention is in the form of mortality statistics, which are of course very unreliable as proof. It is generally agreed that the object of treatment is to give a sufficient amount of antitoxin to neutralize the toxin in the body. Schick, Kassowitz and Busacchi, as a result of their experiments, believe that it is possible to calculate the amount of antitoxin needed according to the body weight of the individual to whom it is to be given. All of this work emphasizes the importance of early

injections. It has been clearly shown that the absorption of antitoxin, whether the serum be injected subcutaneously or intramuscularly, is slow. The most valuable suggestion of the last few years has been the use of the intravenous route in injecting diphtheria antitoxin, and the superior value of this method of injection has been demonstrated experimentally.

THE RELATION OF TOXINS TO DIPHTHERITIC PARALYSIS.—The theory of Erlich that the paralysis is caused by the toxone, while the general poisoning is caused by the toxin, has been supported by recent work. It has been shown that there is no relation between the size of the dose of toxin necessary to produce death and that which produces paralysis. Apparently the power of diphtheria toxin to produce paralysis and to produce death, are distinct features. It has also been demonstrated again that antitoxin never causes paralysis *per se*. The apparent increase of the number of cases of post-diphtheritic paralysis since the introduction of antitoxin is due to the decreased mortality of diphtheria, which allows many severe cases which would formerly have died and which are most likely to have paralysis, to live. Recent work has also shown that the use of antitoxin when given early enough, tends to prevent paralysis. The superior value of the intravenous route was also clearly shown in these experiments.

MECHANISM OF THE CIRCULATORY FAILURE IN DIPHTHERIA.—In all the fatal acute infections, this subject is receiving considerable study at the present day. It is known that in most of the acute infections the phenomena which precede death closely resemble those seen in surgical shock, and they have been attributed to vasomotor paralysis. In diphtheria, however, the symptoms have been almost exclusively attributed to the action of the toxin, either on the heart muscle itself, or on the nervous mechanism governing the action of the heart. Hardly more than a beginning has been made in these studies, but much will probably appear in the course of the next few years. Some recent work of MacCallum tends to show that the death which occurs in the height of an attack of diphtheria is not exclusively the result of direct injury to the heart.

THE TREATMENT OF CARRIERS.—The best means of getting rid of the bacilli in the throats of diphtheria carriers is an important matter, and has been the subject of many suggestions. Up to the present time, no very effectual method which is not objectionable in some way or another, has been found. It is probable that this question will be the subject of much further study. Among recent suggestions there is one of Miller, which recommends spraying the throat with a solution varying in strength from ¼ to 1 per cent of the usual 40 per cent formaldehyde solution. Another method which has been reported to have given good results is the spraying of the throat

with a suspension of the staphylococcus aureus in sa
shall have to wait longer, however, before any one
emerges as preëminent.

PERTUSSIS

(Whooping-Cough)

Pertussis (whooping-cough) is a specific infectiou
contagious, affecting the respiratory tract, characte
of spasmodic coughing succeeded by a prolonged
accompanied by a peculiar sound called the "whoop

ETIOLOGY. THE MICROÖRGANISM.—The sp
whooping-cough is a bacillus, first described by Bo
in 1900, and obtained in pure culture in 1906. U
Mallory, the proof that the Bordet-Gengou bacill
cause of whooping-cough depended upon the follow
organism is found in large numbers, sometimes even i
the sputum of all early cases; 2, similar appearances
not found in other conditions; 3, the serum of who
valescents agglutinates the organism; 4, the serum o
patients gives a complement fixation test specific f

While the Bordet-Gengou bacillus was accepted
whooping-cough by the majority of authorities, its
tion could not be regarded as fully proved. The obs
plete fulfillment of Koch's laws was the absence o
lesion characteristic of whooping-cough. Furtherm
picture produced in monkeys by inoculation with
Bordet-Gengou bacillus, while sometimes suggesti
ficiently typical to be regarded as certain confirmat

In 1912, Mallory and Horner announced the fin
anatomical lesion characteristic of whooping-coug
lesion being the presence of masses of a minute bac
cilia of the bronchi. These organisms morphological
resembled the bacillus described by Bordet and G
was able by intratracheal inoculation to reproduce
in animals. In puppies, the lesions were also rep
tagion. This work supplied the missing links in th
needed to establish the causal relation of the bacill
cough, giving the complete fulfillment of Koch's
possible remaining doubt is whether the Bordet-
represents a single organism, or a number of organisms
phology and cultural characteristics. In view of t
tagiousness of whooping-cough, it is probable that
in the finer biological reactions of the bacilli are no
often been found in different strains of the same org

The bacillus which causes pertussis is a minute ovoid non-motile cocco-bacillus, closely resembling in size amd in appearance the bacillus of influenza, but not requiring the presence of hemoglobin for its growth in cultures. It is Gram-negative, and stains only slightly by ordinary methods, especially in the center of the organism. The organism can be grown at the start only on the potato blood-agar medium devised by Bordet, though after continued cultivation it will grow to some extent on other media. The growth is slow, forming minute transparent colonies. In cultures the organism is round or oval, and resembles a coccus more than a bacillus. It is not resistant toward high temperatures.

The organism is found only in the respiratory tract; its absence in the blood has been demonstrated. That it forms some toxin is shown by the slight inflammatory reaction which it produces, and by the formation of an antibody which gives the complement fixation test. Toxin formation is comparatively slight, and probably plays little or no part in producing the characteristic symptoms of the disease. Mallory has suggested that the action of the bacillus is chiefly mechanical, and this at present seems the most plausible hypothesis. Some writers believe in a neurotic factor in etiology, but this probably plays no more than a possible rôle in influencing the severity of the clinical manifestations.

TRANSMISSION.—The spreading of whooping-cough occurs almost exclusively directly from one case to another. The contagium is air-borne, the bacilli being contained in the moist particles expelled in coughing—the method of "droplet infection,"—and entering with the inspired air. The sputum contains enormous numbers of bacilli, so that a very brief stay in the presence of a whooping-cough patient is sufficient for transmission. The disease is consequently very highly contagious. Indirect transmission, the carrying over of the bacilli by means of healthy carriers, clothing, and contaminated objects, occurs very rarely if at all.

THE INFECTIOUS PERIOD.—Whooping-cough is contagious from the very beginning of the so-called catarrhal stage, and the disease can be and usually is transmitted during the period of one or more weeks before the nature of the disease has been made clear by the appearance of typical paroxysms. The transmissibility of the disease is greatest at this early period, but the disease remains contagious through the earlier part of the convulsive stage. Its communicability diminishes rapidly during the latter part of this stage, and is slight or absent after the paroxysms cease. It is well, however, to regard whooping-cough as still possibly contagious as long as cough is still present.

PREDISPOSITION.—The susceptibility toward whooping-cough is

very great, being comparable with that toward measles and varicella. There is no evidence of any natural or constitutional immunity at any age. Given sufficient exposure, children who have not had the disease are practically certain to acquire it.

This susceptibility is seen at every period of life. Whooping-cough occurs most often in the first three years of life. It is somewhat less common in the first year than in the next two years, but in no other specific infection is infancy so susceptible as in whooping-cough. The disease is often seen in the earliest months of infancy, sometimes in the earliest weeks. It has been described in newborn infants whose mothers had the disease. It is probable that there is no inherited immunity which protects children in the first year, such as is seen in other contagious diseases. The lesser frequency of whooping-cough in the first year is to be explained, not by a lesser susceptibility, but by the fact that young infants are better isolated and protected from exposure, and that many, being first babies, do not encounter exposure at all.

Whooping-cough continues to be comparatively common through childhood, the frequency growing less with increasing age. It is rare in adults. These phenomena are, however, not due to a progressive decrease in susceptibility, but to the fact that in so highly contagious a disease immunity is acquired early in life.

Constitutional peculiarities have no real influence on the susceptibility toward whooping-cough, except that girls appear to be more susceptible than boys. It is often stated that children liable to catarrhal affections of the respiratory tract are more susceptible. This cannot be proven, because all children are so susceptible anyway. Neurotic and spasmophilic children are not more susceptible to infection, but in them the paroxysms are apt to be more severe and the disease to last longer. Debilitated children with low resistance show the influence of constitutional peculiarity in the liability toward complications.

EPIDEMIOLOGY.—As in most of the more highly contagious diseases, the frequency and severity of recognizable epidemics depends on the population of the place. Epidemics are only seen in small or isolated communities. In the large centers of population whooping-cough is present at all times and so large a proportion of the population are immunized that epidemics do not occur.

The disease is somewhat commoner in the winter and spring than in warm weather. This may be due to the greater prevalence of respiratory affections.

IMMUNITY.—An attack of whooping-cough confers a high degree of immunity, so that second attacks are rare. They are seen at times, however, and are rather more frequent than in the exanthe-

mata. Second attacks are almost never seen in childhood, but are commonest in adults who had whooping-cough in childhood, and who are brought into close and long continued contact with children sick with the disease. The greater susceptibility of the female sex is particularly noticeable in connection with these occasional second attacks in adults.

INCUBATION.—The incubation period of whooping-cough is difficult to determine, because the onset of the disease can be less definitely timed than in other contagious affections. It appears to be of varying length. The average length is one week, but the period may be only half this time, or may extend to two weeks. When exposure is suspected, it is safe to assume that if after fourteen days there are no catarrhal symptoms of any kind, infection has not taken place.

PATHOLOGICAL ANATOMY.—The essential lesion of whooping-cough, and the only one peculiar to the disease, is the presence of masses or clumps of bacilli between the cilia of the trachea and bronchi. There is some infiltration of the submucosa with lymphocytes and plasma cells (Mallory), and some invasion of the epithelium with polymorphonuclear leucocytes.

Changes in the blood are constant in whooping-cough, but are not peculiar to the disease. There is a leucocytosis, the lymphocytes showing a marked absolute and relative increase.

The other lesions found at autopsies are either due to some of the mechanical effects of the disease, or to complications. Death is always due to some complication. Among the mechanical lesions are extreme congestion of various organs, such as the meninges, lungs, heart and kidneys; hemorrhages in various regions, such as the eye, or the meninges; emphysema, either of the lung, or of the mediastinal or subcutaneous tissue from rupture of the alveoli. The commonest complications are bronchopneumonia, with its associated lesions, and the lesions of the secondary stage of tuberculosis.

SYMPTOMS.—It is customary to divide the disease into three periods,—the *catarrhal stage*, the *spasmodic stage*, and the *stage of decline*. It must be remembered, however, that such a division is somewhat arbitrary and inexact, as the three periods overlap and run into one another. It is only made for convenience of description.

The symptom complex of pertussis is very constant, varying mainly in the severity of the case. The description as here given applies to a case of medium severity.

THE CATARRHAL STAGE.—The symptoms in the beginning and often for several weeks, are simply those of a bronchial catarrh, or common cold, with cough, and perhaps some sneezing, hoarseness, or reddening of the conjunctiva. These symptoms usually last from one to two weeks, though at times typical paroxysms may appear in

less than a week, or not for four weeks. In the some cases there may be a slight rise of tempera stage the disease cannot be differentiated from an whooping-cough can only be suspected when there exposure. A few cases, which subsequently are rec ing-cough by transmitting the disease, never show toms, but pass through the disease unrecognized however, the cough becomes more violent. The be suspected at this time because the cough begins or less regular intervals by night as well as by da ordinary means of treatment, and also because in sp of the cough, no râles are heard on auscultation of t

THE SPASMODIC STAGE.—The transition from the spasmodic stage is gradual. The cough becomes le more violent. The attacks of coughing become pa occur at more or less regular intervals, the child bei from cough in between. These attacks, sometimes times more gradually, develop into the typical whoop oxysms.

The child often experiences premonitory symptoms, ling in the throat, an oppression in the chest, or a ge anxiety. These cause restlessness, the child runs to holds itself fixed on its chair. Then, after a deep insp a succession of forcible expiratory coughs, occurring in r without any inspiration occurring between them. Duri xysm the face becomes red, the conjunctivae become c the tongue protrudes from the mouth. Finally there c drawn crowing inspiration, in which the air is drawn spasmodically contracted glottis, causing the peculiar attack is, however, usually not ended with this occur immediately repeated, and this may occur a number of t each repetition the lips and tongue become more and m In severe cases, the eyes protrude, cyanosis becomes v and the child looks as though it would die of asphyxia. usually ends with the expulsion of a little tenacious muc older children trickles out of the mouth, and in young ch stays in the larynx. With the expulsion of this mucus, v quently occurs, especially after the severer paroxysn older children recover rapidly from the attack, and often ir resume their interrupted play. Weak children and young left exhausted and bathed in sweat, and require more time fo

The number and severity of the attacks show the widest in different cases. Some children have only four or five i four hours, while others may have thirty, forty, or fifty. Th

of the single attack also varies widely; sometimes the
cough and whoop are only repeated two or three times,
times they may be repeated ten times or more. It is to b
that these variations are seen not only in different child
in an individual case, a fact which must be remembered
the value of any method of treatment.

The duration of the spasmodic stage is also very va
usually three or four weeks, or even more, but may be

The lungs in uncomplicated cases are normal to aus
percussion. There may occasionally be a few coarse br
which disappear temporarily after a paroxysm. In infa
pulmonary emphysema may develop during the spas

FIG. 112

Pertussis during paroxysm. Female, 4 years old

THE STAGE OF DECLINE.—The paroxysms become
quent, then less severe. Vomiting ceases. Finally the
rarely occurs, and has lost its typical character. A sim
cough may, however, persist for a number of weeks lon

The duration of the entire disease is variable; in ave
is from six to ten weeks, but may be three or four mo
uncomplicated cases. A certain amount of loss of w
rule, but this is rapidly recovered in convalescence, if
complication involving the lungs. If during the stag

bronchitis occurs, or even if the child simply catches a simple cold, there is an apparent relapse into the spasmodic stage, the paroxysmal cough returning with all its typical features, and persisting until the bronchitis or cold is recovered from. The same thing may happen after complete recovery, and if a child in the course of the succeeding months, even as long as a year, has any cold or other affection characterized by cough, the cough assumes a paroxysmal character, and even the whoop may reappear.

ATYPICAL CASES.—Very mild cases are seen at times, in which the attacks are rare and not easily recognized as whooping-cough, and in which the duration of the disease is only two or three weeks. In many cases, no whoop is heard at any time, and the paroxysms may be so mild and atypical as not to be suggestive. These mild cases can often be diagnosed only by the presence of whooping-cough in the neighborhood.

Very severe cases of uncomplicated whooping-cough are rare. Occasionally, however, a case is seen which from the onset shows high fever, and marked affection of the general condition through restlessness and loss of sleep. Vomiting becomes very frequent, dyspnea appears, the pulse becomes very rapid, and in younger children, death may occur.

Whooping-cough as seen in the younger infants is often peculiar in its manifestations. The paroxysms are severe, leading to cyanosis, and often producing a brief period of apnea, with disturbance of the consciousness and convulsive movements. At the same time the whoop is absent. the paroxysm ending with gagging and the trickling out of the mucus. Vomiting is less frequent.

SINGLE SYMPTOMS.—There are a certain number of symptoms which do not form part of the constant clinical picture of whooping-cough, but which are seen with more or less frequency. Many of them are the result of the mechanical effect of the severe paroxysms, which produces venous stasis.

In severe cases the heart shows enlargement of the cardiac dulness to the right, due to dilatation of the right ventricle. The pulse in such cases becomes more rapid, and there may be a blowing systolic murmur at the apex. These signs disappear in the stage of decline. After the disease has lasted for some weeks, there is often a certain amount of edema of the face, especially under the eyes. Fever is usually absent throughout uncomplicated cases, except sometimes early in the catarrhal stage. Occasionally there is slight fever throughout the disease.

Ulceration of the frenum of the tongue from pressure on the teeth during the paroxysm, is not uncommon, especially in infants who have only the two lower middle incisors. Involuntary passage of

urine and feces, and sometimes prolapse of the rectum, are occasionally seen during the paroxysms.

The nervous system has a marked influence on the course of whooping-cough. The paroxysms are precipitated by nervous excitement or by an irritation in the throat or the respiratory tract, such as may result either from swallowing or from the inhalation of dust. In nervous and neuropathic children the paroxysms are also more severe, and anything in the surroundings of the child tending to stir up the nervous system may have a marked influence in increasing the severity of the disease.

The digestive system is usually but little affected in whooping-cough, except for the vomiting at the end of the paroxysm. This may, however, be very obstinate, and may seriously interfere with nutrition. Diarrhea is very rare.

Albuminuria, due to congestion of the kidneys, is seen in severe cases.

THE BLOOD.—The changes in the blood of pertussis have been carefully studied in the last few years, and are important in diagnosis. The principal phenomenon is a leucocytosis, the white count being from 20,000 to 40,000, averaging about 27,000. There is an absolute increase in both the neutrophiles and the basophiles, but the increase in the basophiles is the most marked, so that there is a relative increase of the basophiles shown in the differential count. These changes begin in the catarrhal stage, and are most marked in the early part of the spasmodic stage.

COMPLICATIONS.—The most serious and important of the complications of whooping-cough are *hemorrhages*, *bronchopneumonia*, *tuberculosis*, and *disturbance of the nervous system*.

HEMORRHAGES.—Hemorrhages, as a result of the marked venous stasis produced by the paroxysm, are not very uncommon. Epistaxis is the most frequent form, but is seldom severe enough to be threatening. Occasionally blood is coughed up from the pharynx or bronchi. Hemorrhage into the conjunctiva, forming a crescent around the cornea, is relatively not uncommon. Hemorrhages into the eyelids, or into other parts of the skin, are more rare.

Occasionally the mechanical effect of the whooping-cough paroxysm may produce a hemorrhage into the central nervous system, usually involving the meninges, resulting in sudden hemiplegia, monoplegia, or some other disturbance, such as aphasia, facial paralysis, or some disturbance of the sight, hearing, or sensation. The hemorrhage may be fatal. Such cases are fortunately rare.

BRONCHOPNEUMONIA.—Bronchitis of the larger bronchi, as shown by coarse sonorous râles, is so frequent in the severer cases of whooping-cough, that it is usually considered a symptom rather than a

complication. As many children with whooping-cough have no rales at any time, I believe that any bronchitis strictly speaking is a complication, and the bronchitis of the larger bronchi is often a forerunner of a more severe form.

Bronchopneumonia is always preceded by a bronchitis of the smaller tubes, and it is usually difficult clinically to distinguish the two conditions. It is much more common in infants than in older children. The onset of a severe bronchitis in the course of whooping-cough, is marked by the appearance of moderate fever, of ordinary catarrhal cough between the whooping-cough paroxysms, and of a more abundant, greenish-yellow sputum. When bronchopneumonia develops, it is recognized by the appearance of a higher temperature, which is irregular and remittent, and by the appearance of rapid breathing. Often the whooping-cough paroxysms lose their typical characteristics, becoming shorter, with disappearance of the whoop, although the cyanosis may be still more marked.

The signs in the lungs are those of any secondary bronchopneumonia, diffuse crackling râles, with or without areas of dulness and bronchial breathing. The course is that of a secondary bronchopneumonia, but is apt to be severe and of long duration, owing to the weakened condition of the child. Bronchopneumonia is the most frequent cause of death in whooping-cough.

The bronchiectasis and emphysema of the lungs, which are such frequent findings at autopsy, are not often to be recognized clinically. Occasionally, however, there is a rupture of the alveoli which causes an interstitial emphysema of the mediastinum, in which the cardiac dulness is entirely replaced by tympany. Exceptionally there may be a subcutaneous emphysema which, beginning near the neck, may spread widely over the body. Emphysema may be recovered from, or may lead to severe dyspnea and death.

TUBERCULOSIS.—It is generally recognized that children who have recently had whooping-cough, are liable at times to develop some serious form of tuberculosis. This does not mean, as many wrongly suppose, that whooping-cough predisposes children to become infected with tuberculosis. The cases which apparently develop tuberculosis as a complication or sequela of whooping-cough have been infected with tuberculosis at a much earlier date, and when they contract whooping-cough, already have in their bodies the lesions of the primary stage of tuberculosis. The whooping-cough lessens their resistance to the spreading of the disease, and favors the development of the secondary tuberculous lesions. The commonest of these secondary manifestations seen in whooping-cough is tuberculous bronchopneumonia, but tuberculous meningitis, or general miliary tuberculosis, is seen at times.

DISTURBANCES OF THE NERVOUS SYSTEM.—The gravest complications of whooping-cough which involve the nervous system have already been enumerated among the hemorrhages.

In younger children the paroxysms often precipitate a larygneal spasm similar to that seen in spasmophilia, and occasionally eclamptic attacks with loss of consciousness are also seen. Laryngeal spasm with a convulsion may conclude each paroxysm. Eclamptic attacks may even occur between the paroxysms. It is probable that these manifestations are seen mainly in spasmophilic children, the pertussis acting as a precipitating cause. Whooping-cough may prove fatal in cases of spasmophilia with laryngospasmus.

DIAGNOSIS.—The diagnosis of whooping-cough in the spasmodic stage is usually easy, provided that the physician can hear a paroxysm. If, however, the child has no paroxysm during the visit of the physician, a positive diagnosis is impossible. The physician must judge by the description of others, which is apt to be misleading. It is often possible to provoke an attack, either by introducing a spoon or spatula into the mouth as for examination of the throat, pressing strongly downward on the tongue, or by pressure on the larynx or trachea. If a paroxysm cannot be elicited, and if the intervals between attacks are long, the physician is compelled to base his diagnosis on the history. If the typical whoop be present, the description is often sufficiently characteristic for a diagnosis, but if, as often happens, especially in infants, there is no whoop, there may be considerable doubt. The following questions should be asked as having a special bearing on the diagnosis: 1, Does the cough come in long fits without the child drawing breath? 2, Do the attacks come at night as well as by day? 3, Is there any short cough in the intervals between the attacks, or in other words, is the cough always in long fits? 4, Is there vomiting, and if so does it come only with an attack of coughing? 5, Is there any expectoration? It should be remembered that in infants and young children up to the age of eight or ten years, expectoration is almost never seen except in whooping-cough. The diagnosis is supported at times by the absence of physical signs in the lungs in uncomplicated cases, by the absence of fever, by the story of severe congestion of the face in the attacks, by any visible hemorrhages, and by ulcer of the frenum of the tongue.

In the catarrhal stage, and in the spasmodic stage of mild and atypical cases, the diagnosis is much more difficult. Often a diagnosis can only be made when there is recognized whooping-cough in other members of the family, or a history of certain or possible exposure. The nearer the approach to the stage of typical paroxysms, the greater the possibility of suspecting whooping-cough. The examination of the blood often helps in the diagnosis of whooping-

cough in the catarrhal stage. In any cough in children, a high white count, without there being any fever, points toward whooping-cough. There are some secondary infections which give a high white count without fever, but the increase is usually in the neutrophiles, while in early whooping-cough the differential count usually shows a relative increase in the basophiles. Kolmer has reported a series in which whooping-cough in the catarrhal stage was correctly diagnosed by the blood examination in 81 per cent of the cases, and correctly excluded in 71 per cent.

The conditions which most often present themselves in the differential diagnosis of whooping-cough are certain ones involving the air-passages, such as adenoids, pharyngitis, laryngitis, bronchitis and tuberculosis of the bronchial lymph nodes. In the affections of the pharynx and larynx, while the cough may be severe and somewhat spasmodic, it is more frequent and irregular, and typical attacks do not develop. In bronchitis, the attacks of severe coughing come at the very beginning, instead of developing gradually, and are more frequent; also acute bronchitis is accompanied by fever and numerous râles in the lungs. In tuberculosis of the bronchial lymph nodes, the differential diagnosis is sometimes more difficult, as the cough is apt to be distinctly paroxysmal, and there may be expectoration or even vomiting. The whoop is, however, always lacking, and the cough lasts for many months without much change in its character or intensity. The other signs of tuberculosis, such as interscapular dulness, d'Espine's sign, positive von Pirquet reaction, and positive X-ray picture, are usually present in such cases.

PROGNOSIS.—Pertussis is a very serious affection in young infants, and also in older children who are debilitated or poorly cared for. This is because the liability to bronchopneumonia is greater the younger the patient, and both the frequency and fatality of this complication is increased by poor physique. When whooping-cough occurs in older children the prognosis is favorable, provided that they have previously been well and strong, that they are well cared for, and that no complications arise.

In many cases young infants, if their vitality is unusually good, and if they are carefully nursed and made to take a sufficient amount of food, show remarkable powers of resistance during attacks of pertussis.

PROPHYLAXIS.—While it is a fact that in spite of any precautions, almost all children will contract whooping-cough during some period of their childhood, it is not in my opinion ever justifiable to allow a child to become exposed to whooping-cough if it can be avoided. It is particularly important to protect children during the first four years of life, for it is at this age that the tendency to bron-

chopneumonia is greatest. Children with whooping-cough should be strictly isolated, under the same conditions as are used in the prophylaxis of measles, which has the same mode of transmission. It is, however, undesirable that a whooping-cough patient should be confined to a single room, and it is therefore better, whenever possible, to send away the other children of the family, and to convert the house into a whooping-cough hospital, to which the other children can be brought back if they have become infected.

If one waits until the diagnosis of whooping-cough can be made before isolating the patient, the disease will have been already transmitted during the catarrhal stage to all the other children of the family. To approach efficient prophylaxis, it would be necessary to isolate a child every time it begins to cough, and to keep it isolated for several weeks, and this is manifestly impracticable, as colds and coughs are so common in early life, that such a procedure would compel school-children to spend much of their time in useless isolation.

The most practical method, one which I habitually employ with the patients under my care, is to isolate, not the school child with a cough, but the young child whom I wish to protect. When any one of the older children in the family has a cough, no matter how harmless it seems, the younger children, those under five years, are separated at once from the others, and are kept apart as strictly as the circumstances of the family will permit, until the cough has ceased. This will often enable one to protect young children, and to postpone the inevitable day when they must acquire whooping-cough to a period when the disease is fraught with less danger. It is on account of the danger of acquiring whooping-cough that I do not permit my patients to attend kindergartens until they are four or five years old. After this age, the advantages of kindergartens and companionship with children of the same age more than counterbalance the danger of contagious disease. As to how much very young children should be allowed to play with the children of friends, is a question to be determined by how careful the parents of the latter are in questions of hygiene and prophylaxis.

The other children of a family in which a case of whooping-cough has developed, should always be regarded as having been exposed, no matter how brief and slight the contact. They should be regarded as liable to become sources of infection at any time, and should not be allowed to go to school, nor to play with other children. If after the last possible time of exposure, fourteen days have elapsed without any cough having developed, it is safe to conclude that the child has escaped infection.

It is safest to regard the child with whooping-cough as a possible source of infection, as long as it still coughs. If, however, three months have elapsed, and if all the characteristics of the spasmodic

stage have disappeared, continued cough is not an indication for further isolation.

TREATMENT.—The treatment of whooping-cough is to be considered under four divisions, as follows: 1, General hygienic measures; 2, Internal and local treatment of the cough; 3, Treatment of special symptoms; 4, Specific treatment, or vaccine therapy.

GENERAL HYGIENIC MEASURES.—These are considered first, because they constitute the most effective means of influencing the severity of the disease. At least one-half of the cases of whooping-cough encountered in practice require no other means of treatment, and would even do better if no other measures were used. Much harm has been done by the indiscriminate prescribing of drugs in whooping-cough.

Fresh air stands at the head of the list of therapeutic measures in whooping-cough. The effect of being outdoors on the number and severity of the paroxysms is more obvious than that of any other means of treatment. Older children should go out every day even in winter, except on stormy, raw, or dusty days, and should be kept in the open air as much as possible. In the case of young infants and delicate children, some precautions must be taken. I believe, however, that even in winter these children should be taken out in their carriages on all fine days, but care must be taken to clothe them properly, and to guard them from raw damp weather, wind, dust, and sudden changes of temperature. When on account of weather conditions, patients cannot go out, they should be given indoor airings. Only those children who have fever, or are very sick, should be kept in bed, and even these should be put out on a porch or veranda in warm weather, or when weather conditions are favorable.

When children are indoors, more than one room should be used whenever possible, the patients being changed frequently from one well-aired room to another. The sleeping room should be arranged to admit plenty of fresh air. The least closeness of the atmosphere increases the number and severity of the attacks.

Change of climate has been highly recommended in whooping-cough. In infants I do not ordinarily recommend it, as the discomforts and limitations of hotel life are not good for infants, who do better at home. Change of climate is, however, useful in all cases in which during convalescence, cough is unduly prolonged. It is also beneficial to delicate patients in winter. A warm place near the sea-shore gives the most desirable climate.

The feeding of whooping-cough patients is important, and is sometimes difficult on account of the vomiting which accompanies the paroxysms. Whenever vomiting is a symptom, small meals, given more frequently, should be used. It is often necessary to depart

from the usual regularity of the feeding intervals, and to give the food shortly after a paroxysm, so that it will be retained as long as possible. In older children when vomiting is present, the diet should be fluid, chiefly milk, with some thin gruel or beef-juice. Even when vomiting is not prominent, it should be remembered that coarse, dry, solid food irritates the mucous membranes, and the diet should be confined to liquids and soft solids.

The nutrition of the infant is so easily affected that the utmost attention should be paid to the administration of the food. Small quantities of milk carefully modified to suit its digestion should be given at frequent intervals, preferably after the occurrence of a paroxysm, as it is then more likely to retain the milk in its stomach a sufficient length of time for absorption before the next attack. The amount of food which the infant retains in the twenty-four hours is an important factor in the treatment. In infants of from six to twelve months, at least 20 to 25 ounces of milk should be taken and retained in the twenty-four hours. When the amount is lessened to 12 or 15 ounces, the infant's nutrition, as a rule, suffers to such an extent that unless this amount can be increased, a fatal issue is likely to result. Under these circumstances, if the infant is not taking enough food, gavage should be resorted to. If the trouble is entirely the retaining of the food, gavage will sometimes help, as the tube is less apt to irritate the pharynx and precipitate a paroxysm than sucking. In desperate cases, some internal medication must be pushed till the paroxysms are lessened, but such cases are rare.

It is also important in whooping-cough to guard the child from every excessive stimulus to the nervous system All forms of excitement, violent games, excessive noise and so forth, should be prevented.

INTERNAL AND LOCAL TREATMENT OF THE COUGH.—The drugs which have been recommended in the symptomatic treatment of the cough of pertussis are innumerable, and this fact is in itself an evidence that no one of them stands forth as of preëminent value. Whooping-cough is a disease of such irregular course, both as to number and severity of the paroxysms, that no man's judgment as to the efficacy of a therapeutic measure can be of much value. Each physician has his favorite remedies, which he believes to be the only ones of undoubted value, but which differ from those of other writers. I must confess to having no favorite drugs which I believe to be superior to others in whooping-cough. One drug seems to work better in one case, another in another.

I do not believe that drugs should be used as a routine in whooping-cough. Only when the number and severity of the paroxysms are so great as to reduce the strength of the child, to interfere with its nutrition, or to cause it obvious suffering, are drugs indicated.

The most effective drugs in controlling the paroxysms are the opiates, but owing to their other effects, they should only be used in cases of greatest severity.

When drugs are indicated, the physician should try one after another, keeping a careful record of the daily number of paroxysms. Two or three days suffice to test the working of a drug. When one is encountered which appears to produce an appreciably favorable effect, it is better to stick to it. The order in which the drugs are tried is arbitrary, and depends more or less on the fancy of the physician. In going over all the various text-book articles and recent publications, I have found that the drugs can be arranged in an order based on the preference of the majority.

On the basis of majority preference, *quinin* holds first rank in the early stages of whooping-cough. To infants the bisulphate should be given in aqueous solution, made up so that there is 1 grain to the teaspoonful for infants in the first year, and 2 grains to the teaspoonful for infants in the second year. A teaspoonful should be given three or four times a day. For older children the taste must be disguised, and the bisulphate may be given in syrup of sarsaparilla, orange, or yerba santa. The dose should be from 3 to 5 grains of the bisulphate according to the age, three times a day. In children over eight years, the sulphate may be given in capsules.

Antipyrin ranks next, and may be tried when quinin fails. The dose from six months to two years should be 1 grain every four hours, and this may be safely increased to every three hours, and then to every two hours, if no effect is produced. For a child from two to six years, 2 grains may be given every four hours, increasing to every two hours. After six years, 3 grains may be given. Antipyrin may be used alone, or combined with the bromides.

The bromides may be tried next. In the first year, bromide of sodium may be given in doses of 2 grains every four hours; in the second year, the dose may be 3 grains, and to older children 4 or 5 grains every four hours. Bromide of sodium may be given in combination with antipyrin.

Belladonna has been a favorite remedy for whooping-cough in the past. Present day opinion inclines to the belief that it is not often very effective. The dosage will be found in Division II.

It is admitted that the *opiates* are the most effective means of controlling the paroxysms, but owing to their other unfavorable effects, they should only be used in the severest cases, when other means fail.

Codein should be tried first, in the following doses: In the first year, $\frac{1}{60}$ grain; in the second year, $\frac{1}{30}$ to $\frac{1}{20}$ grain; from two to four years, $\frac{1}{15}$ to $\frac{1}{10}$ grain; from four to eight years, $\frac{1}{8}$ to $\frac{1}{6}$ grain; over eight years, $\frac{1}{4}$ grain. These doses should not be repeated

oftener than is necessary. Often a single dose at bedtime, sometimes repeated once later, suffices. No matter how severe the case, codein should not be given oftener than every four hours.

If codein fails, and the case is of the severest type, morphin should be tried, in the doses given in Division II.

Bromoform sometimes works well as a substitute for the opiates. The dose should be from 1 to 5 drops three times a day according to the age of the child.

The *local applications* to the naso-pharynx, and the medicated sprays and inhalations which have been recommended in pertussis, are legion. In my experience neither local applications nor inhalations give the results claimed for them, and I have given up their routine use. For local applications to the naso-pharynx, the preparations most used are silver nitrate in 2 per cent solution, carbolic acid in 1 per cent solution, and resorcin in 1 per cent solution. For inhalations, creosote, carbolic acid, and cresolene are the favorites. The cresolene vaporizer in the room often has a soothing effect upon the child's parents, and can do no harm.

TREATMENT OF SPECIAL SYMPTOMS.—The only serious symptom in the average case of whooping-cough is the vomiting which accompanies the paroxysms, and which may seriously interfere with nutrition. The treatment of this is that of the general disease, and has already been considered.

The slight dilatation of the heart which often accompanies whooping-cough requires no special treatment, except that the children must be kept more quiet. Signs of real cardiac insufficiency are rare in uncomplicated cases and require rest in bed, and stimulation with caffein or digitalis, camphor being used in case of sudden collapse.

The ulcer at the frenum of the tongue is treated by protecting the teeth by a bit of adhesive plaster, and by the application of silver nitrate to the lesion.

The tendency to laryngospasmus and convulsions is best treated by the bromides, given in the same doses as are used in attempting to control the whooping-cough paroxysms. In all such cases, tests should be made for Chvostek's sign, the peroneal reflex, and increased electric excitability, in order to determine whether spasmophilia is the underlying cause of the symptoms. If this is the case, the treatment of spasmophilia should be added to that for pertussis.

Epistaxis is treated locally in the usual manner. The hemorrhages into the skin and conjunctiva require no special treatment. The serious hemorrhages involving the central nervous system are treated as described under cerebral hemorrhage. Mediastinal and subcutaneous emphysema have no special treatment, but if they produce serious symptoms, symptomatic stimulation is employed. The

albuminuria seen at times in pertussis also requires no special treatment.

The serious complications of bronchopneumonia and tuberculosis are treated as described under those diseases.

Prolonged cough in convalescence is treated as is chronic bronchitis, by means of tonics and change of climate.

SPECIFIC TREATMENT; VACCINE THERAPY.—Very soon after the establishment of the Bordet-Gengou bacillus as the presumable cause of whooping-cough, attention was turned to the possibility of a specific method of treatment. The long course of the disease, and the localized character of the lesions, suggested that vaccine therapy was the form of specific treatment most likely to prove of value. Vaccines prepared from cultures of the whooping-cough bacillus were placed upon the market, and have been very widely employed. Recent medical literature is filled with innumerable reports on the results of vaccine treatment in whooping-cough. The majority of these reports are favorable. Some observers believe that the use of the vaccine not only lessens the number and severity of the paroxysms, but also shortens the course of the disease and tends to prevent complications. Other observers are not so sanguine as to the shortening of the course of the disease and the prevention of complications, but believe that the number and severity of the paroxysms are favorably influenced. One encounters many "pleas for the more extended use of vaccine therapy in whooping-cough."

These reports are by no means convincing. It is extremely difficult to estimate the value of any therapeutic measure in a disease so irregular in its clinical manifestations as is whooping-cough. The history of therapeutic endeavor clearly shows that most means of treatment, the results of which are estimated by their effect upon clinical symptoms, are held as effective when first tried. Our minds unconsciously tend to see favorable results from our therapeutic endeavors. The investigators whom one would naturally select as being men having the most critical and impartial minds, have published reports unfavorable to the value of the vaccine in pertussis.

I note a report by Hess of an epidemic of whooping-cough in which four different vaccines were used, including an autogenous vaccine. No one of them showed any effects of value in curing or modifying the disease in this epidemic, in spite of the fact that many cases received early inoculations, or even prophylactic inoculations.

Vaccine therapy in the treatment of pertussis is possibly worthy of further trial. It has been established that it does no harm, and it can be used in any case in addition to the ordinary methods of treatment. The usual dosage is from 100,000,000 to 300,000,000 bacteria, given every other day.

Even if the whooping-cough vaccine has no therapeutic value, it may have some value in prophylaxis. Some writers believe that its prophylactic value has been adequately proven. In Hess' report, twenty children developed whooping-cough in spite of prophylactic inoculation. On the other hand, a greater percentage of cases developed among the unvaccinated than among the vaccinated.

My own present practice is to use prophylactic vaccines in cases known to have been exposed to whooping-cough, and therapeutic inoculation in cases which are running a severe course. I do not, however, allow vaccine therapy to replace other therapeutic measures.

PROBLEMS AND RESEARCH.—The chief problem awaiting final solution has already been discussed, namely, the value of vaccines in the treatment and prophylaxis of whooping-cough. It does not seem likely that conclusive proof will be attained in the matter of vaccine therapy, but the value of vaccine prophylaxis will probably be settled in a comparatively short time.

Considerable work remains to be done in the study of the Bordet-Gengou bacillus and its biological reactions. It is yet to be determined whether the organism represents one or several types or strains. The constancy of the complement fixation test must also be proven more conclusively. Positive tests have been obtained by using the Noguchi hemolytic system, the antigen being prepared from a pure culture of the Bordet-Gengou bacillus grown in ascites-fluid agar. It is possible also that future research will throw more light upon toxin formation and the nature of the antibodies.

New suggestions as to internal and local remedies will probably continue to appear from time to time. Adrenalin has recently been suggested as of value in controlling the paroxysms.

MUMPS

(Epidemic Parotitis)

Mumps is a contagious disease of which the chief symptom is usually an acute swelling of the parotid gland. The disease is undoubtedly a specific infection, as is shown by its marked transmissibility, and its tendency to occur in smaller or larger epidemics. These epidemics are particularly likely to occur in schools and institutions.

ETIOLOGY. THE MICROÖRGANISM.—The organism which causes mumps is unknown. It is probably a filterable virus like that of poliomyelitis. Like other diseases of this class, mumps is characterized by a lymphocytosis.

TRANSMISSION.—Mumps is transmitted directly from an infected individual to a healthy person. The virus can be carried over by healthy persons and on contaminated objects. It is not known

whether actual contact with infected material is requisite, or whether the virus can cross an air-space. In my opinion it is probable that the contagium is not air-borne. It is usually assumed that the virus enters through the mouth, and reaches the parotid gland through Stenson's duct. In view of the occasional localization of the disease in places other than the parotid gland, this must be regarded as questionable.

The contagious period of mumps begins with the first appearance of symptoms, and according to some observers, the disease may be transmitted one or two days before the appearance of swelling of the parotid. The duration of the contagious period is not definitely known, and is probably variable. The infectivity probably ordinarily ceases rapidly during convalescence, but has been known to persist for one or two weeks. The spread of the disease is favored by the frequent occurrence of mild ambulant cases.

PREDISPOSITION.—Some writers state that the susceptibility to mumps is not very great. This I believe to be a careless statement. It is based on the fact that mumps only occurs in a small percentage of those exposed. As the mode of transmission is not thoroughly known, it is impossible to say what constitutes exposure. In scarlet fever only a small percentage of the exposed are infected, and this is known to be due not to a varying susceptibility to scarlet fever, but to the fact that the virus of scarlet fever is not air-borne, and that actual contact with infected material is essential for transmission. In mumps the mode of transmission is probably similar to that of scarlet fever, and accounts for the apparent relative insusceptibility.

Mumps is most common between the ages of five and fifteen years. The disease is very uncommon in infants under two years, although rare cases have been described even in the newborn.

IMMUNITY.—An attack of mumps confers a long-lasting immunity. Owing to the doubt as to the exact mode of transmission, it is impossible to say with certainty how long this immunity lasts, as adults are probably much less frequently exposed than children. Second attacks do not occur in childhood, but in adults are relatively rather more common than with the exanthemata.

INCUBATION.—The incubation is very long, averaging from seventeen to twenty-one days. Family epidemics are consequently often long drawn-out.

PATHOLOGICAL ANATOMY.—Opportunities for pathological examination of the tissues involved in mumps have been very rare. The process appears to consist of edema and congestion in the interstitial tissue of the parotid, and of the tissues adjoining the parotid, accompanied by a certain amount of round cell infiltration. The parenchyma of the gland does not appear to be involved.

In the majority of cases the parotids alone are involved. At times, however, similar changes take place in the other salivary glands. In a certain number of cases, chiefly occurring among adolescents or adults, there is an *orchitis* which is usually classed as a complication, but which is probably rather an unusual localization of the mumps infection. The pathological process is an interstitial inflammation of the body of the testicle, not affecting the epididymis. While the parenchyma of the testicle is not involved in the inflammatory process, the interstitial inflammation may be followed by atrophy of the testicle.

In females, a similar accompanying interstitial inflammation of the ovaries, breasts, or labia majora has been observed. Instances of such a localization of the process are extremely rare, but most of them have been seen in children.

SYMPTOMS.—While there are probably general prodromal symptoms in all cases, they do not attract attention in the milder attacks, and often the first thing to be noticed is the beginning of local symptoms. In the severer cases, the prodromal symptoms are often marked, and last from twelve to thirty-six hours before the appearance of local pain or swelling.

The usual prodromal symptoms are weakness, malaise, anorexia, chilly sensations and fever. The initial fever in mild cases is about 100° F. or 101° F. In severer cases there may be headache, vomiting, pain in the back and limbs, profuse sweating, and a temperature of 102° to 104° F. Nosebleed has been seen in the prodromal stage, and even convulsions have been reported.

The first local symptom is usually pain, which is referred to the area just below the lobe of the ear, and which is increased by movement of the jaws, and sometimes by the presence of sour substances in the mouth. Swelling of the parotid appears soon after, or sometimes simultaneously with the pain. Over the site of the parotid behind the ramus of the jaw appears an indefinite thickening, which is often at first more apparent to the eye than to palpation, and which is best observed by comparing the two sides. Occasionally both sides are affected simultaneously, but usually the involvement of one side precedes that of the other by several days. The swelling increases, feels tense and doughy on palpation, and has no sharply defined boundaries. The size reached by the swelling varies with the severity of the case; in marked cases the tumor may fill the lateral region of the neck between the jaw and the sterno-mastoid muscle, and may extend up on the face as far as the zygomatic arch. The essential feature in diagnosis is the presence of swelling, immediately under the lobe of the ear, and in front of the tragus.

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ATMENT.—There is no specific treatment for mumps, and ase being brief and self-limited, none is needed. The symp- e so few and so little distressing, that no symptomatic treat- required. Children should be kept in bed during the period and constitutional symptoms, and should be kept in the ıtil the glandular swelling has disappeared. The diet should as long as there is any pain on mastication. The swollen ay be protected by a pad of soft cotton. If pain is severe, y be applied, either with a hot water bag, or by an inunction olive oil. Akaline mouth washes should be used frequently. ıtions should be treated according to their symptomatic ns.

LEMS AND RESEARCH.—The central problem in mumps scovery of the organism which causes the disease. A certain of analogy with other diseases in which there is strong evi- at the cause is an ultramicroscopic filterable virus, suggests mps belongs also in this class. The first step in the investi- f the etiology of such infections is the reproduction of the ı animals. Such attempts have been made in mumps with aliva, but up to the present time the results of these investi- emain very incomplete. A transitory tumor of the parotid produced in monkeys with filtered saliva, but not con- o that it is possible that the tumor may represent some sec- nfection.

as been working on the production of artificial immunity ing exposed children with the blood of immunes. The of blood obtained is 6 to 8 c.c., and this, injected intramus- auses no unfavorable reaction. This procedure is promis- umps, as the long period of incubation permits inocu- ne time after exposure. In Hess' cases, mumps did not ı any exposed child thus inoculated.

ood changes in mumps have been thoroughly studied. It found that the lymphocytosis begins on the first day, and or fourteen days. It is believed that these changes may alue in the differential diagnosis from lymphadenitis. Feil- und an increase of lymphocytes in the cerebrospinal fluid which did not show meningeal sypmtoms.

INFLUENZA

(La grippe)

strictest sense, influenza or la grippe is an infectious and tagious disease, due to the *bacillus influenzae* and occurring in epidemics.

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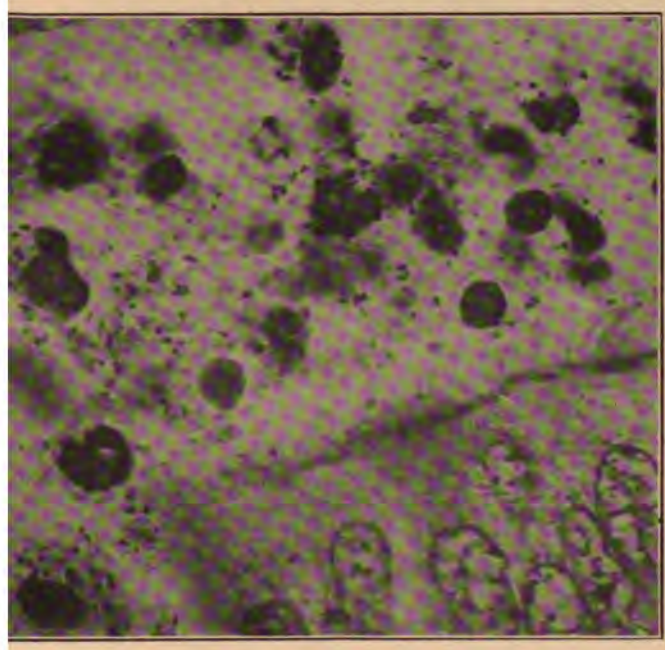
putum it appears as a very minute bacillus, which bipolar staining often resembles a minute diplococ- when present in the sputum, and when the cause of usually very numerous and occur in large clumps or ures the organism is aerobic, and grows best on blood- t appears as minute colorless droplet colonies. It media containing hemoglobin, this being the most in its differentiation from other microörganisms of gy.

FIG. 114



Influenza bacilli in the sputum

ism is found usually in the sputum from the lower air- t is found less frequently in the discharges of the naso- d in the discharge of acute otitis. Still more rarely it the pus of empyema and in the cerebrospinal fluid.

SION.—The disease is transmitted precisely in the same whooping-cough and measles, by "droplet infection." It air-borne contagion, and it is necessary for the individual ted to come into the actual presence of the one already There is no evidence that the disease can be carried upon ed objects.

OSITION.—At the time of the epidemic almost the entire was attacked. There did not, however, appear to be

In young infants,
and prostration ma
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There is a tenden
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VARIATION IN
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a pneumonia
very serious,
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ıts, even when no complications arise, the apathy
nay become extreme, and death may take place
he overwhelming intensity of the infection.
ency to the development of tuberculosis after influ-
ınd recurrent attacks are not uncommon. Out
r of cases in children examined by Schlossmann,
red in 8 per cent, but nephritis was rare. Anemia
ela in young children. Leucocytosis in uncompli-
not present. The mental disturbances and mul-
nmon in adults are not marked sequelae in children.

N TYPE OF THE DISEASE.—It is now generally
ere are distinct types of the disease.

YPE.—In this, the most common form of the dis-
bacillus may attack any part of the respiratory
to the pulmonary alveoli, the symptoms increas-
ording as the latter are approached. In the milder
usual symptoms of acute coryza, fever, and head-
mptoms are accompanied by a far greater degree
n is met with in ordinary attacks of catarrhal
der cases, without complications, may last only
mperature is usually moderate—101° to 103° F.,
n is marked. An annoying, persistent cough is

ıetimes much intensified, may last longer, have a
, and show a greater tendency to complications,
evelopment of otitis media and cervical adenitis.
cases of the pulmonary type, the disease, instead
adily, progresses, and with the development of
, and extreme prostration, may simulate typhoid.
no especial peculiarities, but in young children it
very intense grade, reaching the finer bronchi and
and threatened asphyxia.
nces an influenza pneumonia develops. This
ly lobular. Generally, there are small areas of
diffusely scattered, and often giving rise to no
; than patches of moist rales. The sputum in
enza bronchopneumonia is never "rusty," but
ıerally of a greenish-yellow color. A diagnosis
ding the influenza bacilli in the sputum in large
polynuclear leucocytes. The resolution of such
longed, ending by lysis. This class of cases is
ıuch of the mortality of epidemic influenza arises
ary complications.

DIAGNOSIS.—I
shows a symptom-c
tion, the physician
imply that the cas
influenza. The di
specific organism i
appear in the spu
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should be found v
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TREATMEN
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–If the term grippe be applied to a case which
-complex in any way resembling the above descrip-
ı must remember that the use of the term does not
se is actually one of infection with the bacillus of
iagnosis can only be made by the finding of the
n the sputum. Furthermore, the organism should
tum in such large numbers as to be unmistakable,
luenza bronchopneumonia, clumps of the organisms
ithin the leucocytes. The technic of the examina-
ı has been described in Division II. Although the
l in the sputum are often strongly suggestive,
he identity of the organism can only be obtained
hat it will grow only upon a hemoglobin-containing

—At the present time there is no generally avail-
ment for the disease, and the treatment must be
e general lines of treatment are those of any acute
It is best to isolate the patient during the attack
ecautions as are employed in other air-borne con-
lesirable to fumigate the sick room after the dis-

ıfluenza should be kept in bed and be treated as
tion. The bowels should be opened at the onset
calomel or castor oil. High fever is best controlled
the cold pack. Nervous or subjective symptoms
nall doses of phenacetine. Codein may be added
e. When there is severe and continuous vomiting,
champagne by mouth, and enemata of bromide
dicated. Stimulation is occasionally needed when
ıs of weakness, and is administered as in the other
When the convalescence is prolonged, especially
ncy to prolonged cough, a change of climate is

—Work has been done at the Rockefeller Insti-
on of an anti-influenza serum. Up to the present
only been employed in cases of influenza menin-
tive rarity of cases which are adequately proven
l infections of the respiratory tract, has made
to test the value of serum therapy against this
fection. We shall have to await another wide
ore we can test the value of serum therapy.

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Typhoid fever is,
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PREDISPOSITION.—
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e mammary gland, but by the contamination of
comes in contact with the child's mouth through
e breast-fed infants of typhoidal mothers often

s, through the conditions of its transmission, ex-
ffect more than one member of a family.

—After the fifth year, the frequency of occurrence
about the same in children as in adults. In chil-
ve years of age typhoid is much less frequent than
nder two years, typhoid is rare, especially in the
hen it is very exceptional. It should be remem-
can occur in infants, although it is uncommon.
that some cases of typhoid in infancy are over-
of the atypical clinical picture presented. Con-
not uncommon in children born of mothers sick
n such cases the bacilli gain entrance to the fetal
the placenta. The commonest result of fetal
the fetus and abortion, but a child may be born
and die soon afterward on account of its feeble

to typhoid fever is general, and there is no
ariations in susceptibility or of relative immunity,
he smaller frequency of the disease in infancy
tributed to the fact that infants have less oppor-
adequate contact with infected material.

ugh typhoid fever is a self-limited disease, it
immunity following an attack lasts only for a
l attacks are comparatively common.

ANATOMY.—In childhood the pathological
e in indirect ratio to the age. After the second
sions are constant and the pathology definite.
lvanced and severe lesions of typhoid fever may
well as in the later years of life, yet its char-
young subjects are found to be less definitely
nsist essentially of a hyperplasia of the solitary
yer's patches, and the process, instead of going
ally terminates in early resolution with fatty
lls. Ulceration is absent or slight, and hemorr-
are therefore rare complications in the typhoid

esions in the intestine, hyperplasia of the mes-
ar degeneration of the liver and kidney, and
neration of the heart may be found. Paren-
metimes occur in the peripheral nerves, with

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SYMPTOM
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efflorescence of typhoid fever, the rose-spots, have
ınal importance in the diagnosis from recent bacterio-
ıtions. Various observers have demonstrated the
typhoid bacillus in the rose-spots in a very large
ıe cases examined.

.—The typical clinical picture of typhoid fever in
markedly from that seen in adults. This applies to
of cases. It must be remembered, however, that in-
uently encountered in which the disease runs a course
esembling that characteristic of adult typhoid, and
ın symptomatology and severity which are encountered
occasionally be seen in children. It will be assumed
ader is thoroughly familiar with the general descrip-
fever, which is that of adults, and attention will be
to pointing out the differences characteristic of the
frequently encountered in childhood.

USUAL CLINICAL PICTURE IN CHILDREN

oid fever in childhood resembles more or less the mild
ease as seen in adults. The principal differences are
A shorter febrile period with greater frequency of
a less high range of temperature; absence of the severe
tions of the disease affecting the nervous system;
complications, particularly hemorrhage and perfora-

—In typical cases the child becomes ill with rather
ıs, but the onset, while not sudden, is rather more
ed than is often the case in adults. The symptoms
ıal stage are weakness—the child tiring easily—loss
stless sleep, and at times occasional vomiting. In
there may be headache, which is usually not very
xis is very rare. In this stage high fever is usually
emperature is becoming higher each day, and one of
h especially attracts the attention is that the most
examination fails to reveal a cause for the tempera-
high in proportion to the general constitutional dis-
nany cases the constitutional disturbance is so slight
he fever which attracts attention.

—The temperature curve in childhood bears a general
the classical curve of adults. There is a period of
f temperature, a period of high continuous fever, a
rked daily remissions, and a period of fall by lysis. In
er, all these periods are apt to be shorter, the entire
disease being more compressed. The temperature

in adults. The
apt to pick the
border at the e
careful examin
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not to be felt.
second week.
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lips are often dry and cracked, and children are very
m. The *spleen* is usually palpable below the costal
d of the first or beginning of the second week. With
tion the splenic tumor should be found in the second
ase, but after the second week the spleen is usually
The *rose-spots* appear usually at the beginning of the
n children rose-spots are usually very sparsely scat-
ften to be found only upon the abdomen after careful
younger children they are often absent entirely, and
spots are less constant than in adults, appearing in
o to 70 per cent of the cases. *Abdominal distention*
mon in children than in adults and is seen most often
n. Even when present, it is usually less obstinate
require treatment than in adults. Pain and tender-
men are unusual, although some children complain of
e over the appendix region, but do not show involun-
spasm. *The bowels* in the typhoid fever of children
characteristic disturbance than in adults. In gen-
n is more common than diarrhea, and this is particu-
he younger children. Diarrhea however is seen at
in the severer cases. The stools, even when diarrhea
not so characteristic in appearance as in adults, the
stool being rare. *Bronchitis* of a mild form is present
of cases, especially in children of weak constitution.
e bronchitis is so marked as to lead to an error in
physician being led to believe that the infection is
n the lungs. In weak and rachitic infants, broncho-
develop. *The circulation* in typical cases of typhoid
ife is much less disturbed than in adult life. The
sounds do not usually show signs of weakness suf-
e stimulation. The relative slowing of the pulse in
he height of the fever which is so important a diag-
e typhoid fever of adults, does not appear in children
xth or eighth year; and the same is true of the dicrotic
vous system is much less affected in children than in
ore severe nervous symptoms so pronounced in later
minent in early childhood. Headache of a mild type
n, especially in the early stage of the disease; severe
uncommon. Crying out at night, especially in young
delirium of a mild type are not uncommon. Deep
n fact the chief characteristic of typhoid fever in young
hy. The child takes the nourishment which is given
pecially restless, and usually lies in a half-somnolent
urine often shows a small amount of albumin and
blood corpuscles, especially in cases with high fever.

for; exceptionally
puration of the p
The vomiting c
and is seen only i
especially in neurc
quent, severe and
Otitis Media is t
of children. It is
Bronchopneumon
weak resistance. I
The Circulation.—
and prolonged case
ing of the first heart
carditis and perica
action of the heart
have no serious sign

The Kidneys.—A
typhoid bacilli are e
valescence, but usu
of pyelitis or cystiti
The Nervous Syste
Signs of meningeal ir
and in cases with ri
ties, lumbar punctur
pressure of cerebros
The Skin.—At th
sometimes seen rathe
cases may show vari
course of the disease
children have profu
sudamina, and often
the trunk and extrer
or feet. Decubitus i
other hand, furuncul
valescence.

The Bones.—Periost
children. It usually
not until the lapse of
circumscribed, and m
complication is accom
is often overlooked.

Relapses are just as
in that of adults, and
resemblance to the ori

ally through secondary infection, there may be a sup-
he parotid gland.
ing of typhoid fever is usually comparatively slight,
nly in the earlier stages of the disease. Occasionally,
neuropathic children, the vomiting is much more fre-
and persistent.
a is the commonest complication of the typhoid fever
It is usually of a benign character.
umonia is seen at times, especially in young children of
ce. Its development is often very insidious.
tion.—The circulatory system is only affected in severe
l cases. A toxic myocarditis betrays itself by a shorten-
t heart sound, and by a pronounced tachycardia. Endo-
pericarditis are very rare. Bradycardia and irregular
heart are sometimes seen during convalescence, but
us significance.
ys.—A pronounced nephritis is extremely rare. The
li are eliminated in the urine and persist even in con-
ut usually produce no symptoms. Occasionally, signs
cystitis are found in the urine.
us System.—In older children aphasia is seen at times.
ngeal irritation are commoner in children than in adults,
with rigidity, Kernig's sign, and spasm of the extremi-
puncture often reveals either an increased quantity and
erebrospinal fluid, or a slight increase in the cell count.
—At the beginning of the disease a toxic erythema is
en rather more often in children than in adults. Severe
ow various forms of erythematous eruption later in the
disease. Not infrequently after subsiding of the fever,
e profuse sweating, which leads to the formation of
id often leads to a certain amount of desquamation of
id extremities, which does not involve the face, hands
cubitus is much less frequent than in adults. On the
furunculosis is not uncommon in children during con-

.—Periostitis is seen at times in the typhoid fever of
usually occurs during convalescence, and sometimes
lapse of several months. The areas are usually small,
l, and most frequently found upon the tibia. This
is accompanied by slight if any febrile reaction, and
looked. Osteomyelitis is very rare.
re just as common in the typhoid fever of children as
dults, and the course of a relapse usually bears a close
to the original attack.

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differentiate these two conditions at a very early stage, examination in pneumonia almost always shows a leu-

ms of non-specific *intestinal infection* are seen, in which nal symptoms are not so prominent as in typical cases diarrhea. Such cases may be mistaken for typhoid. s may be in doubt for several days, but the fever in these ıditions is usually of brief duration.

s so common in infancy and early childhood often bears tomatic resemblance to typhoid fever. The differential ı be made at once by examination of the urine.

uberculosis, especially the form which eventually mani- n the symptom-complex of tuberculous meningitis, is ıely difficult to distinguish from typhoid fever. The .y be in doubt for a considerable time, until finally set- appearance either of the splenic tumor and rose spots, e cerebral symptoms. The results of lumbar puncture :ttle the diagnosis before the appearance of the Widal

pticemia, or malignant endocarditis, comes more rarely into ial diagnosis of typhoid fever in children than in adults. certain cases of *rheumatic fever*, in which arthritic symp- ght or absent, the process mainly involving the endo- ich are difficult to distinguish from typhoid fever. Even docardial murmur is present, the physician may be in ıer it does not represent a preexisting endocarditis.

also certain forms of *influenza*, in which the catarrhal ffecting the upper respiratory tract are slight or absent, :ally resemble typhoid fever. Such cases have been "abdominal grippe." They are rare, except in recog- pread epidemics of influenza.

certain cases of typhoid fever in which there is so much nderness in the right iliac region, and so much vomiting, *'icitis* is suspected. In these cases, however, careful ex- vill usually show that involuntary spasm is absent. On nd, there are cases of appendicitis in which fever is promi- ɔcal pain and tenderness slight. The white blood count t importance in the differential diagnosis of these diseases, itis practically always shows a leukocytosis.

:ases of typhoid fever in children the signs of toxic irrita- meninges are so prominent as to suggest some form of *eningitis*. In such cases, lumbar puncture should be in order to settle the diagnosis.

ORY DIAGNOSIS.—There are a number of laboratory tests, ssential to the diagnosis of typhoid fever. Some of these

existence of typh
much earlier in t
by means of bloo
first week in from
Other laborato
Research.

PROGNOSIS.—
good. Statistics
than in adults, a
age. The compl
the intestine or f
parison with tho
varies very much
stages, and a chil
from it.

PROPHYLAX
tion required in p
described in deta
typhoid fever of a
life, it will be as
prophylaxis.

ANTITYPHOID V
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The vaccine r
killed typhoid b
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often used in Bo
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To what exte
children? In cc
character of the
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only under excep
tion of children
conditions:

typhoid fever. It has the advantage of being positive
r in the course of the disease. Some writers state that
f blood cultures a positive diagnosis can be made in the
ı from 90 to 100 per cent of cases.
ɔoratory tests will be considered under Problems and

ɔSIS.—The prognosis of typhoid fever in childhood is
ɪistics show that the disease is much less fatal in children
ults, and that the mortality increases directly with the
complications whether arising from local disturbance of
e or from cardiac and pulmonary disease, are rare in com-
h those encountered in later life. The disease, however,
much in its severity in different epidemics and in different
a child may have a severe type of typhoid fever and die

YLAXIS.—The various measures of isolation and disinfec-
ed in preventing the spread of typhoid fever will not be
n detail. They differ in no way from those used in the
ver of adults, and as the disease is particularly one of adult
be assumed that the reader is already familiar with its
s.

HOID VACCINATION.—Within the last decade, inoculation
phoid fever has been widely employed, especially among
ıd sailors, but also to some extent in civil life. There is
ɔf the preventive power of antityphoid vaccination. Many
ıain to be settled, among them being the duration of the
produced in this way, and the comparative value of vac-
ared in various ways.
ɛcine most commonly employed is a suspension of heat-
hoid bacilli. It has been shown that severe reactions are
that when small amounts of a vaccine of low virulence are
e is little inconvenience.
ıre various methods of using the vaccine. The one most
l in Boston is to give four inoculations at five-day intervals,
being 100 million, 200 million, 400 million, and 600 million
. Proportionally smaller doses are used for children. The
n is made in the interscapular region.
ıt extent is inoculation against typhoid fever indicated in
In considering this question, the comparatively mild
of the disease in early life has considerable weight. I have
ıded antityphoid vaccination in very few cases, and then
er exceptional circumstances. I believe that the vaccina-
hildren should only be recommended under the following
s:

tions. Neverthele
dences of indigest
undigested stools.
leave a large undi

The diet should
in health, with the
children would incl
eggs, butter, minc
child is normal the
by the addition of
to the milk. If th
reduced according
three-hour interval
difficult to overfe
getting them to t
sary, but any form
in exceptional cas

THE BOWELS.—
may be ordered to
If with this routin
ites, the enemata

Diarrhea requi
in twenty-four h
and if this fails, o
should be used.

In childhood ty
ment. When ma
by the rectal tu
should be avoide

HYDROTHERAPY
fever for the con
nervous symptom
has not been sho
of the disease.
whenever the ten
agree with this st
by restlessness or
that hydrotherap
just because the
are no signs wha
be remembered t
well as adults.
on the nervous s
rather than by i

ıeless the physician should always watch for evi-
estion from overfeeding, as shown by vomiting or
ls. It is also best to avoid articles of food which
ıdigested residue, such as fruit and vegetables.
ld be about what a child of the same age would take
he exception of fruit and vegetables. This for older
ıclude milk, cereals, bread or toast, beef-juice, broths,
ıced chicken and ice cream. If the digestion of the
he caloric value of the food can be further increased
ɔf some dextri-maltose preparation, cream, or cocoa,
there are any signs of indigestion, the diet must be
g to the indications. Feedings should be given at
als by day, and one feeding at night. It is usually
eed children with typhoid, the difficulty lying in
take enough. Considerable urging may be neces-
m of forced feeding should not be employed except
ses.

-Constipation is best combatted by enemata, which
be given every other day if the bowels do not move.
e the constipation is accompanied by any tympan-
should be given daily.
es no treatment unless the number of movements
urs exceeds five. Bismuth should be tried first,
ıium in doses appropriate to the age of the patient

npanites is rarely severe enough to require treat-
ted, it may be relieved by turpentine stupes, or
e. Glycerin suppositories and glycerin enemata

—This method of treatment is used in typhoid
ıl of high temperature, and for the relief of such
as restlessness, sleeplessness, or headache. It
to have any influence on the course or mortality
ɔst writers state that hydrotherapy is indicated
rature reaches a certain point. I do not entirely
ment. High temperature is usually accompanied
ne nervous symptoms, and in such cases I believe
s indicated. I do not believe it to be indicated
nperature reaches a certain point, when there
er of any disturbance from the fever. It must
children in general do not bear cold baths as
must be guided by the effect of hydrotherapy
n, and on the general condition of the patient,
fect upon the temperature.

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ting laboratory methods of diagnosis, especially those biological reactions.

ems in transmission and disinfection.

ne therapy and prophylaxis, the principal question now ussion is the comparative value of heat-killed vaccines, s living vaccines, and "sensitized vaccines" prepared by living cultures to the action of immune serum. The use ed vaccines was suggested by Metchnikoff and Besredka. esting to note that in the course of their experiments, they typhoid lesions in a chimpanzee with fecal material from a patient, recovering the organism from the blood. There s been a weak spot in the proof that the bacillus of Eberth ause of typhoid fever, as the disease could not be adequately ed in animals from cultures, and there was a possibility that fever was due to some undiscovered filterable virus, the being a secondary invader. Filtered fecal material failed uce the disease in the chimpanzee, and Metchnikoff and 's work gave the final fulfilment of Koch's laws. Their the superior efficacy of sensitized vaccine was based on the t bacilli from a carrier were not virulent for this chimpanzee, tected him from virulent bacilli, whereas vaccines made of acilli did not protect him. Other observers are working with ed vaccines, and the most recent experimental work tends to nat the serum of persons so treated does not deviate comple- nor give an agglutination reaction, but appears to increase ytosis, and probably contains anti-endotoxins. It further s that the detecting of various specific bodies is no measure ual immunity.

complexity of the problem of the nature of the immunity is ted by this work. It is evident that in typhoid fever immune are many and various, and we know little of their relative tance. The finding by Gay and Claypole of a specific hyper- ytosis on reinoculation of animals already immunized by heat- bacilli, is interesting in connection with the question of the e of immunity and the value of sensitized vaccines.

ere are a number of laboratory methods recently perfected, are mainly used in the research laboratory. The technique e isolation of the bacilli from the feces is still too complicated rdinary clinical use, but has proved of value in the detection of ers. It has been shown that all patients sooner or later give sitive complement fixation test, but there appears to be no rela- between this and other tests based on immune reactions, and as no value in early clinical diagnosis.

everal investigators have also found a cutaneous reaction, which believe to be constantly present during convalescence. They

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which is carried on in the blood of the intermediate host
other being that of *sexual propagation* which is carried
astro-intestinal canal of the definitive host (mosquito).
ages of the sexual forms, however, are also developed in
blood, and it is these forms which when withdrawn by
o carry on the sexual cycle. The paroxysms characteris-
lisease are produced by the *segmentation* of the mature
ns, which occurs at regular intervals, and which is a part
ual cycle. The sexual forms are resistant, latent and
the blood for a long time without producing symptoms.
her been shown that the female sexual forms are capable
ing segmentation without fertilization, and the small
s produced may start a new asexual cycle. This accounts
infections and the *relapses* which are apt to occur in the
the year, or upon removal of a patient from one climate
r.

xual cycle of the tertian parasite, ending in segmentation,
l every forty-eight hours. The asexual cycle of the quartan
is seventy-two hours in length. The estivo-autumnal is
known as tropical or pernicious malaria. The asexual cycle
of the tertian is forty-eight hours in length.

tian parasite first appears in the red blood-corpuscles as a
aline body with ameboid movements. It increases in size
lops fine, dark, pigment granules, which are in constant
due to the ameboid contractions of the protoplasm of the
l parasites. The red cells which contain them become
enlarged and gradually decolorized. Each parasite when
wn nearly fills the red corpuscle. In the stage of segmenta-
ich occurs at the time of the paroxysm, fifteen or twenty
s appear, which invade fresh red corpuscles, and begin the
ain as hyaline bodies.

uartan parasite resembles the tertian, but its ameboid move-
re slower, the pigment granules are coarser, darker, and less
the full-grown organism is smaller, and the red cell in which
anism develops becomes somewhat shrunken about the para-
d of a deeper, old-brass color. It segments into only five
parts.

estivo-autumnal parasite is smaller than the others and con-
ss pigment. The corpuscles containing them become shrunken
ass-colored. About a month after the onset of the attack,
teristic crescentic or ovoid bodies appear, which have coarse
t granules clumped in the center. These are the sexual forms.
tertian form is the one which is by far the most common in this
f the country, and the one which is most influenced by the
istration of quinin; the other form, represented by the quartan,

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SITION.—It is probable that every human being bitten cted anopheles, will develop the disease. In malarial disease is very common in infants and young children, common if not more common than in adults. This is ecause young children are so frequently bitten by mos-

LOGICAL ANATOMY.—There are no especial differences ıe pathological lesions found in the malaria of children which occur in adults. According to Thayer, in *acute* ıalarial fever, on examination with the microscope, the ıpillaries are found to be crowded with malarial parasites. sually a marked granular degeneration of the endothelium ssels.

een is always enlarged, often only moderately. The paren- cyanotic, of a slaty-gray color, and almost diffluent. The e spleen is found to contain enormous numbers of red blood- ;, many of which contain parasites. Great numbers of ls are also seen, some of them being necrotic. The capil- : usually filled with red blood-corpuscles containing the a, while the splenic veins show relatively few, although they ontain large cells enclosing pigment or the remains of red rpuscles.

iver is swollen and has usually a slaty-gray color. The :s are filled with leucocytes, which contain numerous pig- bodies. Relatively few plasmodia are found in the blood- es in the vessels. Areas of disseminated necrosis similar : in other acute infectious diseases have been described.

/essels of the *kidneys* contain relatively few organisms. The .li may be considerably pigmented. There may be marked ation of the epithelium of the capsule, and at times changes parenchyma, especially areas of necrosis of the epithelium of ıvoluted tubules. The other viscera show no special char- ic changes, except, at times, that of melanosis.

IPTOMS.—The symptoms of malaria as the disease occurs in and young children are much more varied and far more un- than those which we are accustomed to meet with in adults. rominent symptom of malaria being the paroxysm, earlier s naturally classified malaria according to the time when the /sms appeared, using the term quotidian when they occurred ntervals of twenty-four hours, tertian when they occurred with als of forty-eight hours, and quartan where they occurred with als of seventy-two hours. Again, the terms *intermittent* and *ent* have been used commonly. The intermittent form is char- zed by entire absence of fever between the paroxysms. The

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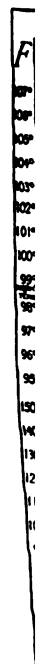

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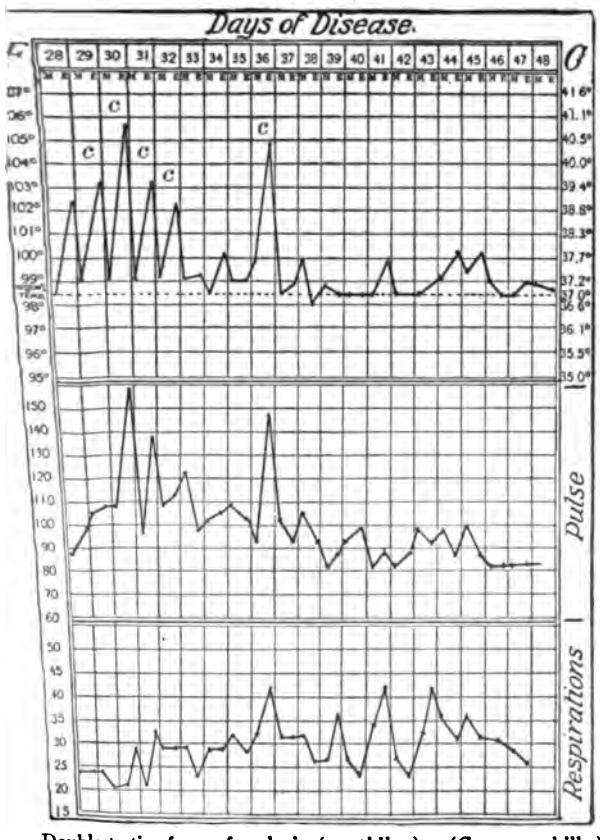
ıer, in cases of quartan fever, through the ripening of of parasites on different days, different combinations ding to the number of sets of parasites. Thus, while in which there is only one generation the intervals be- aroxysms are seventy-two hours, in that in which there

CHART 17



Double tertian form of malaria (quotidian). (*C* means chills.)

generations there may be an interval between the parox- only forty-eight hours, and when there are three generations ay be an interval of only twenty-four hours, thus representing otidian chills described by Mannaberg. The following table s the different types of paludism as they are now understood most recent investigators:

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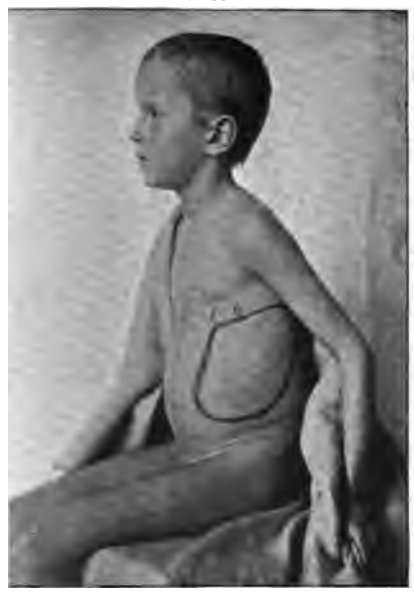
ı fever, and sometimes accompanied by coldness of and a collapsed condition very commonly replaces adult. These symptoms, representing the onset of y often disappear as the disease becomes established, ıce we may meet with the symptoms of some other ısceptibility of the nervous and respiratory systems ːn tends to produce variations in the form and type ı are misleading in regard to diagnosis, the symptoms articular organ often completely overshadowing the ıria, and producing an entirely new clinical picture. often are so indefinite, and the disease frequently liously, that the physician does not see the case until siderable progress and the diagnosis is thus much

FIG. 116



nlarged spleen. Plasmodium malariae found in the blood

ː other symptoms, severe pain in the head and gastric region is met with. In the form in which ual, the prominent symptoms are anemia, loss ıl headache of moderate type. Splenic or hepatic the back, extremities, and neck, and general sia are occasionally observed.

ATION.—The only constant physical sign in ːnt of the spleen. This occurs rather more han in adults, although there is considerable when the splenic tumor can first be detected.

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IS.—Malaria as it occurs in early life is far more diffi-
ose by its symptoms than when the disease runs the
: usually seen in the adult. It is the most protean dis-
e are called upon to deal with in young children, and
o closely so many other conditions we are likely to meet
should always be on our guard, and allow for the possi-
presence of the plasmodium malariae in making a diag-
doubtful case. Whenever there is any periodicity in
ıs, or whenever there is definite splenic enlargement,
ld be suspected. The only rational method of determin-
we are dealing with a case of malaria is by an examina-
ood, which at once settles the question if the plasmodium
. The parasite can usually be found in a patient who
: or no quinin, if the blood is thoroughly examined during
by a person who has had considerable experience. Both
nd the stained specimens should be examined. The
ucocytosis in malaria is an important aid in the exclusion
fever dependent upon septic processes.

SIS.—The prognosis of malaria in children is good,
ıt the child is removed from the malarial district and is
ı quinin. Relapses occur, even after long intervals of
ımunity, and the disease can recur a number of times.

LAXIS.—The value of mosquito-nets from a prophy-
point is significant, and observations in malarious dis-
g the last year or two strongly support this idea. The
patient with malarial fever in a malarious district is a
ınger to those about him is a very important one, the
eing, of course, that one should insist upon such an indi-
ing under a mosquito-net. The importance also of early
gh treatment of all cases of malaria, particularly relapses
malarial season, is evident. In some regions destruction
uito larvae has accomplished a great deal.

MENT.—Malaria is one of the few acute infectious dis-
hich there is a specific drug treatment. *Quinin*, when
ven, will bring about a rapid cure in all cases caused by
parasite, and in most cases caused by the other varieties.

S OF ADMINISTERING QUININ.—Quinin may be given by
rectum, subcutaneously, or intravenously. As a routine,
ority of cases the drug should be given *by mouth*. Infants
ually object to the taste, but they cannot swallow an in-
m of quinin. It is therefore best in infants to use the
in aqueous solution. A convenient preparation is to make
contain two grains to the teaspoonful. If this solution is

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paroxysm is due. The doses have to be rather large. ear quinin well, and the mistake usually made is to give The large dose is given first, in order that if the child has ıcrasy toward quinin, symptoms of disturbance will appear nd the second and third doses may be omitted.
or the three doses before an expected paroxysm, no other given. Used in this way the quinin should entirely abort ysm. Each generation of plasmodia is attacked in this . For example, in a single tertian infection, the quinin repeated once on the day when the next paroxysm would occur. In a double tertian infection, the quinin should ed daily for three days, four days in all. If however the es not entirely prevent the first paroxysm, that generation of should be attacked a third time, the rule being to attack ration once more after the paroxysm has been prevented. he paroxysms have been attacked as described above, I nsider it wise to continue to give quinin in smaller doses sure," as is usually done. If any parasites have escaped ıal onslaught, they will not be destroyed by the smaller en "to make sure," but will only be prevented from develop- causing fresh symptoms. They may thus remain latent in , and may cause a relapse at some distant future time. I ıe practice of continuing quinin in small doses is responsible r incomplete cures and subsequent relapses. My practice *no more quinin* as long as I can keep the patient under close on, with a four-hourly temperature record. This permits tunity for undestroyed parasites to develop and to cause ıs, in which case the original treatment is repeated. Before discharge of the case, the blood is carefully examined as a precaution. Sometimes when the case is about to be released se observation, I give more quinin to make sure, but under umstances I give the full twenty-four hour amount used in nal attack, in three doses at eight-hour intervals, for two or ys.
clinical picture is very indefinite with no recognizable paro- or periodic times when the symptoms show a maximum in- the same quantity of quinin should be given each twenty- ırs as in the table, but the doses must be differently arranged, twenty-four hour amount should be divided into three nearly ortions, which are given three times a day. This should be ed for two days after all symptoms have disappeared, and quinin should be omitted until symptoms reappear, or till ient is discharged.
ria rarely requires any symptomatic treatment, because h there are many functional disturbances and subjective

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DISEASES TENDING TO OCCUR IN EPIDEMICS

ıp includes two diseases which tend to occur in epidemics, ch the essential facts about the mode of transmission are own. These diseases are *epidemic cerebrospinal menin-cute polio-myelo-encephalitis*.

PIDEMIC CEREBROSPINAL MENINGITIS

eningococcus Meningitis). (Cerebrospinal Fever).

n cerebrospinal meningitis refers to an acute infection of ıeninges, which may be due to a variety of microörganisms. : bacteria which produce the lesions of acute leptomeningitis, ıe variety which produces no other disease process in the dy. This variety of meningitis may therefore be consid-*cific* form, and on account of its tendency to occur in epi-ıe term *epidemic* is used in differentiating it from other varieties of cerebrospinal meningitis. These other vari-ıot specific, as the organisms which produce them can also :r disease processes; they will be described in the Division :s of the Nervous System.

)GY. THE MICROÖRGANISM.—The specific organism of meningitis is the *diplococcus intracellularis meningitidis aum*), or, as it is now generally designated, the *meningo-*'he organism occurs in pairs, and closely resembles the s in its biscuit-shaped morphology. It is Gram-negative, ; readily with methylene blue, and with other basic anilin is found in the meningeal exudate, and, in the acute stage ease, is always present in the cerebrospinal fluid obtained r puncture. In recent untreated cases the cocci are both l intracellular; in late or serum-treated cases they are chiefly lar. The coccus is sometimes difficult to grow on arti-ıure media. It grows best at 37° C. on media containing : animal serum, such as blood-agar, or ascites-agar; the best is ascites-agar to which some glucose has been added.

ıeningococcus is extremely sensitive to injurious agencies ıds. It dies out quickly in cultures unless frequently trans-and is very sensitive to light, drying, and even to dilute lution. It has never been found outside the body. It is ıon-pathogenic for animals, except young mice and guinea-

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heir struggle for existence, have the faculty of to time new powers of attack and defence. They reased power of resistance against antagonistic in-"), or an increased power of developing in the tis-irulence). The work of Rosenow with the strepto-ests that an increase of virulence may take place crypts. It is conceivable that at times a similar nce may take place in the meningococci carried in of a healthy carrier. This is the best available ıe peculiar features of the transmission and epidemi-ic cebrospinal meningitis.

ON.—On account of the unknown facts as to trans-ode of infection, the general predisposition toward ngitis cannot be estimated. The disease is much ng children than among adults, although it can occur ıd is not uncommon in young adults. About 50 per es are seen in children under the age of five years, and cent of these in infants under one year.. The youngest e seen was three weeks old.

are usually seen in the winter and early spring, and sporadic cases also occur at this season. In Boston the st prevalent in March, April, and May, and is very rarely summer and autumn.

.—On account of the peculiarities of the incidence of , it is not known whether an attack confers any lasting

LOGICAL ANATOMY.—The lesions produced by the s intracellularis are not specific, and show no essential s from those produced by other organisms. They will be under Meningitis in the section on Diseases of the Nervous

TOMS.—Epidemic cerebrospinal meningitis presents itself ere, acute, febrile disease, with a great variety of symptoms e to the lesions of the central nervous system. These symp-e most conveniently described in the order in which they would d by a physician when called to see a case of meningitis.

HISTORY.—In taking the history the physician should inquire ılarly as to the onset, fever, headache, other subjective symp-vomiting, convulsions, and the mental condition of the patient. *Onset** is usually, though not invariably, extremely sudden. is ordinarily sufficiently marked to have been noted by the

order that the reader may at once gain an idea as to the frequency of occurrence various symptoms and signs, those which occur in more than half the cases are d in *italics*.

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s nearly always present at some time during the disease. n to rigidity, *pain on motion of the neck*, and tenderness to ıre often found. There is frequently marked retraction of and there may be a more or less marked opisthotonos.

pils are usually contracted in the early stages of the dis- e in the later stages they are apt to be widely dilated. The reaction to light is often sluggish, and in very severe cases ɔe absent. Strabismus is a rare symptom. Sometimes, in the later course of the disease, blindness may be detected; oscopic examination shows it to be due to optic neuritis, or congestion of the fundus. Photophobia is often a nota- ·tom.

FIG. 118

Opisthotonos in meningitis

mination of the heart, lungs and abdomen reveals nothing mal beside weakening of the heart sounds, unless some com- ion is present.

ıarked *tonic spasm of the muscles* of the extremities is a common tom. The muscles are involved to a variable extent; at times e limbs are very rigid. Attempts to move the limbs may cause ɜ pain. Muscular tremor is seen at times. Actual paralysis of scle or group of muscles is rare in epidemic meningitis, an im-

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ıses of epidemic meningitis may be divided into four
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ating type, in which the course of the disease is so
l that death occurs in the first twenty-four or forty-
hese patients when first seen are already in an ex-
if not moribund, condition, with profound coma,
ion, and signs of beginning circulatory failure.

ype, in which there is an early development of severe
n which the course of the disease is not so rapid as
ıg type. There is a pause in the progress of the
onset, the patients remaining in *statu quo*. When
tients are either unconscious or in violent delirium,
l condition.

type, in which the patients when first seen are not
are either very apathetic or in mild delirium, with
ıl condition.

e, in which the patients have headache, fever, and
ck, but are perfectly rational, with good general

in epidemic meningitis are apt to reach their
early in the course of the disease.

on deviations from the symptoms described above
, in whom the onset is often less sudden than in
l the signs pointing to irritation of the central
s pronounced. It is not uncommon in infants to
he symptoms of fever and clouding of the men-
of the neck and limbs not being pronounced. In
ere is the very important sign of tense prominence
hich is almost invariably present in all forms of

ıe disease has been much influenced by serum
ription which will be given here applies to the
e as it was observed before the days of serum
is now observed in cases which prove resistant
nent.

; type death invariably occurred within twenty-
ours, and even with serum therapy, most cases
type die before the serum has time to produce

of the disease, after the stormy onset, there
f variable duration in which there is not much
t's condition. In the majority of cases of this
apparently from exhaustion, within the first
ıe of these severe cases, however, the patients
a period of about six weeks of active febrile

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f the disease may be either present or absent, and nnot depend upon any particular symptom or set There are a number of diseases in which symptoms tation are part of the clinical picture. In many of he meningeal irritation may produce not only stimu- of the central nervous system, but may even produce nsion of the cerebrospinal fluid. This condition is as *meningismus* as distinguished from true menin- is no actual infection of the meninges, but only irri- is produced elsewhere. In many cases in which meningismus is present, the clinical picture pre- hat of epidemic meningitis in every way, and they guished from cases of meningitis by the results of

as the distributor of the Flexner serum throws an n the frequency with which other diseased condi- for cerebrospinal meningitis. During the period 142 cases of cerebrospinal meningitis, I was also r serum in 88 cases which had been diagnosed as ngitis, but which proved to be other diseased con- ny form of meningitis. The diseases which thus ed meningitis as to lead to a demand for serum, 48.

TABLE 48

Diagnosed as Cerebrospinal Meningitis on the Basis of Their Symptom

	CASES
nonia	29
ic intoxication	15
iomyelitis	10
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VOLUME 23
NUMBER 5
MAY 1988
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from the above considerations, that *without lumbar agnosis of cerebrospinal meningitis is absolutely without ntific, statistical, or therapeutic purposes.*

FOR LUMBAR PUNCTURE.—The importance of an ac- is in suspected cases of meningitis is so great, that the tion which confronts the physician in every case is : he should perform lumbar puncture. Naturally one o perform a useless lumbar puncture in cases of pneu- ro-intestinal disease. On the other hand, I believe cian is seriously to be blamed if a case of meningitis ve early diagnosis and proper treatment. In every ny of the symptoms described above as being char- ningitis are present, the possibility of this diagnosis nized. The indications for lumbar puncture in such ummarized as follows:

t of a careful history and physical examination, the id definite evidence of the existence of another disease of producing the meningeal symptoms, he is justified condition as the cause of the symptoms, and lumbar dicated. If, on the other hand, the physician cannot evidence of any other disease process capable of eningeal symptoms, immediate lumbar puncture is

tained by lumbar puncture be absolutely clear, epi- nal meningitis can be positively excluded, except ve run so long a course as to be already in the late the fluid be cloudy, the physician can conclude f meningitis is present, and as the form caused by s is the commonest which produces cloudy fluid best to conclude that the case is one of this type. ood-stained, no positive conclusion can be drawn. diagnosis depends upon the results of the labora- f the cerebrospinal fluid, the technic of which has escribed in Division II. For a positive diagnosis, the *diplococcus intracellularis* be found in the fluid. is a marked predominance of polymorphonuclear sm of any kind is found, it is best also to assume poses, that the case is one of epidemic meningitis.

Before the days of antimeningitis serum, the h different epidemics. The mortality at different nt localities has ranged from 50 per cent to 100 ge mortality being about 70 per cent. In cases rious and permanent sequelae were observed in nt. Since the introduction of serum therapy the

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use of serum. The prognosis in such cases depends mainly
r much damage has been done to the tissues in the period
e serum is first given.

TMENT. INDICATIONS FOR SERUM THERAPY.—The ad-
ion of antimeningitis serum is indicated in every case of
cerebrospinal meningitis, except those cases which have
d so far into the chronic stage of the disease that the menin-
is no longer present in the cerebrospinal fluid obtained by
puncture. If the physician has no serum at hand at the time
e makes his first diagnostic lumbar puncture, he must per-
ait until he can obtain the serum, and will have time to con-
diagnosis by the laboratory examination of the cerebrospinal
Whenever it is possible, however, it is best for the physician
called to see a suspected case of meningitis to carry the serum
m, so that he can administer a dose at the time of the first
puncture. It is true that at this time the diagnosis cannot
tively confirmed, but so important is the early administration
serum, that there are certain conditions which warrant its
iate use even before the laboratory examination of the cerebro-
fluid is made. The indications for giving the serum at the
of the first lumbar puncture are the following:

f the fluid obtained by lumbar puncture shows any cloudiness
ut being blood-stained.
When, with blood-stained fluid, the patient is severely ill with
toms strongly suggestive of meningitis.
th either of these conditions the immediate administration of
1 is warranted without waiting for the results of bacteriological
ination. Further injections, of course, are only to be given in
the laboratory examination of the cerebrospinal fluid confirms
liagnosis by revealing the presence of the diplococcus intracellu-
. When the serum is not at hand at the time of the first visit,
ie laboratory examination of the fluid confirms the diagnosis,
m should be obtained and used as soon as possible.

ECHNIC FOR THE ADMINISTRATION OF ANTIMENINGITIS SERUM.—
antimeningitis serum is injected directly into the spinal canal.
the evidence goes to show that the subcutaneous injection of the
im is practically of no value in comparison with the subdural
thod.
The technic of lumbar puncture has been described in Division II.
ien serum is to be administered, as much fluid as possible should
allowed to escape. The withdrawal should be continued until the
id is running no faster than four drops to the minute. While
e fluid is running, a sterile syringe should be filled with the proper
se of serum, which has previously been warmed to about blood

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ensation of the method to be employed in the routine
epidemic cerebrospinal meningitis with serum:

mbar puncture is performed in a suspicious case, be
ect the serum. If the cerebrospinal fluid withdrawn
e the injection of serum immediately and without
acteriologic examination. The next doses of the serum
n only if the *diplococcus intracellularis* has been dem-

withdraw as much cerebrospinal fluid as possible at
e and inject full doses of the serum. Thirty c.c. of
l be injected in every instance in which this quantity
ss has been removed, unless a distinctly abnormal sense
in the spinal canal is encountered after as much serum
ected as fluid has been removed. When the amount of
wn exceeds 30 c.c., introduce a larger quantity of serum
c.c., or even more. In the very severe or fulminating
om 30 to 45 c.c. of serum without reference to the quan-
removed unless abnormal resistance is encountered.
severe or fulminating cases repeat the injection of serum
enty-four hour period, as soon as the symptoms intensify,
condition remains stationary, after the lapse of the first

of average severity make daily injections of full doses
s. If diplococci persist after the fourth dose, continue
s until they have disappeared.
ubjective symptoms including fever and mental impair-
after the diplococci have disappeared, or after the four
een given, and improvement is not progressing, wait
the condition is stationary, and then repeat the four
hould the symptoms have become worse before the
this period, the injections should be resumed imme-

se, which is indicated either by reappearance of the
he cerebrospinal fluid or recrudescence of the symptoms,
s at twenty-four hour intervals are to be repeated and
nt treatment is to be conducted as for the original

n of treatment is to be followed until the patient is free
ms, the diplococci disappear from the cerebrospinal
chronic stage of the disease supervenes. The serum
f some benefit in the chronic stages in which the diplo-
present in the meninges.
been established the injection of serum into the spinal
es little.

In the m
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majority of cases cerebrospinal fluid can be obtained at bar puncture. Occasionally, however, most often in infants, of the most careful technic, lumbar puncture results in a When a dry tap is obtained at the first diagnostic lumbar , not only can serum therapy not be employed, because there urance that the serum is going into the cerebrospinal canal, the diagnosis is left in doubt. In such cases the physician repeat the lumbar puncture in the hope of subsequently g fluid. In the meantime no specific therapy can be used treatment must be wholly symptomatic. Usually, however,

FIG. 121

Tapping the cerebral ventricle

ıps are not encountered until later in the treatment of the case, he physician is proceeding under the assurance that the diag- of epidemic meningitis has been established. Intraspinal in- ns of serum in such cases are apt to be of no use, because if ı can be properly injected into the spinal canal, fluid should be ned by lumbar puncture. In infants with whom this condition ;, I believe that the physician is justified in undertaking more c treatment. The diagnosis of epidemic meningitis having been rmed, the prognosis in a child under two years is absolutely

In a certain
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ber of cases the effect of the serum upon the cere- hown in the progressive diminution and gradual the diplococcus intracellularis in the successive ed. Nevertheless the symptoms of the disease, er, are not notably affected. In such cases it is ne mechanical obstacle exists which prevents the ning the portion of the central nervous system in- ease. Some area is probably shut off, and in this ns continue to multiply and to produce symptoms. I found at autopsy a closure of the foramen of Ma-

FIG. 123

nsertion of the needle through the fontanelle in tapping the cerebral ventricles

by scar tissue, and the ventricles thus shut off were full of cci. In such a case the intraventricular injection of serum also be indicated. Whenever this condition of disappearance organisms from the cerebrospinal fluid without relief of symp- ccurs in an infant, I believe that the cerebral ventricle should pped, and if the fluid thus obtained reveals the presence of the coccus intracellularis, serum should be injected into the ventricles. organisms are found in the ventricular fluid, nothing further be done with serum therapy, and vaccine therapy should be loyed. In older children in whom this condition arises, intra-

ventricular injections are not warranted, and the only spec
ment available is that with vaccines.

When the intraspinal injections of serum are followed by di
ance of the organism from the cerebrospinal fluid and by a pe
fall in the temperature to the normal, the persistence of othe
toms means that the patient is suffering, not from the resul
active infection, but from the results of the organic damage left
by the foregoing infection. In other words, the serum has su
in stamping out the disease, but the damage to the cerebral
is sufficient to cause a continuance of symptoms. In such ca
symptoms may be due to an increased formation of cerebr
fluid, which may persist for a considerable time. The proper
ment of this type of case is frequent lumbar puncture, made
the idea of withdrawing the excess of cerebrospinal fluid, and
relieving the pressure. All specimens of cerebrospinal fluid s
be carefully examined for the presence of diplococci, and if a t
time organisms should reappear, intraspinal injections of serum sh
be begun at once, and continued until the disappearance o
organism, or until the physician is convinced that the chronic
has supervened.

The most resistant type of case is that in which serum the
produces no appreciable effect either upon the symptoms or
the organisms in the cerebrospinal fluid. The question arises whe
we should continue to give the serum indefinitely in such cases
believe that if after four to six successive daily injections no e
whatever is observed, there is little good to be expected from co
tinued serum therapy. This type is seen most often in sporadic
cases. The probable cause of the condition is that these cases are
produced by a resistant or "fast" type of bacillus which is not to
be influenced by serum therapy. These cases should be treated as
described for the chronic stage.

TREATMENT OF THE CHRONIC STAGE.—The chronic stage of epi-
demic meningitis is seen in cases in which the disease has existed for
a considerable time before serum therapy was instituted, and also
in cases which, for one of the reasons given above, are resistant to
serum therapy. In this chronic condition the patients continue to
have symptoms, and the predominating symptoms are those of intra-
cranial pressure. The pupils are usually dilated, headache is often
severe, and clouding of the mentality is more or less marked, accord-
ing to the severity of the case. Any of the other symptoms of the
disease may persist. The temperature is at times normal, at other
times irregularly elevated. Lumbar puncture usually yields an
excessive quantity of cerebrospinal fluid. The fluid during the
course of such a chronic case is usually at times, clear, at other times,

able number of cells. While the polynuclear
te, there is a greater proportion of mono-
the acute stage. The number of meningo-
also varies from time to time; some specimens
of organisms, others contain either no organ-

principal pathological lesion is hydrocephalus,
present in this stage of the disease. Three
in the treatment of these chronic cases: (1)
intraspinal serum injections; (3) vaccine

ıt of fluid obtained by lumbar puncture con-
y excessive, the puncture should be repeated
ich the amount of fluid is not continually
should be performed at less frequent inter-
ng guided by the symptoms shown by the
puncture whenever they tend to grow worse.
t serum at each lumbar puncture in this stage
ı therapy produces its effect comparatively
tant cases more benefit is to be expected from
from the use of serum. It is true that there
g as meningococci continue to be present in
some benefit is to be expected from the use
s I believe that in the first enthusiasm over
equent injections were made in this chronic
ough attention was paid to the treatment of
the relief of pressure. It is wise to give an
serum so long as organisms are present, but
he injections should be much longer than in
isease, and at least two out of three punctures
solely for the purpose of relieving pressure
of serum. In my opinion, frequent lumbar
important measure in the treatment of the
nic cerebrospinal meningitis.

the disease vaccine therapy gives little pros-
arison with serum therapy. When, however,
disease has developed, I believe that vaccine
d as a routine. I have seen a number of cases
ry resistant to serum therapy but in which
llowed by recovery. An autogenous vaccine
s taken from the cerebrospinal fluid of the
ıd should be used in all cases, if the physician
of preparing the vaccine. When autogenous
, a stock meningococcus vaccine may be tried.
50,000,000, and the doses should be gradually

along the spine
this procedure.

Of the sympt
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meningitis.

Spasm or rigi
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In many cases,
of the disease. T
irregular pulse, or
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As to alcohol, the
it any stimulant v
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The earliest clini
1840, who called i
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ıe, there is no evidence pointing toward any value in
ɜ.
ptoms which call for special treatment, the most promi-
ain, which when severe requires morphin, sometimes in
doses. Other nervous symptoms, such as delirium or
are treated with bromides, trional, sulphonal, or chloral.
ɜ drugs, I should place the bromides in the first rank in

rigidity of the limbs is the second symptom of impor-
ɾing treatment. The bromides are decidedly the best
ɔmbatting this symptom, and should be given in doses
large to produce a visible result. If the bromide is vom-
ot well taken by mouth, or disturbs digestion, it may be
ɛctum. Convulsions also are best met by the administra-
ɔmide, preferably by the rectum. If the convulsions are
severe, and repeated, chloral should be given by rectum
n to bromide. At times prolonged severe convulsions
ıalations of ether.
y cases, stimulants are required at some time in the course
ɜease. They are indicated when there is weak, rapid, or
pulse, or stertorous respiration. I believe that caffeine is
drug under these circumstances. At times digitalis or
ɜhus produces good results. Strychnin should not be used,
case of collapse following the administration of the serum.
ɔohol, the weight of modern opinion appears inclined to deny
ɪmulant value. Nevertheless, I believe alcohol is frequently
ble addition to the treatment of cerebrospinal meningitis,
ɪrly in prolonged cases, with marked emaciation. Possibly
e lies in its aiding the nutrition.
he residual paralysis, which was formerly a common sequel
ɛmic meningitis, but which is only rarely seen under serum
ɣ, massage, warm baths, and friction should be employed;
ity should be used only when all symptoms of central irrita-
ɪve subsided.

POLIOMYELOENCEPHALITIS

(Infantile Paralysis). (Acute Anterior Poliomyelitis).

s is an acute infectious disease with characteristic lesions in
entral nervous system, due to a filterable virus, and occurring
in epidemics and sporadically.
ɛ earliest clinical studies of the disease were those of Heine in
, who called it "*infantile spinal paralysis*." Knowledge of its
ɪathological anatomy began with Charcot's observations on the
rior horns of the spinal cord, and it was from these lesions that

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TRANSMISSION.
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Neither of these
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Problems and Rese

Whether the thec
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uncommon, and wh

ıan beings and monkeys, the virus is present in the
ı the mesenteric lymph nodes, and in the tonsils
as a strong elective affinity for the tissues of the
›hatic systems, and is highly concentrated in the
; in human beings is not present either in the blood
›inal fluid, but has been found in the blood of ex-
ted monkeys. It has been demonstrated in the
persons who have been in contact with poliomye-
nts, and it is probable that it can exist in a virulent
›iod in such virus carriers. Carriers, are, however,

·The mode of transmission at the present writing,
7) is still unknown. There is probably little doubt
the disease comes from some preëxisting human
ıl obstacle encountered in studying the mode of
fact that the *portal of entry* for the virus has not
The virus can leave the body in the secretions
d nasal cavities, and the presence of the virus in
;ts that the portal of entry may be the lymphatics
his theory has not been proven, although some
monkeys by scarification and inoculation of the
have succeeded.
held as to the mode of transmission in polio-

y is that the disease is spread by *personal con-*
ıgiousness of the disease suggests that the con-
ıdirect than direct. The demonstration of virus
t the means of transmission in indirect contact
ıy person, but in view of the longevity of the
y, transmission by contaminated objects remains

›ry is that the disease is transmitted by means

ıeories has been conclusively proven to be true.
d against each theory will be reviewed under
ch.
/ of personal contact is true or not, it is a fact
›t very contagious. It is very rare that a case
.litis can be directly traced to a preëxisting
›n of cases in epidemics, in which the country
›pulated areas often show a greater prevalence
he densely populated centers, is suggestive of
giousness. More than one case in a family is
ı such instances do occur, both children are

but since 1907 t
were 6,000 cases
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This year, 1916,
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PATHOLOGI
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ve been seen in most civilized countries. There Swedish epidemic of 1911.

disease has, since 1907, been epidemic every of the country. Some of these epidemics have paratively limited localities, or to the country t a certain city and its surroundings. The 16 in New York, which involved the city and ate, is the second visitation since 1907. The n Boston and its vicinity in 1909. Since then l smaller epidemics in other parts of the state. isease is very prevalent in Boston, throughout setts, and in the adjoining states.

ANATOMY.—The virus of poliomyeloencep- a predilection for the tissues of the central t is there that the characteristic lesions of the ght. The primary lesion is a form of lepto- ts in a round cell infiltration, which is most cular lymph spaces of the arteries which enter and spinal cord. This perivascular inflamma- ry lesions, such as edema and hemorrhage, in ssels. The changes which take place in the e secondary to the vascular lesions of the dis- ty is determined by the extent and intensity ement. The changes which occur in the nerve degenerative. The reason that the anterior rd are most involved is, that the arteries which r; the reason the cervical and lumbar enlarge- the most frequent seat of lesions is, that those abundant blood supply. Following the degen- nerve cells occurs a parenchymatous degenera- fibres. Any part of the central nervous system he lesions of poliomyelitis, and the distribution variable. It is true that in the majority of is the part most involved, but exactly the same may be seen in any part of the brain, involving and the white matter. In fatal cases it is usual f the entire cord with its pia mater, of the entire d pons, of the basal ganglia, and often of the The extent of involvement of the tissues of the m is always much greater than would be sup- cal manifestations, and even in comparatively areas are affected. It is quite evident from this term *poliomyelitis* is in no way descriptive of he pathological process, and that the term *polio-*

THE ACUTE
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THE PARALYTI
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of the acute stage
ence, it usually a

—The onset of the disease is sudden and in
nset of any acute infection, such as tonsillitis
ation of the acute stage, that is, the period
nptoms, is from one to seven days. In the
s paralysis appears at some time in the first
te stage really overlaps the paralytic stage,
d to last as long as the fever, which usually
even days.

he acute stage are very variable. In some
ed except the most usual symptoms of an acute
, loss of appetite, and apathy or irritability.
the symptomatology of the acute stage, is seen
h much resembles that of meningitis. There
ons between.

ptoms of the acute stage may be subdivided
Symptoms which are manifestations merely of
out definite connection with the part of the
mptoms of meningeal irritation. 3. Symptoms
f the nerve cells, namely, the paralytic symp-

symptoms in the first group are the following:
sness, *apathy*, and *general muscular weakness*.
nptoms in the second group, those of meningeal
e, *rigidity of the neck*, *delirium*, *stupor*, *convul-*
sphagia, *sluggish pupils*, *general pain*, *pain on*
a, and *cold extremities*. The symptoms of the
lysis and the attendant disturbance of the re-
described under the *paralytic stage*.

ered that no symptom or group of symptoms is
stic of the disease. The symptoms enumerated
sent or absent, and when present may exist in
nation. In the majority of cases, before the
is, the physician is confronted by certain symp-
an acute infection, possibly with meningeal
tion of these symptoms is usually not over a
e more than twenty-four hours. The tempera-
to 106°, and the fever curve is in no way char-

TAGE.—During the acute stage of the disease,
e type to be described below, signs of paralysis
scles appear. The period at which paralysis is
variable; it may appear at any time in the course
m the first to the seventh day. In my experi-
ears within the first three days. It is rather

muscles of the
also that at tim
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the child is una
of respiration a
Paralysis of the

Anterior pol
Left
Female, 9

The paralysis
due to a lesion
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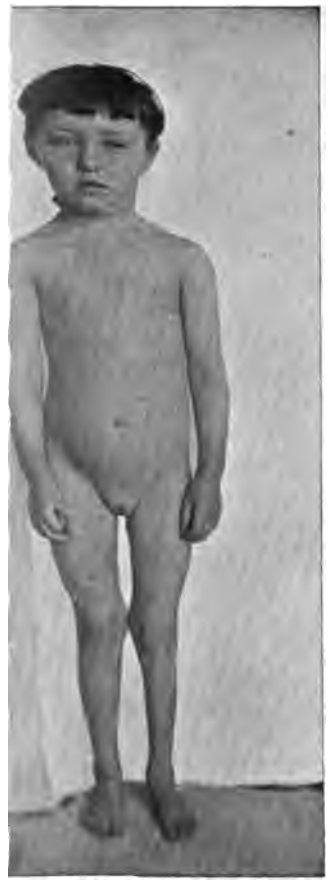
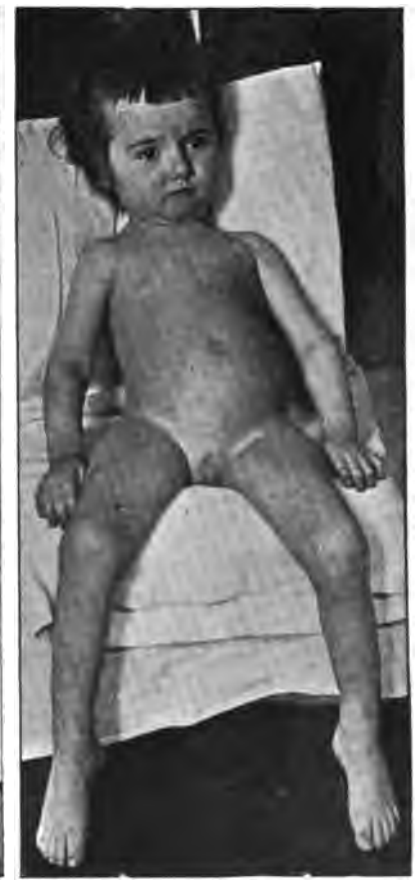
In the great n

ck, abdomen, and back. It must be remembered
there is paralysis in the neck, abdomen. and back,
s of the extremities, and that only single muscles
. When the deep muscles of the neck are affected,
le to hold up its head. When the accessory muscles
re affected the breathing is wholly diaphragmatic.
diaphragm is rare in this type.

FIG. 124



Anterior poliomyelitis
Left leg
Female, 9 years old



Anterior poliomyelitis
Abdominal muscles, left side
Female, 2½ years old

he paralysis in this form of the disease is characteristic of that
to a lesion of the lower motor neuron, and is accompanied by
s of the deep reflexes and flaccidity, and is followed by the reaction
degeneration and muscular atrophy.

In the great majority of cases of this type, the maximum disability

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ıerve
Para-

three oculomotor nerves (third, fourth, and sixth
shown by strabismus, double vision, and some-
he eyelid. The first sign of paralysis of the hypo-
ially difficulty in talking, with a "pudding-mouth
:amination, difficulty in using the tongue is seen.
:ases there is difficulty in swallowing, which may
. that no food can be swallowed. The vagus is
.volved, but I have seen cases in which the first
.asal voice with regurgitation of liquids through the
sis of the muscles of the pharynx, and also cases in
mptom was loss of voice from paralysis of the move-
cal cords.

y exist alone or with a paralysis of the extremities.
cases of this form which I have seen, the paralysis
sively bulbar, or else only paralysis of the deep mus-
: was present in addition. Bulbar paralysis is some-
n extension downward of the process to the diaphrag-
I have never seen a fatal ending in cases characterized
f the facial and oculomotor nerves without paralysis
ossal nerve. When the latter nerve is affected, the
ile often recovered from, is also often fatal. When
t occur in bulbar paralysis, the prognosis as to complete
function is very favorable.

PHALITIC TYPE.—This form is seen when there is ex-
vement of the cerebrum. The clinical picture is very
: in general this type resembles the more acute forms of
meningitis, which are sometimes seen in infants. The
.ally with fever and vomiting, after which there is rest-
lirium, and finally increasing apathy, which may deepen
. Convulsions are not uncommon in this type, and the
eaction is often sluggish. The paralysis is difficult to
he cerebral form of poliomyeloencephalitis. It is due to
the upper motor neuron, and is of the spastic type. Spastic
is difficult to detect on physical examination in infants.
ysis may not be very marked, and the amount of muscular
very variable. The most careful physical examination
e the physician in doubt as to whether the spasm which is
is only symptomatic of meningeal irritation, or whether
.ctual paralysis. Careful observation on the part of a trained
it will often reveal that some movements are not properly
ed. Nevertheless, this type is extremely difficult of diag-
nd is often diagnosed as tuberculous meningitis, the true
of the disease only becoming evident when recovery ensues.
y of the neck and Kernig's sign are usually absent in this
n contradistinction to the meningitic form described below.

Other common sy
tenderness of the
sign, photophobia,
from delirium to
in every way that
opment of a defi
body, is the only
of paralysis is by
in cerebrospinal m
is, that in cerebr
and variable from
the poliomyeloenc
constant. Also p
epidemic form of
forms, depends u
involved in the pr

THE ABORTIVE
tion of the aborti
exist, and in the
of the disease, it
In this type the g
tions, may be p
good deal has be
symptoms, name
recognition of
symptoms, howe
ence is strongly
localizations for
absence does no
Most cases of the
go with any of t
is usually only
The diagnosis, b
lumbar puncture

THE BLOOD.—
the blood in poli
a decrease in th
have reported an
no change. In e
changes in the bl
penia, or at any
with a relative ly
to be much less
of practical value

ns are pain in the back, rigidity of the neck, a tendency to opisthotonos, positive Kernig's gish pupils and a mental condition varying r. The clinical picture presented resembles idemic cerebrospinal meningitis. The devel- aralysis which may involve any part of the o the correct diagnosis, and even the presence ans a positive sign, because it may be present itis. One point which I have found of value l meningitis the paralysis is usually shifting to day or even from hour to hour, whereas itis paralysis, once it has developed, is usually sis is a comparatively rare symptom in the ngitis. The prognosis in this form as in other whether or not the respiratory centers become . The majority of cases of this type recover.

.—It is very difficult to give a clinical descrip- rms of the disease. We know that such forms nce of an epidemic, or of a marked prevalence netimes possible to recognize an abortive case. l symptoms of the disease with all their varia- t without the development of paralysis. A itten about the importance of a triad of early weating, irritability, and hyperesthesia, in the ive cases of poliomyeloencephalitis. These re by no means constant, although their pres- vor of poliomyeloencephalitis when no other fectious process can be demonstrated. Their out an abortive form of infantile paralysis. tive type present a clinical picture which might ognized acute infections, and infantile paralysis cted because of the presence of an epidemic. er, can often be confirmed by the results of

od deal has been written about the changes in eloencephalitis. Some observers have reported cocyte count during the acute stage. Others rease, while still others have reported little or imental poliomyeloencephalitis in monkeys, the appear to be more constant. There is a leuko- , an absence of leucocytosis in the acute stage, ocytosis. The changes in human beings appear tant and are not sufficiently pronounced to be the diagnosis of the disease.

to swallow both sol
diaphragmatic brea
of the child to hold
motion, whether ch
perform all the volu
child to attempt; th
of the limbs; the c
carefully noted, pa

Whenever the ph
is present, the diag
be settled by the re

The question ari
encephalitis can be
paralysis. The ans
a diagnosis based u
be made. This sta
tive types of the di
or when an epidem
sibility of poliomye
or in abortive cases
of lumbar puncture

LABORATORY DI
fluid obtained by
able evidence in
acute stage of the
the characteristic
cally constant. T
is, a fluid without
enables the physi
in the acute stage
and an increase in
brospinal fluid, it
encephalitis or of
two conditions th
features of the cas
demonstrated in th

The fact that th
by lumbar punctu
poliomyeloencepha
lumbar puncture is
be considered, wha
ture in a suspected
thoroughly establis
upon an early dia

ınd fluids; the character of the respiration,
; being particularly significant; the ability
s head; the resistance of the limbs to passive
erized by flaccidity or rigidity; the power to
y movements which it is possible to get the
esence or absence of pain on passive motion
cter of the reflexes. If all these points be
sis when present, will not be overlooked.
ın is in doubt as to whether or not paralysis
; in the acute stage of the disease can only
of lumbar puncture.

whether a clinical diagnosis of poliomyelo-
de before the appearance of a recognizable
to this question is most emphatically that
the history and physical examination cannot
ınt also applies to the diagnosis of the abor-
. When poliomyeloencephalitis is prevalent,
ists, the physician can often suspect the pos-
cephalitis before the appearance of paralysis,
e can only confirm his suspicion by the results

SIS.—The examination of the cerebrospinal
ar puncture gives the most valuable obtain-
liagnosis of poliomyeloencephalitis. In the
ıse, whether or not paralysis has developed,
ırances in the cerebrospinal fluid are practi-
nding of a normal cerebrospinal fluid, that
in formation and with a normal cell count,
definitely to rule out poliomyeloencephalitis
ıe disease. When, however, a fibrinous clot
ıumber of lymphocytes are found in the cere-
ıs that the case is either one of poliomyelo-
ulous meningitis. In deciding between these
sician must be guided by the other clinical
d by whether or not tubercle bacilli can be
id.

ımination of the cerebrospinal fluid obtained
of such great importance in the diagnosis of
does not necessarily mean that diagnostic
cated in every case. The question must now
the indications for diagnostic lumbar punc-
of infantile paralysis? If the disease had a
specific therapy, the value of which depended
s, there would be no doubt that diagnostic

1. *Meningitis.*—
pronounced to be
gitis, diagnostic l
that lumbar punc
of infantile paral
symptoms without
It must be remem
and constant par
latter disease, alt
gitis. The result
between infantile
tuberculous.

The distinction
meningitis and th
often very difficul
the tubercle bacill
be remembered th
the bacillus is not
tuberculous menin
favor of meningiti
be made except f

2. *Other Acute*
same initial symp
nosis depends upo
signs of some oth
Later in the
alitis from the fol

3. *Multiple Ne*
from the polyneu
ing points in poly
the paralyses are
disturbances are
has a peripheral n
is apt to be more
symmetrical.

4. *Acute Myeli*
pyramidal tracts
supplied from the
flaccid paralysis o
the lesion. The
verse myelitis, sen
nounced in the lo

5. *Myatonia Co*
infantile paralysis,

ever meningeal symptoms are sufficiently
ıtible with the possibility of epidemic menin-
puncture is always indicated. This means
hould be performed in the meningitic type
nd also in all cases which have meningeal
alysis characteristic of poliomyeloencephalitis.
however, that the development of a definite
inclines the diagnosis strongly toward the
it is not wholly incompatible with menin-
lumbar puncture will differentiate clearly
ysis and all forms of meningitis except the

reen the more acute types of tuberculous
cephalitic type of poliomyeloencephalitis is
umbar puncture does not help here unless
ound in the cerebrospinal fluid. But it must
th ordinary routine methods of examination
d in more than 10 per cent of the cases of
The complete absence of paralysis is in
n many cases, however, a diagnosis cannot
he outcome of the case.

ions.—Poliomyeloencephalitis may have the
of these conditions, and the differential diag-
development of paralysis or of the essential
ection.

e, we have to distinguish poliomyeloenceph-
g conditions:

—This condition is often to be distinguished
ype of the disease. The principal distinguish-
is are the following: The slower development;
complete at the beginning; objective sensory
y more marked and last longer; the paralysis
opography rather than a radicular topography,
l in the extremities, and is often bilaterally

In this condition the involvement of the
lly gives a spastic paralysis in the muscles
below the site of the lesion, while there is a
muscles supplied from the cord at the site of
differential point, however, is that in trans-
disturbance—particularly anesthesia—is pro-
part of the body.

ta.—I have seen this condition mistaken for
account of the markedly flaccid condition of

bar and encephal
favorable as to c
spinal type. In
sis, contracture,
man states that
of the cases su
series the perce
was 13.5 per ce

Anteri
Flail
Male,

PROPHY
ures which s
the unknow
ease. I beli
of transmiss
should be t

bes, while less favorable as to life, are more
restoration of function than is the common
vere cases of the latter, some degree of paraly-
formity, is left behind permanently. Wick-
e recovery can be counted on in 20 per cent
from paralysis. In Lovett and Sheppard's
f complete recovery in the paralyzed cases

FIG. 125

tis
d

Anterior poliomyelitis
Talipes equinus on right side
Male, 11½ years old

It is difficult to prescribe prophylactic meas-
taken in poliomyeloencephalitis, on account of
connected with the transmission of the dis-
ver, that in a disease with an unknown mode
precaution based on the available evidence
hile transmission by personal contact is not

in which the diseas
great enough to w
As to further n
definite data upo
possibility, not su
min may prevent
it can at least do
acute stage. Sim
entry, hexamethyl
exposed. Weak s
in the nose and t
No method of
has yet become su

TREATMENT.

treatment of pol
treatment of the
eral follows the lin
From the first on
During the acute
that of any acut
period of six we
Attempted move
the ganglion cell
the trouble and t
if too much mov
development of
cles, which in chi
form impossible
pain, tenderness,
should continue
symptoms have

The symptoma
very similar to th
The principal sy
ally bromides ar
vulsions. Stimul
able course. Th
during the acute
no good after th
be avoided not o
during the period
paralysis. Local
tirely useless in a
to the comfort of t

is prevalent, but the danger of infection is hardly
rrant such advice to the community at large.
easures in prophylaxis, there are as yet no very
which conclusions can be based. There is a
ported by definite evidence, that hexamethylena-
the escape of the organism from the body, and
no harm to give this drug to patients during the
nilarly, as the nasopharynx may be a portal of
lenamin may be given to those who have been
solutions of peroxide of hydrogen used as a spray
throat, may have some prophylactic value.
producing an active immunity by vaccine therapy
sufficiently practical to warrant its routine use.

T.—At the present writing, no specific method of
oliomyeloencephalitis is generally available. The
e disease must be wholly symptomatic, and in gen-
lines of any acute infectious disease of similar severity.
onset of symptoms, patients should be kept in bed.
te stage, the management of the diet and nursing is
ute infection. Patients should remain in bed for a
weeks, and should be kept as immobile as possible.
vements of the paralyzed limbs causes stimulation of
ells in the cord, and consequently tends to increase
d to interfere with the process of repair. Furthermore,
ovement be permitted, there is a tendency toward the
of muscular soreness or lameness in the affected mus-
children are constantly contracted in the effort to per-
le movements. If, at the expiration of six weeks, any
ness, or pain on motion is still present, the patients
ue to remain quiet in bed until three weeks after these
ave subsided.
tomatic treatment of the acute stage of the disease is
to that described for epidemic cerebrospinal meningitis.
l symptom which requires treatment is pain. Occasion-
es are required for the relief of muscular spasm or con-
timulation is of no avail in cases which run an unfavor-
. There is no drug which should be used as a routine
acute stage of the disease. Hexamethylenamin will do
fter the appearance of the paralysis. Strychnin should
l not only during the acute stage of the disease, but also
period of six weeks' rest which follows the appearance of
Local applications to the head, neck, or spine, are en-
less in affecting the course of the disease, and do not add
nfort of the patient except in a few cases with severe headache.

has been brought
other portal of en
There is also sor
theory based on
directed at preve
contact, have beer
of the prevalence
The following
the theory that th
contact. In the f
of the diseases kr
through the infect
of poliomyeloence
is present in the
vidual to individua
The second argur
incidence of the d
are more prevalen
in houses, schools,
disease is the st
some epidemics t
the country distri
other epidemics,
of the disease co
immunity of son
on the ground
proportion of th
other means of c
The following
is transferred by
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of this theory is
tremely difficult
which is strongly
dence of all kinc
another rodent o
Against the ins
to be present in
does not precluc
become contamin
Transmission by
proved. The pri
it is as yet entire
At the present
theories may be t

rward to show with certainty that there is any
y.
e epidemiological evidence in favor of the contact
ertain epidemics in which prophylactic measures
ting the distribution of the disease by personal
followed apparently by an appreciable diminution
of the disease and by limitation of its spread.
onsiderations have been brought forward against
e disease is transferred by direct or indirect human
irst place, the nature of the virus differs from that
nown to be spread through personal contact, and
ted secretions of the mouth and nose. The virus
phalitis markedly resembles that of rabies, which
saliva and yet which is not transferred from indi-
al, except through the agency of a punctured wound.
ument against the contact theory, is the summer
disease. Most diseases spread by personal contact
nt in winter, when the population is most congested
ls, and so forth. The lack of contagiousness of the
strongest point against the contagious theory. In
the maximum prevalence of the disease has been in
stricts, where personal contact is least intimate. In
s, the statistics showing the distribution and spread
constitute evidence against the contact theory. The
ome districts after an epidemic cannot be explained
l of immunity acquired through infection of a large
the population, and is an argument in favor of some
f distribution than that by personal contact.
ng points are in favor of the theory that the disease
by some other agency than human contact, possibly by
nsects, or both. The most important evidence in favor
y is the summer incidence of the disease, which is ex-
cult to explain under the human contact theory, but
ongly suggestive of transfer by insects. Statistical evi-
kinds has been brought forward in support of one or
ent or insect, but none of it is finally conclusive.
he insect theory is the fact that the virus does not appear
nt in the blood of human beings. This fact, however,
reclude transmission by means of insects, as they may
ntaminated with infected material from the mouth or nose.
ion by means of the stable fly has never been conclusively
The principal argument against the insect theory is, that
t entirely unsupported by experimental evidence.
present time we can only conclude that either one of these
ay be the true one as to the mode of transmission of polio-

V. DISEASES

This group inclu
organism is a strep
variety of patholo
lesions which are i
ease produced by
tion in the stricte
research, however,
a single type of org
varieties with dif
rheumatic fever an
ial varieties in the
liar manifestations
as specific infectio

(Acute Articular

Rheumatic fe
characterized by
and joints. Th
text books to d
disease as it oc
marily and chie
cations. It is
in whom the n
chiefly articular
having too wide
surgeons has co
articular lesion
are interested ir
the articular le
infectious arthr
disease of the jo
infection of wh
rheumatic fever
infection, the w
but being used
typhoid fever ar
Rheumatic fe
children and in

OF DOUBTFULLY SPECIFIC CAUSE

udes two diseases in which the causative micro-
ptococcus. As the streptococcus can cause a great
ogical processes in various parts of the body, with
in no way specific, it is doubtful whether any dis-
this organism can be considered a *specific* infec-
test sense of the term. The tendency of modern
r, is to regard the streptococcus as representing not
rganism, but a group which includes many *strains* or
differing virulence and pathogenic peculiarities. If
and erysipelas are eventually shown to be due to spec-
he streptococcus group which produce only the pecu-
ons of these two diseases, they will rightly be regarded
tions.

RHEUMATIC FEVER

ar Rheumatism). (Polyarthritis Rheumatica Acuta).
(Inflammatory Rheumatism).

fever is an acute, non-contagious, infectious disease,
by inflammatory non-suppurating lesions in the heart
The term "acute articular rheumatism" used in most
describe the disease is based on the pathology of the
occurs in adults, in whom the manifestations are pri-
hiefly articular, the cardiac lesions appearing as compli-
is a bad term for the disease as it occurs in children,
ne manifestations of the disease are not primarily nor
ular. The term rheumatism is further objectionable in
wide and ill-defined a signification. From the orthopedic
as come the term "infectious arthritis" as describing the
esions of rheumatic fever. The orthopedists, however,
ted in the classification of articular lesions only, and while
lar lesions of this disease belong in the general group of
arthritis, rheumatic fever is not chiefly or exclusively a
the joints, but gives a clinical picture suggestive of a specific
of which arthritis is only one manifestation. The term
c fever is used here to describe such a presumably specific
, the word rheumatic denoting no special theory of etiology,
g used for convenience in the same way as scarlet fever and
fever are used to describe similar infections.
matic fever occurs with approximately equal frequency in
and in adults. It is, however, a disease that differs widely

PORTAL OF EN
dence that the po
is the tonsils. T
rheumatic fever
Furthermore, stre
rheumatic fever
tonsils of rheumat
the organisms are
sillitis is so frequ
be considered on

Transmission f
tically no rôle in
örganism of such
contagion are en
infection. We m
affecting the viru
constitutional res
that the virulence
of the body may
with other bacter
tonsils are an esp
is very suggestiv
search.

PREDISPOSITIO
in the occurrenc
various clinical
is the tendency
vidual. There
heredity as an et

In addition t
is probable that
ing toward infec
possible predisp
and wet, poor
disease occurs at
Children are inf
adults. The dis
and is so extrem
the fifth year t
the years precec
adult life. The
childhood, is un
on local conditio

IMMUNITY.—T

RY AND TRANSMISSION.—There is abundant evi-
tal of entry for the rheumatic fever streptococcus
ne frequency of the association of tonsillitis and
nas long been a recognized clinical observation.
ptococci showing all the peculiar properties of the
organisms, have been frequently isolated from the
tic fever patients. Except in certain rare instances,
not found in other possible portals of entry. Ton-
iently associated with rheumatic fever, that it may
ne of the clinical manifestations of the disease.
from one patient to another appears to play prac-
in rheumatic fever. The streptococcus is a micro-
ch wide distribution, that direct transmission and
entirely unnecessary in explaining the occurrence of
must look to other factors, among which are factors
irulence of the streptococci, and factors affecting the
resistance of the individual. The view of Rosenow
nce of streptococci, and their affinity for various parts
nay be altered by various factors, such as symbiosis
cteria, and variations in oxygen pressure, and that the
especially favorable place for the action of such factors,
stive, and will be discussed under Problems and Re-

ITION.—That there is an important *constitutional factor*
rence of infection with rheumatic fever is suggested by
ical features of the disease. Prominent among these
ncy of the disease to recur frequently in the same indi-
ere is abundant clinical evidence of the importance of
an etiological factor.
ion to a permanent constitutional factor in etiology, it
that there are a number of etiological factors predispos-
infection which act from outside the body. Among such
redisposing causes are damp dwellings, exposure to cold
poor hygienic surroundings, and insufficient food. The
curs at all seasons, but is most common in the spring months.
are infected with rheumatic fever fully as frequently as are
The disease is very uncommon under the age of five years,
extremely rare in infancy as to be almost unknown. After
year the disease is seen with increasing frequency, and in
s preceding puberty it is somewhat more common than in
fe. The explanation of the immunity of infancy and early
od, is unknown. It may be constitutional, or may depend
l conditions in the throat.

UNITY.—There is no evidence of any lasting immunity con-

tained by so many
suggests that the o
deposited by the blo
the coronary arterie

SYMPTOMS. T
matic fever, we find
variably with fever
by pain, tenderness,
an analysis of 223 c
the mode of onset

Fever and a	
Fever and c	
Fever and b	
Fever only	
Fever and s	
Fever and c	
Unknown	

From this it ap
mode of onset is
onset with sympto
mon. By cardiac
ously developing e
but actual sympto
dyspnea, and ort
onset with arthrit
one mode of onset
The number of ca
onset by the occu
vague constitution
with the practical
fever in children n
eases which do not
toms at the onset,
days in doubt, un
often happens, th
infectious disease,
are not illuminati
among his possibi
symptom accompa
In consideration of
in a small proporti
or tonsils is found

investigators. The distribution of the nodules rganisms reach the heart valves, not by being ood flowing through the cardiac chambers, but by es.

THE ONSET.—As to the mode of onset of rheu- d that in the adult the disease begins almost in- r and arthritis, usually polyarthritis, characterized ss, redness, and swelling of the affected joints. In cases of rheumatic fever in children, I found that t was as follows:

TABLE 51

Mode of Onset in Rheumatic Fever

	CASES
nd arthritic symptoms	88
nd cardiac symptoms	82
nd both arthritic and cardiac symptoms	25
only	18
and sore throat	6
and chorea	1
own	3

t appears that, although a little the most common t is with symptoms referable to the joints alone, an mptoms referable to the heart alone is almost as com- rdiac symptoms are meant not the evidence of insidi- ing endocarditis disclosed by the detection of a murmur, mptoms of cardiac weakness, such as precordial pain, d orthopnea. It is also notable that, although the rthritic symptoms alone is commoner than any other onset, yet it existed in only 40 per cent of all the cases. of cases in which the disease manifested itself at the e occurrence of fever only, with or without slight and itutional symptoms, is of special interest in connection ractical question of diagnosis. It shows that rheumatic ldren must be placed in that class of acute infectious dis- do not necessarily show characteristic or localizing symp- e onset, and in which the diagnosis must often be for several ubt, until such symptoms have developed. When, as so pens, the physician is confronted with a beginning acute disease, in which the physical examination and history lluminating, he must not forget to number rheumatic fever is possibilities. The onset with sore throat as the only accompanying the fever in six cases, is also of interest. deration of the fact that children only complain of sore throat all proportion of cases in which inflammation of the pharynx ls is found on physical examination, it is very possible that

the back and limbs,
joints. Such cases a
are almost never gen
The profuse acid swe
tively uncommon in
childhood.

ARTICULAR MANIF
fever in children de
of the disease which
cases, was the compa
dren suffering from a
to as being rheumati
to the Children's Ho
(45 per cent) had j
mildness in compari
are the objective ma
paratively infrequent
often being so little
ness to pressure is
and swelling were pr
ing joint symptoms,
fever in the series.
is also very brief, a

This frequent gre
arthritic symptoms,
fever and no cons
rheumatic fever in
toms, even when co
the parent to "grow
manifestation is a
limb, the disability

As to the number
tion. Only one joi
to one joint more o
series). Usually m
is in general fewer
The ankles are the
second and the wri

In this descriptio
fever I have laid s
children, but I do n
never occurs in child
Cases are seen in w
treme pain on motio

without pain on motion or tenderness in the
are frequently diagnosticated as grippe. There
neral symptoms referable to the nervous system.
eating, so commonly seen in adults, is compara-
n children, especially during the earlier years of

IFESTATIONS.—The joint symptoms of rheumatic
deserve detailed mention. The first peculiarity
ich struck my attention in analyzing a series of
mparative infrequency of joint symptoms in chil-
n an acute infection which other evidences pointed
natic fever. Out of 223 consecutive cases admitted
Hospital suffering from rheumatic fever, only 102
d joint symptoms. A second point is their great
parison with rheumatic fever in adults. Not only
e manifestations of swelling, redness, and heat com-
quent, but the pain on motion is much less severe,
ittle as to cause only a slight limp, and the tender-
e is often but slightly marked, or absent. Redness
re present in only about 50 per cent of the cases hav-
oms, and in only 20 per cent of all the cases of rheumatic
ries. The duration of the joint symptoms in children
ief, averaging a little less than two days.
nt great mildness and extremely short duration of the
toms, combined as they so often are, with very slight
constitutional symptoms, account for the fact that
er in children is so frequently overlooked. The symp-
hen complained of by the child, are often attributed by
"growing pains," and in other cases, in which the only
n is a slight limp in walking or complaint on using a
ability is attributed to a strain.
number of joints affected in children, there is wide varia-
one joint may be affected, and the process is confined
more often in children than in adults (10 per cent of my
sually more than one joint is affected, but the number
al fewer than in adults, a general polyarthritis being rare.
s are the joints most frequently affected, the knees being
d the wrists and hands third.
description of the arthritic manifestations of rheumatic
ave laid stress on the general and typical peculiarities in
but I do not wish to convey the impression that the disease
urs in childhood in the same severe arthritic form as in adults.
e seen in which there is high fever, polyarthritis, with ex-
in on motion, tenderness, redness, and swelling, and in which

there were act
are the ordina
and precordial
and cyanosis in
ical interferenc
to the myocard
which is the mo
The term "brol
symptoms in a
course of an ac
the term implie
of a damaged l
the symptoms i
rather an acute

The valvular
comparatively li
the one most fre
special peculiarit
on to effusion.
which is of imp
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were actual cardiac symptoms. These symptoms themselves the ordinary ones of cardiac weakness:—dyspnea, palpitation precordial pain in the milder cases, with the addition of edema cyanosis in the severer ones. *The symptoms are not due to mechan- interference with the circulation because of the valvular lesion, but e myocarditis which usually accompanies the endocarditis, and h is the most common of the rheumatic cardiac lesions* in early life. term "broken compensation," so often used in describing cardiac otoms in adults, is not applicable to the symptoms seen in the se of an acute attack of rheumatic fever in childhood, because term implies that the cause of the symptoms is the overtaxing damaged but "compensated" heart. In children the cause of symptoms is not such overstraining of a compensated heart, but r an acute infection of the myocardium.

e valvular lesions show no important peculiarities, and are of aratively little clinical significance. The mitral valve is by far ne most frequently affected. Pericarditis in children shows no *l peculiarities* in symptomatology. The majority of cases go effusion. It is not the character of the cardiac symptoms is of importance, but their severity, obstinate duration, and r to life. They are accompanied by fever and evidence of infection, and I believe the frequency of death from heart se in children is not due to the overstraining of chronically ged valves, but to the liability of children to acute rheumatic tion of the heart, causing myocardial weakness.

e cardiac symptoms of rheumatic fever in early life may precede, mpany, or follow arthritic manifestations.

very important peculiarity of rheumatic fever in children lies e occurrence of cases in which the only localization of the infec- is the cardiac—*the primary acute rheumatic endocarditis of child- —cases which run a course characterized by fever and cardiac symp- only.*

CURRENCES.—Finally must be mentioned one of the most im- nt peculiarities of rheumatic fever in childhood, *the liability to ence of the infection with varying manifestations.* It is not nmon for these recurrences completely to dominate childhood. is no special order for the various clinical types to appear, and attack may be mild or severe. At one time the symptoms are y cardiac, at other times mainly articular, at still other times bination of both types. Any attack may be accompanied by rditis or followed, accompanied, or even preceded by chorea. y attack the severity of the cardiac symptoms may lead to a ending.

CASIONAL MANIFESTATIONS.—Next to the lesions of the heart

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es are characterized by the sudden development of tonic spasm the sterno-cleido-mastoid, by marked tenderness and pain on mo-n, and by the fact that the spasm disappears spontaneously within ew days. Other forms of myositis are less common, and usually ct the muscles of the extremities.

Pleurisy, usually dry, characterized by pain on breathing and ghing, and by the appearance of a pleuritic friction rub, is seen times, especially in prolonged cases with cardiac manifestations. itonitis is very rare.

The Subcutaneous Nodules found at times in cases of rheumatic er are undoubtedly a specific manifestation of the disease. Their roscopic anatomy is very similar to that of the submiliary nodules the heart, although they are much larger. They were described t by Barlow and Warner, as "oval, semi-transparent, fibrous lies like boiled sago grains." These nodules vary in size from that a large pin's head to that of a small bean, or larger. They come rops, and may last only a few weeks and then disappear, or may sist longer. They are found most frequently at the back of the ows, at the sides of the ankles, and above the patellae, but may ur on the backs of the feet and hands, along the spine, and in other ts of the skin. They can be seen when the skin is tightly drawn, are more easily detected by palpation.

When present, these subcutaneous nodules are diagnostic of rheu-tic fever. Judging by published reports I should say that they seen much more commonly in England than in this country, probably also more often in some parts of this country than in ers. They are rarely seen around Boston.

There is also an undoubted connection between certain forms of thema, of which *erythema nodosum* is the type, and rheumatic r. It has not, however, been established that rheumatic fever ction is the sole cause of these lesions. Both *erythema multiforme purpura* are frequently associated with rheumatic fever. These ditions, however, are known to be symptomatic of a number of ased conditions, rheumatic fever being among the number, and cannot be considered as specific manifestations of the latter ase.

UMMARY OF THE CLINICAL TYPES OF RHEUMATIC FEVER.—It is cult to divide a disease with such varied and changing symptoms distinct clinical types. The following division is based upon character of the onset and the severity of the case; it must be embered that at any time in an articular type of case cardiac ptoms may develop, and vice versa, and that even the mildest s after recovery usually are left with a permanently damaged t:

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Rheumatic fev

ations: Joint symptoms no matter how slight, even if they be
indefinite pains in damp weather, or growing pains; any cardiac
ptoms; chorea; frequent attacks of tonsillitis; any of the other
ifestations of rheumatic fever. The importance of the previous
ory is due to the tendency of the disease toward recurrences.
physical examination is also of the greatest importance. The
s to be looked for are signs of endocarditis or pericarditis, signs
rthritis, anemia with a history of rapid development, and sub-
neous nodules.

n the other hand, if the patient is under the age of five years
probability of rheumatic fever is greatly lessened, and if under
years, the probabilities are strongly against the disease.

he combination of pains in the joints on motion and fever with
len onset, usually means rheumatic fever, unless some other
e, such as another recognized acute infection, can be found.
ere is redness, swelling, and tenderness to pressure, the diagnosis
rengthened, but these signs are usually absent in the younger
lren. If more than one joint be affected, this also strengthens
diagnosis of rheumatic fever, but it must be remembered that
articular involvement is not uncommon in early life. If there
idence of a cardiac lesion, the diagnosis is almost certain.

he cases which present the clinical picture of fever and cardiac
ptoms are much easier of diagnosis. When in such cases the
sical examination shows signs of a cardiac lesion, and when there
o evidence of any other recognized infectious disease, such as
theria or scarlet fever, the diagnosis is practically certain. Never-
ss such cases are often diagnosed as chronic endocarditis with
ken compensation," especially if there has been a previous
k leaving a chronic valvular lesion. The occurrence of a fresh
k of rheumatic fever is overlooked, because the physician regards
carditis as a complication of arthritis, and associates a fresh
k with fresh articular symptoms. The commonest cause of
ac symptoms in childhood is a fresh infection localized in the
, and when such symptoms are accompanied by fever, this
osis is almost certain.

e type characterized by fever without definite localizing symp-
is the most difficult of diagnosis. The diagnosis is usually
ssible until a murmur develops in the heart, or until arthritic or
ac symptoms appear. I have often seen such cases diagnosed
ippe. It is important to recognize the possibility of rheumatic
in any acute infection with indefinite manifestations. When
is a previous history of any rheumatic fever manifestations,
isease should be strongly suspected even before definite symptoms
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eumatic fever is more frequently overlooked than confounded

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e immediate prognosis becomes more doubtful, and depends upon e severity of the cardiac disturbance. The physician must remem- r that the signs of weakness of the heart are not due to relative er-exertion, but to an acute infection of the heart, and therefore atment with rest and digitalis cannot touch the cause. The ogress of the infection is very difficult to predict. If there is only ecordial pain, palpitation, and moderate dyspnea, the outlook is rly good. If there is severe dyspnea, marked dilatation, edema, signs of pericarditis, the outlook is very doubtful. A great many ildren die in the acute stage of rheumatic fever involving the heart, d the most common cause of death from cardiac failure in child- od is not broken compensation, but rheumatic fever infection. In my series of cases, the immediate mortality of 209 cases in which e rheumatic fever infection affected the heart, was as high as 20 r cent. This of course was hospital mortality, and is perhaps ghtly higher than it would be outside. Nevertheless, when one nembers that the heart is involved in 90 per cent of all cases of eumatic fever in childhood, and that the immediate mortality of ese cases may be as high as 20 per cent, one begins to realize the iousness of the disease in childhood.

ULTIMATE PROGNOSIS.—What is the outlook for a child who sur- ves his first attack of rheumatic fever? During the remaining ars of childhood such a patient is constantly in danger on account the tendency of the disease to recur one or more times. In my ies there was more than one attack in 76 per cent. In each of ese subsequent attacks, the disease may assume a severe type and ove fatal. In a series of 180 cases followed till they were fourteen ars old, 85 died subsequently, a mortality of 47 per cent. The use of death in most of these cases was a fresh acute infection, hough a few died from broken compensation.

After adult life is reached, the outlook is entirely different. The es in my series which entered adult life with damaged hearts were iety-five in number, and of these only seven died in the next ten ars, a mortality of a little over 7 per cent. Here we come to a re favorable side of the picture. Not only is the mortality low in ldren who enter adult life with hearts damaged by rheumatic er, but the disability caused by such damaged hearts is surpris- ly slight. The danger of cardiac disability, attacks of broken mpensation and so forth, in adults who acquired their cardiac on in childhood, is much less than in adults who acquired it adult life. This will be further discussed in the Division on seases of the Heart.

To summarize the prognosis of rheumatic fever, one would say t it is a dangerous disease in childhood, with constant danger m recurrence and cardiac failure. If, however, the child escapes

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at the rheumatic fever streptococci produce acids, and indeed senow has found marked acid production in his cultures. The ring of alkalies is therefore legitimate symptomatic treatment. e acetate or citrate of potassium or the bicarbonate of sodium may used in doses proportioned to the age of the child. The object to make the urine alkaline, and the amount of alkali given must increased if the desired effect is not obtained.

There is no doubt that the *salicylates* have a specific action in ntrolling the symptom of pain in rheumatic fever, whether the pain referred to the joints, to the heart, or to the muscles. The question to whether the salicylates are etiotropic, that is, whether they upon the specific cause of the disease in such a way as to hasten covery, is one about which much difference of opinion exists. Many thorities believe that the salicylates have such action, and many icles are written advocating the superior value of one or another the various forms of salicylate. The evidence on which these nclusions are based is all of the unsatisfactory, purely clinical riety. No other infectious disease caused by a bacterium has been found to be specifically influenced by any drug, and the ecific action of the salicylates in rheumatic fever is contrary to all alogy. Even the supporters of this view qualify their statements admitting that salicylate therapy does not have much action in stening recovery from cardiac involvement. The chief evidences the value of the salicylates in apparently hastening recovery have en obtained in the severe polyarthritis of adults. We must admit at the question is still an open one.

Insofar as children are concerned, the joint manifestations tend disappear rapidly, and the cardiac manifestations to persist obnately. I do not believe that continued use of any form of salilate will hasten the recovery in cardiac cases. Consequently, I lieve that *in children the salicylates should be used mainly in the nptomatic treatment of pain*, although there is no objection to giving all doses for a longer period provided that they do not disturb stomach. My preference is for the *sodium salicylate* as a rou-e, given in doses proportioned to the age of the child as shown in vision II. If salicylate of soda is ineffective or disturbs the stom-, *aspirin* should be substituted. Other substitutes to be tried in ublesome cases are oil of wintergreen, salicin, salol, and salecetin.

The articular symptoms of rheumatic fever in childhood rarely quire further treatment than the salicylates. In very severe cases embling the adult type, protection of the limb from the weight the bedclothes, and hot fomentations to the joints, should be emyed as in adults.

The treatment of the cardiac symptoms of rheumatic fever will more fully described under diseases of the heart. The essential

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Nevertheless the diplococcus rheumaticus was not generally ac-
pted as the specific cause of rheumatic fever, particularly in this
ntry, where the observers who obtained negative cultures were
the majority. It was a known fact that many varieties of infec-
n are attended by articular and cardiac complications, and this
s used as an argument against the specific etiology of rheumatic
er. It was shown experimentally that streptococci in general
ve a certain affinity for the joints, and that both arthritis and
docarditis could be produced with strains of streptococci obtained
m sources other than rheumatic fever lesions, and this was advanced
favor of the second view outlined above, that rheumatic fever is
specific tissue reaction against various forms of infection, strepto-
ccus infection in particular. Nevertheless this work really favored
e specific etiology of rheumatic fever, because the lesions produced
various forms of streptococci were not constant in character, and
fered markedly from the lesions produced by organisms isolated
m rheumatic fever patients, and those obtained with the diplo-
ccus rheumaticus. The differences correspond quite closely to
e differences observed clinically in rheumatic fever on the one
nd, and streptococcus arthritis on the other.

The principal objection, beside the negative findings of many skilled
servers, to the acceptance of a specific organism as the cause of
eumatic fever, was the fact that there were at first no important
rphological or cultural characteristics which served to distinguish
e "diplococcus rheumaticus" from other varieties of streptococcy
ntinued study of the streptococcus group, however, has recentli.
en attended by marked progress in separating the various strains
streptococci one from another.

The work of Rosenow is the most important contribution to the
bject of streptococcus infection in general, and rheumatic fever
particular, which has yet appeared. He found that changes in
ygen pressure exercised an important influence on the growth of
croörganisms of the streptococcus group. On the basis of grada-
ns of oxygen pressure he devised a technique by which he got
re cultures from the joint fluid in sixteen out of nineteen non-fatal
ses of rheumatic fever, and from the blood in five out of eight cases.
The strains of streptococci obtained in this way all differed in one
spect or another from ordinary strains of pus-producing strepto-
cci, and from the *streptococcus viridans*. When injected into
imals they did not produce pus, and exhibited an affinity for the
docardium, pericardium, and synovial membranes. Rosenow
rther observed a tendency of these various strains of streptococci
localize in experimental animals at sites corresponding roughly to
sites of the lesions in human cases whence they were obtained. Thus
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e may eventually prove amenable to vaccine treatment. Still ater is the possibility of the successful use of vaccines in preventing dangerous recurrences of the disease.

ERYSIPELAS

Erysipelas is an acute infectious disease, characterized by an ammation of the skin, spreading through the lymphatics.

ETIOLOGY. THE MICROÖRGANISM.—The cause of erysipelas is *streptococcus vulgaris hemolyticus* (common *streptococcus pyo-es*). This organism is the most common type of pus-producing ptococcus, and is found in most cases of general streptococcus ticemia, in many localized purulent inflammations, and in certain er localized inflammatory lesions. There is no known means of inguishing the streptococcus which produces erysipelas from the ptococci associated with these various pathological processes. nsequently erysipelas cannot be considered a specific infectious ease, and under a strict classification should be described under the eases of the skin. Nevertheless the pathological anatomy and ical manifestations of erysipelas are so peculiarly characteristic, so much like those of a specific infection with general constitu-nal symptoms, that the disease can be most conveniently and kingly described in connection with the specific infections. Also, most recent research has raised the question whether the strepto-cus group of organisms cannot be further subdivided upon a basis acquired selective affinity for certain tissues and organs. It is sible that erysipelas may be due to a strain of *streptococcus hemo-cus* which has acquired properties which cause it to produce the uliar lesions of the disease. For these reasons I have felt justified leparting from a strict basis of classification in order to describe sipelas in this Division.

MODE OF INFECTION.—The streptococcus enters through some on of the skin or mucous membrane. In the newborn the usual tal of entry is the umbilicus. In later infancy the portal of entry sually some small lesion of the skin, particularly on the face, such night be produced by a scratch, insect bite, a patch of eczema, excoriation or fissure about the nose, lips, or ears. In older chil-n the most common portal of entry is the mucous membrane of nose, which has become excoriated from rhinitis or trauma. At ages erysipelas may follow the infection of a wound or an ulcer. merly infection through vaccination or circumcision was fairly mon, but with modern precautions it is extremely rare.

The streptococcus is an organism of such wide distribution, that ct transmission from one case to another plays practically no t. Formerly, before the days of asepsis, when conditions of un-

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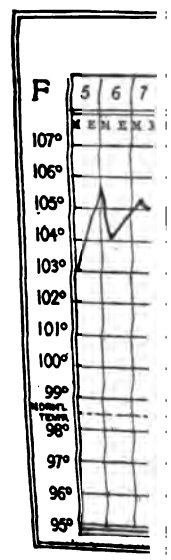

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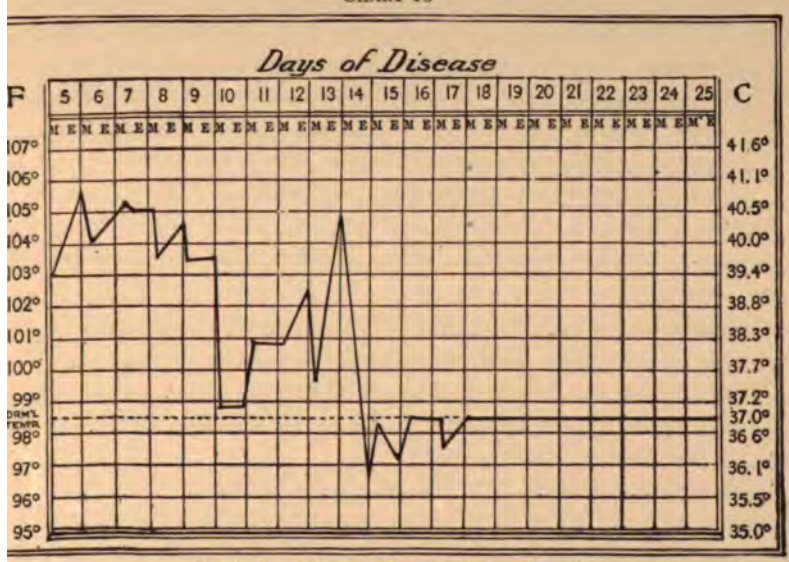
rysipelas has been found in these organs, but only such changes nay occur from a continued septic infection.

YMPTOMS.—The incubation period varies from a few hours even days. The onset is usually sudden, with fever, and marked stitutional disturbance.

RYSIPELAS OF OLDER INFANTS AND CHILDREN.—Except in the ier months of infancy, the clinical course of erysipelas is very ilar to that of adults, but in general milder and more favorable. initial chill is usually absent, and constitutional disturbance is marked, although vomiting is usually present, and occasionally rium. The temperature is high at the onset, is usually continuous slightly remittent for from four to eight days, and then falls by is or rather rapid lysis.

CHART 18



Erysipelas of legs. Female, 6 months old

The skin shows a sharply bounded area of redness on the face, nk, or extremities. This area is swollen, hot, often tender, and surface is tense and often glazed. Vesicle formation is common. facial erysipelas the features are characteristically altered by the elling. The spread of the disease is rapid, and the boundaries ntinue to be sharply defined. The swelling and redness usually appear, and desquamation begins, in the area first affected, while disease is still spreading at the margin. The adjacent lymph les are usually swollen and tender.

The spread of the disease may cease abruptly at any time, with all of temperature. Occasionally the process may progress further,

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iculty in differential diagnosis. Neither condition shows the rp raised boundaries characteristic of erysipelas. There are, vever, intermediate types between erysipelas and lymphangitis, ecially on the scalp.

PROGNOSIS.—The prognosis depends upon the age and general dition of the patient. In strong, well nourished infants, and in er children, the prognosis is good, in general better than in adults. ere cases with high fever, intense inflammation of the skin, great lessness, delirium, and somnolence, leading to death, are rare. n the newborn, in very young infants, and in weak, poorly nour- d infants, the prognosis is very grave. In the newborn the dis- e is almost invariably fatal.

PROPHYLAXIS.—Cleanliness, and the aseptic treatment of nds and ulcers, are the chief measures of prevention against ery- las. Such measures are particularly important in the care of umbilicus in newborn infants. It is well not to allow children h open wounds or abrasions to come into contact with cases of sipelas.

TREATMENT.—The treatment of erysipelas is in general that ny acute self-limited disease. Severe cases with circulatory de- ssion require appropriate stimulation. None of the drugs formerly ocated for internal use has given evidence of any real value.

For local application, almost every remedy in the pharmacopeia been advocated. The disease is so extremely erratic in its course, t no convincing evidence can be obtained as to the value of any ticular remedy. The favorite preparation is *ichthyol ointment* ten to twenty-five per cent strength. This is spread on muslin, l covered with gutta-percha tissue to prevent drying. A fresh plication is made daily.

n severe cases, vaccine therapy may be tried. There is no posi- e evidence of its value, but it can do no harm. An autogenous cine, made from a culture taken from the lesions, should be used enever there is anyone within reach who is master of the technique. herwise, a stock streptococcus vaccine must be used.

PROBLEMS AND RESEARCH.—The chief problem in erysipelas to do with the possibility of a specific therapy. This must end upon the continued study of the streptococcus group of micro- anisms. The difficulties attending this work have already been mmarized under Rheumatic Fever.

The serum treatment of streptococcus infections has certainly proved successful. The weight of evidence is against its value. s is probably due to the multiplicity of strains which compose streptococcus group, and to the probability that a serum is only ctive against its own particular strain.

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CHRONIC INFECTIOUS DISEASES

TUBERCULOSIS

ɔsis is an infectious disease, caused by the *bacillus tubercu-*
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ɔus infection may occur at any age. Owing to the fact
hology and clinical manifestations in early life are widely
m those of adult life, tuberculosis was formerly believed
ı infancy. This has been shown to be untrue, and it has
ecognized that tuberculosis is the most important chronic
nfancy and childhood.

s statistics have been collected in recent years tending
frequency of tuberculosis in the various years of infancy
ɔd. These tabulations show a wide variation according
ity in which they were collected, the European figures
ly higher than those from this country. Moreover, such
e no really exact information as to the frequency of tuber-
fancy and childhood. Some tabulations are based on the
leaths from tuberculosis per 10,000 of the living. These
not, however, an accurate measure of the frequency of
in early life, but are rather a measure of its tendency
tal ending at the different ages. It is well known that
especially liable to severe acute forms of tuberculosis,
ddle childhood various subacute and chronic secondary
do not tend to end fatally are particularly common.
of deaths from tuberculosis per 10,000 of the living is
ıe first year of life, and remains high during the second
the second year it falls rapidly, reaching a minimum in
etween five and ten years of age. After this age it again
ıg in the period of twenty to twenty-five years approxi-
ame figures as those seen in the first year. It continues
a maximum is reached at sixty years.

ulations are based on the percentage of autopsies at
culosis has been found on careful examination. Here
ıtistics are no accurate measure of the general frequency
sis among children, because the tuberculosis may be the
ntributary cause of death, or it may be an accidental
ng no bearing on the cause of death. The figures in
a very low percentage of tuberculosis at autopsy in the

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arly life, on the fact that in the great majority of cases in erculosis appears in adult life, the original infection was in childhood, and on the fact that the normal adult appears paratively immune. This explanation does not seem en-clusive. Postmortem statistics show that infection with s is rare in the first three months of life, but shows a great frequency as the infant begins to come more in contact rroundings, and from this time on, the frequency of tuber-lings at autopsy increases steadily. It seems most prob-he human being in general is predisposed toward the tuber-ction, in the sense that when exposed to virulent bacilli, ntract the disease. Exposure becomes frequent as soon nt comes in contact with his surroundings, and continues quent throughout childhood. Consequently, tuberculosis acquired in childhood, not because children are more sus-ut because exposure to infection usually occurs before s reached. The apparent immunity of adults to fresh infec-e explained on the hypothesis that these adults may have fected in childhood, and having successfully resisted the ve acquired an immunity. The variations in the fatality explained by the hypothesis that resistance, not against ut against the spread of the disease in the body, is developed childhood. For a proper understanding of the phenomena losis in early life, it is essential to distinguish sharply esistance against the occurrence of infection, and the re-hich all human beings develop after the occurrence of through the defensive mechanism possessed by their bodies. r resistance, in my opinion, does not exist, or at most, is rtant factor. The latter resistance is all-important.

RY.—It has been believed from early times that an in-disposition toward tuberculosis is a most important factor. is based on the frequency of tuberculous infection in the tuberculous parents. The most modern views on tuber-ny that any specific susceptibility toward tuberculous can be transmitted by inheritance. The frequency of is in the children of tuberculous parents can be explained atisfactorily on the basis of *exposure to contagion*. There nce that the puny offspring of parents who have suffered rculosis, or other chronic or constitutional diseases, are eptible to the original tuberculous infection. It is how-able that in such children the capacity of developing re-rough their defensive mechanism is impaired.

Y AND CROWDING.—This is an important predisposing he tuberculosis of early life. Wherever human beings live

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ll-born, or die in the early months of infancy. Such infec-
little bearing on the clinical tuberculosis of infancy and
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r INOCULATION.—Tuberculosis can be transmitted by direct
on, as is shown by numerous instances. The disease has
mmunicated by the bite of a tuberculous patient, during the
ircumcision, and during obstetric practice. In comparison
er modes of transmission, infection by direct inoculation is

AMINATED FOOD.—The milk of tuberculous cattle can transmit
osis to the young child. The frequency of bovine infection
ntaminated milk is, as already stated, widely variable. In
n experience, milk infection is rare, but well-authenticated
from some localities suggest that it may be quite common.
pasteurized milk of tuberculous cattle will certainly not
t tuberculosis to every child fed with that milk, and probably
n to the majority. On the other hand, it should be remem-
hat there is always danger of such transmission, and care
always be taken to avoid this possibility, either by pasteuriza-
milk supply, or by using milk of properly inspected cattle.

TAGION—Contagion is by far the most important factor in
nsmission of tuberculosis. Tubercle bacilli are discharged
greatest numbers in the sputum of adults suffering from pul-
tuberculosis. They are discharged in smaller numbers and
h greater dilution, in the urine, feces, and milk, and in the
ge of tuberculous abscesses. The sputum is the chief source
ger. Owing to the peculiar pathology of tuberculosis in early
e tuberculous child is relatively of very slight importance as
ce from which the infection is conveyed to other children.
n is not expelled in early childhood, and few if any bacilli
pelled in coughing. Indeed, the lesions of tuberculosis in early
e of such a character, that the tubercle bacilli are usually kept
oned, and cannot be conveyed to other children. Only such
n as have some discharging tuberculous lesion are really dan-
as sources of infection, and even in them the danger is less
rom the adult consumptive.

as been shown that tubercle bacilli may be conveyed directly
one individual to another, through the medium of the moist
expelled in coughing, or by kissing. Such drops may be ex-
in a fine spray to a distance of eight or ten feet. From this
, and from carelessness in disposing of the expectoration, cloth-
ed-clothes, carpets, hangings, handkerchiefs, and similar articles
e child's surroundings may be contaminated by virulent bacilli
moist state. Sputum may become dried without the bacilli

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han are the mesenteric, and show more frequent evidences
y involvement when both sets are tuberculous. This objec-
een answered on the theory that the tubercle bacilli entering
he intestine, pass through the mesenteric lymphnodes, and
ocalized in the bronchial lymphnodes as a point of least
. This explanation is highly improbable, and is not sup-
any adequate evidence. All the evidence points toward the
equency of infection with bacilli of human origin, taken
alation.

nsil stands third in frequency as a portal of entry, and may
e more frequent than the intestine, especially in later child-
eports show wide differences. Neither Albrecht nor Gohn
single instance of primary tonsillar infection in his large
autopsies. Other observers have reported the frequent
a primary lesion in the tonsil. Whether the existence of
us cervical adenitis always means infection through the
whether the infection is conveyed to the cervical lymph-
m a primary infection in the lung, is still an open question.
able that infection through the tonsil is commoner in child-
n in infancy, and that this explains its rarity at autopsy,
requency with which the tonsils removed by operation are
erculous.

n through the skin, through the mucous membrane of the
nose, and through that of the genito-urinary tract, has
rved, but instances are rare.

PATHOLOGY

RIMARY LESION.—One of the most prolific subjects of
in the tuberculosis of early life is whether the tubercle
auses a lesion at the portal of entry, or whether it passes
he mucous membrane without the formation of a local lesion,
ng a primary lesion in the lymphnodes. The chronic tubercu-
rly life is essentially a lymphnode process, and the char-
pulmonary tuberculosis of adults is rare in childhood.
s fact has come the widely accepted teaching that the prim-
is in the lymphnodes. Von Pirquet, basing his opinion on
rches of Albrecht and Gohn, states definitely that there is
primary lesion at the portal of entry, and that when it is not
primary lesion in the lung has been overlooked. While this
is perhaps not definitely settled, I have become convinced
Pirquet's statement is very close to the truth. In my experi-
careful search be made, a primary lesion may usually be
the portal of entry. It is often so inconspicuous as to sug-
it might easily have been overlooked, and I believe that

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Hary ulcerations of the intestinal mucosa which are caused allowing of tuberculous sputum, and is recognized as being because either the mesenteric lymphnodes only are tuber- they show a more advanced process than the lymphnodes est. Also, in cases with primary tuberculosis of the intes- haracteristic primary lesion is found in the lung.

tonsil, the primary lesion consists in conglomerate tubercle , and is usually not recognizable macroscopically.

FIG. 129

Section through the primary lesion of tuberculosis in the lungs is to be noted that the alveolar outlines are completely obliterated

ROUTES OF INVASION.—The tubercle bacilli tend to om the primary lesion, and to invade the body of the host. e various routes by which this invasion takes place. The are the commonest: 1. By the lymph channels; 2. By tension; 3. By the air passages; 4. By the digestive canal; e blood.

rst extension of the tuberculous infection is probably one nost invariably occurs in infants and children, namely, to hnodes by way of the lymph channels. While there has ie evidence afforded by animal experiments that the bacilli s through one set of lymphnodes to locate in another, this

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oung children do not expectorate, but swallow this sputum.
ible that in some cases tubercle bacilli from this source
tonsils or other lymphoid tissue of the pharynx, and are
by the lymphatics to the *cervical lymphnodes*, where they
rocess similar to that seen in the bronchial lymphnodes.
known in what proportion of cases tuberculous cervical
presents a secondary, and in what proportion a primary
hrough the tonsil. The bacilli in the swallowed sputum
to the intestinal canal, where they may cause tuberculous
of the mucous membrane. These secondary ulcers are
ultiple, and in appearance are indistinguishable from the
ntestinal lesion. From this *tuberculous iliocolitis*, whether
or primary (bovine), the bacilli again following the lymph-
are carried to the mesenteric lymphnodes where they cause
chronic lesions. Further spreading may occur by direct ex-
the peritoneum, in which case *tuberculous peritonitis* is seen.
the bacilli may enter the blood stream in large numbers.
rticularly liable to occur in infancy, and occurs less and
as children grow older. When it occurs, the bacilli are
seminated throughout the body, and give rise to a more
neral *acute miliary tuberculosis*, which is a severe and fatal
icularly associated with early life. The miliary tubercles
und in the lungs, spleen, and liver, and at times in various
ns. The tubercles are often carried to the brain, causing
us meningitis, or *acute miliary tuberculosis of the meninges*,
very apt to determine the character of the clinical mani-

lren grow older, there is less tendency toward escape into
stream of a large number of tubercle bacilli, but occasionally
y escape in small numbers, or possibly singly. Probably
illi thus escaping do not find an environment favorable
eir development, and are destroyed. At times, however,
in some remote part of the body, where they can develop,
the reaction of the tissue again gives rise to chronic tuber-
ions. Examples of such extension are the tuberculosis of
and joints, the solitary tubercles sometimes seen in the
rain, and chronic tuberculosis of the genito-urinary organs.
grams show the routes of invasion of the tubercle bacillus
od.

lerstand the phenomena of tuberculosis in childhood, it is
look at the condition from the point of view of the parasite
from that of the host. It is the law of life for the parasite
ust enter the body of the host, and must also escape from
in order to infect another individual. The tubercle bacillus
ild encounters comparatively little resistance at the portal

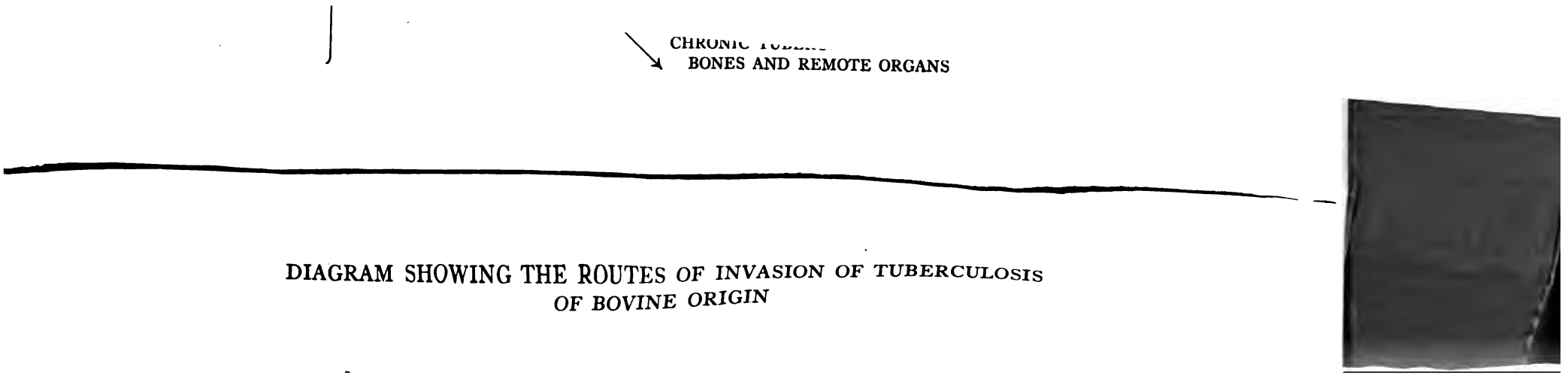
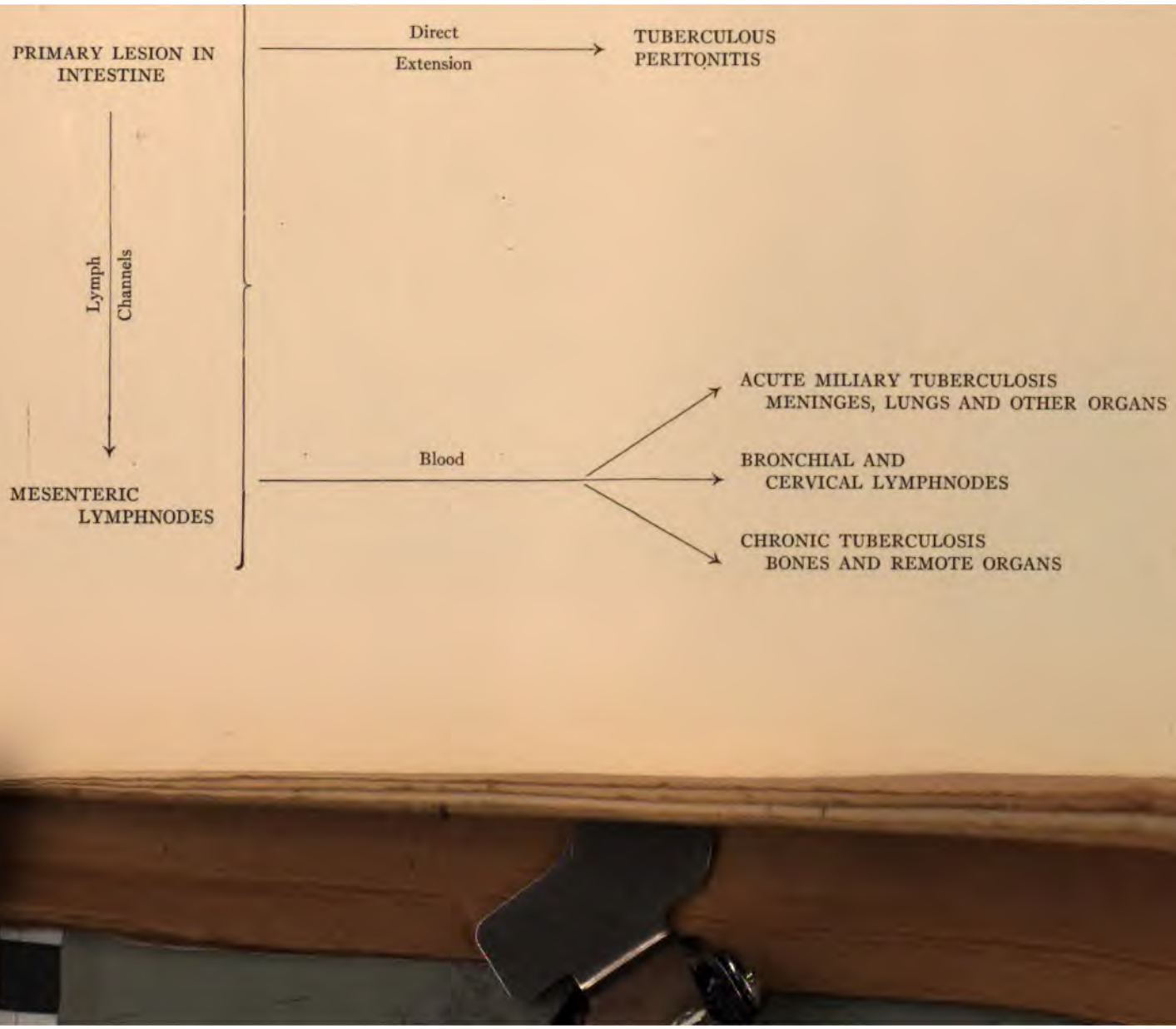


DIAGRAM SHOWING THE ROUTES OF INVASION OF TUBERCULOSIS
OF BOVINE ORIGIN


PRIMARY LESION IN INTESTINE
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Lymph
Channels
ACUTE MILIARY TUBERCULOSIS
MENINGES, LUNGS AND OTHER ORGANS
Blood
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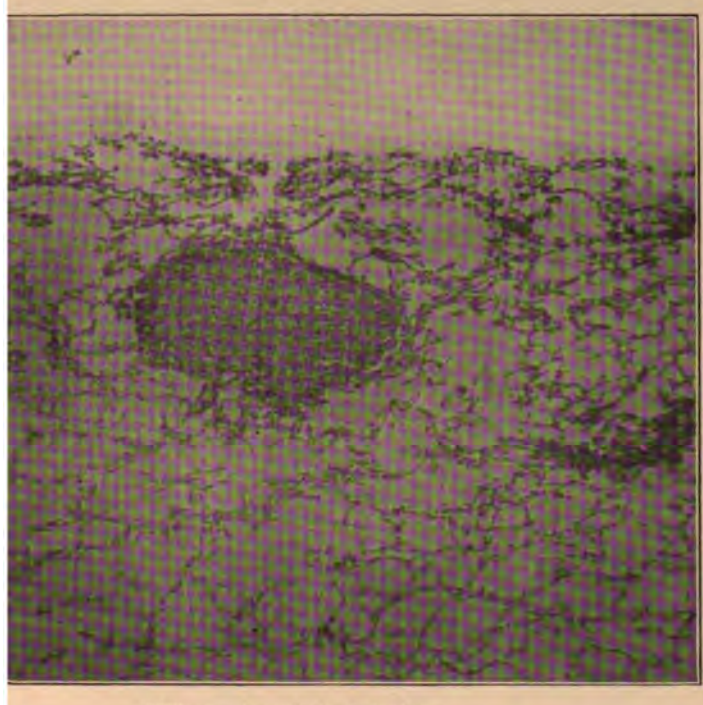
stion as to whether the chronic pulmonary tuberculosis epresents a fresh aerogenous infection, or a lighting up fection acquired in childhood, is still a subject of discussion. ty of European observers (Behring, Hamburger, Römer, t), and many in this country believe the latter theory to one. Hamburger divides tuberculosis into three stages, presented by the primary lesion and the associated pro- lymphnodes, the second represented by all the secondary luced by invasion, and the third represented by the chronic lesions, such as the pulmonary tuberculosis of adult life, onic tuberculosis of the bones and joints.

FIG. 131



Miliary tuberculosis of the lung
sion representing extension of the infection through the blood. It is at the alveoli surrounding the tubercle are free from exudate.

MY OF THE LESIONS. THE LYMPHNODES.—The lymph- ally involved when tuberculous infection occurs through are usually spoken of as bronchial, but include three dis- os. The first surrounds the trachea, the second is situated rcation of the trachea and surrounds the primary bronchi, hird follows the course of the bronchi into the lung. These es are enlarged to a varying degree. Sometimes all three

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ratively rare secondary lesions. The pneumogastric and laryngeal nerves may be involved in the inflammatory The trachea, bronchi, and esophagus may be compressed l by ulceration. There may be ulceration into the air or a mediastinal or retropharyngeal abscess may be formed.

UNG.—The lung is the organ most frequently involved in culosis of infancy and childhood. Four distinct lesions in the lungs, the first being the primary lesion already The three secondary lesions are,—1. Miliary tubercu- Tuberculous bronchopneumonia, and 3. Phthisis.

FIG. 133

Tuberculous bronchopneumonia
lesion from extension through the air passages beginning necrosis in the rounding alveoli filled with cellular exudate.

liary tuberculosis of the lung, the infection is carried to the sue through the blood stream. The tubercles may be found some part of the lung, or may be of general distribution. e seen both upon the pleural and upon the cut surfaces, and as small, circular areas, varying in size from that of a pin- that of a pin-head or larger, of yellowish, greyish, or "sago appearance. Microscopically these tubercles are clear cut, re is no exudate into the surrounding alveoli, unless there is

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scopic examination shows that these cheesy nodules usually tubercles in the walls of the small bronchi. The alveoli ling the focus become filled with inflammatory exudate, to ne tuberculous caseation extends, thus forming the larger Still larger areas are formed by the conglomeration of sev- rete areas.

FIG. 135

Large tuberculous ulcer of cecum

nic pulmonary tuberculosis, or phthisis, is a rare lesion in od. There is usually much old fibrous tissue formation about ion, which in the center shows areas of caseation, softening, ty formation.

PLEURA.—The commonest lesion seen is a deposit of miliary es upon the visceral pleura. Second in frequency is the ion of dense fibrous adhesions between the lung and the chest The pleura may be markedly thickened with some caseation. ne cases a fresh fibrinous exudate is seen at autopsy. Serous y is comparatively rare in infancy and early childhood. Empy-

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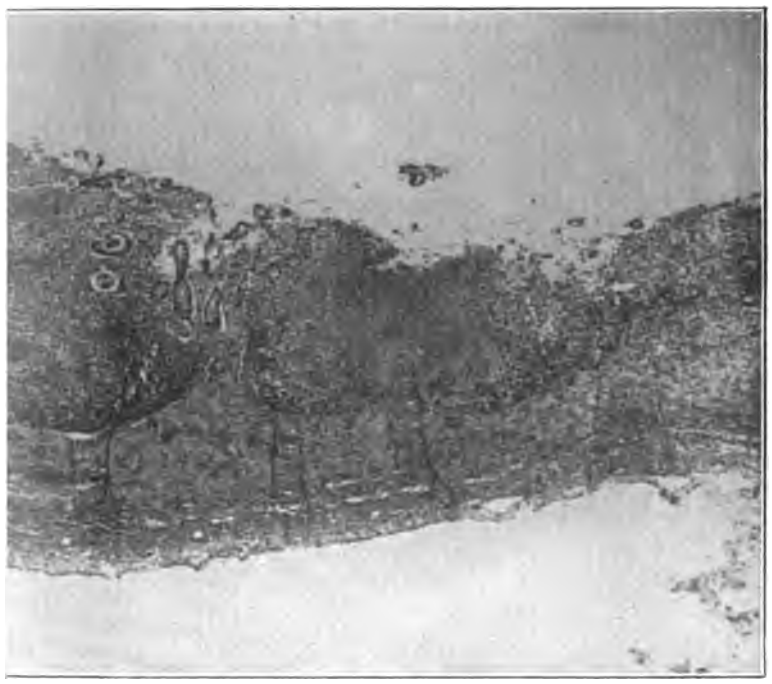
›neumothorax, and pyo-pneumothorax are all very rare lesions tuberculosis of early life.

INTESTINE.—The fully developed lesion in the intestine is a ulous ulceration. In their early stages, small yellowish tuber- ›pear upon the mucous membrane, usually in the region of the s patches. The ulcers are irregular in shape, the long axis at right angles to that of the intestine. They have raised, nined edges, with ragged, greyish bases covered with granula-

FIG. 137



Tuberculous ulceration of the intestine
is ulcer represented the primary lesion in this case, but is histologically indis- ishable from secondary intestinal ulcers originating from swallowed sputum.

. On the peritoneal surface, there is a congestion, and usually llection of small yellowish nodules over the seat of the ulcer. ulcers may involve only the mucosa, or may extend into the er layers. Perforation is comparatively rare, and when it does r, is not into the general peritoneal cavity, as a localized inflam- on forms adhesions. The ulcers may heal by fibrous cicatriza- causing puckering of the intestinal wall, and sometimes narrow- ›f the lumen.

›ere is no anatomical distinction between tuberculous ulcera- of the intestine representing the primary lesion, and that second-

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strewn with discrete or conglomerate tubercles, and shows
of inflammation. There is congestion, some fibrin forma-
a profuse serous exudate, which usually gives rise to a
scites. This form is caused by extension of the infection
ns of the intestine or of the mesenteric lymphnodes.
nmonest form is produced in the same way, by extension,
ch less acute than the ascitic form. There is tuberculous
ion of the peritoneum, in the products of which efforts
take place, by the formation of fibrous tissue. The result
rous adhesions, which bind the intestinal coils one to an-
l to the omentum, viscera, and abdominal walls. There
rous, sero-purulent, or purulent exudate. There is usually
)rous thickening of the omentum. In the fibrous exudate
he intestines, and in the mesentery and omentum, there
seous nodules, or large caseous masses, some of which may
ning, and may even cause abscesses opening externally.

METASTATIC TUBERCULOSIS.—These lesions are caused by
ission through the blood stream of tubercle bacilli, which
nic tuberculosis in various parts of the body. The com-
m of this variety of tuberculosis is the well-known tuber-
ase of the bones and joints. Other examples are the
)ercles sometimes seen in the brain and liver.

MILIARY TUBERCULOSIS.—This is a common termination
)us infection in infancy and early childhood. It is caused
)read general dissemination of the tubercle bacilli through
stream. Numerous small miliary tubercles are found in
ans. The organs most commonly involved are the lungs,
, and spleen. At times miliary tubercles are found in
, adrenals, heart, and other organs.
ns in the brain, which primarily involve the meninges,
ing. In some cases, the surface of the brain is more
kly studded with miliary tubercles, without evidence of
ween the tubercles. This is strictly a miliary tuber-
he meninges, rather than a tuberculous meningitis, al-
two conditions have the same clinical manifestations.
es there is in addition to the miliary tubercles more or
e inflammatory exudate, showing itself either by cloudi-
he course of the vessels, or by the actual formation of
)osits. This is actually a tuberculous meningitis. In
ase, it is probable that each tubercle is formed by bacilli
the blood stream, while in the latter case, there is local
the infection from one area to another.
esses are apt to involve the base of the brain more than

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notable symptoms point toward the abdominal cavity. They described under the diseases of the organs involved.

-joint Tuberculosis.—This form represents extension of the ulous process through the circulation. Bacilli lodging in bony set up a chronic tuberculous process, of which the clinical stations are distinctive. It will be described under diseases bones and joints.

ary Tubercle of the Brain.—By similar extension, chronic ulous processes may be set up in other parts of the body. In cases these solitary tubercles have no distinctive clinical symp- and form only a part of the chronic tuberculosis of early life.

FIG. 139

Tuberculous dactylitis

itary tubercle of the brain, however, gives the clinical manifesta- is of brain tumor, and will be described under diseases of the brain.

hthisis, or Chronic Pulmonary Tuberculosis, corresponds to the d stage of European writers, and is usually recognizable clinically. will be described among the diseases of the lungs.

Tuberculous Bronchopneumonia, is the commonest secondary lesion infancy and early childhood. It is usually clinically recognizable a bronchopneumonia, and will be described under that heading.

Tuberculous Pleurisy, is similarly clinically evident as a pleurisy, d will be described under that heading.

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berculous infection. While such definite symptoms are of the st value in making a diagnosis of tuberculosis in a child, they aid in the recognition of the disease in its early stages.

ERAL SYMPTOMS.—There are three general symptoms associated chronic tuberculosis in infancy. These are *fever*, *emaciation*, *nemia*. Fever is more commonly absent than present, unless ion of the infection has occurred. The temperature can show teristic evening rises, but this is unusual, and fever, when t, is usually irregular, and often the temperature shows but elevation above the normal.

e most common symptoms are those of failing nutrition. The ts may be pale and thin, and do not gain in weight. Changes e composition of their food, made with the object of making gain in weight, are unavailing. They are apt to show a par- r difficulty in the digestion and absorption of fat. Finally, begin to lose steadily, and become still more anemic. They lso often subject to bronchitis. It must always be remembered ever, that chronic tuberculosis is quite compatible with a normal in weight. In my experience, emaciation and anemia are e often absent than present. In more than half the autopsies he Infants' Hospital of infants dying from tuberculosis, the gen- nutrition was good.

he primary lesion does not often give rise to recognizable physi- signs. The principal lesion in this chronic stage of the disease uberculosis of the lymphnodes. The following symptoms are ciated with tuberculosis of the bronchial lymphnodes, but it t be remembered that any or all of them may be absent.

OUGH.—When present, the cough is loud, ringing, and metallic, is sometimes paroxysmal. In some cases it may strongly suggest inning whooping-cough. In whooping-cough, however, the cough nges its character, and becomes associated with characteristic oxysms, with whoop, and with vomiting. The cough of bronchial nd tuberculosis does not change.

EXPIRATORY DYSPNEA.—This symptom, while much more often ent than present, is occasionally quite marked and characteristic. is caused by pressure of the enlarged lymphnodes upon the bronchi d trachea, and is seen most frequently in young infants. The erity of this symptom is very variable. In marked cases the piration seems prolonged and forcible, and is accompanied by a d stridor, while inspiration is hardly perceptible. The rate of spiration is not increased to any extent, and the expiratory stridor ay be scarcely perceptible when the child is quiet, but appears ter crying or coughing.

INTERSCAPULAR DULNESS TO PERCUSSION.—The enlarged bron-

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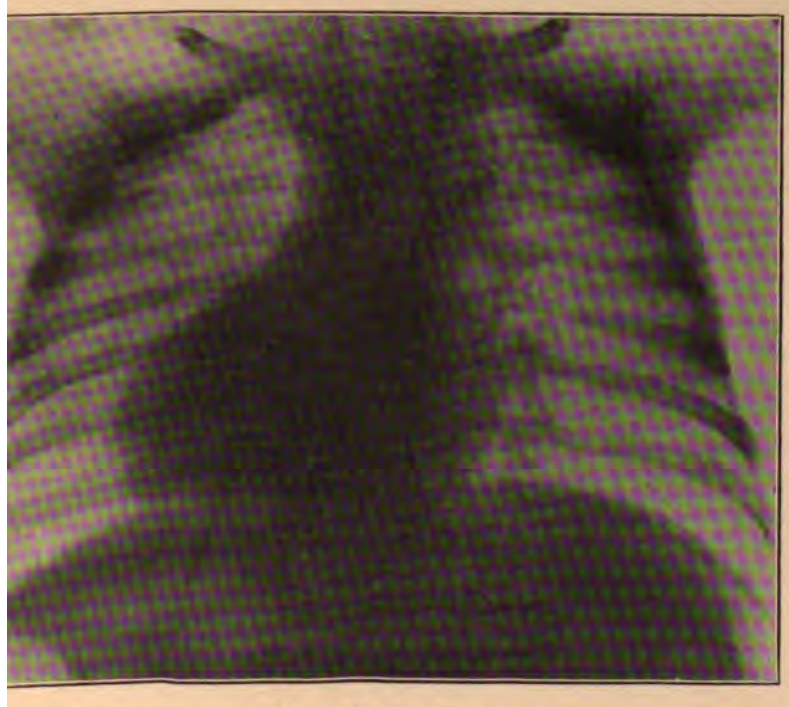
and primary bronchi, this bronchophany is heard over the orsal vertebrae. This boundary as described by d'Espine r older children, and in them any bronchophany over the orsal vertebrae points towards bronchial lymphnode enlarge- In infants under two years of age, the limit of normal bron- y must be placed somewhat lower. In a series of cases at nts' Hospital coming to autopsy, several have been observed n bronchophany extended down as far as the third dorsal ver- which did not show enlargement of the bronchial lymphnodes. other hand, no case in which bronchophany was found below

FIG. 142



Chronic tuberculosis
Showing caseation of the peribronchial lymph nodes

third dorsal vertebra failed to show enlargement, and most tuber- us cases showed distinct bronchophany over the fourth, fifth, sometimes even the sixth spines. In infants, therefore, with nchophany between the seventh cervical and third dorsal spines, sign may be considered doubtful. With bronchophany below third dorsal vertebra, the sign is positive, and points strongly ard enlargement of the bronchial lymphnodes.

The significance of this sign in the diagnosis of chronic tubercu-

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TUBERCULIN REACTION.—In an individual infected with
sis, there are formed certain specific antibodies which are
distributed throughout the body. These antibodies,
ey encounter tubercle bacilli, or a preparation of tubercle
ch as tuberculin, cause the liberation of toxic products as
of the reaction. These products are formed chiefly, first,
te of the introduction of tuberculin; second, in those areas
ere are tuberculous products, or encapsulated bacilli, in
nsequently there is an increased quantity of antibody;
the site of a previous introduction of tuberculin. The irrita-
n these toxic products gives rise to local reactions. Further-
re may be a general reaction, caused by the formation of
ction-products throughout the body, or by the absorption
roducts from tuberculous areas. The local reaction in tuber-
reas, and the general reaction only occur when a sufficient
of tuberculin is introduced into the general circulation.
llowing methods have been employed for eliciting the tuber-
ction:

cutaneous Injection.
ra-cutaneous Injection. (Hamburger's Stich-reaction.)
taneous Inoculation. (von Pirquet's Test.)
taneous Inunction. (Moro's Test.)
njunctival Application. (Calmette, or Wolff-Eisner Test.)

bcutaneous test causes a general reaction. It is carried out
g a subcutaneous injection of a measured dose of Koch's old
n, and taking frequent observations of the temperature.
ve result is shown by a distinct febrile reaction. This test
en is fully as reliable as in older patients, but has very little
on in childhood for the following reasons: If no previous
cal reaction has been made, there is no means of estimating
itiveness of the patient, and if there happens to be present
mount of antibody, the introduction of tuberculin into the
circulation may cause an exceedingly severe reaction, with
er and general toxic symptoms. Furthermore, it can only
oyed in cases without fever. It should never be employed
the previous testing of the local reaction. If the local reac-
positive there is no need to test the general reaction. If all
ctions are negative, under ordinary circumstances there is
ation for further testing. In an exceptional case, such as,
mple, one in which there are clinical evidences of some lesion,
the nature is doubtful, and the local reaction is negative,
eral reaction can be tried.
urger's intra-cutaneous test is carried out by injecting through
eedle a measured quantity of tuberculin into the superficial

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quet test is the one which, from every consideration, is best to routine use in infants and children.

s *inunction test* is carried out by rubbing into the skin a ion of tuberculin made up to fifty per cent strength with us lanolin. A mass of this about the size of a pea is rubbed minute into the skin of the back or abdomen, over an area t five square centimeters. The reaction is positive when a eruption appears over the area in from twelve to forty-eight There is no evidence that this test is any more delicate than n Pirquet reaction. I have used it once, in a patient who d to being scratched with a sharp instrument.

ette's *ophthalmic test* is carried out by introducing into the eye of a one per cent tuberculin solution. It is positive when a ctivitis appears. It is decidedly not to be recommended for children. Severe inflammation of the eye has been known to its use.

Diagnostic Significance of the Tuberculin Reaction.—There is difference of opinion as to the significance of the tuberculin n in infancy and childhood. In general, it is regarded as g its greatest value in early life. It is necessary to distinguish en the significance of a positive reaction, and that of a negaeaction. The tuberculin reaction is undoubtedly specific, and seen in individuals who have never had tuberculosis. A positest is evidence of the existence in the body of specific antibodies st tuberculosis, and therefore that the patient at some time een infected with tuberculosis. The antibody content is greatest e years immediately following infection, or in those following rudescence of activity, or reinfection. It is for this reason that eaction is considered to have its greatest value in childhood. it is probable that considerable time is required for tubercle i to be entirely eradicated from the body, and that in children infected, living bacilli are still present, even if they are imprisand inactive. In cases showing clinical evidence of some n which might be caused either by tuberculosis, or by some r infection, a positive reaction does not necessarily mean that the n is tuberculous. It simply means that there is a tuberculous s somewhere, and increases the probability of any doubtful lesion g tuberculosis.

positive tuberculin reaction means that there is somewhere in child a tuberculous focus, containing living bacilli. Consequently, *he chronic, primary stage of tuberculosis in infancy and childhood, sitive reaction confirms the diagnosis.*

weak or doubtful reaction suggests that at some time the child been infected with tuberculosis, but that the process may be

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against it, except in acute miliary tuberculosis, in some active of clinical tuberculosis, in weakly, poorly nourished infants, measles and possibly in other acute infections.

SPINE'S SIGN AND INTERSCAPULAR DULNESS.—As tuberculosis commonest cause of enlargement of the bronchial lymphnodes ly life, the presence of d'Espine's sign is of great significance gnosis. Interscapular dulness or low spinal dulness generally with a positive d'Espine's sign.

ENTGEN-RAY EXAMINATION.—The Roentgen examination, in xperience, gives the most reliable information as to enlargement e bronchial lymphnodes of any diagnostic method. In many the diagnosis of chronic latent tuberculosis based solely on the y examination was later confirmed at autopsy.

e following table gives the frequency of the three important of latent tuberculosis in a series of twenty-five cases in which rculosis was found at autopsy at the Infants' Hospital:

TABLE 53

Diagnostic Signs of the Chronic Stage of Tuberculosis in Infancy

CASES	D'ESPINE	X-RAY	VON PIRQUET
1	o	o	o
2	?	?	+
3	o	o	+
4	?	?	o
5	+	+	o
6	+	+	o
7	o	?	+
8	?	+	o
9	+	+	+
10	+	+	+
11	?	+	o
12	?	+	+
13	+	+	o
14	+	+	+
15	+	?	o
16	+	+	+
17	o	o	+
18	+	+	o
19	?	?	+
20	+	+	+
21	+	+	+
22	+	+	o
23	?	+	o
24	+	?	+
25	+	+	o
Totals	+ 14 ? 7 o 4	+ 16 ? 6 o 3	+ 13 o 12

In general, the presence of any of these three signs is sufficient warrant a diagnosis of probable chronic tuberculosis. Such a agnosis is probable enough to warrant treating the case as one of ıronic tuberculosis. The presence of either d'Espine's sign or a ɔsitive roentgenogram, makes the diagnosis practically certain.

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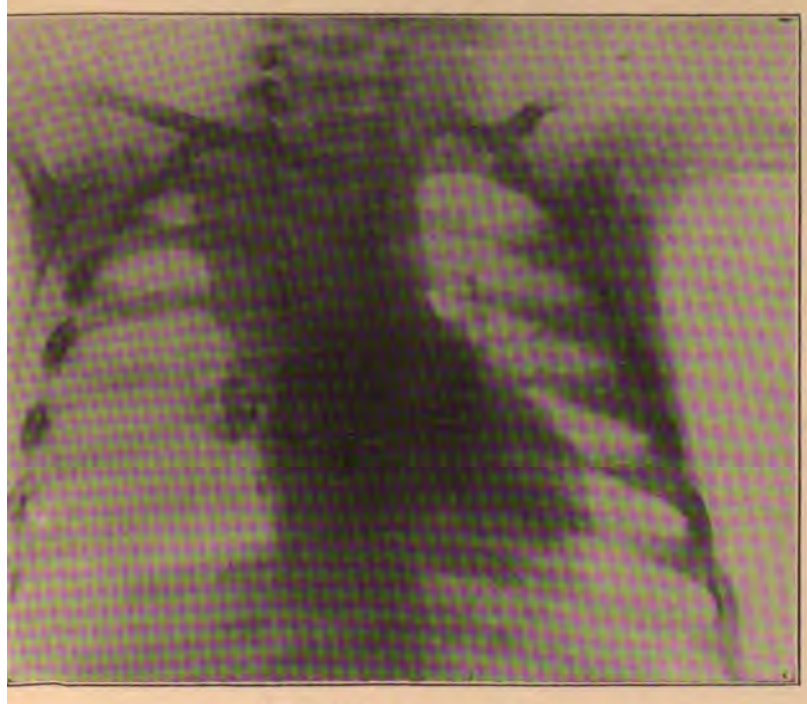
t them, but is not in itself the cause of death. Such children ften die from malnutrition without tuberculosis being present. prognosis depends mainly on the tendency to severe acute lary manifestations, and this tendency depends upon the age patient. The younger the child, the greater the tendency to acute manifestations, such as general miliary tuberculosis, is certainly fatal, or tuberculous bronchopneumonia, which ngerous. As the child grows older, the tendency toward general ry tuberculosis first diminishes, and then the tendency toward rculous bronchopneumonia is lessened. In later childhood there

FIG. 144



Tuberculosis. Primary stage
The plate shows caseation of the mediastinal and right peribronchial lymphnodes

still persists a tendency toward secondary manifestations, but these are not of so fatal a character. Tuberculous bronchopneumonia, if it occurs, is less acute and severe; tuberculous peritonitis, pleurisy, cervical adenitis, and bone-joint tuberculosis, become relatively more common.

In any child in the chronic latent stage, there is a possibility of recovery without the process spreading beyond the lymphnodes. In any child thus infected there is at all times throughout childhood

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children who survive the tuberculosis of childhood, recovery be complete, or phthisis may develop in later life. There are liable statistics on the liability to late chronic pulmonary tuberis.

ROPHYLAXIS.—The chief danger of infection in the child s from the consumptive adult. The ideal measure in prophy- is not to allow the child to live in the same house with anyone wn to be infected with tuberculosis, especially an adult consump- nor to allow it to visit or receive visits from such an individual. h strict isolation of the child is not always practical, especially n the tuberculous patient is a parent. Much can be accomplished insisting that the consumptive parent shall undergo proper sana- um treatment. When it is impracticable to completely separate child from a tuberculous parent, the greatest care should be taken destroy the tubercle bacilli expelled, and to prevent the child from aling the spray thrown out in the coughing. All sputum should carefully collected in proper receptacles and destroyed by germi- les or fire. When the tuberculous mother sees the child, it is tter for her to visit the child's room than for the child to visit her om, and she should try to avoid coughing in the room if possible. ne should never nurse her child, and should avoid kissing it on the outh. She should have her special eating and drinking dishes, hich are boiled after use. Any other tuberculous member of the ousehold, if it cannot be arranged that he or she shall live else- here, should be excluded from all contact with the child. Under no ircumstances should such a person enter the child's room, and it s better for the child not to come to meals, if a consumptive be pres- nt. No wet-nurse, nurse, or other caretaker, should ever be em- loyed, who has ever shown any evidence of tuberculosis. Other children, who have no signs of active tuberculosis, are not a source of danger, but if they have active signs, they should be excluded from contact with healthy children. It is better that children be not allowed to live in a house ever known to have been inhabited by a consumptive. If this cannot be avoided, the whole house should be most carefully cleaned and fumigated with formaldehyde.

While milk infection is rare in comparison with infection by exposure, the danger exists, and is preventable. If possible, only milk of known good conditions of production, from tuberculin tested cows, should be used. Whenever the least doubt exists as to the source of the milk, pasteurization should be employed.

In the case of children known to have been exposed to tuberculous infection, the greatest care should be taken. Parents should be impressed with the fact that care means watchfulness rather than coddling, and that plenty of fresh air is essential in the bringing up

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ool. In many cases it can be arranged to have a governess essons in the open air.

e question usually arises whether a change of climate is advis- in cases of tuberculosis in childhood. It is an open question ner the influence of climate is specific, in the sense that the dis- s more favorably combatted when the patient lives in a certain te, or whether the influence of climate is due to the fact that in climates, or in places where everybody is living outdoors, resh air treatment can be more thoroughly carried out. It is probable that much of the benefit of climatic and sanatorium ment in tuberculosis is due mainly to the thoroughness of the air treatment, and partly to the influence of suggestion. Never- ess, the winter climate of the Atlantic Coast and the Central States, uding the cities of Boston, New York Philadelphia, Cincinnati, Louis and Chicago, is not favorable to the outdoor treatment uberculosis. It is in general cold and damp, and very variable, , snow, extreme cold, or thawing weather, following each other rapid succession. Under such conditions, the carrying out of the en air treatment is difficult, and requires the most constant care d watchfulness. Even the summer climate of the New England ast has such wide variations of temperature, that care is required. r these reasons, a change of climate is advisable, whenever the rcumstances of the family permit. Suitable winter climate may found in Southern California, Texas, New Mexico, Florida, and ne inland parts of South Carolina and Georgia. The Adirondacks nd Colorado have good summer climates.

In spite of the advantages of a suitable climate, home treatment very satisfactory when properly carried out. It is not advisable, nerely for the supposed benefit of climate, to take children from good omes, and put them into hotels and boarding houses. The majority f tuberculous patients must be treated at home, and the treatment an be carried out properly in the homes of the well-to-do and middle lasses. The results of proper home treatment are quite as good as hose of climatic or sanatorium treatment. The advantages of sanatorium treatment are by no means as great in children as in adults. Children need not be sent to a sanatorium except in those cases in which the fresh air treatment cannot be carried out at home, and this condition is only encountered among the poor. Such sana- toria would have to be state institutions. The various tuberculosis clinics which have been organized in the large cities, cooperating with various charities and social service agencies, are becoming most effec- tive in dealing with the problem of tuberculosis among the poor.

DIET.—Careful attention to the diet of tuberculous patients is most important. The successful treatment of tuberculosis is largely

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robably be many years before we have definite evidence. It ver, a specific treatment offering a theoretical prospect of and I believe its employment in children has been too much d. Properly carried out, it can do no harm, and I believe d be more widely tried.

essential feature of the treatment is to begin with very small A dose no larger than one millionth part of a gramme of old lin should be used to begin with ($\frac{1}{1000}$ milligramme), and se gradually increased. The dose may be increased by giving much more as the preceding dose at each injection. The ons should be given twice a week. The tuberculin should be well diluted, and in order to avoid giving too concentrated ns as the dose is increased, the dilution may be kept constant following scheme:

TABLE 55

heme for the Therapeutic Use of Tuberculin, in Increasing Doses and Constant Dilution; Injections Twice a Week

Using 1:1,000,000 Solution of Tuberculin

	QUANTITY OF TUBERCULIN SOLUTION		TOTAL QUANTITY OF FLUID INJECTED
1	1 ccm.	in	10 ccm.
2	1.5 ccm.	in	15 ccm.
3	2 ccm.	in	20 ccm.
4	3 ccm.	in	30 ccm.
5	4.5 ccm.	in	45 ccm.
6	7 ccm.	in	70 ccm.

ne above scheme is then repeated using a 1:100,000 solution of rculin, and is repeated a third time with a 1:10,000 solution. three stock solutions of tuberculin are made up with sterile nal saline solution, and the same is used as the diluent.

f at any time in the course of the treatment, there should be any ile reaction, the same dose is given at the next injection, and ever again appears, tuberculin treatment should be given up. the above scheme is completed, the patient is taking $\frac{7}{10}$ milli-mme at the last dose. The next dose should be 1 milligramme d this, if not followed by fever, should be repeated twice a week for weeks more.

TREATMENT OF ACUTE MANIFESTATIONS.—When a child with berculosis develops fever, it is evidence of increased activity in e chronic lesions, or of extension of the process to form one of the condary manifestations. It is advisable to put the child to bed, at the fresh air treatment should be continued. If after several eeks, there is no other symptom than fever, and no evidence of any econdary manifestation, and if the vigor of the child has not been npaired, it may be allowed out of bed, and a carefully regulated

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When they do occur, the symptoms are also very
is rapidly progressive emaciation. The tempera-
nd fluctuates from 99° or 100° F. to 104° or 105° F.,
The respirations are often accelerated beyond what
by the fever, and physical signs are markedly absent
ne pulse is rapid. Certain cases of this class simulate
y presenting symptoms of apathy, headache, slightly
and tympanites.

.—In infancy the diagnosis can rarely be made.
shows emaciation out of proportion to the symptoms
especially when accompanied by fever otherwise in-
iary tuberculosis may be suspected. The suspicion
l if the d'Espine sign is positive, or if Roentgen Ray
the chest shows evidence of enlarged bronchial lymph-
tuberculin reaction in miliary tuberculosis is almost
ive.

hildren, the diagnosis is less difficult in the cases simu-
id fever. The fever, though often continuous, is less
in typhoid. The spleen is less frequently enlarged, rose
sent, and the disease shows no tendency to improve after
ur weeks. The finding of signs of enlarged bronchial
s, is valuable confirmatory evidence.

OSIS.—No evidence has ever been found at autopsy
evious acute miliary tuberculosis had healed. Therefore
osis must be considered absolutely fatal. In practice, a
nosis in this disease can never be given, on account of the
isting doubt of the accuracy of the clinical diagnosis. The
may mention his suspicion of the presence of miliary tuber-
nd state that it is a fatal disease. If he mentions his suspi-
should also state that it is only a suspicion, and cannot be more
ch, and should encourage hope that his suspicion is wrong.

ATMENT.—If miliary tuberculosis is present, no treatment
of any avail, and should be entirely symptomatic and pal-
The cases in infants resembling infantile atrophy must be
l as gastro-intestinal disorders. In the cases resembling ty-
fever, the treatment should be that of typhoid.

SYPHILIS

philis is a chronic infectious disease, caused by the *treponema*
lum (*spirocheta pallida* of *Schaudinn*).

TIOLOGY. The Microörganism.—The specific organism of
ilis was discovered by Schaudinn in 1905, and was called by him
spirocheta pallida. It has since been shown to belong in the

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d with syphilis is closely connected with the mode
e antibodies. It is the results of the struggle of the
nism of the host against the biological needs of the
n syphilis, as in many other diseases, appear before
picture of a "disease."
course of syphilis is strongly suggestive of such a
long incubation period, the localized primary lesion,
generalized secondary manifestations, the increasing
calization of the lesions with each recurrence of mani-
circumscribed character of the tertiary lesions, the
virus at different stages for different tissues—all these
re suggestive of a contest in which each side has many
varies its method of attack or defence. It is such a
in syphilis, as in tuberculosis, constitutes the picture
infectious disease.

SION.—Infection with the *treponema pallidum* is always
from a person already infected. Such transmission can
before birth, during birth, or after birth. When infection
re or during birth, the disease is described as *congenital*
When infection occurs after birth, the disease is described
syphilis.

before birth is by far the commonest source of syphilis
and childhood. The disease is transmitted always from
ic mother to her offspring; it cannot be transmitted from
r to the ovum by means of the spermatozoa. Belief in
ion from the father to the offspring has formerly been wide-
nd many arguments have been brought forward in support
heory of *syphilis ex patre*. The most important support for
ory is the fact that the mother of a syphilitic infant very
ly shows not the slightest trace of syphilitic manifestations.
nevertheless immune to infection from her offspring, as Colles'
tes. This immunity has been explained in various ways by
who wish to avoid the conclusion that it necessarily means
on of the mother. That immune bodies can pass from the
o the mother in such quantity as to produce a lasting passive
nity, or that antigen can pass from the fetus to the mother
ch quantity as to produce a lasting active immunity, is con-
to all the evidence provided by modern immunological research.
are forced to the conclusion that *in syphilis, immunity means
tion*. Furthermore, the majority of the mothers of syphilitic
nts, even though *clinically* free from the disease, nevertheless
evidence of antibodies in the blood which mean infection (Was-
nann reaction), and in the maternal portion of the placentae of
h mothers spirochetae can frequently be demonstrated. Finally,
view of our knowledge of the effect of early infection before the

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akes place in the first half of pregnancy, the syphi-
e fetus are not very typical. When, however, abor-
n the latter half of pregnancy, the syphilitic lesions
and it is from such cases that one can best learn
e syphilitic virus upon human tissues, when their
nce are at a minimum. These tissue changes are
d typical of syphilis. When, however, infection
g pregnancy, the child is born alive, and the powers
ich are rapidly developed by the tissues continually
racter of the specific tissue reaction. Nevertheless,
on retains its fundamental characteristics, the modi-
the result of a changed local susceptibility and of a
veloped resisting power.
ntal lesions consist in two processes:

llular infiltration.
of development.

lar infiltration is the most characteristic histological
of syphilis. When the infection is acquired early,
nfiltration is most likely to involve the visceral organs,
the spleen, liver, kidneys, lungs, pancreas, thymus, and
growth of cells has its origin in the interstitial tissues
the smallest blood-vessels, and it is in the perivascular
he spirochetae are most numerous. In a later stage
s lead to connective tissue overgrowth with an eventual
contraction.

ition of development characteristic of syphilis is caused
infiltration and hyperplasia of the interstitial connective
re is a corresponding hypoplasia of the parenchyma.
pically the syphilitic tissue changes show themselves
st part only in an increased size of the affected organ,
of the spleen and liver. In some organs, however, the
c appearances are more characteristic, for example, in the
re the over-development of interstitial tissue is accom-
marked fatty degeneration and desquamation of the alveolar
, which can actually fill the alveoli, so that the cut surface
g presents macroscopically a peculiar homogeneous yellow-
appearance—the so-called *white pneumonia*. Another
which the lesion is particularly characteristic is that of the
of the long bones. These changes are almost constant
philitic fetus, still-born infant, or one dying soon after birth.
n is described as the syphilitic osteochondritis or epiphysitis.
pically the epiphyseal line appears to be broader and more
in color than normal. Microscopically the appearances
at calcification, while irregular, is not diminished, but the

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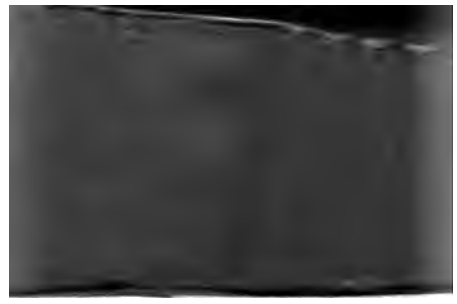
tion of the nasal mucous membrane. The nasal
rrowed or stopped up, and the result is difficult nasal
h manifests itself in a peculiar snuffling sound. The
y be sufficient to interfere with nursing. On exam-
nose all that is seen is a swelling of the mucous mem-
larly in the posterior part of the nares. In the begin-
n is slight or absent, and one never finds the profuse
rge which is characteristic of an ordinary cold. Later
a purulent discharge which may be streaked with blood.
is by no means seen in all syphilitic infants. When it
owever, it is seen either at birth or shortly after birth,
ppears later than the fourth week. It usually persists
eeks and then disappears, but it may be very obstinate,
months. The appearance of snuffles so early in life is
e the attention of the mother, so that a question as to
ance of this symptom forms an important part of the
every case of suspected syphilis. It must be remembered,
hat absolute reliance cannot be placed upon the mother's
this question, and that the snuffles may have been produced
cause other than syphilis. When the snuffles are actually
by the physician, so that he need not depend upon the
t of the mother, they are usually of marked diagnostic
nce. The characteristic deformity of the nose, marked
g of the bridge, which appears at a later period of infancy,
bly to be attributed to an earlier syphilitic rhinitis with its
pon the development of the nasal bone and cartilage.

eruption of vesicles upon the hands and feet is the so-called
ic pemphigus or *pemphigus neonatorum*. The appearance is
rounded vesicles varying in size from that of a small pea to
a cherry upon an inflammatory base. The contents of these
s are at first serous, but later become cloudy and purulent.
calization of the lesions upon the palms of the hands and soles
e feet, and on the plantar surfaces of the fingers and toes, is
y characteristic. The syphilitic pemphigus is always a mani-
tion of an early developing infection. It is not present in the
rity of cases of congenital syphilis, and is less common than
syphilitic rhinitis. When present it usually appears at birth.
s always an early sign, and very rarely appears later than the
rd or fourth week. Occasionally, when an eruption appears late,
icles are seen on other parts of the body than the hands and feet.
e vesicles of syphilitic pemphigus soon become broken, leaving
e denuded corium sharply marked out from the epithelial remains
the margin of the lesions. This early eruption is to be sharply
istinguished from the later cutaneous manifestations. It is always
f very bad prognostic significance.



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estations of congenital syphilis. In general the
divided into two principal groups:

ltration.
ped eruption.

infiltration of the skin is simply the appearance in
he same process of diffuse cellular infiltration which
tological lesion of syphilis in the other organs of the
ly difference is that the skin is involved somewhat
ourse of the disease. Clinically this tissue-change
as a thickening and hardening of the skin. Diffuse
the skin is a special peculiarity of congenital syphilis,
en in the acquired form of the disease.

s these lesions may involve the skin of the whole body.
ever, the lesion is confined to certain particular parts
The regions most commonly affected are the face and
es, particularly the hands and feet.

ce the syphilitic tissue-changes lead to certain clinical
have always been associated with the diagnosis of con-
ilis. In the first place there is a peculiar yellowish
face which is difficult to describe. The lips often lose
ion border, their color merging into the pallor of the
in.

n is particularly marked about the mouth, nose, and
ne process may be so pronounced as to cause the skin
ts elasticity. In such cases, wrinkles and fissures appear
rts where movement of the skin is most active. This is
of the well-known *rhagades*, superficial or deep radiating
the lips, which constitute one of the most important of
ta of congenital syphilis. When the entire face is involved
cellular infiltration it presents a peculiar stiff, mask-like
e.

cases the involved areas of the face may show an inflamma-
ening of the skin. The inflammatory process may go fur-
ducing a *crusted eruption* very similar to impetiginous
Such an eruption is seen most frequently about the nose
th, but may appear on the forehead or scalp. The crusts
lly easily removed and leave scarcely reddened areas which
bleed.

continued infiltration of the skin naturally interferes with
rition of the hair. This is the cause of the *alopecia* which
times seen in congenital syphilis. It may involve the eye-
eyelids, and scalp. The syphilitic alopecia usually occupies
ne front half of the scalp, in marked contrast to the alopecia
ordinary restless or rachitic infant.

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take on a peculiarly characteristic yellowish-brown
which eventually disappears.

st sign of congenital syphilis is the sudden outbreak
-papular syphilide, it is a favorable prognostic sign.
uption suggests that the human organism has acquired
g power against the syphilitic antigen. Indeed, the
he efflorescence, the better is the outlook. Such cases
igns of visceral changes, such as enlargement of the
en. When, on the other hand, the circumscribed exan-
and is preceded by other signs of syphilis, the outlook
orable.

FIG. 146

culae, ulcers and bullae on the soles of the feet. Male, 2½ months old

the circumscribed eruption of congenital syphilis becomes
This is a somewhat different process from the syphilitic
, which was mentioned as one of the early signs of the
It is true that the syphilitic pemphigus may appear for the
later in the course of the disease, but the eruption is always
rom the start, and its presence is always the sign of a severe
When the circumscribed eruption of the later stage be-
stular there is usually a regular progression from macule
, and from papule to pustule.

MUCOUS MEMBRANES.—In the early manifestations of con-
yphilis which occur in the first three or four months of life,
ous membranes are affected very infrequently in comparison
skin. Mucous patches are more characteristic of the re-
hich take place later. At times, however, even in early
mucous patches can be found upon the tongue, lips, or soft
Hoarseness or aphonia due to a similar process in the
is seen at times.

BONES.—Lesions of the bones, on the other hand, are not
non among the early manifestations of congenital syphilis.

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ıusing extreme pain on motion; (2) extension of the infiltra-
ıe muscles, interfering completely with their movement;
tion of the diaphysis from the epiphysis.
›ther parts of the long bones are affected, such as those
knees, shoulders, wrists, or ankles, the chief clinical mani-
beside psuedo-paralysis is diffuse bony thickening near
ɜ. These swellings are often acutely tender. Roentgeno-
the syphilitic osteochondritis give a very characteristic
The line of the epiphysis is broadened, irregular, and in-
Next to the diaphysis there is a lighter zone corresponding
:anulation tissue which is formed in this situation. Finally
ɛ increased areas of shadow about the end of the diaphysis
nding to irregularly formed bony tissue, while the shaft
ows a line of bony thickening along the periosteum.

FIG. 148

Syphilitic dactylitis
Showing the thickening of the periosteum

ıe syphilitic *dactylitis* is another characteristic bony lesion seen
.rly life. This process begins very gradually and does not cause
:r pain or interference with function. The affected phalanx
lly shows a marked olive shaped swelling in the region of the
ıle joint or of the proximal phalanx; occasionally the distal
ıanges are also affected. The thickening is entirely in the peri-
ıum, and the joints and soft parts remain unaffected. This process
ally begins in early infancy and usually involves more than one
ıanx. The appearance closely resembles that of a tuberculous
tylitis, the chief differential point being that the syphilitic process
'er leads to suppuration or fistula formation.

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syphilitic virus. The manifestations now take on a dif-
determined by the newly acquired attacking powers
ite and defensive powers of the host. When syphilitic
ns appear after a period in which the infant seemed to
ee from symptoms, it is called a relapse. These relapses
al syphilis usually occur during infancy or in the first
ildhood, that is, during the first four years of life. Occa-
lapses appear somewhat later, still retaining the mani-
characteristic of infancy. When, however, the renewal
ntest is postponed until about the period of the second
the manifestations are not spoken of as relapses, but as
yphilis of childhood.

relapses of infancy and early childhood the manifestations
sease are not so general in character, but are more sharply
. Single lesions, however, are apt to be of greater extent.
y of eruptions may again appear upon various parts of the
his stage of the disease, more or less resembling the maculo-
sylphide. They are apt, however, to appear suddenly, to
ery short course, and to disappear rapidly. The most char-
ic manifestations of the relapses of congenital syphilis seen
ncy are:

lucous patches on the mucous membranes.
ondylomata on the skin.

Mucous Patches.—These manifestations are seen most often
the second to the fourth year. Ulcers and mucous patches
be found upon the mucous membrane of the lips, cheeks, ton-
and soft palate, and on the sides and under surface of the
ue. They are superficial, and of variable size. Infiltration is
narked, but is present to a greater or less degree. Both ulcers
mucous patches have infiltrated edges, but the plaque in this
ation rises above the level of the mucous membrane, while the
rations are considerably depressed. They are both character-
of syphilis. Their locality is determined somewhat by the
sence of such irritants as sharp teeth pressing against a portion
the tongue. The secretion of all these lesions of the mouth and
s is highly infectious.

2. *The Condylomata* characteristic of this stage are exactly like
ose seen in acquired syphilis. They are reddened, moist, papular
sions of the skin which vary in size from that of a pea to that of
fifty-cent piece. They grow rapidly in all dimensions. The sur-
ace often shows indentations and fissures. The favorite situation
f these lesions is around the anus and genitals.

Gummatous lesions may occasionally appear in connection with
these early relapses, but are more characteristic of the late syphilis

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is in childhood which are quite distinct from gumma
d which are sometimes seen in children in whom no
mma formation is found. These are:

c periostitis of the tibia.
ial keratitis.
son's teeth.

: PERIOSTITIS.—In the syphilitic periostitis of the tibia,
ne is thickened in its entire length, the over-lying skin
ched, shiny, and occasionally slightly reddened. The

FIG. 150

ate manifestations of congenital syphilis in a boy 6 years old, showing periostitis of both radii and both tibiae with an area of softening in the lower third of the right tibia

ling may or may not be painful to palpation. The impression
n is that of a solid spindle-shaped swelling. The edge of the
a may be lost in the swelling, or may be palpable; in the latter
: it is apt to present little roughnesses and indentations.
This inflammatory process in the periosteum may subside or may
come gradually ossified. In the latter case, the result is a perma-
at deformity of the tibia, the so-called sabre leg.

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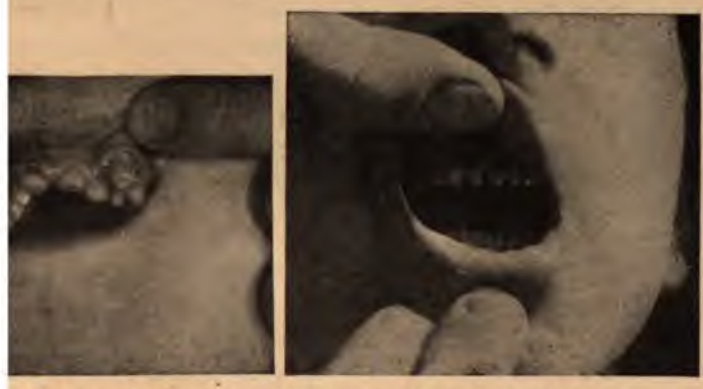
rstitial keratitis occurs most frequently in female sub-
ıost common between the ages of ten and fifteen, al-
occur much earlier, and according to Fournier, may
met with at birth. Complications may arise in the
choroiditis, and retinitis.

s's TEETH.—The first set of teeth in infants with con-
s may show the same markings which are characteristic
dentition, but usually have nothing characteristic about
ıow a lack of nutrition, a condition which may arise
:her morbid processes.

FIG. 152



nifestations of congenital syphilis, showing the typical notched and pegged teeth of the second dentition

nd set of teeth, however, presents certain characteristics.
acteristics are shown especially in the two middle upper
which the cutting edge of the tooth is worn away, leaving
ıurface. The teeth are also apt to be somewhat far apart,
child grows older, to assume a peg shape. The especial
tics of syphilitic teeth were first described by Hutchinson.
ar shape of the teeth is not always present in syphilis,
it appears it is very suggestive of the disease. Fournier
attention to the frequent involvement of the molars. The
may be replaced by yellowish pegs which are free from
These may soon wear down to the surface of the body of
presenting a smooth crown with yellow center and narrow
vhite. Absence of canines and wide separation of the in-
often caused by congenital syphilis.
tion to these three signs of the first importance, there are
her manifestations occasionally seen which are particularly
istic of late syphilis in childhood.

KULL.—In the late form of congenital syphilis the bones

preservation of some of the functions and com-
The rapid diminution of some symptoms and
iers are characteristic. The presence of syphilis
iarked improvement under antisyphilitic treat-
points in the diagnosis. Syphilitic endarteritis
ta of the base have been observed in infants as
ths.

CLINICAL.——The clinical diagnosis of congenital
on the recognition of pathognomonic signs of the
in which such signs are clearly evident, the diag-
lt. Unfortunately all the manifestations of con-
re not pathognomonic, and the signs observed by
any time may be of doubtful significance. Again,
the disease may not be present at the time when the
ler observation, and the history obtained from a child's
er wholly reliable as a means of diagnosis. Neverthe-
ants who are undersized, who lack vitality, or who have
with nutrition, a painstaking search should be made for
stations of congenital syphilis.
ilitic rhinitis with its snuffles actually comes under
n of the physician in the early weeks of life, it can
tinguished from the snuffles of adenoids or a common
y of snuffles is suspicious, but by no means diagnostic.
higus neonatorum is pathognomonic. Enlargement of
the early weeks, while, perhaps, not absolutely diag-
strongly suspicious; when it occurs later, other causes
led, such as rachitis or leukemia.
allor of the face, and the shiny, glistening soles of the
e characteristic of the stage of cellular infiltration of
are strongly suspicious, if not pathognomonic. If in addi-
are present, the diagnosis is certain. When the pro-
e has advanced to the formation of a crusted eruption,
of syphilis is not so certain.
lo-papular syphilide, when typical, is unmistakable.
comes from the fact that there may be very few lesions,
a single spot upon the forehead or upon one of the
When the circumscribed eruption is not actually ob-
the physician learns of it from the history, its diagnostic
ch less, and the history can only be termed suspicious.
circumscribed eruption takes a less typical form, as in
with pustular lesions, the case falls into the suspicious
arious forms of eczema and dermatitis must be con-
ossibilities. The previous history as to syphilitic mani-
of great importance in such cases.

three groups further laboratory investigation should not be made as a routine measure. In the tory investigation, while perhaps not absolutely vertheless be made. It will give positive con- f the correctness of the diagnosis, and no purely ever absolutely certain. If the case is to be y only, the laboratory diagnosis, while not abso- ertainly desirable. But if the case is of such a to call for treatment with salvarsan, laboratory blood is positively indicated. Except in a very mergency, *no case of congenital syphilis should be* son unless the *presence of a positive Wassermann* established.

roup, the testing of the Wassermann reaction is ted. The diagnosis can only be made with certainty of the Wassermann reaction, and certainty of diag- in the treatment of congenital syphilis.

ER MANN REACTION.—The technic of obtaining the Wassermann reaction in infants has been described in At least 1 c.c. of serum is required. Sufficient blood qua ntity of serum can best be obtained from the longi- in nfants. In older children the blood can be obtained e o the ear, or by making a short incision in the heel. ic f the Wassermann reaction or complement-fixation nilis is too complicated for ordinary clinical use. It re- pecia ly equipped laboratory, and a worker thoroughly the se of the test. There are now laboratories in all the s of this country sufficient in number and equipment for nanc e of all the tests which may be required. When blood one o f these laboratories, reports will be sent back promptly.

STIC VALUE OF THE WASSERMANN TEST.—According to the ven y Noguchi, the most distinguished investigator of the ann eaction in this country, a positive test is obtained in al sy hilis in 96 per cent of cases. It is probable that the ative ests were obtained in cases examined very early after These figures probably apply to congenital syphilis as mani- n inf ncy and the early years of childhood. Noguchi gives cent as an average for late latent syphilis, but this applies quired syphilis. In the latter part of childhood congenital s is in a late latent stage. There are no figures on the Wasser- reacti on at this stage, but they probably lie between 63 per and 9 per cent.

sitive Wassermann tests have been reported in rare instances iseases other than syphilis. Among these diseases are yaws,

complished in all stages of congenital
sually be found in discharging lesions
crapings from condylomata and from

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se is India ink. A drop of the sus-
glass slide and mixed evenly with a
best quality. A thin smear is then
same way that a drop of blood is pre-
is dried in air, and examined at once
The spirochetae appear as glistening,
ls standing out upon a homogeneous

sis of congenital syphilis depends mainly

f the infant.
yphilitic manifestations.

naturely born, the prognosis is very un-
its even though born at full term, are
physical development and vitality, the
are bad. Such infants are extremely
liable both to severe nutritional dis-
nt infections, among which broncho-
ause of death. The prognosis is most
ear normal at birth, and who develop
of life, showing no syphilitic manifes-

of the infant, and the character of the
largely dependent upon the period of
occurred. This in turn depends to a
of the disease in the mother. When
acquired or an untreated syphilis, the
worse than when she has had the dis-
treated. It is well known that the ten-
emature birth diminishes in the course
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...neosalvarsan should be used in the treatment of ...of congenital syphilis. In early life the *treponema* ...s in the visceral organs, where it produces great ...is the cause of the serious prognosis of the disease. ...ance of the body from the ravages of the invading ...ularly indicated in infancy. In all cases in which ...tion of the patient or the particular syphilitic mani- ...such a character as to bring the prognosis into the ..., treatment with neosalvarsan should be begun with-

...pe of congenital syphilis in which there is any question ...isability of salvarsan treatment, is that in which the ...ar normal in the early weeks, the first manifestation ... being a comparatively late outbreak of the circum- ...ous eruption. In such cases the prognosis was always ...er the old mercurial treatment, although the complete- ...anence of the cure as measured by the Wassermann ...r to be less than under salvarsan treatment. How- ...pleteness and permanence of cure under mercurial ...ends on the thoroughness with which the treatment ...This must enter into consideration. If the circum- ...patient are such that the physician is in doubt as to ...nged mercurial treatment will be thoroughly carried ...recommend salvarsan treatment even in these appar- ...e cases. If, on the other hand, the physician can insure ...curial treatment in this type of case, it is wisest not ...he slight risk which attends salvarsan treatment. The ...the treatment in the relapsing stage of the disease also ...he prospects of thorough mercurial treatment. This ...cularly favorable to treatment with mercury properly

...rity of cases of the late syphilis of childhood, the treat- ...iodide of potassium. Only when some lesion proves ...sistant to this treatment, or when there is a lesion in ...on as to threaten life, is neosalvarsan indicated.

OF NEOSALVARSAN.—Except in the newborn, the routine ...alvarsan for intravenous administration in infancy is ...In the newborn it is from 0.03 to 0.05 gramme according

fortunately this cannot be accomplished in all stages of congenital syphilis. The organism can usually be found in discharging lesions of the skin, and often in the scrapings from condylomata and from lesions of the mucous membranes.

The best routine use is India ink. A drop of the suspected material is placed on a glass slide and mixed evenly with a drop of fluid India ink of the best quality. A thin smear is then made with another slide in the same way that a drop of blood is prepared for staining. The smear is dried in air, and examined at once th an oil-immersion lens. The spirochetae appear as glistening, very, corkscrew-like threads standing out upon a homogeneous ck background.

ROGNOSIS.—The prognosis of congenital syphilis depends mainly hree factors:

The general condition of the infant.

The character of the syphilitic manifestations.

he treatment.

Syphilitic infants

When the infants Prematurely born, the prognosis is very unbelow the normal infants even though born at full term, are for their normal in physical development and vitality, the o nourish, and to survival are bad. Such infants are extremely , and to intercurrent infections, among which bronchoia stands first as a cause of death. The prognosis is most in infants who appear normal at birth, and who develop in the earlier weeks of life, showing no syphilitic manifesntil a later period.

he general condition of the infant, and the character of the manifestations are largely dependent upon the period of at which infection occurred. This in turn depends to a ent upon the stage of the disease in the mother. When r has a recently acquired or an untreated syphilis, the the child is much worse than when she has had the dis- time, or has been treated. It is well known that the tenmiscarriage or premature birth diminishes in the course r after treatment, and there is a progressive diminution in ty of the disease in successive infants born at full term. infant, the later the appearance of syphilitic manifestations, er the prognosis. Infants who show signs of the disease at sually die. The ominous nature of the syphilitic pemphigus ady been mentioned. Infants in whom the signs are mainly visceral syphilis, and who never show pronounced cutaneous tations, give a very unfavorable prognosis. On the other hen the first manifestation of the disease is a pronounced

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s, with preservation of some of the functions and com-
others. The rapid diminution of some symptoms and
e of others are characteristic. The presence of syphilis
l the marked improvement under antisyphilitic treat-
ortant points in the diagnosis. Syphilitic endarteritis
gummata of the base have been observed in infants as
n months.

IS. CLINICAL.—The clinical diagnosis of congenital
nds upon the recognition of pathognomonic signs of the
cases in which such signs are clearly evident, the diag-
difficult. Unfortunately all the manifestations of con-
lis are not pathognomonic, and the signs observed by
at any time may be of doubtful significance. Again,
of the disease may not be present at the time when the
nder observation, and the history obtained from a child's
ver wholly reliable as a means of diagnosis. Neverthe-
fants who are undersized, who lack vitality, or who have
with nutrition, a painstaking search should be made for
estations of congenital syphilis.
hilitic rhinitis with its snuffles actually comes under
ion of the physician in the early weeks of life, it can
istinguished from the snuffles of adenoids or a common
ory of snuffles is suspicious, but by no means diagnostic.
phigus neonatorum is pathognomonic. Enlargement of
n the early weeks, while, perhaps, not absolutely diag-
y strongly suspicious; when it occurs later, other causes
luded, such as rachitis or leukemia.
pallor of the face, and the shiny, glistening soles of the
are characteristic of the stage of cellular infiltration of
strongly suspicious, if not pathognomonic. If in addi-
es are present, the diagnosis is certain. When the pro-
face has advanced to the formation of a crusted eruption,
s of syphilis is not so certain.
ulo-papular syphilide, when typical, is unmistakable.
y comes from the fact that there may be very few lesions,
y a single spot upon the forehead or upon one of the
When the circumscribed eruption is not actually ob-
the physician learns of it from the history, its diagnostic
ch less, and the history can only be termed suspicious.
circumscribed eruption takes a less typical form, as in
s with pustular lesions, the case falls into the suspicious
Various forms of eczema and dermatitis must be con-
ossibilities. The previous history as to syphilitic mani-
s of great importance in such cases.

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he three groups further laboratory investigation should not be made as a routine measure. In the ratory investigation, while perhaps not absolutely nevertheless be made. It will give positive con- of the correctness of the diagnosis, and no purely is ever absolutely certain. If the case is to be ury only, the laboratory diagnosis, while not abso- certainly desirable. But if the case is of such a as to call for treatment with salvarsan, laboratory ne blood is positively indicated. Except in a very emergency, *no case of congenital syphilis should be rsan unless the presence of a positive Wassermann established.*

group, the testing of the Wassermann reaction is ted. The diagnosis can only be made with certainty of the Wassermann reaction, and certainty of diag- l in the treatment of congenital syphilis.

MANN REACTION.—The technic of obtaining the Wassermann reaction in infants has been described in t least 1 c.c. of serum is required. Sufficient blood antity of serum can best be obtained from the longi- infants. In older children the blood can be obtained of the ear, or by making a short incision in the heel. of the Wassermann reaction or complement-fixation is too complicated for ordinary clinical use. It re- ally equipped laboratory, and a worker thoroughly use of the test. There are now laboratories in all the this country sufficient in number and equipment for ce of all the tests which may be required. When blood of these laboratories, reports will be sent back promptly.

VALUE OF THE WASSERMANN TEST.—According to the by Noguchi, the most distinguished investigator of the reaction in this country, a positive test is obtained in philis in 96 per cent of cases. It is probable that the tests were obtained in cases examined very early after figures probably apply to congenital syphilis as mani- ncy and the early years of childhood. Noguchi gives as an average for late latent syphilis, but this applies syphilis. In the latter part of childhood congenital a late latent stage. There are no figures on the Wasser- on at this stage, but they probably lie between 63 per per cent.

Wassermann tests have been reported in rare instances other than syphilis. Among these diseases are yaws,

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NOSIS.–The prognosis of congenital syphilis depends mainly
factors:

general condition of the infant.
character of the syphilitic manifestations.
treatment.

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below the normal in physical development and vitality, the
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to nourish, and are liable both to severe nutritional dis-
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DOSE OF NEOSALVARSAN.—Except in the newborn, the routine neosalvarsan for intravenous administration in infancy is me. In the newborn it is from 0.03 to 0.05 gramme according

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o infants by inunction and by the mouth. In the great majority f cases, inunction is the preferable method. I usually order a mixure of mercurial ointment and vaselin in equal parts. Of this a quantity the size of a pea, which contains about gr. x of mercurial ointment, is rubbed daily into the skin. The place of inunction should be changed from day to day. The best localities are the palms, soles, axillae, and inner aspect of the thighs.

The only objection to inunctions is when the family is to be kept n ignorance of the treatment. In such cases the gray powder may e given by mouth in doses of gr. ½ four times a day. Other prepaations of mercury offer no advantages in early life.

FIG. 153

Intravenous injection in infancy
Injection of Salvarsan into the longitudinal sinus of a syphilitic infant
A—— L—— before treatment

Mercurial treatment should be continued until the Wassermann reaction becomes negative. It should never be used continuously for a long period, but with occasional periods of about a week of intermission.

TREATMENT WITH IODIDE OF POTASSIUM.—This is used in the later stage of the disease, or when any gummatous lesions are present. The drug is well borne by children. The usual dose for older children is from one to two drachms daily, divided into three doses. In infants the daily amount is from fifteen to twenty grains.

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which suggest that the immune reaction is of a particularly complicated nature, and consequently a particularly fascinating problem awaits further research in this direction. Owing to the comparatively recent discovery of the specific microörganism which causes syphilis, and to the still more recent success in growing the organism in pure culture, very little progress has been made up to the present time in the study of the antibodies formed by the host in its contest against the *treponema pallidum*. Almost nothing is known of the antibodies which are undoubtedly formed in syphilis. The varying nature of the clinical manifestations of the disease at different stages is sugges-

FIG. 154

Congenital syphilis
A—— L—— after one injection of salvarsan

tive of a varying virulence on the part of the infecting parasite. Nothing, however, is known as yet about these various factors. The research of the next decade will probably be largely occupied with these problems.

That antibodies are actually formed in syphilis, there can be no doubt. Nevertheless some very interesting light has recently been thrown upon the nature of the Wassermann reaction in syphilis, which apparently shows that the reaction is not dependent upon specific antibodies in the ordinary sense of the term. In the complement-fixation tests which have been used in other infections, it has been shown that the reaction depends upon the fixation of complement to antigen by means of a specific complement-binding anti-

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SSION.—Syphilis may be acquired from the mother
the disease subsequently to the birth of the child.
take place by nursing, by kissing, or by some other
ical contact. The disease may be contracted in the
m wet-nurses. Also, a syphilitic child may transmit
one who is healthy, both by kissing, or other form of
tact, and indirectly by means of infected utensils such
cups, spoons, clothing, and so forth. In former days
virus was used in smallpox vaccination, syphilis was
ransmitted in this way, but since the general introduc-
ne virus, the disease has almost never been acquired by
. There have been cases in which the disease was trans-
he rite of circumcision, from the mouth, or from the in-
f the operator.

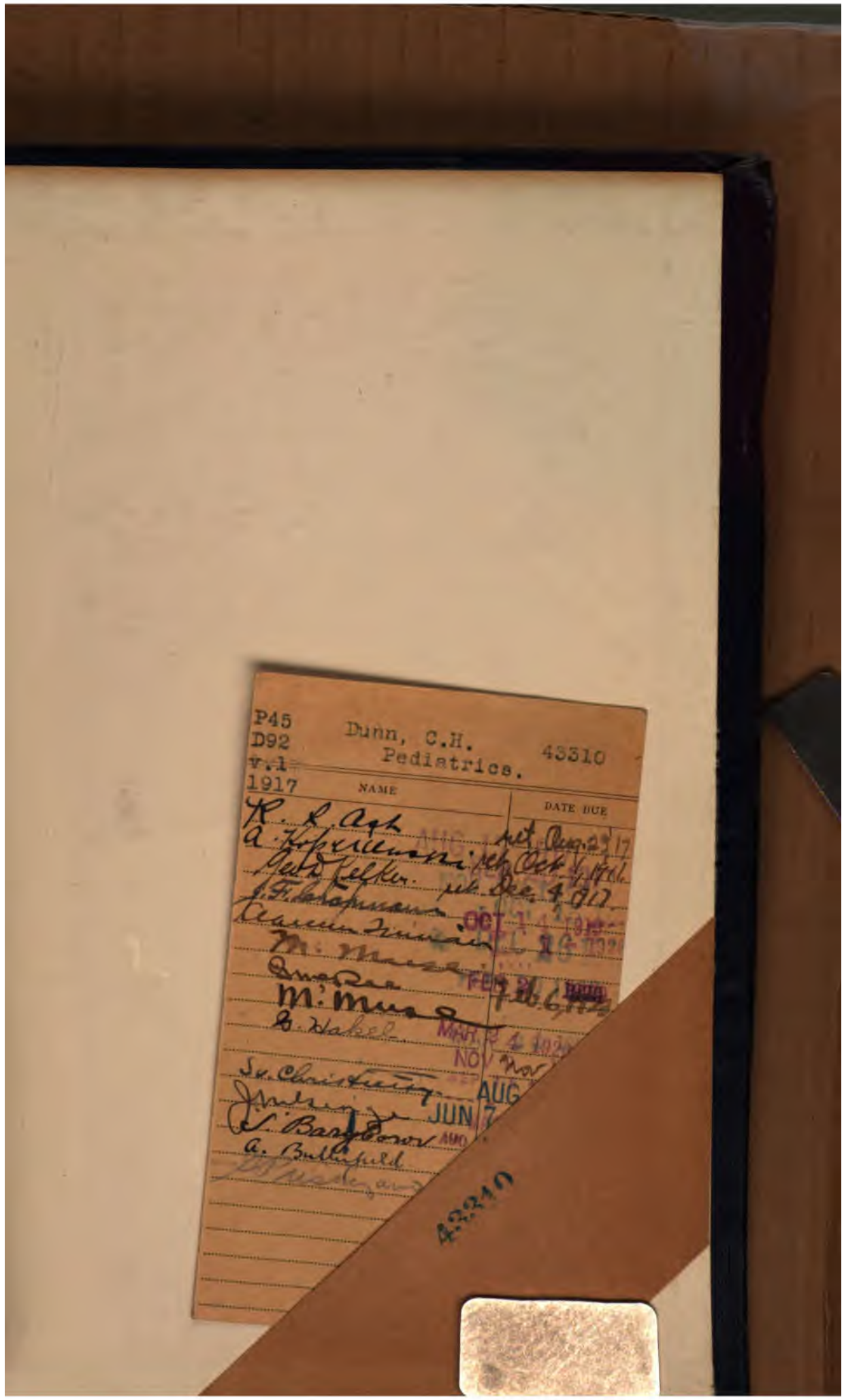
OMS.—The symptoms of acquired syphilis in children
way from those of the same disease in adults, and will
cribed here in detail.

OSIS.—The recognition of the disease as belonging to the
orm depends mainly upon the finding of a primary lesion
nt of inoculation. While in some cases the secondary lesions
nble those of congenital syphilis, the diffuse cellular infiltra-
he skin is never seen in the acquired form of the disease.

NOSIS.—As compared with the congenital form of syphilis,
ired form is much milder and more favorable.

TMENT.—The treatment of acquired syphilis is the same
of the congenital form of the disease.

End of Volume I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