# DISEASES OF CHILDREN

BY

EDWIN E. GRAHAM, A.B., M.D.

PROFESSOR OF DISEASES OF CHILDREN IN THE JEFFERSON MEDICAL COLLEGE; PEDIATRIST
TO THE JEFFERSON MEDICAL COLLEGE HOSPITAL AND TO THE PHILADELPHIA GENERAL
HOSPITAL, PHILADELPHIA; CONSULTING PEDIATRIST TO THE TRAINING SCHOOL
FOR THE FEEBLE-MINDED AT VINELAND, N. J.; MEMBER OF THE
AMERICAN PEDIATRIC SOCIETY, ETC.

Illustrated with 89 engravings and 4 Plates
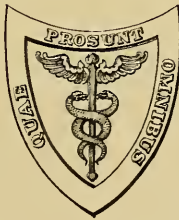
LEA & FEBIGER
PHILADELPHIA AND NEW YORK
1916

R J45
G762

TO

THE MEMORY OF MY

BROTHER

JAMES GRAHAM, M.D.

PREFACE.

In the preparation of this work the aim of the author has been to make it represent the most modern views upon each subject discussed, and to present these views in such a way that they may be immediately available to the busy practitioner as well as easily intelligible to the medical student.

The treatment of each disease is given in full, and the physician engaged in general practice will find herein the precise management of a typical case of any disease which he is called upon to treat. In addition, the clinical examination of the sick child is carefully considered, and its anatomy and physiology are discussed in detail.

In the consideration of each subject a special effort has been made to make the book thoroughly up to date, and to each chapter has been allotted the amount of space its importance appeared to warrant, only a few of the rarest diseases having been omitted. As, however, the book has been written from the practical, and not from the theoretical standpoint, recent, and unconfirmed suggestions and theories have not, as a rule, been considered.

Sufficient space has been devoted to pathology, and a larger amount, proportionately, to symptoms, diagnosis, and treatment. The illustrations and x-ray plates are all from the author's private and hospital cases, except where credit has been given to others.

From long experience in teaching both students and practitioners, it has seemed to the author most important that the processes of normal digestion be thoroughly understood before any attempt be made to study the various disturbances and diseases of the gastro-intestinal tract, hence the subject of normal digestion has been considered in a special chapter.

The subject of infant feeding has received particular attention; the construction of milk mixtures, usually a vague subject to both the general practitioner and the student, is carefully explained, and the calculation of caloric and percentage feeding has been illustrated by formulas reduced to ounces.

Diseases of the gastro-intestinal tract have been presented in full, and some of the most advanced ideas concerning diagnosis and treatment have been incorporated. Food injuries, chronic constipation, pylorospasm, and pyloric stenosis have received special consideration, a careful differentiation being made between the two latter affections.

Special features of the book to which the author desires to call the attention of the reader are the chapters on Infant Mortality, Heredity and Environment, Puberty, Fresh Air, and Diseases of the Liver, Spleen, and Thymus Gland. To each of these subjects is allotted a special chapter in view of their ever-increasing importance to the practising physician.

The Diseases of the Nervous System have been discussed in eighty pages. A careful consideration of these diseases belongs in a book on Pediatrics, and the physician interested in children will find here a broad field for study. To Infantile Paralysis the author has devoted ten of these pages.

A special chapter has been devoted to Dentition, for the author regards this as a normal and physiological process in the course of normal development. Particular attention is also called to the articles on Diphtheria, Influenza, Pertussis, Poliomyelitis, and Enlargement of the Thymus Gland.

The author here acknowledges his indebtedness to the following physicians connected with the Pediatric Department at Jefferson Medical College: Dr. Julius Blechschmidt, Dr. R. L. Engle, Dr. Joseph Fleitas, and Dr. W. H. Johnston.

I wish to express my appreciation to my publishers, Messrs. Lea & Febiger, for their earnest coöperation during the entire period of the book's preparation.

E. E. G.

Philadelphia, 1916.

CONTENTS.

CHAPTER I.

THE NORMAL INFANT AT BIRTH.

CHAPTER II.

NORMAL DEVELOPMENT OF THE CHILD.

CHAPTER III.

The Clinical Examination of Sick Children.

CHAPTER IV.

Infant Mortality.

CHAPTER V.

Heredity

CHAPTER VI.

Congenital Malformations.

CHAPTER VII.

Diseases of the Newborn.

CHAPTER VIII.

Infant Feeding.

CHAPTER IX.

Normal Digestion.

CHAPTER X.

Fresh Air in the Treatment of Disease

CHAPTER XI.

Dentition

CHAPTER XII.

Rickets.

CHAPTER XIII.

Diseases of the Gastro-intestinal Tract.

CHAPTER XVI.

DISEASES OF THE BLOOD.

CHAPTER XVII.

DISEASES OF THE DUCTLESS GLANDS.

CHAPTER XVIII.

Diseases of the Bones and Joints.

CHAPTER XIX.

Diseases of the Genito-urinary System.

CHAPTER XX.

DISEASES OF THE SKIN.

CHAPTER XXI.

Diseases of the Ear.

CHAPTER XXII.

The Specific Infectious Diseases.

CHAPTER XXIII.

RHEUMATISM.

CHAPTER XXIV.

SCURVY 763

CHAPTER XXV.

THE NERVOUS SYSTEM.

CHAPTER XXVI.

Puberty.

DISEASES OF CHILDREN.

CHAPTER I.

THE NORMAL INFANT AT BIRTH.

General Appearance.—The physical proportions of the normal infant at birth show a striking contrast to those resulting after developmental adjustment. The head appears large in proportion to the size of the body; likewise, the shoulders, arms and upper chest, when compared with the pelvis and legs.

The child's body is well rounded, being covered with a goodly layer of superficial fat. The head may be covered by a moderately thick growth of hair, although this is by no means always the case; strong, vigorous children showing not infrequently at birth a very small growth of hair. The head is often misshapen, as a result of pressure during labor.

The face of the infant in the first few months of life is small in proportion to the cranium, and is as 1 to 8; at the age of five years, as 1 to 4; and in adult life as 1 to 2 or $2\frac{1}{2}$. The rapid growth of the inferior maxillary bone has much to do with the increased facial size. The chest of the infant is barrel shaped, the anteroposterior and transverse diameters being about the same.

The abdomen is very prominent, being much larger proportionately than later in life. This is due to the large size of the liver in infants and the small size of the pelvis.

The feet of the infant are distinctly arched, but this is not always apparent on account of the large amount of fat often present.

The nails project beyond the fingers, their borders being very brittle.

The color of the body immediately after birth is that of venous blood, as a result of stasis during the period of intra-uterine pressure. As this is removed, and respiration becomes established, the oxygenation of the blood soon manifests itself by a hyperemic redness, resulting within a few days in exfoliation which persists for about a week.

Skin.—At birth the delicate integument of the infant is frequently covered with a material, the vernix caseosa, a secretion of its own sebaceous glands, with exfoliated epithelium and lanugo. The latter is the term applied to the soft, downy hair covering the body of the fetus, and persisting for a long time after birth. The sweat glands of the normal infant are comparatively inactive for several weeks after

2

birth, while the functional activity of the sebaceous glands is marked, often resulting in seborrhea of the scalp. The skin on the scalp is thicker than elsewhere on the body and adherent to the occipito-frontalis beneath it.

Head.—At birth the head is capable of being easily molded, often resulting in elongation due to birth pressure, and frequently giving it a grotesque appearance. This plasticity, on the other hand, also permits of rapid adjustment of the cephalic contour, so that extensive disproportions often disappear soon after birth. The fontanelles, or membranous spaces between the bones of the skull, are the results of incomplete ossification, and are important landmarks in pediatrics as they are in obstetrics.

The largest, the anterior fontanelle, is situated at the junction of the coronal, sagittal, and frontal sutures; it is rhomboid in outline, with the apex projecting anteriorly.

The posterior fontanelle, triangular in shape, hardly exists as an opening at birth, the bones usually lying quite close together. The Wormian, or supernumerary bones, are frequently encountered along the sutures and at the fontanelles.

The base of the skull in the newborn differs from that in the adult, in that there are no mastoid processes; on the other hand, the base of the skull is well ossified, notwithstanding that, during fetal development, the base of the skull is poor in ossification as compared to the vault.

Thorax.—At birth the thorax is extremely compressible, the average circumference of the chest being thirteen inches. The clavicle is the first bone to ossify, and also the one most frequently fractured during parturition. The humerus at birth is almost entirely ossified, although the extremities of the bone are composed chiefly of cartilage.

The knee-joint is supplied with numerous bursæ, distributed among the tendinous attachments of the various muscles.

Circulatory System.—At birth, the average weight of the heart is about two-thirds of an ounce. The thickness of the right ventricle is very nearly the same as that of the left, the ratio being 6 to 7. The left ventricle, however, grows very much more rapidly than the right, so that at the end of the second year the ratio is 2 to 1, which is nearly that of the rest of childhood.

In the fetus the connection between the pulmonary artery and the aorta is called the ductus arteriosus. There also exists an opening, the foramen ovale, between the auricles, and there is likewise a valve which guides the blood from the inferior vena cava through the foramen ovale. This valve becomes atrophied after the normal circulation of the child has become established. The fetal circulation is characterized by the fact that arterial blood flowing from the placenta enters the fetus by means of the umbilical vein.

Post-natal Circulation.—The transition of the fetal circulation to that after birth is characterized by several well-defined features. As respiration becomes established, and the pulmonary circulation

begins, the patulous foramen ovale and the ductus arteriosus gradually close, the expansion of the lungs and the filling of their bloodvessels inaugurating the independent circulation by reason of the effect of the increased blood-pressure in the aorta upon the valve-like ductus arteriosus. Furthermore, the blood which is emptied into the left side of the heart exerts a mechanical pressure upon the valve closing the foramen ovale.

The interruption of the placental circulation, together with the ligation of the umbilical cord, finally causes an atrophy of the ductus venosus and umbilical veins, converting them into a strand of fibrous tissue, the round ligament of the liver.

Arteries.—The relation between the size of the heart and the diameter of the arteries in the newborn is inverse to that of the adult. In the latter the heart is quite large compared to the diameter of the arteries, while in children the heart is small and the caliber of the arteries is large. It is manifest, therefore, that the blood-pressure in children is relatively low; on the other hand, it is quite high in the pulmonary circulation, on account of the larger caliber of the pulmonary artery as compared to that of the ascending aorta.

Blood.—After birth, and before ligation of the umbilical cord, a certain amount of blood enters the child, resulting in plethora. Soon after birth, however, an equilibrium is established, due to excretion of fluids and a concentration of the blood. There is consequently a high color index, which soon disappears. The red blood corpuscles are present in all sizes, nucleated ones being frequently present for several days after birth. The blood of the newborn contains more leukocytes than that of the adult. The number of erythrocytes in the newborn and in very young children varies from 4,340,000 to 6,500,000.

Pulse.—The activity of the circulation is very much more pronounced at birth and early infancy than in later life. It is estimated that the entire circulatory cycle in the infant is completed in twelve seconds. The pulse is slightly more frequent in females than in males. At birth, and even during infancy, a very rapid and irregular pulse may be compatible with good health; it may be noted even during sleep.

Digestive System.—The organs of the digestive system in the newborn and in infancy possess peculiarities which markedly differentiate them from those of the adult. This is true not only of diseases of these structures, but is inherent in their anatomy and physiology. Among the most pronounced peculiarities of the mouth are the dryness and thinness of the oral mucous membrane, the deficiency of the salivary secretion at birth as well as its weakness in enzymosis. The absence of prehension is compensated by the suction produced by the lingual musculature and pressure of the cheeks. The stomach is small, almost vertical, and the fundus is practically undeveloped.

The gastric juice contains essentially the constituents of that of the adult, while the pancreatic juice, on the other hand, is incomplete in its action on fats. The intestines are relatively longer than in the adult and poor in the development of Lieberkühn's and Brunner's glands.

The liver in the newborn is large and vascular, the volume being greater than that of the two lungs combined, the ratio not being reversed until the advent of puberty. The bile is poor in inorganic salts, excepting iron.

Respiratory System.—The caliber of the nasal passage in the newborn is very small. The larynx is, likewise, very narrow, which is important to appreciate in considering intubation in an infant. The trachea at birth is flattened anteriorly and posteriorly, and remains so for some time, notwithstanding the distention from respiration. The surfaces of the cartilaginous rings are in apposition until the trachea becomes further developed.

Lungs.—At birth the color of the lungs is a pinkish gray; the direct chemical action of the blood and the repeated inspiration of air soon changes that color to one of mottled gray and black. While before birth at full term the position of the lungs is toward the posterior portion of the thorax, the expansion induced by birth soon causes them to cover the pleural portion of the pericardium and occupy their permanent position in the thoracic cavity. The weight of the lungs before birth is 48 grams, while after the complete establishment of respiration their weight is increased to 80 grams.

Ductless Glands.—The thymus gland in the newborn is situated in the anterior mediastinum behind the manubrium of the sternum, and reaches full development about the end of the second year. It is generally composed of two lobes which are in apposition in the middle line. After the second year it loses its identity by fat substitution, or entirely disappears.

The thyroid gland in the newborn is relatively very large, the two lobes being united transversely by an isthmus. The parathyroids are present behind the lateral lobes of the thyroid gland.

The bronchial glands, lymphatic in character, consist of several groups; one in close relation with the trachea, another at the bifurcation of that structure; others at the roots of the lungs, known as the hilus glands, while still another group is in intimate relation with larger bronchioles as they enter the lungs.

Genito-urinary System.—The kidneys of the newborn are relatively large and do not increase in size as much as the lungs or heart. At birth the kidneys are practically at the acme of their functional capacity, and the suprarenals are relatively larger than in the adult and are extremely vascular.

The bladder in the newborn is oviform in shape, the smaller end being directed upward, the bladder descending as the pelvis develops. At birth the bladder is capable of holding from 2 to 4 drams.

Both the ovaries and testicles are at first abdominal organs, and only in the course of the development of the child, begin to occupy their respective positions.

The uterus at birth is about one inch in length, and possesses no fundus. The cervix, on the other hand, is thicker and longer than the remaining portion.

Nervous System.—In the newborn the nervous system is in a very rudimentary condition. The brain is large, watery, and very soft, and shows but little differentiation of gray and white matter. Likewise, the spinal cord is of soft consistency, the anterior horns being more fully developed than the other structures.

Organs of Special Sense.—At birth, and for some time thereafter, the sclerotic coat of the eye has a bluish color, due to the underlying choroid being seen through the slightly transparent sclera. There is no coördination in the excursions of the extraocular muscles. The refraction of the eye at birth is usually hyperopic. The power of fixation is also absent. While the power of convergence may be present, it will not produce contraction of the pupil for some little time after birth. The color of the iris is usually blue or bluish gray. Photophobia in the presence of strong light is a constant characteristic of the newly born.

The ear and acuity of hearing in a newborn babe are very poorly developed, due to the shortness of the external meatus and the absence of bony formation. It is surprising, however, how soon the acuity of hearing in childhood reaches its acme.

Taste.—This sense is very acute at birth, the newborn having been observed on frequent occasions to signify its appreciation or dislike for sweet or sour substances respectively. The slightest alteration in accustomed food is quickly detected in infancy, a bottle often being refused when there has been the smallest possible variation in an accustomed food mixture.

Tactile Sensation.—This is developed at birth, more especially at the lips and tongue, where its utility is manifest for the purpose of nursing.

Mammary Glands.—At birth the mammary glands are normally congested and somewhat swollen, and in many instances secrete a milk-white fluid. They are from one-third to one-fifth of an inch in diameter.

Temperature.—The temperature of the newborn and of the young infant is exceedingly unstable. At birth the rectal temperature is from 98.4° to 100° F., fluctuating between these points for several weeks. The variation in the temperature is easily understood when one considers the large surface for radiation and the facility with which heat is disseminated from the easily dilated capillaries in the infant. A great many observations have shown that the temperature of infants begins to rise in the forenoon, reaches its fastigium in the course of the afternoon, and then declines. The temperature is at its minimum during the night and early morning hours, and at its maximum in the early afternoon.

CARE OF THE NEWBORN.

Bathing.—As soon as possible after birth the child should be bathed. In order that no delay may occur during the bath, all preparations should be made beforehand. The time of giving the bath should be

fixed at a certain hour each morning, neither just before nor after feeding, nor immediately preceding the taking of the child out of doors. The temperature of the room should be 72° F. In winter, if possible, the child should be bathed before an open fire, care being taken not to place the infant in any line of draught.

The best basin is the one divided in two portions, and supported at a convenient height by an iron frame. A small, fine sponge should be used to cleanse the face, corners of the eyes and ears; a small piece of soft linen or muslin to cleanse the nose. The head is now washed with pure castile soap, the soap cleansed off carefully and the head dried, the child's head, during this portion of the bath, being allowed to fall back on the hand of the nurse, care being taken to prevent soap getting in the eyes. The water in the other portion of the basin is now used on the body, with castile soap, special care being given the axillæ, groins, genitals, and anal region.

The child is now quietly lowered into its tub—for infants the one of rubber, for older children the ordinary tin tub—and allowed to kick and move its body freely for two or three minutes. The baby is then rubbed and dried quickly with warm towels; its clothing being all previously arranged, it is quickly dressed and returned to its crib. This bath should be given every day, care being taken that the rubber tub does not become overheated, as severe burns may result from the baby being placed in the tub if the rubber has become too hot by being stationed near an open fire.

The temperature of the bath is important, as a child's circulation is easily depressed, and coldness and blueness of the extremities easily produced. The temperature of the bath at different ages is shown in the following table:

At birth, 98° F.
At two weeks, 96° F.
At one month, 94° F.
At one month to six months, 92° F.
Six months to one year, 90° F.
One year to two and one-half years, 86° F.

The ordinary bath thermometer is easy to read even by an untrained nurse. It is protected by a wooden cover and does not sink; it is indispensable in a well conducted nursery.

Clothing.—The clothing of all infants and children should be loose, especially around the neck, chest, abdomen, and pelvis. In order that the chest may develop properly and the normal action of the lungs be not restricted, it is absolutely essential that no compression whatever of the thorax be permitted. The proper performance of digestion, the motor activity of the stomach and intestines, and the normal circulation and functions of all the abdominal organs may be more or less interfered with unless quite loose garments are worn. The wearing of diapers tightly drawn and pinned is an undoubted factor in the development of pelvic deformities.

All clothing should be supported from the shoulders and not from the

chest or hips. Care must always be taken to keep the extremities warm, especially the hands and feet.

The abdominal band can be dispensed with after the second month. The knitted binder fitting the body snugly and provided with arm-holes is the best form to use.

In summer, gauze or very light flannel underwear is the best. A child must, however, be dressed with a view to its circulation; a thin delicate baby, with poor circulation, requires more clothing than one whose body is well covered with a plentiful supply of fat.

At night, infants should wear a shirt of cotton or wool, a napkin, cotton stockings, and a long outer garment of light flannel, made with a drawing string or buttons at the bottom, and sufficiently wide not to confine the legs. This insures the lower extremities being covered during the night.

The infant or long clothes consist of a binder worn during the first two months; shirt with sleeves, napkin, long stockings, flannel petti-coat, white petticoat, a long dress, and light flexible shoes or knitted socks. Diapers should not be too heavy or cumbersome; one of the best materials is birdseye.

All shoes should be large, broad-toed, and made rights and lefts. If not so made the child's feet are turned from their normal line, the axis of the great toe changed, and the normal movement and growth of the feet impeded. A light and graceful step depends largely upon the strength and elasticity of the toes. The soles of children's shoes should be flexible, for if stiff the active movements of the muscles of the feet are interfered with. These muscles are especially active in children.

All clothing must be worn with due regard to climate and sudden changes in the weather. The tendency, undoubtedly, is to bundle children up too much. It is not advisable to change the weight of the underclothing too frequently. A heavier or lighter coat is a much better arrangement for changes in the temperature. In winter, the head, and, in quite cold weather, the ears, should be covered with a woolen cap. In summer, the ordinary hat of light straw with broad brim is the best.

Sleep.—The bedroom should be large, airy, and sunny. The curtains should be of muslin or linen to insure easy and frequent washing. The light may be excluded by dark shades. All superfluous and heavy draperies should be dispensed with, and the floor covered with light, inexpensive rugs or carpet.

The temperature of the infant's room should not fall below 60° F. during the night. The day nursery and sleeping apartment should always, if possible, be separate rooms. Each child should have a separate bed, and, if practicable, older children should room alone.

During the first two months of life the child requires about twenty hours of sleep out of each twenty-four. As it grows older the amount of sleep required gradually becomes less. From the age of two months to six months, sixteen hours; at one year, fifteen hours; and at eighteen

months, thirteen to fourteen hours. A child two and a half years old should sleep twelve hours, a portion of this being a nap during the day, and if possible this sleep during the day should be kept up until the child is five or six years old, and should be taken in the open air. At night the windows of the sleeping room should be kept wide open.

Infants should be put to bed between six and seven o'clock; older children, between seven and eight o'clock. Regularity of sleeping should be instituted at birth, and much depends upon training. Healthy children, if kept in their own crib, in a quiet, dark room, not handled unnecessarily, and only disturbed to be fed, bathed, clothed, and to have soiled linen changed, will rarely fail to obtain the prescribed number of hours of sleep.

Slight causes may, however, keep the child awake. An overheated or poorly ventilated room and too much bedclothing are among those causes most frequently overlooked. If there is decided restlessness in sleep, or a marked reduction in the amount of sleep, it usually indicates illness of some kind and should be carefully investigated.

The crib should be provided with high sides or an inexpensive wooden fence, otherwise the child, after the tenth month, may be severely injured by falling out of its bed. A good set of springs, a moderately firm mattress, preferably of hair, and a small, flat hair pillow not over three inches thick is the best suited for the baby. The mattress, to be kept clean and sweet, must be protected by a rubber sheet.

Exercise.—The infant if put in its crib without bed covering and its clothing loose will kick and move the arms vigorously, giving evidences of its pleasure by sounds as expressive as words. Occasionally, by placing the child on its abdomen the spine will be seen to bend vigorously and the head be bent backward, while the arms are often used to advantage. At the age of eight to nine months the infant usually begins to creep, a process that calls into action almost all the muscles of the body.

When one year to fifteen months of age, infants usually stand alone and begin to walk. It is not wise to encourage the child to stand alone, or to assist it much in walking, unless one is sure that its failure to walk is due to its timidity, and not to lack of strength. Many delicate, rachitic children are left with more or less permanent bowing of the legs as the result of an overproud parent teaching his child to walk too soon.

The habit of propping up in their coaches infants of twelve months or less is most unwise. The spinal column, being largely composed of cartilage at this age, is utterly unfitted to support the superimposed weight. As a result of this increased flexibility of the spine the child leans to one side, the spine becomes bent, and this unnatural position may be kept up for hours, the child even possibly falling asleep, and thus the foundation is often laid for a future spinal curvature. For the same reason, infants when carried should, if possible, be kept in a horizontal or semiprone position; or if held more or less erect the back

should be well supported or the body allowed to fall forward against the chest and shoulders of the person holding it.

The time when a baby should be taken outdoors depends largely upon the time of the year when it was born and the climate. A child born in the winter or fall in a climate such as Philadelphia, where the winters are moderately severe, should not be taken out before the age of two months. Then on mild, sunshiny days with little wind the baby may be taken out for a half to one hour, well bundled up in its coach, and its face protected by a veil.

When two months old, children may, for the same length of time, be wheeled in a coach in the nursery, dressed as if for outdoors, the windows being raised and all doors closed to avoid draughts. This makes the change from the nursery to the outside air less abrupt.

Children born in warm weather may be taken out when three weeks old. The eyes of all infants should be well protected by a parasol from the direct rays of the sun. It is safer to keep the baby indoors in damp weather, or on days when the thermometer falls below 20° F., especially if there is a strong wind. Older children usually get sufficient exercise in their play, which is to be preferred to long walks, the latter being often fatiguing. When possible, gardening is a most useful mode of exercise, each child having its own little plot for plants and flowers.

A roof garden may be made to suit the purse of the poor or rich. If used in summer it is a great boon to those unable to keep their children for a long period out of the warm, close air of the city. It is also of great practical use in winter.

Fresh Air.—The infant and young child should be given an abundance of fresh outside air, and if the child's normal bodily temperature is preserved, and the hands, feet, and ears kept warm, no injury from fresh outside air need be feared. It is always advisable, when possible, to protect the child from draughts, but a free circulation of air through the nursery and bedroom is always desirable. An open fireplace is of great advantage, insuring a withdrawal of air from the room. The window ventilator, consisting of a board fitting in the window with the pipes directed upward to admit fresh air, answers admirably. All rooms occupied by children should be thoroughly flushed out once a day, during the child's absence.

A temperature of 66° to 70° F. is suitable for the nursery, the former being better than the latter. The child when six weeks old, warmly clad, may be taken into a room at 70° F., and, being carefully kept out of draughts, a window may be raised and the temperature allowed to fall to 60° F. This may be done for one hour, morning and afternoon. The temperature at two months may be reduced to 50° F., and at the age of three months to 40° F. When the child is taken out of doors in cold weather a veil should always be worn, the hood of the baby carriage should always be kept raised, and the coach always turned so as to protect the infant from cold winds. No

harm but great benefit results from these daily airings, and the old prejudice about taking cold should not be considered.

Hot water heating is cleanly, does not dry the air, and is to be preferred. If a hot-air furnace is used the radiators should be covered with cheesecloth.

Eyes.—The eyes at birth, in the normal infant, are fully developed. As soon as possible after birth they should be well washed with a saturated solution of boric acid, and if any suspicious vaginal discharge is present in the mother a few drops of a solution of nitrate of silver, 2 grains to the ounce, should be carefully dropped into each eye and the eyes then washed with normal salt solution. The eye-wash of boric acid should always be used twice daily during the first week of life, as mild forms of conjunctivitis, non-gonorrheal in character, but caused by the entrance into the eyes of discharges from the vagina or rectum of the mother, are not uncommon.

The mouth should be very gently cleansed with a piece of gauze dipped in a saturated boric acid solution, care being taken not to injure the delicate buccal mucous membrane. During the first two weeks of life the mouth should be cleansed just before each nursing. This is of advantage to both mother and babe, as during this period the nipples of the mother and mouth of the child are especially liable to become the seat of disease.

Rash.—The skin of the newborn babe is easily irritated, and causes quite incapable of doing any harm in older children may produce erythema, eczema, or intertrigo in infants, especially in the folds of the groins and genital region. Perfect cleanliness, the use of castile instead of irritating soaps, keeping the folds dry, and using a dusting powder of starch, lycopodium, or zinc oxide, will usually be found all that is necessary to prevent the development of these annoying conditions.

Genitals.—The genitals should be kept clean, but not washed oftener than the rest of the body, unless soiled by vaginal or rectal discharges. In boys the prepuce should be examined and retracted daily, if tight or long, until the parts are in a normal condition. Circumcision is often necessary. It usually removes more or less local irritation. It may do much good—if properly performed it can never do harm.

It is important that the child should be taught to exercise control of the bladder and rectum quite early in life. When six months old the child should be placed in its chair for about three minutes or less, preferably just after each feeding. If this rule is adhered to systematically the majority of children will, at the age of one year, have learned to control the discharge of both urine and feces. This is of advantage to the child, as the evacuation of the bladder and bowel at certain regular intervals is important, and the training of the child itself is also of benefit.

Anal Region.—During the first year of life and often until the end of the second year, if the child has not been so trained, the nurse is compelled each day to devote considerable time to the washing of the

napkins, and such time is perhaps taken from the hours which the child should spend in the open air. The nurse, if relieved of this washing, is also, in my experience, much more cheerful and manageable. Again, it is certainly not hygienic for a child to be wearing articles of clothing soiled by urinary or fecal discharges any longer than is necessary.

Vaccination.—All children should be vaccinated during the first year of life; after the third month is preferable; and better, if possible, during the cool than the hot months. If the arm is properly protected from infection by an appropriate dressing the symptoms produced by vaccination are slight and temporary. Vaccination should be deferred in a child who is frail, ill, or suffering from any skin disease.

CHAPTER II.

NORMAL DEVELOPMENT OF THE CHILD.

As a rule, it may be stated that for a child to develop according to normal standards, certain conditions such as heredity, environment, birth, food, and hygienic surroundings, should be up to the normal or usual standards. Ideal conditions are unnecessary, but the better the conditions are the greater is the probability that the infant will develop properly.

Normal development differs greatly in races and in the same race under different environment. In the monkey tribe it requires from six to ten years to reach full intellectual and physical development. In certain African races the span of life is only thirty years, and the children of these savages are very precocious. They walk very early, and reach puberty and full intellectual development at the age of ten years.

Caucasian infants of civilized countries are the least precocious of all races, requiring twenty-five years to attain full mental development; and, as the developmental period is the most important one in the life of the individual, it follows naturally that we cannot be too careful that the individual lives during this period under conditions which will best promote his mental and physical progress.

There are certain portions of the body that develop rapidly in size, but acquire their complete functional activity slowly. A striking example of this is seen in the infant twelve months old, whose brain is almost two-thirds as large as the adult's, but the functions of this brain require years of careful training for their development. It is well for us to appreciate that normal children come from normal parents, and that much can be accomplished by a better physical, mental, and moral training of the masses. If the physical nature alone is developed we produce a race of athletes. If the mentality only is cultivated we develop a people among whom prodigies and precocious youths are the rule rather than the exception. If only the moral side is developed the race becomes narrow-minded and fanatic.

How many children are systematically weighed and measured to see if they are growing and developing normally? and how much time do parents, as a rule, give to the study of their child's growth and development? Normal development is more likely to be found where systematic medical examinations of the child are made. This is now becoming the rule among the better educated, and school physicians are of much assistance in detecting both acute and chronic conditions among those who are, perhaps, less often taken to a physician. All children who are compelled to work should be pronounced by a

physician to be fit and capable of working before being employed, and the law concerning age limit and hours of employment should be strictly enforced.

During school life many factors combine to retard the normal growth of children, both boys and girls. The daily sessions are too long, and the concentration of attention demanded by lengthy recitations is also too great a tax. Recesses do not come frequently enough to relieve the inactivity and tension of the child's muscles. Opportunities to relieve the bladder and rectum are most important, and should be provided for. Sometimes, through timidity, the child will fail to obey such calls until reduced to an agony of nervousness. There are also too many hours of home study; the child needs most of these hours for play or family association; and during her hours out of school the little girl should not be taxed with sewing, knitting, painting, or too many piano lessons. The free hours would be far better occupied in winter by gymnastics or skating, and in summer by swimming or cycling.

During the long summer vacation the child should not be obliged to carry out a course of study prescribed by the school before closing, such as the reading of certain stipulated books, the writing of compositions, etc. As a rule, school teachers fully appreciate the importance of hygienic measures, but cannot personally apply these principles to the welfare of the pupils. In the early morning hours the mind is most alert to permanent impressions, the muscles most capable of doing their hardest tasks. After the first fatigue drop at noonday the blood tension and temperature increase steadily until their highest point at 6 P.M. From this time on the natural craving of adult or child is for rest and sleep. Sleep is necessary; even the young mammal toward evening will suckle or eat, then play, and settle itself to sleep.

High blood-pressure we know precludes sleep; yet in this period the plastic body of the child often suffers lasting injury by being driven to evening study. Thus we defeat the very purpose toward which all education is directed—namely, the production of the highest degree of efficiency. The system of cramming for examinations, which so closely follow each other as to be no gauge of the child's advancement, is another pernicious factor that adds to the general high tension. The fact seems to be often overlooked that there is an education higher and far better calculated to fit the child for life than the mere imparting of a mass of facts. The inculcation of moral principles, which the child may have no opportunity of learning elsewhere, forms a much more stable foundation.

The physical conditions upon which the activity of the mind depends are very complex, and it certainly seems only reasonable to believe that, during the years in which the bodily growth is most rapid, there should be a corresponding reduction in the amount of mental and physical work imposed upon the child. This is necessary even if the child is apparently perfectly able mentally and physically to do the work.

It is an interesting fact that bright boys are, as a rule, taller and heavier than dull boys, and it is also well established that, as the age increases, brilliancy in most studies decreases, and that the ability to learn and absorb quickly is an attribute of the young. Normal children should be studied more, and the normal standard is best ascertained by the careful study of the physical statistics of a large number of children. These are often of great value, and frequently furnish important information both to the physician and educator. Special attention is necessary during the two years before puberty, as in both boys and girls this is the period of most rapid growth in both height and weight, the age being from twelve to fourteen years for girls and from fourteen to sixteen for boys.

Height.—Heredity is a distinct factor in height, the children of tall parents being, as a rule, taller than those of short parents. Rachitic children are, as a rule, shorter than those of the same age who are not rachitic. It is also of interest to note that often during acute febrile conditions the child may show a rapid growth in length, notwithstanding a decided loss in weight, malnutrition evidently affecting the weight but not the height. Children grow most rapidly during the first year, and especially during the first three months of life. The average length of boys at birth is $19\frac{3}{4}$ inches (49.5 cm.), and of girls slightly less, $19\frac{1}{4}$ inches (48 cm.). During the first two years the growth is about the same for both girls and boys. From the third to the twelfth year girls grow more slowly than boys. After the twelfth year, owing to the earlier advent of puberty, the girl grows more rapidly and overtakes the gain made by the boy. The boy develops the more rapidly from the fourteenth to the sixteenth year, and again passes the girl in height.

The growth of the child during the first year is 8 inches (20 cm.), in the second year 4 inches (10 cm.), in the third year 3.2 inches (8 cm.), in the fourth year 2.8 inches (7 cm.), from the fifth to the eleventh year the child grows from 2 to $2\frac{1}{2}$ inches (5 to 6 cm.) each year, and at the age of puberty both girls and boys grow from 2 to 3 inches (5 to 7.5 cm.) each year for two or three years.

During the day the child decreases in height and gains in weight, during the night he increases in height and loses in weight.

At the age of five years the child is twice as tall as at birth, and at fourteen years three times as tall as at birth.

Insufficient food and poor hygienic surroundings usually retard both growth in height and weight; whereas acute illness often results in a growth in height, notwithstanding a loss in weight.

Weight.—A delicate, premature, or undersized infant should be weighed every day; infants of normal weight at birth should be weighed twice a week during the first six months, during the second six months once a week, during the second year every two weeks, and a careful record should be kept of these weighings. It is necessary to appreciate that a gain in weight does not always signify an improvement in the general, physical, and nervous condition. The tissues may be soft

and flabby, the bones rachitic, the child anemic and neurotic, and yet the scales indicating a gain in weight might divert one's attention from these conditions, unless care is taken to note that a gain in weight to be satisfactory must go hand in hand with normal development in every other respect.

If fed on high percentages of starches and sugars, they may gain in weight but the tissues are apt to be soft and flabby. The importance of a failure to gain in weight is evident. It signifies either that the different food elements are deficient in quantity or quality properly to nourish the infant, or that all or one of them is not adapted to the child's digestive powers; again, the child may be digesting its food properly, but some other condition, such as fever or loss of sleep, may be so increasing the metabolic processes that no gain in weight results.

In other cases the quantity and quality of the food offered may be correct, but the infant fails to take a sufficient amount. Bottle-fed babies are much more likely to develop gastro-intestinal disturbances than those who are breast-fed, and the problem of a child with stationary or decreasing weight must be studied from the stand-point of that individual child, and the different causes, such as food, the personal and family history, hygienic surroundings, gastro-intestinal or other diseases considered. The weight curve of the bottle-fed baby is usually below that of the breast-fed infant, and irregularities in this curve are more frequent.

In order to avoid errors in the weight chart a child should always be weighed in the same clothes, the fewer the better, on the same scales and at the same time of day. The lightest weight is usually reached by the third day, the loss in weight during the first three days being accounted for largely by the small amount of breast milk received by the infant and the loss of meconium and urine. During these first few days water should be given freely, and while it is not wise during this period, either from the stand-point of the child or the mother, to force it to nurse too frequently, still it is a well-established fact that the sooner the child begins to receive an abundant supply of breast milk the sooner does this initial loss of weight cease.

The presence of the colostrum corpuscles is also an element in the weight loss, as the loss of weight is largest when the colostrum corpuscles persist. The average loss during these first three days is about six to eight ounces, and this should all be regained by the end of the first week. If, after the third day, the weight still continues to fall, or even remains stationary, it points to either a deficient secretion of breast milk or, perhaps, some abnormal condition in the infant of an inherited or acquired character—among the former syphilis or some congenital malformation, among the latter sepsis and gastro-intestinal or pulmonary disease. Such a child must be studied most carefully in order as early as possible to correct or at least modify the cause of its failure to gain. This is of importance, as an unnatural loss of weight during the first few days or weeks of life is often regained very slowly and with difficulty.

The normal increase in weight in the child during the first year is remarkable. It should double its birth weight when five months old, and treble it at one year. The infant starting with a small initial birth weight has less vitality than the heavier baby, the rule being that the greater the weight the greater the vitality. Ordinarily the baby small at birth will present during its first year a weight chart with a lower curve than the one shown by the infant heavier at birth. To this rule there are, however, many exceptions, as it is quite common to find the child small at birth, if normal and healthy, gaining so rapidly in weight that at five months and one year its weight is equal to the normal weight of the infant heavier at birth. The accompanying chart (Fig. 1) shows the usual weight curve during the first two year in a child of average birth weight.
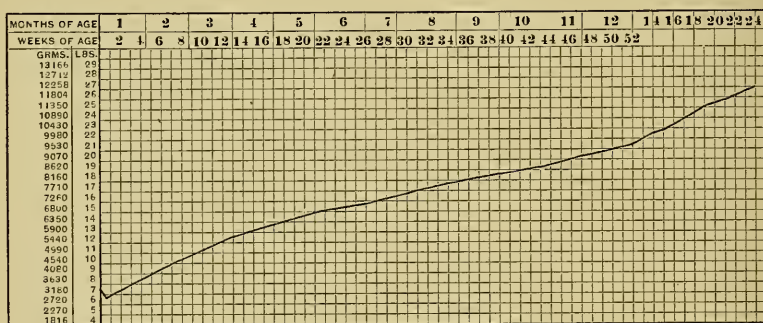
Fig. 1.—Weight chart.

This, of course, represents only the average weight. There is, however, a distinct tendency for both the light and heavy infant at birth to conform closely to this curve, provided the child is normal and healthy. This rapid gain in weight, in conjunction with the great amount of heat lost by the infant's body, necessitates an amount of food which is proportionately large for the child's age, especially when one considers the excessive metabolism of the growing baby. The production of heat required by an infant is much greater in proportion than that required by the adult, because the infant has a smaller body but a greater skin surface relatively than the adult. If this heat is not produced, the body temperature of course cannot be maintained.

The weight gained during the second year is six pounds, the third year four and one-half pounds, the fourth year three and one-half pounds, from the fourth to the eighth year four pounds each year, and from the eighth to the eleventh year six pounds each year.

The following tables give the average height and weight of both boys and girls from birth to sixteen years:

HEIGHT.

Age.	Boys.	Girls.
Birth	19¾ inches	19¼ inches
1 year	28½ "	28 "
2 years	32½ "	32¼ "
3 "	35 "	34½ "
4 "	38 "	37¾ "
5 "	41 "	40½ "
6 "	43½ "	43¼ "
7 "	45½ "	45¼ "
8 "	47½ "	47 "
9 "	49½ "	49¼ "
10 "	51½ "	51½ "
11 "	53½ "	53½ "
12 "	55 "	55½ "
13 "	58 "	58¾ "
14 "	60 "	60 "
15 "	63 "	61½ "
16 "	65 "	62 "

WEIGHT.

Age.	Boys.			Girls.		
Birth	7 pounds 8 ounces			7 pounds 2 ounces		
5 months	15 "			14 "	4 "	
1 year	21 "			20 "	6 "	
2 years	27 "			26 "		
3 "	32 "			31 "		
4 "	37 "			36 "	4 "	
5 "	41 "			40 "		
6 "	45 "			44 "		
7 "	49 "			48 "		
8 "	54 "			52 "	8 "	
9 "	60 "			58 "	8 "	
10 "	66 "			64 "		
11 "	72 "			70 "	4 "	
12 "	79 "	12 ounces		80 "		
13 "	88 "			90 "		
14 "	97 "			100 "		
15 "	108 "			108 "		
16 "	119 "			113 "		

The child who is increasing normally in weight is twice as heavy at the age of six years as it was at one year, and at the age of thirteen years has doubled the weight of six years.

The failure of an infant to gain in weight is often the first indication to the physician that the child is not developing normally, and should be a sufficient reason for making a careful examination of the infant's food and life; if the examination is successful the cause of this failure to gain will be discovered, perhaps long before any evidence of deficiency in the quantity or quality of the food, or any signs or symptoms of indigestion or other illness have become sufficiently marked to attract attention. In fact, it is impossible for any one to decide except by the use of the scales just what progress the baby is making, and the failure to weigh the baby systematically often accounts for nutritional disorders being unnoticed and not treated sufficiently early.

With the possible exception of the first two years of life, the growth which occurs at puberty is the most important. It is the time of the

3

most rapid growth and development of the entire body, of the greatest increase in both height and weight, and at this period we find also the most rapid development in chest measurements, in lung capacity, and in the muscles of the arms and legs.

The custom of putting children to work, especially in ill-ventilated apartments, during this critical period cannot be too strongly condemned, as such children invariably show subnormal development. Statistics carefully collected on a large scale in England prove that in children who work half time in the mills subnormal development is as follows: At the age of eleven years, 7.5 per cent.; twelve years, 11.2 per cent.; thirteen years, 15.7 per cent.; fourteen years, 19 per cent.; fifteen years, 26.5 per cent. Could anyone wish a more striking example of the injurious effects of child labor?

Head.—At birth, especially if labor has been difficult or prolonged, the infant's head is often elongated in its anteroposterior diameter. This condition, the result of pressure during labor, is usually of no pathological significance, and, as a rule, disappears in the course of a few weeks. The larger the child the larger should be the head; and, while a certain amount of variation from the normal may be of no special importance, still an infant's head considerably above the normal size should at least suggest the possibility of hydrocephalus, rickets, or possibly cretinism.

If the infant is much below normal weight its head will, of course, be smaller than normal, and this is the case in premature infants, although in premature infants the head is large in proportion to the thorax. A head much smaller than normal in a well-developed baby at full term should suggest microcephalus, and possibly mental deficiency. In deciding as to the probability of the head being of normal size, it must be borne in mind that the maximum circumference of the head and of the thorax during the first two years of life correspond very closely. After this period the chest increases more rapidly in size, as is shown by the following table:

	Circumference of head	Circumference of thorax
At birth	13.5 inches	12.5 inches
6 months	17.0 "	16.5 "
1 year	18.0 "	18.5 "
18 months	18.5 "	19.0 "
2 years	19.0 "	19.5 "
5 "	20.5 "	21.5 "
10 "	21.0 "	25.0 "
15 "	22.0 "	30.0 "

The anterior fontanelle always exists at birth, and may vary in size from one-half to two inches laterally, and from two to three inches longitudinally. It changes very little in its diameters until the ninth month, when it gradually begins to unite, and is usually entirely closed by the eighteenth month. If the child is robust, and the head of normal size, the early closure of the fontanelle usually is of no significance. The failure of the suture to close until a few months later

than normal may simply indicate malnutrition; but, if the head is unnaturally large, may point to rickets, hydrocephalus, or cretinism.

Owing to the lack of ossification the bones of the skull in the infant are very soft and the skull may even become misshapen by allowing the infant to lie continually on one side.

The principal sutures of the head are, as a rule, ossified at the seventh month, although a delay of one or two months is of no special significance.

Neck.—A casual examination gives one the impression that the neck of the infant is short. In reality the cervical portion of the bony spine is longer proportionately than in the adult. The apparent shortness is due to the high position of the sternum, the large deposit of superficial fat in this region in the infant, and the failure of the young child to hold its head erect.

Spine.—In the young infant the spinal column presents the sacral curve and a long posterior curvature or convexity extending above the sacrum. The normal spinal curves develop as age advances, the curve in the cervical region appearing when the baby is able to hold its head erect, and the curves in the lumbar and dorsal spine developing when the child is able to walk. The spinal cord descends to the third lumbar vertebra in the young infant, a point lower by one vertebra than its lowest point in the adult.

The spine in the infant and young child, owing to the large amount of cartilage and comparatively small amount of bone cells, is very flexible, and can therefore bend or be bent to a degree much greater than is possible in the adult. Its greater flexibility and lack of ossification render it more susceptible to slight and temporary influences of an injurious nature than is the case with the adult bony spine, and predispose the infant and young child to spinal deformities. A line drawn between the anterosuperior spinous processes of the ilia passes over the spine of the fourth lumbar vertebra, and is a useful landmark in lumbar puncture.

Thymus Gland.—The thymus develops during fetal life from the third visceral pouches on either side, and thus forms a bilaterally symmetrical tubular organ stretching out between the thyroid and upper limits of the heart. The epithelial elements are more and more encroached upon by surrounding vascular and lymphatic tissue, so that at birth the gland consists largely of the latter, containing only epithelial remnants in the form of so-called Hassal's corpuscles. The thymus gland grows up to the second or third year of life, when its weight varies between 7 and 27 grams (0.2 and 0.9 of an ounce); Friedleben gives it at 27 grams as compared with 14 grams (0.5 of an ounce) at birth. Atrophy of the thymus begins after the second or third year and progresses slowly until puberty. From puberty to early adult life the atrophy progresses rapidly, and at adult life very little thymus tissue remains.

The thymus consists usually of two flat slender lobes joined in the middle by delicate connective tissue. It moves up and down with

respiration. At birth it is of a faintly red color, moderately soft, and loosely enclosed in a thin capsule which in its turn is closely connected with the surrounding structures, the trachea, the arch of the aorta, the pulmonary artery, the superior vena cava, both innominate veins, the recurrent and pneumogastric nerves, also the upper part of the epicardium and the apices of the lungs. The gland usually reaches up into the episternal notch, but not necessarily so, even when enlarged. Its close proximity to so many important organs, and the fact that the distance between the manubrium and the vertebral column in infants is little more than 2 cm., accounts for the very serious disturbance which may arise from an abnormal enlargement of the thymus.

The thymus is a ductless gland supplied by the vagus and sympathetic nerves. Researches as to its function are not yet conclusive. During the last fetal months it plays a part in the making of blood. It is claimed also that it has an internal secretion similar to that of the thyroid which influences the general growth and development of bones, and perhaps also the blood-pressure. Complete extirpation of the gland in dogs has resulted in osteoporosis, osteomalacia, and rachitis, possibly as a result of acid intoxication which has interfered with a deposit of lime salts in the bones or has dissolved some already deposited.

The condition of the thymus gland seems also to be an index of the general condition in infants, as in 18 cases of marasmus the average weight of the gland was found at autopsy to be only 2.2 grams (33.9 grains) as against 18 grams (three-fifths of an ounce) in the normal. As a possible explanation of *idiotica thymica*, it seems interesting that Bourneville at postmortem found the gland absent in 25 out of 28 mentally weak children.

Thorax.—The chest of the child is barrel-shaped and gradually changes to a dome-shape at puberty. The anteroposterior and transverse diameters are nearly equal at birth, but after the third year the transverse increases more rapidly up to puberty than the anteroposterior. The lungs, on account of the shape of the chest, lie more posteriorly than in the adult. The walls are very elastic, as the spine, ribs, and sternum are largely cartilaginous. The chest walls are also thinner, owing to their slight muscular development; and in normal infants the superficial tissues are composed largely of fat. The high position of the diaphragm, the large thymus, and possibly dilated stomach and bowels tend to lessen the size of the thorax.

In newborn infants the maximum circumference of the chest is about three-fourths to one inch less than the maximum circumference of the head; at six months it is one-half inch less than the head; but at one year the thorax not only equals the head in size, but its maximum circumference is now one-half inch greater than the head. During the first three years the difference in the size of the head and thorax is very slight, a fact which is often of assistance in suspected microcephalic or hydrocephalic conditions. A very fat child will, of

course, have a large chest; on the other hand a small thorax should always be looked upon as an indication of imperfect development, and should suggest the necessity for regulated calisthenics. Rickets, empyema, emphysema, cardiac disease, and, in older children, Pott's disease or lateral spinal curvature, cause more or less thoracic deformity. "Trichter-brust" is a funnel-shaped depression at the lower end of the sternum. It may be congenital or the result of rickets; in the former case the lower end of the sternum is very much retracted; in the latter other evidences of rickets are usually present.

If after the second year the chest is smaller than the head, it may indicate that the head is enlarged, as in rickets or hydrocephalus, or the unnatural smallness of the chest may be the result of disease of the lungs or of some interference with respiration, such as may be produced by adenoids. A long, narrow, and flat chest suggests the tubercular type, while a pigeon-shaped breast points to rachitis, an asymmetrical chest suggests pleural effusion, pneumothorax, or scoliosis, while precordial bulging may indicate heart disease.

From birth until the seventh year the most noticeable change in the thorax is the increase in its tranverse diameter; during these years the chest also greatly increases in its vertical length, while from the sixth to the ninth year the vertical diameter develops more rapidly than the transverse. At puberty there is again a rapid increase in the vertical diameter, especially in boys. The infant's shoulders are small and the ribs more nearly horizontal than in the adult. By the end of the second year the sternum has a number of centers of ossification, but is still largely cartilage; the manubrium and gladiolus are fairly well ossified, while the ensiform cartilage ossifies more slowly.

Fetal Circulation.—The circulation *in utero* is carried on without any oxygenation of the blood by the lungs of the fetus. The oxygenated blood from the placenta, carried by the umbilical vein, enters the fetus at the umbilicus, and then passes to the under surface of the liver of the fetus where it gives off two or three branches to the left lobe. Farther on, at the transverse fissure, it divides into two branches, the larger of these joining the portal vein and entering the right lobe; the smaller, the ductus venosus, joining the left hepatic vein at the point where the latter empties into the inferior vena cava. All the blood entering the fetus by the umbilical vein except that portion which passes through the ductus venosus traverses the liver, and empties by the hepatic veins into the inferior vena cava.

The inferior vena cava, which contains also the blood deoxidized by its passage through the lower extremities, empties into the right auricle, and, guided by the Eustachian valve, passes through the foramen ovale into the left auricle. Here it mixes with the small quantity of blood which, having served to nourish the lungs, is emptied by the pulmonary veins into the left auricle. The blood passes from the left auricle through the mitral orifice to the left ventricle, and from the left ventricle through the aortic valves into the aorta, and is in large part conveyed by the carotid and subclavian arteries to the head

and upper extremities which, thus receiving the major portion of the oxygenated blood, are especially well-developed at birth. A small portion of this blood, however, passes into the descending aorta.

The blood from the head and upper extremities is returned by the veins to the superior vena cava and into the right auricle, where it mixes with a small portion of the blood emptied into the right auricle by the inferior vena cava, the major portion of this latter blood having passed directly through the foramen ovale into the left auricle. The small portion of blood from the inferior vena cava that does not pass through the foramen ovale, mixing with the blood that enters the right auricle by the superior vena cava, passes through the tricuspid orifice into the right ventricle, and from the right ventricle into the pulmonary artery.

As the lungs require only a small portion of blood to nourish them, the amount distributed to them by the right and left pulmonary arteries is not large, and, after performing its function of nourishing the lungs, it empties into the left auricle by the pulmonary veins. The major portion of the blood entering the pulmonary artery passes through the ductus arteriosus into the descending aorta, mixing with the small quantity of blood which passes into the aorta from the left ventricle. This blood containing a small proportion of oxygen passes down and supplies the viscera of the abdomen and pelvis and the lower extremities, the major portion of it, however, being conveyed by the umbilical arteries to the placenta, where it is again oxygenated. The small portion of blood passing to the lower extremities explains their comparatively small size and lack of development at birth.

The important points which one should remember in the infant and fetal heart are: (1) the opening between the two auricles, and (2) in connection with this opening the large size of the Eustachian valve. Situated as it is on the left side of the opening of the inferior vena cava, it serves to guide the blood through the foramen ovale from the right into the left auricle.

The heart of the newborn occupies a vertical position until the fourth month. After this period it gradually assumes an oblique position. Its size as compared with the body is at birth as 1 to 120, at the second month as 1 to 50, and in adult life as 1 to 160. Early in fetal life the auricles are larger than the ventricles, the right auricle being larger than the left. Near the end, however, of intra-uterine life, the ventricular portion becomes the larger, the left ventricle as the period of birth approaches becoming thicker than the right.

As soon as the child breathes, an increased amount of blood from the pulmonary artery passes into and through the lungs, and they at once assume the function of oxygenating the infant's blood.

After the fifth month of intra-uterine life, the lumen of the ductus arteriosus gradually decreases, and its rapid obliteration as a blood-vessel is therefore quickly accomplished after birth. At birth only a small portion of blood passes through the ductus arteriosus, and its lumen, already considerably lessened, quickly becomes obliterated,

no blood passing through it after the sixth to the tenth day. The foramen ovale rapidly closes and becomes practically impervious to the passage of blood at about the tenth day after birth. A small slit-like opening, however, usually persists for some months; in fact, some weeks before birth the foramen ovale gradually becomes smaller as the result of a septum which is slowly closing the orifice. With the ligation of the cord the blood ceases to flow through the umbilical arteries, clots form in each artery, and, becoming organized, result in the closure of their lumina. The umbilical vein is also obliterated, having become the round ligament of the liver. The time required to complete the circulation in the infant is at birth twelve seconds, at five years of age fifteen seconds, at fourteen years eighteen seconds; in the adult it is twenty-two seconds.

Pulse.—The frequency of the heart's action is shown in the following table:

	Pulse.
Before birth	150
At birth	130 to 140
First year	115 to 130
Second year	100 to 115
Third year	90 to 100
Fourth to seventh year	85 to 90
Seventh to fourteenth year	80 to 85

The pulse, if possible, should always be taken when the infant is very quiet, or, better, if asleep. Under perfectly normal conditions its rate varies much more than in the adult, and the slightest movement will increase the rapidity of the heart's beat; the more violent the movement or excitement the greater will be the increase. An increase of from twenty to thirty beats in the minute may easily follow any excitement or unusual exertion, the slightest cause being enough to disturb the rate and force. The rapidity of the pulse is of less significance than its force. An infant ill from any cause may have a pulse of 150 to 175 or even higher and be in no danger, while the same pulse rate if lacking in force would be of much more serious consequence. The pulse wave of a healthy infant shows dicrotism, which becomes more marked in cardiac disease and in all acute infectious diseases.

Respiration.—Respiration is more rapid in infancy and early life than in the adult, although it tends to approach the adult type earlier than does the pulse. The rhythm during infancy varies greatly, even in the perfectly normal and healthy baby. Respiration in the infant is of the abdominal type, the muscles of the chest being poorly developed, while the muscles of the abdomen and diaphragm are well developed. The abdominal or diaphragmatic type of respiration persists until about the tenth year in girls and the eleventh year in boys, when it gradually tends to change to the costal type. In the infant respiration may for a few moments be quite superficial, perhaps changing a few seconds later and becoming deep. The period between inspiration and expiration also often varies. While a disturbance of the

respiratory rhythm is of no significance in infants, and, in fact, occurs regularly in the normal infant, it is of importance to appreciate that this is a phenomenon of infancy only. In the child of two years or older a disturbance of the rhythm or the rapidity of respiration often suggests the possibility of brain or lung disease. Comparatively slight causes—a moderate fever, excitement, violent crying, or slight muscular exertion—will in the infant cause not only a change in the rhythm but also a marked increase in the respiratory rate.

The frequency of the respiration is shown in the following table:

At birth	35 to 50 per minute
At first year	28 "
At 2 years	25 "
At 5 "	22 "
At 10 "	18 "

Temperature.—The temperature of an infant or small child should always be taken in the rectum; the temperature so taken is more reliable than that in the axilla or groin, and, owing to the liability of breakage, the method is safer than when taken in the mouth. The heat centre in the young is evidently not fully developed, and slight causes easily disturb the equilibrium of the temperature. It is almost invariably higher in infants than it would be in adults, the two suffering from the same disease. Infants produce more calories in proportion to the body weight than adults, but give off more heat in proportion to their size. They are, therefore, less able than adults to stand extreme cold.

The temperature of the infant falls from 1.5° to 2° during and after the first bath, and requires from twelve to twenty-four hours to return to the birth temperature. In weak or premature infants the fall may exceed 1.5° to 2°, and the temperature may with difficulty return to the original temperature at birth. This, of course, is a strong argument for the omission of the bath in such children, and, in addition, usually indicates the necessity for the employment of external heat.

Slight variations in the temperature invariably occur even in normally developed and healthy infants, and, if not exceeding 0.5° F., are of no significance. The bottle-fed baby ordinarily shows a more irregular temperature than the infant raised at the breast. During sleep the thermometer often registers from 0.2 to 0.5 of a degreee less than when the infant is awake and active. Very slight causes or mild illness will often produce in the infant or young child an elevation of from 1° to 3°. Excitement, unusual exertion, crying, or too many clothes on infants in hot weather will often cause a similar rise.

If the temperature remains continuously high it usually indicates some serious illness. An intermittent temperature may or may not be associated with illness of much consequence. It is extremely important, however, to appreciate that, while variations in temperature in the infant may be and often are produced by slight causes, one must not go to the other extreme, and become careless as to the danger which may be associated with such changes.

Fever may occur as a part of many of the illnesses from which an infant or young child is suffering, and yet the symptoms of this illness may be vague and uncertain. The fever occurring in measles previous to the appearance of the rash, the temperature during the first few days of typhoid, and the fever associated with otitis media may be of great significance, and yet the symptoms be, possibly, very obscure. The broad rule of never making a diagnosis until the child has been carefully examined from head to foot, not forgetting the throat and skin, is often the only means of deciding as to the significance of a fever.

Subnormal temperatures are sometimes the result of allowing the thermometer to remain too short a time in position, or from being taken in a moist axilla. In premature or delicate infants also the temperature is often below the normal, and in cases of marked malnutrition subnormal temperatures are the rule and not the exception. The temperature is often subnormal in children with congenital and acquired heart disease. In older children a subnormal temperature is associated with a number of pathologic conditions, of which may be mentioned diabetes insipidus and mellitus, Addison's disease, and myxedema.

It is an interesting fact that in many cities the maximum outdoor temperature occurs at 2 to 3 p.m., and that the maximum temperature in dwellings, that is, indoors, is almost always at its height at a considerable number of hours after 2 to 3 p.m., perhaps at 8 p.m., or, possibly, well into the night or early next morning (3 a.m.). Observations taken on a large scale in poorly ventilated and small apartments show that under such conditions the maximum temperature indoors very often occurs late in the evening or late in the night. Children sleeping in such rooms should, of course, never be bundled up even if the night air outside is cool.

Muscular Development.—While the muscles of the infant are fairly well-developed, coördination does not exist. Glycogen is present in the muscles in small amounts, about 0.5 of 1 per cent.; there is also a small amount of grape sugar. The electrical response to both galvanism and faradism is diminished at birth, and the response to electrical stimulation is more slowly elicited, the period of latency being longer. Motion is, to a degree at least, reflex, and is more or less associated with a sense of feeling or touch.

During birth the only muscle liable to injury is the sternocleidomastoid. In breech presentations a blood clot with, perhaps, a tearing of some of the muscle fibers may take place. A swelling the size of a robin's egg may be noticed, usually on the anterior aspect of the muscle; it is, as a rule, absorbed spontaneously in the course of a few weeks, although shortening of the muscle and torticollis may follow.

The hands at birth seem to show considerable muscular development, as illustrated by the strong grasp of the newborn infant. At three months the infant will hold its head erect fairly well, and at the age of six to seven months it can be placed on a large firm mattress or on a blanket on the floor, or, still better, in a pen, and left to learn

to creep. At eight or nine months old, if normally developed, it will have acquired the power to move its body from one place to another, either by rolling, pushing, or creeping. An infant should always lie on a firm mattress, and often be placed on its abdomen as well as on its back, and invariably be loosely dressed so as to allow free movements of its neck, arms, body, and legs.

At the age of ten months a child usually begins to grasp a chair or the side of its crib or pen, and to draw itself up on its knees. At the age of twelve months it should be able to walk with assistance. Failure to walk at twelve or thirteen months may be due to timidity, poor bony and muscular development, or to mental deficiency. It must be remembered that it is wise to give the infant all possible freedom of movement and exercise, and to allow it voluntarily to creep, to stand, and to walk. Unusually large and heavy children often walk late; this may be more to their advantage than otherwise, as their bony and muscular systems are less firm and resistant than normal, hence less able to support the child in the upright position.

After the ability is acquired to stand alone some children quickly learn to walk alone; others, if timid or not very robust, may not walk alone for several months after the period of walking with assistance. In my opinion it is rarely necessary or advisable to encourage or to teach a child to walk. A child normal, physically and mentally, will naturally walk as soon as it is able to do so; in fact, the tendency should be more to restrain the child than to urge it on.

Inability to grasp objects firmly in the hand at birth, to reach out for objects at the fourth or fifth month, to hold its head up at four or five months, to change its position at eight or nine months, to sit up at one year, or to stand at fifteen months, means usually that there is some physical or mental defect, and should at least make it imperative to examine most carefully into the mental as well as the physical condition of the child.

According to Schlossmann, the infant in proportion to the weight of its body performs an amount of work about equal to that of an active adult, and the food required by an infant varies as does the food of an adult in direct proportion to the amount of muscular work performed.

Nervous System.—Until the last few months of fetal life the cord extends to the end of the cervical canal. At birth, owing to the more rapid growth of the bony spine during the last few months of intrauterine life as compared with the cord, the end of the clonus medullaris reaches to the third lumbar vertebra. In the adult the cord terminates at the lower end of the first lumbar vertebra. The average weight of the brain in the healthy newborn child is 12 to 13 ounces (350 to 370 grams), and increases very rapidly, in the first nine months of life attaining one-third of its maximum weight, another third at about the age of two and a half years, and its final weight at about the twentieth year. The frontal lobes of the brain of the newborn are poorly developed, and the island of Reil is less distinctly outlined than later in life. Heredity affects especially the weight of the cerebrum.

The cerebellum averages two-thirds of an ounce (20 grams) in weight at birth, and when fully developed about five ounces (140 grams). At the age of six months its weight increases to about two ounces (60 grams), and at two years to three and one-half ounces (100 grams). Its subsequent growth is much slower and its full development of five ounces (140 grams) is reached at about the same time as that of the cerebrum, 20 years. The weight of the boy's and man's brain is greater than that of the girl's and woman's brain at corresponding ages.

The spinal cord of the newborn averages about one-tenth of an ounce (3 grams) in weight, and in the adult about nine-tenths of an ounce (27 grams). It weighs two-tenths of an ounce (6 grams) at five months, three-tenths of an ounce (9 grams) at one year, and four-tenths of an ounce (12 grams) at the age of two years.

The excitability of motor nerves and muscles is very faint during the first two months, and the sensory nerves respond very slightly to electrical stimulation during this period, the face at birth being absolutely irresponsive to this stimulation. At birth the ganglion cells lack all the characteristic adult features, this being most marked in the cerebral hemispheres. Pigment is absent at birth and does not develop in certain portions of the nervous system until years afterward. At the age of one year brownish pigment is deposited in the locus cæruleus. The pigment of the vagus and substantia nigra begins to appear at the fourth year, and at the sixth pigment is found in the posterior spinal ganglia, but does not appear in the spinal cord until the seventh year.

Many nerve fibers do not develop their myelin sheaths until after birth. This is most marked in the cerebral hemispheres; for while at birth the full proportion of myelin is present in the spinal cord, the cerebrum, cerebellum, medulla, pons, and quadrigeminal bodies possess absolutely no myelin.

In the fetus of eight months the tactile and muscle sense tracts are the only ones supplied with myelin sheaths. At full term the pyramidal tracts, the olfactory, the visual tracts, and the corona radiata possess more or less of their myelin sheaths. The lack, or almost total absence, of brain function at birth is quite likely due in great part to the absence of myelin in large brain areas. The tendon reflexes are present in the premature infant, and during the first year of life are more marked than in the adult. After the infant is ten days old the cutaneous, abdominal, and the plantar reflexes are very active. The Babinski reflex is normally present during the first six or eight months of life.

Special Senses.—The pupils of the newborn respond normally to light, but the infant is not believed to have at birth the power to fix objects, and the power of accommodation does not appear until one month of age. The reflex closing of an eyelid at the approach of a finger is well developed at birth, but the true optical reflex of winking is not seen until about the seventh week. At the age of one month the child is capable of fixing its eyes upon an object, and at four months can follow a moving object with its eyes.

In children born before full term the swelling of the Eustachian tube often prevents the development of hearing for some days. Although the newborn infant cannot hear, the ability to do so develops within a few hours to several days after birth, hearing depending upon the time of entrance of air through the Eustachian tube into the internal ear.

The sense of taste develops early, as is evidenced by the fact that a very young infant will often detect slight differences in its food; or will, perhaps, refuse water when accustomed to having a small amount of sugar added thereto.

The sense of smell is little if at all developed at birth, but develops rapidly in the first few days of life. The sense of touch is one of the first to appear, and is always present at birth, the tactile tracts being supplied with myelin at the eighth month of fetal life. Appreciation of touch is especially noticeable in the lips, the introduction of any object into the mouth being usually sufficient to produce the reflex act of sucking. The newborn infant does not feel pain, is quite insensitive to the prick of a pin, and is probably entirely unable to appreciate the difference between external heat and cold, although heat and cold, as in the douche or plunge, exert a reflex influence. Sucking, swallowing, and ocular movements at birth probably do not depend upon any conscious mental effort, but are largely or entirely inherited reflexes.

Speech begins to develop at about the tenth month, varying more or less in different children, and depending to a certain extent on the child's surroundings and management, but the tendency to induce infants to talk is certainly not to be encouraged.

Stomach.—When the stomach is empty the pylorus occupies the lowest position, being found in the continuation downward of the midsternal line, or slightly to the left of this line. The cardia occupies a position on the left of the tenth dorsal vertebra, and is about one to one and a half inches (2 to 3.5 cm.) above the pylorus. The fundus is usually only fairly well developed at birth, and the capacity of the stomach at different ages is represented in the following table:

At birth	1 ounce	30 c.c.
At two weeks	2 ounces	60 c.c.
At one month	3 "	90 c.c.
At two months	3½ "	100 c.c.
At three months	4 "	110 c.c.
At four months	4¾ "	125 c.c.
At five months	5¼ "	140 c.c.
At six months	6 "	160 c.c.
At seven months	6¾ "	180 c.c.
At eight months	7¼ "	200 c.c.
At nine months	8¼ "	225 c.c.
At ten months	9 "	250 c.c.
At eleven months	9½ "	275 c.c.
At twelve months	10 "	290 c.c.

The capacity of the stomach, of course, varies normally within considerable limits. The capacity of the infant's stomach at different ages is best obtained by estimating the average amount of milk in

ounces usually taken by a child of a certain age or size. Children of the same age often differ greatly in size, and the larger the child the larger, as a rule, is the stomach. In deciding upon the amount of food to be given to a child at a single meal it must be remembered that a portion of the food taken passes out of the stomach during the swallowing of the meal, and that the tendency of the stomach is to empty itself of any food remaining from a previous meal as soon as the food of a subsequent meal is introduced, the rapidity with which this is accomplished depending largely upon the composition of the food and its amount. If a milk mixture contains a high percentage of fat, it tends to pass more slowly out of the stomach than a mixture with less fat and more protein and sugar.

In the young breast-fed infant the stomach empties itself in one and one-half to two hours, this depending upon the size of the meal. An infant fed on the bottle will take an hour longer to empty its stomach than does the breast-fed baby; that is, two and one-half to three hours. The normal reaction of the stomach at birth is neutral or acid, but is always acid after taking either breast or cows' milk, owing to the secretion of hydrochloric acid. This is present one and one-half to two hours after a meal in 0.1 per cent. solution in healthy breast-fed infants. In bottle babies free hydrochloric acid may not be found, owing to the greater ability of cows' milk to combine with acids. The contents of the stomach of the breast-fed baby, containing more free hydrochloric acid, are therefore more antiseptic than in the bottle-fed. Lactic acid is also present in the infant's stomach during digestion. A fat-splitting ferment is also found, but its presence is probably of comparatively little importance. The milk, sugar, and water are the first elements of the food to pass through the pylorus, and are followed in turn by the albuminoids and the fats. Only a small amount of salts, sugar, and protein are absorbed from the stomach. Rennin is normally present in the stomach, and produces coagulation of the casein, the hydrochloric acid dissolving the curds. The coagula of cows' milk are larger and firmer than those of human milk, and are dissolved with more difficulty than are the lighter curds of human milk.

Intestine.—The length of the intestine in the newborn is about seven to twelve feet (200 to 400 cm.). The muscular coat is, as a rule, poorly developed, in the infant, whereas the solitary follicles and Peyer's patches are relatively better developed. According to Orban and Weinland, a ferment lactase is found in the secretion of the small bowel capable of decomposing lactose.

The pancreas of the newborn has a moderate diastatic action, as well as a distinct fat and protein-splitting function.

CHAPTER III.

THE CLINICAL EXAMINATION OF SICK CHILDREN.

In examining an ill child, it is absolutely essential never to hurry. It may be necessary to coax, amuse, or divert an infant; but the primary idea is not to frighten it, and the presence of any stranger in the room is often sufficient to upset the delicate nervous organism of an infant or even an older child. The early portion of the visit should be taken up by questioning the mother or nurse, first carefully eliciting the family history, subsequently the personal history of the child, and then the history of the present illness.

Family History.—The family history should include the possibility of syphilis, tuberculosis, alcoholism, or rheumatism; the number of pregnancies; the number of children living and in health; or, if sick, from what they are suffering; the number of children dead and the causes of death. The hygienic conditions of the child's previous environment should be inquired into. Has it been an open air or a coddled baby? Has it been recently exposed to any communicable disease? At what age was the first tooth cut, and how many teeth has it now? When did the child creep, stand alone, and walk? Was the child asphyxiated at birth, did it breathe and cry immediately after birth, or were efforts to resuscitate it necessary? How long was it breast-fed, wholly or in part, and what exactly was the strength of the modified milk mixtures or other food given it? What has been its diet since weaning? If any records of the child's weight have been kept, they should be carefully studied.

All this information may be secured in a few minutes, and included in the child's history at the first visit; the few minutes so employed are not wasted; once obtained, it becomes a permanent record, and is often of paramount importance in establishing a past or present diagnosis. Furthermore, during this time the infant or older child becomes accustomed to the physician's presence.

Inspection.—If possible the child should always be examined when asleep. The natural instinct of the mother or nurse is immediately to arouse the child upon the physician's arrival. Much information may, however, be secured by observing the child while he is asleep, and by studying the facial expression, which may indicate a quiet, relaxed, nervous system, or pain and tension. The child's mental powers can be gauged and compared with well-known standards.

Note the child's general development, the appearance of the head, whether hydrocephalic, microcephalic, or asymmetrical. Observe whether there is a bulging or sunken condition of the anterior fontanelle.

The child's color should be noted; cyanosis may suggest congenital heart disease; puffiness of the eyes a possible acquired endocarditis or nephritis; pallor an anemia, probably secondary. Is the face pinched and toxic, or is it of normal contour and good color? Inspection may disclose the absence or presence of enlarged glands of the neck. It may also show any abnormality of development in the contour of the abdomen and chest. A bulging in the precordia may suggest cardiac hypertrophy, or a systolic dimpling a previous pericarditis with adhesions.

The development of the upper extremities should be compared with that of the lower, and the comparative size of the head and thorax noted. The eye should be examined as to the presence or absence of jaundice, also strabismus, nystagmus, or ptosis. The pupils are examined as to their size and their reaction to light. The history should include the presence or absence of pain, discharge, or odor in the ears. Many cases of restlessness, sleeplessness, and high fever may be in this way cleared up. Any nasal discharge should be noted. In very young children this may suggest syphilis, especially if it be persistent and blood-tinged. In an older child a nasal discharge may mean diphtheria; or, if it recurs frequently, possibly adenoids. If the discharge is recent, one-sided, and persistent the presence of a foreign body should be suspected.

The respiration should be carefully observed as to whether it is rapid, or slow and irregular. Pneumonia may be suggested by the former and meningitis by the latter. Adenoids will be thought of if the child is a mouth breather, or if it snores. If there is cough (and in a child under two years of age there is, of course, no expectoration) a specimen of sputum may be obtained by passing a curved probe wrapped with cotton well back into the pharynx. Rachitis should be looked for in the spine, thorax, and extremities; the presence or absence of spinal or joint deformities which are not rachitic may assist in forming a diagnosis of tuberculosis.

The posture of the child is sometimes significant. Continual lying on one side is suggestive of pleurisy with effusion. Lying immovably on the back with the legs drawn up points to peritonitis. Sitting up in bed with the head thrown back indicates laryngeal obstruction. An eruption or desquamation may suggest one of the exanthemata; or, if desquamation alone exists, recent scarlet fever or syphilis. Syphilis may also be indicated by disease of the matrix resulting in suppuration and exfoliation of the nail. The dorsum is arched, and the nail appears as if it had been pinched by a pair of forceps, *i. e.*, claw-shaped. Inspiratory dyspnea is shown by retraction of the suprasternal notch, the supraclavicular and intercostal spaces, and retraction of the abdomen. It should be noted whether the abdomen is distended or retracted. Cyanosis is suggestive of congenital heart disease, especially if combined with clubbing of fingers and toes.

Palpation.—Palpation should always precede percussion, and usually precedes auscultation. The anterior fontanelle should be palpated.

Is it large or small for the age of the child, and does it bulge or is it tense? Are the cranial sutures open or closed? In early infancy, if the child is emaciated and marasmic, the posterior portion of the head may show the presence of craniotabes (thinning of the infantile skull in spots) which suggests rickets or lues.

Palpation may reveal the presence or absence of enlarged glands of the neck, or change in the size of the thyroid. An increase in tactile fremitus would suggest lung consolidation, pneumonic or tuberculous, while a decrease might indicate pleural effusion. Palpation may disclose the existence of a cardiac thrill or murmur, and an enlarged or displaced heart can be diagnosed by displacement of the apex beat. The apex beat in the newborn infant may be felt higher than in the adult, this being partly due to the higher position of the diaphragm. On account of the greater breadth of the heart as compared with that of the chest, the apex is external to the mammary line, and remains so until the fourth year, and from this time to the ninth year it is in or near the mammary line. During the first year the apex beat is found in the fourth and, subsequently, as a rule, in the fifth intercostal space. A feeble apex beat does not in itself prove anything; it is sometimes hardly perceptible, even in healthy children, but the case is entirely different if it becomes weak or disappears during the course of some disease, when it indicates either imminent heart failure or the formation of pericardial exudation.

The normal liver extends about one inch below the free border of the ribs, and if it is enlarged the increase in size may be disclosed by palpation. The spleen, if of normal size, is not palpable; hence, if demonstrable by palpation it must be enlarged. Bimanual palpation of the abdomen, the child lying on its back with the knees flexed, may enable one to recognize foreign masses in the abdomen; or, better results can sometimes be secured by digital examination per rectum. Bimanual examination with the finger in the rectum is often of advantage. By this method we may detect abdominal growths, enlarged glands, or a calculus.

Dropsy in the face, abdomen, or extremities may be determined by palpation. Kernig's sign, *i. e.*, inability to extend the lower leg if the thigh is flexed on the abdomen, and Babinski's reflex, *i. e.*, extension of the great toe with spreading of the adjacent toes upon stroking the sole of the foot from the heel to the toes, preferably on the inner side, with a moderately sharp instrument such as a toothpick, may be of more or less assistance in confirming a diagnosis. It must be noted that the Babinski reflex is of value only in children over one year of age.

Auscultation.—Auscultation in the child should, as a rule, follow palpation. Sometimes, however, it will be advisable to perform auscultation before palpation. Some children may be more willing to submit to the former than to the latter. It is well to auscultate the posterior surface of the chest first, and to use a stethoscope with a small bell when auscultating the axillæ.

Percussion.—Percussion follows auscultation. It should always be performed gently, the posterior aspect before the anterior. On comparing the two sides of the chest, the area of relative cardiac dulness and the area of absolute cardiac dulness in the infant and older child may be mapped out. The area of relative cardiac dulness is at the upper boundary of the second interspace, or the lower border of the second costal cartilage, at the left margin of the sternum. From this point the line of dulness extends in a curved direction outward and downward, the extreme left limit being at or slightly beyond the mammary line in the fourth interspace. On the right side, the line of dulness extends downward from the second interspace in a slightly curved direction along the parasternal line. The lower border is undeterminable on account of the liver.

The area of absolute or superficial cardiac dulness is that part of the heart not·covered by the lung, resembling in shape the same area as in the adult, but being relatively longer. Its upper limit is the upper border of the third intercostal space, sometimes the third costal cartilage; it extends to the left to a point between the parasternal and the mammary lines, and to the right as far as the left border of the sternum.

Percussion enables one to detect the presence of fluid in the abdomen, and auscultatory percussion (percussing in the ordinary manner and listening over this area with a stethoscope) is often of advantage in pulmonary, cardiac, and abdominal conditions.

Mensuration.—Mensuration enables one to compare the size of the head, thorax, and abdomen, although it must be remembered that temporary conditions often produce marked variations in abdominal measurements. The circumference of the head in comparison with that of the thorax, from birth up to fifteen years of age, has been tabulated on page 34.

The length of the child is of importance. Infants and younger children are best measured by placing them in the recumbent posture, with a card or book vertically at the head and feet, then measuring the intervening space. For older children a measuring rod placed against the wall or the ordinary measuring rod that is attached to weight scales may be employed.

The cry of the child may be of more or less assistance in locating the seat of the disease; the cry of pain may be sharp, acute, and accompanied by some attempt at localization, with contraction of the features; whereas young infants may be only restless and irritable. The cry of fright, as when a stranger enters the room, begins when such a person arrives and ceases when he leaves. The cry of pneumonia is short and catchy, *i. e.*, an expiratory grunt; that in laryngitis is croupy; in extreme exhaustion or marasmus we hear only a feeble whine. A moaning cry suggests intestinal disease. In scurvy the child's cry is sharp, and occurs especially when it is touched.

CHAPTER IV.

INFANT MORTALITY.

The modern tendency in medicine is to concentrate each individual's efforts within comparatively narrow limits; if possible, to add some new facts or theories to present knowledge. The sum total of all the additions and advances has revolutionized medical knowledge in the last twenty years. Countless intelligent and zealous physicians working along different lines and in special departments of medicine have made it difficult, if not impossible, for any one to keep fully abreast of the times in all branches of medical science.

Perhaps this tendency in modern medical study has led to the neglect of certain broad principles of hygiene, diet, fresh air, bathing and general methods of living; these, being every one's business, have become no one's business.

John Gardner, surgeon, London, wrote in 1838 an interesting pamphlet on "Why So Large a Number of Children Perish." He appreciated certain physiological differences between a child and an adult, and under "Dentition" wrote:

The true nature of the effect of this natural process on the health and life of children is much misapprehended. In a healthy body, the teeth are always cut without suffering, and not far wide of the ninth month. The passage of the teeth through the gums produces a slight excitement, which is not a deviation from health.

Benjamin J. Crew, of Philadelphia, wrote in 1882 an excellent article on "The Care of Deserted Infants," which was read before the assembly meeting of the Philadelphia Society for Organizing Charity in March of that year. In this article he strongly advocates a combination of "placing out" and "asylum plan" for these infants, and quotes statistics which clearly prove how great is the reduction in the mortality in infants under the plan of treatment advocated.

J. Brendon Curgenven, M.R.C.S., London, 1867, writing on "The Waste of Infant Life," states that the excess of infantile mortality occurs in laboring people. The poorer and lower classes show a mortality of 35 to 55 per cent. under the age of five years; the educated and well-to-do, only 11 per cent. He analyzes the Registrar General's report, and shows clearly the causes of this excess of 24 to 44 per cent. of deaths. Reference will be made to these statistics later.

In an article written by Dr. William Farr, over thirty years ago, he stated that the mean annual death rate of infants under one year in some of the principal countries of Europe was as follows: Out of one thousand infants there died yearly in Sweden 141.8; in Denmark, 137.5; in England, 182.6; in France, 223.2; in the Netherlands, 237.5; in Spain, 249.6; in Italy, 273.3.

The annual death rate in one thousand children under five years, according to the same authority, was: In Norway, 40.9; in Sweden, 51.4; in Denmark, 52.7; in England, 67.6; in Belgium, 74.9; in France, 79.2; in Prussia, 82.4; in Holland, 91.2; in Austria, 104.0; in Spain, 111.7; in Italy, 113.5. The United States census for 1910 shows that 265,000 babies died during their first year and 53,000 in their second year.

A. Brothers, B.S., M.D., in 1896, in his article on "Infantile Mortality during Child-birth and its Prevention," states that in the four years, 1889 to 1892, the total number of births in New York City was 173,126, and that during this period of four years 16,888 children born at term had died within the age of one month. Ten per cent. of the children, therefore, are lost before they reach the age of one month.

Collective statistics from sixteen European cities embracing 1,439,056 children show that 10 per cent. of those born alive die within the first four weeks of life. Eröss' statistics show that the greatest number of deaths occurred on the first day of life, and that the deaths diminish day by day. According to Eröss, 54.24 per cent. of the deaths among children within four weeks after birth are due to congenital debility.

Dr. Shaw, in the *Albany Medical Journal*, 1913, states that in the United States 300,000 babies die annually before they reach the age of twelve months. The ratio of infant mortality to births is 150 to 1000; in other words, one baby in seven dies before it is one year old.

In New York State the death rate in these infants is 121 to 1000; in Vermont, 145 to 1000; in Burlington, Vt., it is 230 to 1000; in London, England, 90 to 1000; in Great Britain, 130 to 1000; in New Zealand, 62 to 1000; in Chili, 331 to 1000; and in Russia, 240 to 1000.

Phelps states that in England and Wales, in 1912, the death rate in infants born alive was 95 to 1000.

William Moore, in a paper read before the Dublin Obstetrical Society in 1859, states that the proportion of deaths throughout England, under all ordinary conditions of life, is believed to be one in six within the first year. To parallel this proportion of mortality, we must pass on to those dying between the ages of 80 and 85.

Dr. D. Meredith Reese, of New York, reported at the meeting of the American Medical Association in May, 1857, that nearly 50 per cent. of the total deaths in large cities occurred in children under five years of age. In New York City in the fifty years, 1804 to 1853, the whole mortality was 363,242, including stillbirths, and during this period 176,043 children under the age of five years died—nearly 49 per cent. of the entire number of deaths.

M. Bertillon stated before the Academy of Medicine of Paris that in a period of ten years there had been in France 9,700,000 births; of those born 1,500,000 died within the first year of life.

Morse makes the statement that 85 per cent. of all infants who die are bottle-fed babies, and that 90 per cent. of all deaths due to diarrheal conditions occur in those who are bottle-fed.

J. Maule Sutton, M.D., of London, in 1872, drew attention to the influence exerted on infant mortality by the social status of the parents. His figures give a mortality of 77 per thousand for children under one year of age in urban population; and a lower percentage for rural population. These same districts, excluding the upper-class births, gave a mortality of 158 per thousand. He studied the infant mortality among the children of the farmers of Devonshire and Norfolk, two agricultural counties, and found it to be 95 per thousand in the farming class; the rate among the children whose parents were not farmers was 130 per thousand.

John S. Parry, M.D., of Philadelphia, in 1871, quotes Dr. A. Jacobi as saying that "of 100 infants born alive to the gentry of England (1844) there died 20; to the working classes, 50. In the aristocratic families of Germany there died in four years 5.7 per cent.; among the poor of Berlin, 34.5 per cent. In Brussels the mortality, up to the fifth year, was 6 per cent. in the families of capitalists, 33 among tradesmen and professional people, and 54 among workingmen and domestics." Quoting de Villiers, he further writes that "the mortality among the children of the workingmen of Lyons is 35 per cent., and in well-to-do families and agricultural districts it is 10 per cent."

Dr. George Reid, in 1906, in London, at the National Conference on Infant Mortality, in considering social status as an etiologic factor, divides the working class into three groups: (1) Those among whom the proportion of employed, married and widowed females between eighteen and fifty years of age reached or exceeded 12 per cent.; (2) those among whom the proportion was 6 to 12 per cent.; (3) those among whom the proportion was below 6 per cent. The decades, 1881 to 1890, 1891 to 1900, and the four years, 1901 to 1904, were studied. The infant mortality was always highest in group 1 and lowest in group 3. The average yearly infant mortality rates of group 1 were 195, 212 and 193; group 2, 165, 175, 156; group 3, 156, 168, 149. These statistics point out in no uncertain manner the fact that the infants of women employed in industrial and manufacturing plants during the time of their married life and motherhood are born into this world with less chance of battling with the problem of living than those whose mothers are not compelled to perform this kind of work. The wives of farmers may and often do perform hard work, but it is done more or less out of doors, and not in the vitiated and contaminated atmosphere of a mill or factory.

Helle examined into the social status of the parents of 170 infants dying in Graz during 1903 and 1904; 112 infants who died had very poor parents; 49 children had poor parents; 9 had well-to-do parents, and no deaths occurred among the children of the rich; the percentage of the four classes being 65.9, 28.8, 5.3, 0. The general infant mortality in Graz has markedly decreased in the last twenty years, while the mortality due to gastro-intestinal lesions does not show any marked diminution.

In Brün, a city of 110,000 inhabitants, the health statistics for fifteen years show that the general infant mortality during this time

decreased very much, while that due to gastro-intestinal lesions changed very little.

In Berlin, 1903, Newman investigated 2701 infant deaths. Where the families were in one-room dwellings he found 1792 deaths; in two-room dwellings, 754 deaths; in three-room dwellings, 122 deaths, and in larger dwellings, 43 deaths. It seems to be an established fact that the percentage of deaths among infants of the poor largely exceeds the mortality among the infants of the rich.

The hygienic surroundings of the infant—city or country life—are factors which play an important part in the sum total of infant mortality. In England and Wales, 77 per cent. of the whole population is urban; fifty years ago the population was equally divided between urban and rural districts. A considerable portion of this urban population lives in small towns, more closely resembling country than city life. In the year 1904, in England and Wales, 59.1 per cent. of the people lived in large towns of over 20,000 inhabitants; in 1801, only 16.7 per cent. lived in large towns.

It seems clear to me that this tendency to live more in large towns has much to do with the stationary infant mortality in England and Wales. City life means for the parents, often, long hours of work in a factory or mill; living in a small house in a small street, often poor food, and not uncommonly dissipation of drink and perhaps immorality.

Epidemic diarrhea is mostly a disease of large towns and cities. It can be positively stated that geological strata, character of soil and climate have nothing to do with infant mortality, nor is it entirely a question of poverty. Overwork, poor hygienic surroundings, and poor housing seem to be two powerful factors causing infant deaths. Density of population *per se* may and does mean a good deal in causing deaths in infants. Urban England has a higher infant mortality than rural England. However, in first-class modern houses, the population may in a given area be dense, but the infant death rate may be small if other factors are present, as good hygiene, food, fresh air, healthful occupations and good social status.

If a town is distinctly industrial or manufacturing, the mortality invariably exceeds that of the town where the occupations have more of an agricultural tendency. Table 1, by Newman, shows the infant mortality in the country of Wiltshire, in which there are no large towns; it shows also that even under favorable conditions the city mortality exceeds the rural mortality; it also shows the high mortality of large towns, and the mortality of England and Wales, and rural England and Wales.

TABLE 1.—INFANT MORTALITY RATES IN WILTSHIRE AND ENGLAND AND WALES, 1900–1904.

Districts.	1900.	1901.	1902.	1903.	1904.
County of Wiltshire . .	94.0	93.7	97.23	85.63	95.99
Urban districts (Wilts.) .	95.6	106.8	93.63	89.27	100.32
Rural districts (Wilts.) .	115.7	83.8	99.89	82.76	92.52
England and Wales . .	154.0	151.0	133.0	132.0	145.0
Large towns in England .	172.0	168.0	145.0	144.0	160.0
Rural England and Wales	138.0	137.0	135.0	118.0	125.0

In these two countries the highest infant mortality occurs in large towns; next, in large towns and rural districts; and the lowest mortality in rural districts.

Table 2, by Newman, shows the remarkable difference in infant mortality in three agricultural counties, five mining and manufacturing counties, and three towns where textile industries and mining are largely followed.

TABLE 2.

Age.	Of 100,000 infants born, the number surviving at each age.			Annual death rates per 1,000 living in each successive interval of age.		
	Three rural counties: Herts., Wilts., Dorset.	Five mining and manufacturing counties: Staffs., Leic.,Lancs.,W.R. Yorks, Durham.	Three selected towns: Preston, Blackburn, Leicester.	The three rural counties.	The five mining counties.	The three towns.
At birth	100,000	100,000	100,000	213	331	382
3 months . . .	94,820	92,051	90,874	75	154	· 240
6 months . . .	93,068	88,574	85,574	61	128	180
12 months . . .	90,283	83,081	78,197			

This table covers the three years, 1889 to 1891, and shows that of 100,000 infants born in the rural counties 10,000 died; in the manufacturing counties 17,000 died, and in the manufacturing towns, 22,000. An important point to notice in this table is that the town rates are most in excess of the rural rates in the later months of the first year of life, showing clearly that the congenital conditions, atrophy and immaturity, can be left out of consideration, and that the continuous ill effect of town life finally kills many children that have made a strong but useless struggle against their environment. Epidemic diarrhea plays a powerful part in this sacrifice of infant iife in those towns where textile industries, manufacturing and mining, flourish.

The deaths occurring during the first year of life are very unevenly distributed. This applies to all countries, and all statistics that I have been able to find prove it absolutely. The greatest percentage of deaths occurs in the first three months of life, and I believe that this percentage is increasing and not decreasing. In London during the years 1839 to 1844, 24,354 infants died during the first three months of life, an infant death rate of 68 per thousand. In the same city in the years 1898 to 1903, 56,963 infants died during the first three months of life, a death rate of 72 per thousand. According to Newman, there has been, in recent years, an increased percentage of infant deaths in England and Wales during the first three months, and a slight decrease in the percentage of deaths during the last six months of the first year. Newman asserts that infants die more from immaturity at the present time, and that consequently more infants begin life with less vitality than in former periods. He also states: "Children under twelve months of age die in England today, in spite of all our boasted progress and in spite of an immense improvement in

the social and physical life of the people, as greatly as they did seventy years ago."

The report of the Registrar General of England, for 1903, shows for England and Wales, 51.4 per cent. of infant deaths in the first three months; 19.9 per cent. in the second three months, and 28.7 per cent. in the last six months of the first year of life. In the year 1904, in Berlin, 53.6 per cent. of deaths of infants under one year occurred in the first three months. These deaths are, of course, distributed over the entire calendar year, and this observation consequently is not contradicted by the fact that the greatest number of deaths was in the hot months. Births occur in each month with fair regularity, and the congenital conditions which contribute so largely to this mortality in the first few weeks of infant existence are consequently distributed with fair regularity throughout the year.

Morse classifies the causes of infantile mortality as follows:

	Per cent.
Prematurity, congenital debility, congenital defects, accidents at birth	25
Acute gastro-intestinal diseases	25 ⎫ 40
Diseases of nutrition	15 ⎭
Acute respiratory diseases	25
Acute infectious diseases	3
Tuberculosis	2
Syphilis	1
Unclassified	9

The following table shows the causes of infant mortality in Philadelphia during the year 1915:

Disease.	Under one year.	One to two years.
General diseases	118	195
General diseases, non-epidemic	181	82
Diseases of the nervous system	113	17
Diseases of the circulatory system	27	8
Diseases of the respiratory system (pneumonia excepted)	145	45
Pneumonia, all forms	598	293
Diseases of the digestive system (diarrhea and enteritis excepted)	124	32
Diarrhea and enteritis	1168	213
Skin diseases	34	1
Diseases of the genito-urinary system	10	7
Diseases of the locomotor system	2	2
Malformation and early infancy	1560	9
Violence	40	25
Injuries at birth	99	. . .
Ill-defined	1	8
Totals	4220	937

During the year 1914 there were 41,063 births reported in Philadelphia and 1929 stillbirths registered as deaths. The causes of infant mortality in Philadelphia for the year 1914 were as follows:

INFANT MORTALITY—AGE PERIODS UNDER ONE YEAR.

Diseases.	Total.	Under 1 week.	1 week to 1 month.	1 to 3 months.	3 to 6 months.	6 to 12 months.
Total	4981	1125	750	839	930	1337
General diseases, epidemic . .	208	2	12	30	41	123
General diseases, non-epidemic .	179	16	25	30	47	61
Diseases of nervous system . .	81	20	11	15	20	15
Diseases of circulatory system .	10	3	1	4	. . .	2
Diseases of respiratory system (pneumonia excepted) . . .	220	11	35	45	55	74
Pneumonia, all forms . . .	733	19	80	150	173	311
Diseases of digestive system (diarrhea and enteritis excepted)	138	6	12	22	32	66
Diarrhea and enteritis . . .	1474	14	143	303	442	572
Diseases of genito-urinary system	14	2	3	6	1	2
Skin diseases	32	. . . .	10	10	1	11
Diseases of locomotor system .	3	. . . .	. . .	1	. . .	2
Malformation and early infancy	1736	946	395	206	106	83
Violence	50	6	5	12	12	15
Injuries at birth	101	80	16	5	. . .	. . . .
Ill-defined	2	. . . .	2	. . .	. . .	. . . .

Living at birth	41,063
Stillbirths registered as deaths	1,929

The added deaths from gastro-intestinal disease, occurring as they do in great excess in the hot months, cause the great increase in the total infant mortality for the heated term.

Schereschewsky gives us the following figures to show the effect of heat on infant mortality:

The number of deaths in Berlin in July and August of 1910 (a cool summer) was 1439, while in July and August of 1911, which was a very hot summer, 2050 infants died. The universally hot summer of 1911 in Europe caused a clearly demonstrable rise in the number of deaths in infants in every locality where statistics were compared.

The following table of deaths from diarrhea and enteritis shows the increase in infantile mortality during the summer months of 1914 in Philadelphia:

MORTALITY IN INFANTS UNDER ONE YEAR OF AGE.

	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Breast-fed . .	8	9	13	9	10	7	8	28	22	18	11	10
Artificially fed	21	14	15	13	16	4	155	148	104	56	36	24
No data as to feeding . .	9	18	21	17	27	50	138	161	71	73	36	34
Totals .	38	41	49	39	53	61	301	337	197	147	83	68

The outside and home employment of mothers is a factor in infant mortality that was appreciated long since, and led Sir John Simon in 1856 to state that "infants perish under the neglect and mismanagement which their mothers' occupation implies." In Dundee a large percentage of the female population of girls and married women work in the jute and hemp factories. The labor is unskilled, the wages small and the hours 6 A.M. to 6 P.M. These women and girls are, as a class, subnormal in weight and general physical development; many

of the children are born and raised in houses containing only one or two rooms, or in large tenements, where overcrowding and, usually, uncleanliness exist. In the ten years, 1893 to 1902, the infant mortality was 176 per thousand births; in 1904, out of 174 deaths, 125 were due to prematurity and immaturity, and over 49 per cent. of the deaths occurred in the first three months of life.

In England the Factory Act of 1901 states: "An occupier of a factory or workshop shall not knowingly allow a woman or girl to be employed therein within four weeks after she has given birth to a child." This is positive legislation of a far-reaching character. If the hygienic conditions of air, light and cleanliness were only adequately controlled by law in these mills or factories, and such provisions for sanitary surroundings as are needful were insisted on, much could be done to remove the injurious influences of this class of employment. Much has already been accomplished in this direction, but much still remains to be done.

In Kearsley, a town of Lancashire, of 9500 population, the infant death rate increased from 179 per thousand in 1894–1903 to 192 in 1903, and 229 in 1904; and this is due, according to J. C. Eames, M.D., medical officer of the town, to the town having "developed into more of a manufacturing district."

In Mulhouse, Mr. Dollfus, who owned a large cotton mill, established a fund to which all the married women subscribed, and he personally contributed. Each woman subscribing received from the fund sufficient for her support during the two months following her confinement. On resuming work at the end of this two months, she was granted time at midday to return home and care for her baby. This procedure alone reduced the infant mortality more than 50 per cent.

In 1876 there was established in England a Society for Nursing Mothers. The object of the Society is to save the child's life by preserving the health of the mother. The mothers are cared for in institutions for several weeks before confinement, being well fed and housed; but, what is more important is that, during the first year of the child's life, the mother is cared for wholly or in part, as it is necessary. A physician and nurse visit her at her house and give her the assistance she may require. Each month the child is weighed, thoroughly examined, and if sick is always taken care of. The Society has cared for over forty thousand children, and the saving of infant life has been very great.

In Paris since 1904 the Coullet dining-rooms have gone one step ahead of anything done, as far as I know, in America. They have established restaurants in the poorer districts of Paris; any woman who is nursing a baby is given free of all cost two good meals each day. They feed the mother, and the mother nurses the baby.

Since all empires are built of babies, unless a change in the trend of statistics of infant mortality shall take place, our future generations will fail to develop physically and numerically along the lines which are both normal and natural. Race suicide is not a theory, but a fact.

France is actually facing slow extinction; its birth rate is smaller than that of any other European nation. The trouble and expense incident to the care and rearing of children does not appeal to all women of the present day; motherhood is not always synonymous with wifehood. A high birth rate is usually, but not invariably, linked with prosperity. The foreign-born population of the United States has apparently a larger percentage of children than the native population, but this excess of fecundity is probably no more than that which usually is found in urban populations in poor districts, and the high infant mortality commonly found among this foreign element more than reduces its growth to the level of native-born Americans.

The Royal Commission in New South Wales, quite recently appointed, after careful study and thought, decided that the main factor in the reduction of the birth rate was: "A diminution in fecundity and fertility in recent years; due to the deliberate prevention of conception and destruction of embryonic life, and to pathological causes consequent on the means used, and the practices involved therein." The remedy for this is not easy to apply; all nations are becoming more extravagant in their methods of living—automobiles and babies may be incompatible possessions—if we have one, we must often renounce the other.

Infanticide by neglect or intention undoubtedly causes the death of many hundreds of infants each year. Women in the poorer walks of life should be urged to nurse their children, entirely or in part, as long as possible. Part breast- and part bottle-feeding is much better than all bottle feeding; weaning should never be done prematurely unless by the advice of a physician. Women with illegitimate children should be kept in the hospital and made to nurse their babies until after the third month. After this age, the child is better able to withstand the perils of artificial food, and the mother, from her association with the child, has perhaps become sufficiently fond of it to make an effort to protect its life. If a child is born, and no one is with the mother at the time of its birth, the danger of death at the time of delivery is greatly increased, and the secrecy of birth may induce the mother, under certain conditions, to destroy the child's life.

The giving of opiates to children, either in the form of paregoric or of a soothing syrup, is pernicious and should always be absolutely forbidden. Overlying, either by accident or design, is in certain portions of this and other countries very common; an infant, of course, should never sleep in the same bed with its mother.

Infant life insurance and burial clubs cause the death by neglect of many; statistics prove that a much greater number of children insured and in burial clubs die than of those children in the same cities and towns who are without such insurance. Coroners' inquests should be rigid and impartial, and if there is any question or possibility of infanticide, the case should be thoroughly investigated and proper punishment imposed on the guilty. Of 864 children dying under one week of age in Philadelphia, inquests showed, according to Parry, that 94 were "stillborn," 210 died from "unknown causes," 293 from

"asphyxia," 62 from "exposure and neglect," and 22 from "want of medical attention." In these cases the coroner's physician believed that the majority of those which he examined were murdered.

The death rate per thousand is well known to be much higher among illegitimate than among legitimate children. An interesting fact, which is perhaps not always appreciated, is that in large cities the death rate among illegitimate infants is much greater than in country districts. In Glasgow in 1873 the death rate for illegitimate infants was 293 per thousand; for legitimate infants, 154 per thousand. In London in 1902 the death rate of illegitimate children was almost twice as great as the death rate of legitimate children. The infant death rate of London, as a whole, exceeds the rural infant death rate by about 20 per cent. The deaths among illegitimate infants in London exceeds the rural death rate among illegitimate infants by over 50 per cent.

According to Dr. Norman Kerr, in 1894 the proportion of female inebriates in England had increased greatly in the few years preceding 1894. He asserts that prison experience shows a distinct increase in excessive drinking among women. According to the annual death rates from alcoholism in England and Wales, per million living, from the year 1875 to 1904, the mortality due to inebriety is distinctly increasing. The average for every five years from 1875 to 1904 was: 1875 to 1879, 25 deaths per million living; 1880 to 1884, 29 deaths per million; 1885 to 1889, 36 deaths; 1890 to 1894, 50 deaths; 1895 to 1899, 58 deaths; 1900 to 1904, 71 deaths. In studying these figures it seems as if there can be no reasonable doubt that alcoholism is increasing among the women of England and Wales, although some allowance should probably be made for the more accurate diagnosis of recent years. Dr. Scott, quoted by Newman, believes that alcoholism is increasing among the women of Scotland.

Alcohol is a distinct poison to children, but the number of deaths caused by the giving of alcohol direct in any form to children is certainly very small in the United States. It has, however, been clearly shown that suffocation in bed and overlying are twice as common on Saturday as on any other night in the week; and the prevalence of drinking among the poor on that night is proverbial. An alcoholic mother rarely supplies her baby with a good breast milk, and what is perhaps more important is the fact that the milk from such a mother may even contain alcohol. Alcoholism among women is perhaps increasing in America, but it is surely less common than in England.

Systematic nursing and medical care are wonderful aids in the prevention of infant mortality, and account largely for the difference between the infant mortality of the rich and poor. Home treatment, or perhaps better say maltreatment, of very young infants often destroys what little chance of life the infant would otherwise have had. The poor and ignorant classes often call "the doctor" too late to save the patient.

It is not an easy task to form an accurate idea of how many or what proportion of infant deaths are due to congenital causes and those

diseases which, if not actually congenital, still leave the child more susceptible to their development than is the child whose parents are free from such diseases. Herbert M. Rich, in 6866 deaths under one year of age, found 23.2 per cent. to be due to malformations, congenital debility and premature birth.

In Philadelphia during the year 1915, 70 deaths were recorded in infants under one year of age as due to syphilis, and 4 deaths in infants from one to two years of age from the same cause. During this same year there were 848 premature births in Philadelphia, a large portion of which, it is reasonable to suppose, were due to syphilis, which is certainly responsible for many premature and early deaths.

During 1911 and 1912, in New York City, there were 132,776 births, of which 6749 were stillbirths.

The following figures are from the records of the Board of Health of New York City:

	No. of births.	Stillbirths.	Illegitimate births.	Deaths one month and under.
1911 . .	66,537	3438	1559	8223
1912 . .	66,249	3311	1541	7675

In London and in most English cities the mortality from prematurity and atrophy is about 45 per thousand, these deaths almost all occurring in the first three months of life. Smallpox, malaria, typhoid fever and tuberculosis are all diseases that may and do exert an influence on the infant. Lead, mercury and phosphorus may have a distinct antenatal effect, and the influence of alcohol has already been alluded to. In fact, any and all toxemias may influence the child during intra-uterine life.

The table on page 61 gives the causes of death in babies less than one month old during the year 1912 in New York City.

Gastro-intestinal diseases are not only the most common diseases of infancy and childhood, but they are also responsible for more deaths than any other class of diseases. Infantile diarrhea is especially apt to occur in the first year of life, although very common in children under two years of age. Hot weather, bad feeding and poor hygiene are the chief etiological factors. It is often seen in epidemics, is very dangerous, and is the common cause of deaths among infants in cities in summer. Epidemics have often been traced to infected milk, although one must remember that milk may be infected at the farm, in transit, or by the consumer.

Cases of this disease are rare among the rich, compared to the number one sees among the poor; they are rare in the country, compared with the number seen in the cities. The diarrheal death rate is, as a rule, highest in those countries where the infant mortality is greatest. Taking 42 of the largest German cities, in the years 1904–5–6, there occurred 67,633 deaths of infants under one year of age; of these 28,422 were due to gastro-intestinal disease.

The records of the Bureau of Health of Philadelphia for the year

1915, show the number of deaths from diarrhea and enteritis, as follows:

	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Under one year	32	27	42	54	47	49	201	269	217	122	69	39
1 to 2 years .	5	6	8	7	5	10	37	60	44	15	9	7

This gives a total of 1163 deaths in infants before the end of the first year, and only 213 deaths during the second year.

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
Erysipelas . . .	1	2	4	3	0	3	3	2	0	0	1	0	19
Congenital debility, icterus,atrophy,pre- maturity, marasmus	120	113	120	95	134	111	125	121	121	121	102	105	1388
Gangrene . . .	0	1	0	0	0	0	0	0	0	0	0	0	1
Syphilis . . .	6	4	6	9	4	3	3	2	3	4	8	5	57
Gonococcus . . .	0	0	0	0	1	0	0	0	0	0	0	1	2
General diseases, pur- pura hemorrhagica, diabetes	1	0	0	0	0	0	0	0	0	0	0	0	1
Convulsions . . .	8	0	3	1	2	5	3	4	0	4	0	2	32
Bronchitis . . .	13	6	8	2	4	0	2	3	3	5	5	5	56
Pneumonia . . .	28	26	14	26	6	8	10	10	10	21	26	22	207
Influenza . . .	1	0	0	0	0	0	0	0	0	0	0	0	1
Pertussis . . .	0	0	0	0	0	0	0	0	0	0	2	0	2
Diarrhea, enteritis .	4	6	13	11	11	7	14	13	14	10	4	7	114
Diseases of stomach	0	0	0	0	0	0	0	0	0	0	0	3	3
Hernia and intestinal obstruction . .	0	1	1	0	0	0	0	0	0	0	0	1	3
Congenital malfor- mations . . .	13	11	20	3	17	12	16	11	8	17	15	13	156
Diseases of skin and adnexa	1	0	1	1	0	1	1	0	0	0	0	1	6
Diseases of kidney .	0	0	0	0	0	0	0	0	0	0	0	1	1
Diseases of liver. .	0	0	0	0	0	0	0	1	0	0	0	0	1
Meningitis . . .	0	0	0	0	0	0	0	0	0	0	1	0	1
Softening of brain .	0	0	0	0	0	0	0	1	0	0	0	0	1
Tetanus	3	0	0	0	0	0	0	1	0	0	0	0	4
Causes peculiar to infancy, umbilical hernia, atelectasis, forceps injury . .	43	47	50	38	45	31	35	37	20	45	31	37	459
Homicide, piercing, cutting . . .	1	0	0	0	1	0	0	0	0	1	0	0	3
Homicide, other means	1	0	4	0	2	1	0	0	0	0	1	0	10
External violence .	2	1	1	0	0	1	0	0	0	0	0	0	5
Accidental drowning	1	0	0	0	0	1	0	0	0	2	0	0	4
Absorption of delete- rious gases . . .	1	1	0	0	0	0	1	0	0	0	1	1	5
Acute poisoning .	0	0	0	0	0	0	0	0	0	0	0	0	0
Acute abscess . .	0	0	3	0	0	0	0	0	0	0	0	1	4
Diseases of organs of locomotion . . .	0	0	0	0	0	0	0	0	1	0	0	0	1
Diseases of lymphatic system	0	0	0	0	0	0	0	0	0	0	0	0	0
Unspecified causes .	0	1	0	0	0	0	0	0	0	0	0	0	1

In certain American cities it has recently been clearly proved in many instances that this enormous summer death rate, due to the diarrheal diseases, can be reduced. Clean milk must be provided for the poor at a nominal cost, and this milk must be properly modified for children of different ages and conditions. A campaign of education among the mothers of this poor class must be carried on persistently and continuously; visiting nurses must be supplied; and fresh air and improved hygiene must be insisted on. It is not asserting too much to say that a reduction of 50 per cent. in summer infant mortality may be accomplished by these means.

It is an old truth thoroughly appreciated by American physicians

that breast-fed infants do well, whether they belong to the rich or the poor, but I do not believe that it is appreciated how great a difference exists in the infant mortality between breast-fed and bottle-fed infants. In the year 1903, 4075 infants died in Munich; of these 83 per cent. were artificially fed. In Berlin since 1885 the census gives the character of the feeding of all living children. Taking the five years, 1900 to 1904, only 9 per cent. of the deaths occurred in breast-fed babies, and Budin has shown that only about 9.5 per cent. of the infant mortality in Paris occurs in breast-fed children. Of course, breast feeding is usually associated with other favorable factors, and bottle feeding often combined with many unfavorable associated conditions, but the figures are truly startling.

The importance of breast-feeding as a means of reducing the infant mortality rate is clearly shown by the statistics which follow:

In New York City more than 85 per cent. of all deaths in infants occur in the bottle-fed. In Boston, in the year 1911, 74 per cent. of all infants over two weeks of age who died were artificially fed, and in a series of 1000 cases of infants studied by Armstrong, in Liverpool, 22.8 per cent. of the artificially fed babies died in their first year, and only 8.4 per cent. of the breast-fed. In 1908 the Health Department of New York City reported that of 1000 fatal cases of enteritis only 90 were in breast-fed infants.

In war time the infant mortality often declines in manufacturing centres, in spite of the fact that the general mortality rate increases. During the siege of Paris, 1870–71, it is claimed the general mortality rate doubled, yet the infant mortality rate declined 40 per cent. Under such conditions infants do not die, and why? In times of war or great industrial depression the poor woman, having no work, stays at home and nurses her baby, and the child lives. In prosperity she works all day, gives her baby the bottle, and it dies.

This is borne out by recent statistics compiled in Paris where, since the beginning of the present war, the general infant mortality has diminished, more babies are born at full term, and fewer are abandoned. These conditions must be attributed to the special protection extended to expectant mothers and to infants during the first year of the war.

In England this work of saving the babies has received a great impetus since the outbreak of the war, more infant welfare stations having been established than ever before in the same period of time. During the first eight months of the war 100 new schools for the instruction of mothers were founded, many of which furnished meals for both expectant and nursing mothers, while 200 more baby stations were planned for; all this in towns with a population of 20,000 or more, to say nothing of what the smaller places may have done.

It is both interesting and instructive to note that any considerable variation in the infant death rate in any locality is almost invariably linked with a corresponding change in the diarrheal death rate, the mortality from other causes changing, as a rule, comparatively little.

The factors contributing to infant mortality are so many and varied

and the difficulties in controlling these harmful influences are so great that at the present day one is forced to admit that, while the preventable death rate is very large, still among the poor there must necessarily be a high death rate.

Bronchopneumonia and true pneumonia also cause not a small proportion of deaths during infancy, the majority of fatalities from these diseases occurring in the crowded tenement districts. During the year 1915 the deaths from these diseases in Philadelphia were as follows:

Under one year.	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Bronchopneumonia.	59	37	53	55	37	23	20	18	15	30	27	58
True pneumonia .	21	26	19	24	12	6	11	3	8	5	10	21
One to two years.												
Bronchopneumonia.	16	15	23	15	20	10	20	18	15	30	27	58
True pneumonia .	10	11	19	21	13	7	4	2	1	4	7	12

Total deaths from bronchopneumonia under one year of age, 432
Deaths from bronchopneumonia one to two years, 182
Deaths from true pneumonia under one year of age, 166
Deaths from true pneumonia one to two years, 111

Several years ago the mayor of Huddersfield, England, offered a gift of $5 to every child born in his town that lived to the age of twelve months. All classes, rich and poor, were included; the mortality in the Huddersfield district was immediately reduced more than 50 per cent.

In Yonkers, N. Y., a campaign was undertaken in 1894, having as its object the reduction of the infant mortality rate. The physicians of Yonkers, aided by the public press, established milk stations, and instituted and carried out a campaign of education among mothers. A sanitary inspection of the tenement district was adopted, and nurses were appointed to visit the sick. The Board of Health also passed a regulation requiring in all new tenements a sufficient amount of light and air. The deaths from digestive diseases were reduced more than 50 per cent. Dr. S. E. Getty believes that, of all the means employed, the most important was the establishment of the milk dispensaries.

Holt, in the following table of statistics, shows the decrease in infant mortality as the result of a campaign waged in New York City by its Board of Health.

1880	. . . .	228 deaths per 1000 infants born alive
1902	. . . .	168 " " " "
1908	. . . .	144 " " " "
1911	. . . .	120 " " " "
1912	. . . .	109 " " " "
1913	. . . .	102 " " " "

That a general propaganda against infant mortality has been vigorously pushed all over the United States is shown by the census, of 1880 and of 1890. In 1880 the general infant mortality of the United States was 246 per thousand; in 1890 it had fallen to 159 per thousand, and during the same period it is gratifying to note that the infant mortality in cities decreased from 303 to 184 per thousand. In 1914, there were reported 204 infant welfare stations in 39 cities and towns of the United States. This is surely a record to be proud of.

The Register of Records of the Department of Health in New York City shows the reduction in the annual death rate of children under five years of age, and this is largely attributed to the use of pasteurized milk. The figures are as follows:

1891	. . . .	96.5 per 1000				125.1 per 1000	
1901	. . . .	61.3 "		July and August		76.2 "	
1911	. . . .	43.8 "				46.3 "	
1913	. . . .	37.3 "				38.8 "	

Since 1910 the deaths among infants under one year of age in greater New York have dropped as follows:

In 1910	16,215
In 1911	15,053
In 1912	14,289

In France, 1874 to 1893, the average infant mortality was 167 per thousand. Ten years later, in 1903, it was only 137 per thousand. In Paris it was only 101; wonderful Paris has the smallest birth rate and the lowest death rate of any large European city.

The physicians of the United States have accomplished much in the last ten years, and yet when we consider how remarkably successful have been the efforts directed to save infant life, should we not, as the representative body of the American profession, feel chagrined that we have not accomplished more? Certainly 50 per cent. of all infant deaths at the present day are preventable.

Hospitals for infants have been established all over the world, and we are establishing new ones almost daily, and yet some physicians question whether they do good or harm. Going back to the year 1871, we find that 29.82 per cent. of all the children born in Philadelphia died before the end of their first year. In the same year, in the found-ling ward of the Philadelphia Hospital, 73.65 per cent. died. The death rate among the foundlings was 43.83 per cent. more than among infants of the same age of Philadelphia. These children were, as a rule, in fair health on entering the Hospital. Of these infants, 74.69 per cent. died from diarrheal diseases, and only 25.31 per cent. from all other causes. At this period, the records of the foundlings' ward in the Philadelphia Hospital were about the same as the records from the foundling hospitals in other large American cities. Dr. A. Jacobi, at this date, had the courage to point out publicly the enormous mortality in the foundling institutions of New York, and as a con-sequence was asked to resign from the staff of the Hospital with which he was connected.

Van Ingen reports a series of 738 cases of infants in an institution in New York City; 22 per cent. of all that died succumbed during the first month. The total mortality during the first month among all the babies born in the institution was only 8 per cent., but 48 per cent. died before the year was ended.

Dr. Knox, of Baltimore, reports a series of 200 cases of infants in

different institutions in that city in which 80 to 90 per cent. of the deaths occurred before the end of the first year, and this was the actual total of those who remained in the institutions. The 10 per cent. that survived had practically all been sent away from the institutions into the care of foster mothers.

What is the condition in our infant hospitals at the present day? My personal opinion is that in the modern infant hospital, where the air space is ample, and the windows are kept open day and night; where the milk is the best and the milk-room thoroughly up to date; where the ratio of nurses to patients is not less than one to five; where the sick are promptly isolated from the well, and "mothering" is understood—that in such a hospital the physician is an optimist and not a pessimist.

Many of the infants will die, but most of them will live. If one considers that many of those who die are "weaklings" on admission, and that the greatest proportion of deaths occurs in the first three months of life, one is apt to believe that the best of the modern infant hospitals are worthy institutions and should be supported. Much depends on the character of the feeding; but, here as ever, the truth that breast feeding is better than bottle feeding is well exemplified.

Of 300 infants admitted to the Dresden Children's Polyclinic in 1900 to 1901 there were 53 deaths. All the deaths, 53 in number, were among the bottle-fed babies. Among 93 breast-fed babies, during the same period, in the same hospital, there was not a death. Breast-feeding is surely a powerful measure with which to combat death.

According to the census of 1900, the infant mortality per 1000 in the United States was in those States where registration was in force:

	Per 1000 births.
District of Columbia	274.5
Rhode Island	197.9
Massachusetts	177.8
New York	159.8
Connecticut	156.8
Maine	144.1
New Hampshire	172.0
New Jersey	167.4
Vermont	122.1
Michigan	121.3

The census of 1900 shows the returns for infant mortality from many cities and towns of the United States. The infant mortality in some of these cities is very high, over 400 per 1000 in Charleston, S. C. A number of them show a mortality above 300, and more than 100 cities exhibited an infant mortality above 175 per 1000.

The important point to be decided is as to the influence which has been exerted on this infant death rate in recent years. Have we been able to reduce in any appreciable degree this great and unnecessary waste of infant life? A careful study will show that there has recently been a great saving of life, and much will surely be accomplished in the future.

In 1903 the infant mortality of France was 137. In the previous twenty years it was 167, and yet this death rate ought to be still more greatly reduced, for we know that Ireland has an infant death rate below 100. Norway in 1902 had an infant death rate of 75, and Sweden 107 per 1000.

Of all European countries, Russia has the highest infant death rate, 270 per 1000. Germany has the next highest, averging in recent years a little over 200 per 1000. Medical science and skill have reached a very high plane in both these countries, and infant mortality has been greatly reduced during the last thirty years. It is to be hoped that the useless waste of life in these two countries will quickly be much more distinctly diminished, and it is believed they will soon show as great a reduction as has been brought about in France.

There has been a great decrease in the proportion of infants dying under one year of age in the United States during the last twenty years. The infant mortality in 1880 was 246 per 1000; in 1890 it had fallen to 159 per 1000. During the same period the mortality in the cities of the United States fell from 303 to 184 per 1000.

Many figures might be quoted showing that in recent years infant mortality has distinctly lessened. In London in January, 1908, the deaths of infants under one year of age to 1000 births was 115. According to George B. Mangold, U. S. Department of Commerce and Labor, the infant mortality in New York City in 1891 was 241.9 per 1000; in 1900 it was 191.7 per 1000; and in 1906 it was 167.8 per 1000. In the same city the death rate of children under five years of age was 96 per 1000 in 1891; in 1896 it had fallen to 77.5 per 1000, in 1900 the mortality under five years of age was only 67 per 1000; and in 1904, it was 54 per 1000. In the same community the deaths from measles, scarlet fever and diphtheria have become distinctly less; and diarrheal diseases in small children have decreased 62 per cent. since 1881.

School nurses are now provided and medical inspection of schools is today well recognized and practised in many of our large cities. According to Dr. W. M. L. Coplin: "A necessity for medical attention was detected in 27,481 children in the schools of Philadelphia in 1905, and 31,544 children in 1906." Dr. A. C. Abbott, formerly chief of the Bureau of Health of Philadelphia, shows that in the years 1903–5 a distinct decrease in the infant death rate occurred. Philadelphia shows a very decided decrease in infant mortality since 1897.

Thomas A. Buckland, City Chemist for St. Louis, states that there has been a decrease in infantile mortality in that city since 1904. W. Ernest Wende, M.D., Health Commissioner of Buffalo, states that infant mortality is decreasing in that city. Samuel E. Allen, Health Officer of Cincinnati, states the proportion of deaths of children under two years of age to the total mortality has decreased considerably since 1886. In the year 1886 it was 32.56 per cent.; in the year 1906 it had decreased to 21.92 per cent.

Milwaukee and Minneapolis and the nine largest cities in the State

of New York, according to George W. Goler, M.D., show a decrease in infant mortality:

The following have occurred to me as being important factors in lessening infant mortality:

Abatement of nuisances.

Milk inspection: milk dispensaries; visiting nurses.

Free antitoxin.

Improved sanitation.

Good food.

Education of girls and married women in the duties and requirements of motherhood.

Maternity fund in all industrial establishments where married women are employed.

Care of poor pregnant women before and after confinement.

Laws carefully protecting all children who are cared for by private individuals, apart from their parents; rigid enforcement of these laws.

Elementary principles of hygiene taught in all schools, public and private.

Nursing of all babies, as far as possible, by their mothers.

Sending children to the country in summer.

Pasteurization of milk during the hot months.

Farming out, under proper medical supervision, of foundlings and institution infants, and the appointment of nurses to visit these infants regularly.

CHAPTER V.

HEREDITY.

We derive certain characters from our parents, and certain characters from our progenitors other than our parents. The entire body is, as it were, built up of myriads of units, each unit inherited from some ancestor. It is important to remember that the child inherits not only certain physical characters, as height, features, color of eyes, etc., but that mental and moral characters are also inherited, such as moral sense, ambition, industry, activity; and there is strong evidence to prove that a trait or fundamental principle absent in both parents cannot be present in their child, also that some defect that is marked in both parents, especially if there is consanguinity of the parents, is apt to be present in their children. For instance, it has been estimated that almost one-third of the children of deaf-mutes, if there is consanguinity in the parents, are deaf, whereas only one-tenth of the children of such parents are deaf when the parents are not blood relatives. Deafness may arise from a great many causes, and the possibility of this cause being found in both parents is much more likely if they are consanguineous.

The inheritance of one mental or physical property does not depend upon the inheritance of others; for instance, one child in a family may have straight hair, another curly hair, while both may have blue eyes. In fact, when the parents and grandparents are known, many physical characteristics of the children may be explained. An inherited condition—for instance, hemophilia—may be transmitted by an apparently normal person.

Man begins as a single cell, the fertilized ovum, and the organism is built up by cell division. As evolution proceeds environment assists in producing certain changes; i. e., certain characters, which may formerly have been essential, have, owing to changes in environment, become less useful or, in fact, only a hindrance, and these characters which are no longer of any use or assistance tend to disappear. As development progresses the environment, if it is to suit accurately the new individual, must also change.

The embryo of man shows at a certain stage of its embryonic life an arrangement quite analogous, as far as the large bloodvessels are concerned, to that found in the gills of fish. This would tend to indicate that the progenitors of man were more or less aquatic animals; but, the human embryo not needing any apparatus similar to the gills of fish, these have disappeared except for such scant evidences of their former existence as the branchial clefts and arches. Changes of this type, called regressive, are of course brought about only slowly,

and are in marked contrast to other characteristics spoken of as progressive, which are in keeping with the evolution of the individual and its changed environment. By this method of regressive and progressive changes which take place in all of the organs and tissues of the body the gradual development of the higher animals occurs.

Children may, and not uncommonly do, show mental and physical traits which were not present in either parent, but were inherited from some ancestor. All that environment can do is to modify the inborn characters. Environment and training are certainly the direct causes of acquired characters in the individual, and, if the same environment persists, these characters may appear in the offspring. If, however, the special environment that has brought about the acquired character is not present, the acquired character usually ceases to reappear in the offspring. Characters disappear sooner or later when they cease to be the subject of selection. Variations occur in all directions, and it is the environment which is influential. If the transmission of acquired characters has played an important part in evolution it has come about by the selection of inborn variations.

Acquired characters may be transmitted, but it is not a common or usual occurrence; the evidence or proof that acquired characters are frequently inherited is insufficient; but as most competent observers attach a certain amount of weight to this idea, it may be accepted tentatively as an occasional fact, and its acceptance and belief may do good by stimulating the better side of our natures. Undoubtedly acquired characters are, as a rule, simply normal variations and the natural transmission of such variations; if suited to the individual's environment, they are permanent and progressive; if not adapted to the environment they are more or less rapidly eliminated. In order to believe that the inheritance of acquired characters is common or usual, one must also believe that it is possible for this acquired character to exert some specific and definite action on the germ cell itself, and it is certainly improbable that some characters, developed perhaps in middle adult life, could produce this effect.

Environment can only modify characters; it can not originate them; and it is self-evident that each specie is in the environment best adapted to its development. Use will develop both mind and body, and environment is certainly the cause of acquired characters developing or appearing in an individual, but environment rarely, if ever, produces changes in the inborn characters.

It is important to appreciate that diseases in the parent, such as syphilis, rheumatism, or acute alcoholism, do produce mental and physical diseases in the offspring; but the diseased condition or taint or inherent weakness is in these cases transmitted directly to the germ cell, and normal heredity differs from diseased heredity as normal tissue differs from diseased tissue. If this diseased offspring is not placed under the best environment and given proper medical treatment it tends to be eliminated, and disappears either in the first or succeeding generations. If the environment and treatment are proper and

suitable, it may in the first or later generation return to the normal standard of its progenitors. Heredity is constructive, not destructive; it tends to build up, not to tear down; and will, if assisted, always do its full share toward restoring the individual to the normal for its species.

Variations are not always of the same kind or type, and the persistence or disappearance of these variations depends on the environment. The descendants tend to inherit these variations, some in a greater, others in a less degree, and with changing environment and selection certain new characters, as sight and hearing, are evolved. Variations are of common occurrence, but need environment to complete the process of evolution. As environment changes, the species must change, else it would be destroyed.

The effect produced by environment is clearly shown by the well-known experiments of Nägeli. He removed some Alpine plants from their usual location and transplanted them to the rich and fertile soil of the Botanical Gardens in Munich. Under the new environment the plants and foliage became much more luxuriant, and the seeds from these plants also produced this new and abundant growth. After thirteen years of such cultivation and luxuriant growth the plants differed greatly from their original stunted and sparse forms. When, however, these plants were removed back to the original poor soil of their Alpine environment, they quickly lost all their acquired luxuriance of foliage and growth, and returned to their former stunted and dwarfed condition. This experiment adds much weight to the theory that acquired characters can and often will be inherited if the offspring be placed in the same conditions of environment as the parents were living in when the change in their characters occurred; and while it tends to show that these acquired characters can undoubtedly be caused by environment and can be transmitted, still, there was no permanency to these acquired characters except while the changed environment persisted.

It is well known that the cells of the central nervous system do not multiply after birth; they of course develop, and gradually assume certain functions; but no new nerve cells are produced, and, once destroyed, they are never reproduced. Evolution in the higher animals as seen in man means an *increase* in the size of the brain and a consequent increase in the intelligence; but we have as yet no proof of the possibility of parents being able by acquiring brain cells to transmit any added number of brain cells to their offspring. The difference between man and the higher apes is especially noticeable in the brain, and all records of man show that he has not changed any in brain capacity.

In the consideration of the characters inherited from our immediate parents and progenitors, it is of course true that changes in environment accomplish much, and that characters acquired by the parents may appear in the offspring, during many or all succeeding generations, if the offspring live under the same conditions as the parents, and

the environment does not change. But it is, nevertheless, true that man has changed very little, if any, in stature and brain capacity during the past 5000 years. As far back as the reindeer age, skeletons of men and women show height and brain capacity to have been about the same as in the highest types of man today, and the skulls show no changes in the jaw or frontal bones. The characters of a race undoubtedly change very little from generation to generation; individuals differ sufficiently in looks, voice, external appearance and temperament to be recognized and grouped, and these slight variations always occur in races.

The individual inherits one-half of his characters from the two parents, one-fourth from the four grand-parents, one-eighth from the great-grandparents, one-sixteenth from the great-great-grandparents, and so on. This is true not so much in an individual case as when large numbers of individuals are observed in the aggregate.

Galton traced the family histories of parents of unusual mental capacity, and proved conclusively that these superior and unusual mental characters were inherited, and the offspring of such parents were superior mentally to the offspring of parents mentally inferior. Parents with unusual mental powers will transmit such powers to their offspring, even if the mental powers of the parents have not been developed by educational methods and environment—hence the mental powers of a child are largely decided before he is born. This does not imply that the individual's mental powers may not be improved by education and environment; but the mental powers of the child will be the same whether the parents were educated or not. The hope of improving the race by giving special care to the weak and feeble-minded is fallacious, and marriage and procreation by such parents can only tend to lower the general tone of the race.

Heredity has been defined as "the inheriting of certain qualities or tendencies, or the tendency manifested by an organism to develop in the likeness of its progenitors."

"Degeneracy is the absence or loss of that degree of development or energy seen in the ancestry of the organism." Heredity is a tendency to develop the type of the ancestor. Degeneracy is a tendency to a lower type than the ancestor. All evolution results from variation and heredity. If type a is to become b by evolution, some individuals of a must vary toward b, and this variation must be inherited and transmitted until a finally becomes b. The ovum and spermatozoön must bear in themselves all the characters that are inherited from their parents and ancestors.

Environment is a potent cause in the production of variations. Heredity should and does imply merely a single link in a long chain. Degeneracy may be inherited or it may be acquired; it is a mere accident in a long line of heredity.

CHAPTER VI.

CONGENITAL MALFORMATIONS.

BRAIN AND SPINAL CORD.

MENINGOCELE, cephalocele, and hydrencephalocele are herniæ of certain portions of the cranial contents, either through an abnormal opening in the bony skull or along one of the lines of suture or fissures. The openings through which the cranial contents protrude are found in the occipital, nasal, parietal, and temporal regions.

Etiology.—The etiology is rather obscure. Hörnke and Hertwig produced similar deformities in some animals by chronic poisoning of the parents before impregnation, and in others by injuring the ovum. Von Bergmann claims that a misturning of the cerebral sac is the important factor in its causation.

Pathological Anatomy.—The opening in the skull is usually small, and the dura mater does not protrude, only the arachnoid and pia mater escaping from the cranial cavity. The membranes are often greatly thinned by the distention of the contained fluid. Small tumors are the more common, and are less apt to contain brain tissue.

Meningocele.—A meningocele contains the internal membranes of the brain, and these membranes are usually, but not invariably, distended with fluid. The opening into the brain is generally small. Fluctuation is present in the tumor, but not pulsation.

Symptoms.—Bimanual palpation, one hand on the tumor, the other on the fontanelle, will disclose increased tension in the fontanelle, unless the opening into the cranial cavity has become occluded. If the sac does not rupture, and is not of large size, a meningocele may produce few, if any, symptoms. If, however, it becomes progressively larger and the walls thinner, the nutrition of the enveloping skin and scalp tissue becomes impaired, and infection and bursting become more likely.

Diagnosis.—The diagnosis is usually easy. The location of the tumor is significant; its partial reduction by pressure and fluctuation, if present, help to confirm the diagnosis. If, in addition to this, there is pulsation and pedunculation the diagnosis is rendered practically certain.

Treatment.—Surgery offers the only hope for the patient. This consists in the removal of any fluid the tumor may contain, and the closure of the hernial opening. It must be borne in mind that young children stand long operations badly. If hydrocephalus is present, the result is apt to be unfavorable. If the tumor is small, this adds to the probable success of the operation. As a rule, the result of the

Fig. 2.—Meningocele in a child aged four months.

Fig. 3.—Meningocele.

Fig. 4.—Meningocele, child aged seven months.

operation in meningocele is much more favorable than in encephalocele or hydrencephalocele.

Encephalocele.—This consists of not only the membranes of the brain but also of brain tissue. The tumor does not connect with any of the ventricles, and, if fluid is present, it is in the outer portion of the sac, immediately beneath the skin and underlying tissues. The opening into the skull is usually larger than in a meningocele, and the tumor is generally small and not pedunculated as in a meningocele. Pulsation is often present, and the tumor is with difficulty reduced by pressure; cerebral symptoms commonly follow efforts at reduction.

Treatment.—The treatment is entirely surgical. The smaller the tumor the better is the prognosis; although, as surgery presents the only hope, the child should be given the chance for its life that surgery offers. Much depends on the child's physical condition, the maintenance of all its vital forces, and on the care of a skilled pediatrician, as well as upon the surgical technic employed. Unless such combined care is possible, surgical interference is useless.
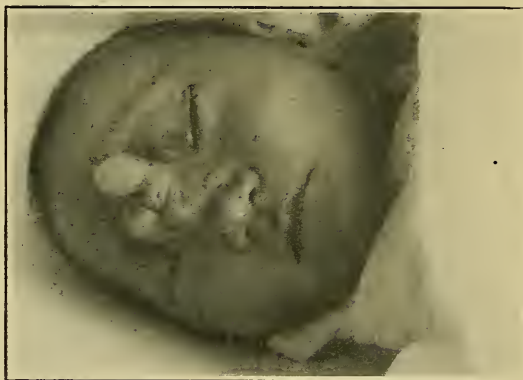
FIG. 5.—Encephalocele. child three days old.

Hydrencephalocele.—This consists of a portion of brain tissue enclosing a cavity filled with fluid which communicates with a lateral ventricle, the entire mass being covered externally with brain membranes. The tumor can not be reduced, is usually of large size, and is apt to be pedunculated. The fluid in this form is in the interior of the mass, and gives a sense of deeper fluctuation than in the other two forms; moreover, this form may be associated with hydrocephalus. It is the most serious of all brain herniæ, and offers no hope of cure by surgical means. The diagnosis of this variety from meningocele and encephalocele is important, as operation offers some hope in the last two conditions.

Acute Hydrocephalus.—Acute hydrocephalus is associated with inflammation of the base of the brain, and is, as a rule, tubercular.

An increase of fluid is always found in acute meningitis of all forms, but the quantity of effusion into the ventricles is usually small. As a consequence of the small amount of effusion in acute hydrocephalus, associated as it is with meningitis, the head rarely attains any great size. It is usually the result of tuberculous meningitis, is the most common form of brain inflammation seen in children, and is almost invariably fatal. Tuberculous meningitis will be found described in full under "Diseases of the Nervous System."

Chronic External Hydrocephalus.—Chronic external hydrocephalus is rare as compared with the internal form. It may be the result of hemorrhage of the meninges, pachymeningitis, or congenital maldevelopment of the brain, the latter cause producing the most typical cases. The convolutions are flattened and atrophied from the pressure of the fluid situated outside of the brain substance between the dura and arachnoid. If present in large and increasing quantities the fluid may cause a progressive enlargement of the head, similar to that seen in the internal variety of the disease; usually, however, the amount of fluid is small, and the distention and pressure only slight.

Fig. 6.—Chronic internal hydrocephalus, child aged seven months.

Chronic Internal Hydrocephalus.—Congenital hydrocephalus is almost always of the internal variety. In this form the lateral ventricles are distended with fluid, the brain substance being stretched and thin because of the great pressure. The fontanelles are much increased in size as the result of a large amount of contained fluid, and the bones of the head are more or less forced apart.

Etiology.—Evidences of hydrocephalus may be present to a marked degree at birth, but more often they are only slight, if present at all, and commonly the infant does not show any manifestations of the disease until it is some weeks of age. Among the systemic causes capable of producing the disease may be mentioned alcoholism, syphilis, heredity, and trauma during pregnancy. Among the local causes are meningitis, disease of the choroid plexus, leptomeningitis, the pressure of brain tumor, and obliteration of the foramen of Magendie. The disease may be congenital or acquired. In a certain proportion of cases hydrocephalus will be found associated with rickets, and in my experience these cases are the most hopeful. Cases have

been reported where the hydrocephalus has followed a successful
operation for spina bifida or encephalocele. The salt content of the
cerebrospinal fluid is very low and is of the natrium variety. The
protein elements in the fluid are also very low.

Pathological Anatomy.—The brain shows an accumulation of fluid
in the ventricles and the septum lucidum partly destroyed by pressure.
The brain is anemic, and it may be difficult to differentiate between
the gray and the white matter. The ependyma is generally thickened
and anemic. Other congenital deformities may also be present. A
chronic meningitis, usually basilar in type, may be found, and the
choroid plexus may show inflammatory changes. The foramen of
Magendie may be obliterated, and a brain tumor may be present.
The specific gravity of the fluid varies from 1005 to 1007.

Symptoms.—The most noticeable symptom is the large size of the
head, which may be so large as to cause the death of the child during
its birth. The forehead is bulging, the anterior fontanelle widely dis-
tended, and the frontal and parietal bones are often greatly separated.
The anterior fontanelle is tense and bulging, the eyes protrude, and
the cornea is partly covered by the lower eyelid, the white sclera show-
ing above. The skin covering the bones of the head is stretched, and
the superficial veins dilated. The head is wedge-shaped, the apex
being formed by the chin. The eyes have a downward direction as the
result of pressure upon the orbital plates of the frontal bone, and
nystagmus and strabismus may occur. The hair is scanty and dry,
and there is often inability to hold the head erect. The mental powers
are usually more or less deficient, although if the fluid accumulates
slowly a fair degree of mentality may be present. Atrophy of the
optic nerves was observed in a few of my cases but, as a rule, only in
those in whom the pressure was extreme. The arms and legs may be
more or less rigid, and the hands tightly closed; or the extremities
may be relaxed and flaccid. The reflexes are usually exaggerated.
The pupils are, as a rule, equal and are more apt to be contracted than
dilated. Convulsions occur in a fair proportion of cases. A slight
increase in the hydrocephalic fluid very often produces marked symp-
toms, providing the bones of the cranial vault are firmly ossified;
whereas, in certain cases, if the anterior fontanelle, frontal, and
parietal sutures are still more or less membranous, the bony walls
are somewhat elastic, and the symptoms may be very slight.

Diagnosis.—It has been demonstrated by Theobold Smith, Arthur
I. Kendall, and many others that there is an available carbohydrate
in the normal cerebrospinal fluid that directly influences the activity
of the bacteria. Toxins are not produced in the cerebrospinal fluid
in the presence of this carbohydrate, and the carbohydrate that the
normal cerebrospinal fluid contains is generally believed to be a form
of non-fermentable dextrose. This carbohydrate of the normal
cerebrospinal fluid is the first element attacked by the bacteria after
their entrance into the central nervous system. The flora which

ordinarily is represented by the infectious agent belongs to the group which prefers a carbohydrate to a protein diet. The first proof, therefore, of pathogenic microbic invasion is found by the absence of this copper-reducing body from the cerebrospinal fluid. This is practically true in all forms of brain inflammation except in the tuberculous variety of slow development. A copper-reducing body is absent from the cerebrospinal fluid in other forms of meningitis. This finding antedates by some hours, and possibly by a longer period than this, the demonstration of bacteria in the fluid. It is, therefore, of assistance in diagnosing the presence of a meningitis in its very earliest stages. Moreover, if the hydrocephalus is due to a previously existing meningitis, the reappearance of the copper-reducing body after its earlier disappearance is evidence of the subsidence of the meningitis and consequent improvement in the prognosis. In a fair proportion of cases hydrocephalus seems to be the result of a chronic inflammation of the ependyma.

A rachitic head is about the only condition which resembles hydro- cephalus. In rachitis the head is square, and the vault is more flat than in hydrocephalus, where it is apt to be rounded. The rachitic head does not show bulging fontanelles; moreover, other evidences of rickets are usually found in the bones of the chest or extremities. Systematic head measurements will, if the case is hydrocephalus, generally show a gradual increase in its circumference.

Prognosis.—The child usually becomes progressively weaker, suffers from headache, vomiting, and may possibly have convulsions. Death in the large majority of cases results from some intercurrent disease. The child may die as early as the sixth month, and it rarely lives to be more than seven or eight years of age. Cure has apparently taken place in a few cases by spontaneous evacuation through a fissure into the nasal cavity. Permanent drainage of the lateral ventricles seems to offer the best prospect of cure in well-marked chronic cases.

The prognosis as to life depends largely upon the rapidity with which the fluid accumulates. In those cases in which the fluid rapidly increases in amount, death may occur at six months or one year with all the symptoms of hydrocephalus present in severe form. If, however, the fluid accumulates very slowly, there may be few, if any, symptoms and the prognosis as to life is much better, the child living to be six or eight years of age. Few cases that show a progressive enlargement of the head live beyond this period. If the fluid ceases to accumulate at any age life may be indefinitely prolonged, the child being usually more or less mentally and physically defective.

Treatment.—In some of my cases in infants a cure has resulted when hydrocephalus has been associated with rachitis, and after one or more lumbar punctures the infant has remained perfectly well. At least three such cases have now continued to be well for periods varying from two to three years. Lumbar puncture has often in my

experience caused a marked temporary lessening in the symptoms, especially headache and vomiting. Many other operations have been devised; fine drains or a silver tube have been placed between the subarachnoid space and ventricles. This is practically autodrainage, and out of a number of cases operated upon some have shown marked improvement. The operation of puncturing the corpus callosum has been employed, with the object of relieving brain pressure by establishing an opening between the ventricles and subdural space of the brain cord. Some excellent results are reported to have followed this operation. Permanent drainage seems to offer the greatest hope for the permanent cure of this condition. Treatment by drainage of the cisterna magna, as recommended by Kopetzky and Haines, especially for meningitis, is of course applicable to cases of hydrocephalus. Better results from operation are obtained in those cases where the accumulation of fluid is very slight than in those where it increases rapidly in quantity. If there is even a vague suspicion of syphilis being the cause of the condition, the child should be given a thorough course of iodide of potassium and mercury by inunction. Fresh air, proper food, and the best hygienic surroundings possible assist in keeping up the general health and nutrition.

Caput Succedaneum.—The most common malformation is the swelling of that portion of the head which represents the presenting part. It consists of an effusion into the soft tissues of the scalp outside the periosteum. It is rarely seen following a rapid or easy labor, but the more tedious and prolonged the labor the more marked, as a rule, is the caput succedaneum. Its most common location is on one of the parietal bones extending backward to the occiput. The effusion is absorbed spontaneously, and requires no treatment. Care should be taken carefully to cleanse the portion of scalp tissue overlying the swelling, and to protect this portion from any infection which might follow slight trauma of the scalp occurring during labor.

Cephalhematoma.—Cephalhematoma is a tumor filled with blood, produced by a rupture either before birth or during labor of some of the vessels of the cranial periosteum. It is most often found over one of the parietal bones, and is usually spontaneously absorbed. It is commonly found outside of the skull, although it is occasionally situated inside the cranium. The variety outside of the skull is called external, and the form within internal; the external is by far the more common. In the external variety the blood is between the periosteum and the skull, or between the periosteum and the occipitofrontalis muscle; in the internal form the extravasation takes place between the dura mater and the skull.

Etiology.—A common cause of cephalhematoma is difficult and prolonged labor, and it may also follow the application of the forceps. It is most common in vertex presentations, although occasionally observed in breech cases. Poorly developed bloodvessels and abnormal conditions of the blood in infants are predisposing causes. Another predisposing cause is a weakened or badly nourished state in the mother.

While it is true that difficult labor and the application of the forceps have much to do with the formation of cephalhematomata, still the factors of bloodvessels, blood, and nutrition are also operative. In proof of this may be mentioned the fact that cephalhematoma has been recorded in infants delivered by Cesarean section and also in prematurely born, hence small infants. It is present in about one and a half per cent. of all cases.

Pathology.—It is commonly found over one of the parietal bones, its position usually depending upon the portion of the head that presents, although breech presentations may show the tumor in the occipital region. One, two, or three tumors may be found in the same patient. As the hemorrhage is from the periosteum the swelling, on

Fig. 7.—Double cephalhematoma in an albino, aged three weeks.

account of the close attachment of the periosteum at the sutures, is always limited to the area represented by the bone. Slight hemorrhage occurs in the scalp. Beneath the cephalhematoma is felt a crater-like opening which upon pressure at the external border gives a sensation of crackling, this crepitus evidently resulting from pressure upon the bony cells and the thin blood-clots which rapidly develop along the edges of the tumor; that is, at the line of separation of the periosteum. In cases of cephalhematoma complicating severe injury to the head, trauma of the soft parts, or depressed fractures, infection may be present, and possibly meningitis. The tumor usually entirely disappears in from five weeks to three months, and the smaller the mass the more rapidly, as a rule, it is absorbed. After its absorption,

a firm irregular thickening usually remains for a considerable length of time at the site of the tumor.

Symptoms.—A cephalhematoma usually appears during the first five days of life, and gradually increases in size for a period of from six to ten days, then tends to become smaller. It may vary in size from the smallest swelling to one as large as a goose egg. Fluctuation is present, but the mass can not be reduced in size by pressure, since no portion of it can be returned within the skull. Violent crying produces no effect on its dimensions or tenseness, and it is soft and elastic to the touch. Unless infection has occurred, there are no local or constant signs of inflammation, and pulsation is very rarely present.

Diagnosis.—Cephalhematoma may be diagnosed from herniæ cerebri by the fact that the latter always occur along one of the suture lines, at a fontanelle, or at some opening in the skull; whereas cephal-hematoma beginning beneath the periosteum is always limited to the area of the bone. Meningocele and encephalocele are partially reducible, and symptoms of cerebral pressure commonly follow efforts at reduction; in addition to this, crying may increase their size. None of these conditions, of course, prevails in cephalhematoma. A depressed fracture does not show a tumor above the normal cranial vault as does a cephalhematoma, and the ridge that surrounds a cephalhematoma is elevated above the level of the surrounding bone, and has a crater-like center; caput succedaneum is not limited by sutures, and is rapidly absorbed within a few days; moreover, it is usually present at birth, while cephalhematoma generally develops after birth. If it is absolutely necessary to verify the diagnosis of cephalhematoma, an aspiration needle may be inserted into the tumor, and the presence of pure blood be demonstrated, but this puncture, must be performed only under the most rigid rules of antisepsis.

Prognosis.—The prognosis is favorable, absorption usually taking place in from six weeks to three months. If a large hematoma has formed within the skull, it may, unless rapidly absorbed, produce pressure symptoms and lesions.

Treatment.—Uncomplicated cases require, as a rule, neither local nor constitutional treatment. Care must be exercised that the tumor be not injured by pressure or manipulation, and that the scalp is kept clean and free from possible infection. Any local injury or infection of the soft parts, bones, brain membranes, or brain substance would necessitate appropriate surgical treatment. An internal cephalhema-toma producing symptoms which persist calls for surgical interference. Under strict antisepsis a small incision may be made in the scalp, the sac opened, the blood expressed, and an antiseptic pad firmly applied. This has the advantage of securing the immediate removal of the blood, and prevents the thickening that may persist after absorption without operation.

Anencephalia.—Anencephalia means, literally, absence of brain; and the monsters to whose condition this term is applied are born with the vault of the cranium missing, owing, it is believed, to the

production of an abnormally sharp cranial flexure in the embryo. Usually a reddish, fleshy mass, which may contain rudimentary brain

Fig. 8.—Anencephalia.

Fig. 9.—Anencephalia.

tissue, is found lying upon the basal bones. In very rare cases there is no brain tissue whatsoever (Figs. 8 and 9).

6

These fetuses are usually of the female sex. Their bodies are well-developed. The head appears to sink down between the well-formed shoulders, while the face is turned upward; the eyes are protruding. The base of the skull is narrow, the nose broad and flat, and the mouth is held partly open, which gives the monster a toad-like appearance.

During the last few weeks of pregnancy the pressure of the anencephalic head not infrequently causes extreme irritability of the bladder in the mother, and at delivery the broad shoulders may render podalic version necessary. The face usually presents.

These monsters rarely go on to full term. They are not extremely rare, and most physicians with a large obstetric practice can recall at least one case within their own experience. Formerly several classes of these monsters were recognized; but today they are all included in the term anencephalia.

Fig. 10.—Hare-lip and cleft palate in a infant five months old.

HARE–LIP AND CLEFT PALATE.

Hare-lip.—It is a well-known anatomical fact that congenital malformations of this type are physiological at one period in the development of the fetus, and are the remains of fissures that are primitive and naturally present in the normal infant at a certain stage of the process of development.

Etiology.—Many theories have been advanced as to the causation of these conditions. Alcoholism in the parents, producing a lack of developmental power in the fetus, is a probable cause, this lack being either mechanical or pathological. Heredity undoubtedly plays an important role, and is by many considered a most important factor. An increase of intracranial pressure, adhesions, deficient amniotic fluid, and amniotic bands, are also probable causes. A failure of the globular processes to join will also produce a fissure in the upper lip. The second upper maxillary processes uniting with the anterior margins of the head fold form later the upper maxillary bone, and a portion of

the head fold also assists in the formation of the nose and intermaxillary bone. The failure of any of these structures to develop properly, or the lack of proper union of these different structures, will cause the congenital malformation of hare-lip.

Pathological Anatomy.—The fissure may be of the lip only, and either one or both sides may be involved. The deeper soft parts, that is, the upper maxillary processes, may or may not be involved, and the palate may be normal or cleft. It may extend into the nose, or may be merely the slightest drawing in of the lip. The fissure is rarely if ever in the median line, and the cleft may be large or small. Either single or double hare-lip may be associated with cleft palate, although double hare-lip with cleft palate is the more common. The fissure is situated either between the canine tooth and the second incisor, or between the first and second incisors. The altered position of the intermaxillary bone may cause the teeth to develop at a right angle to the fissure, or the teeth may be directed almost straight outward.

Symptoms.—These infants often have great difficulty in nursing, as it is frequently almost impossible for them to produce a partial vacuum in the mouth, hence they are unable to cause the milk to flow from the nipple; moreover, the milk is hard to swallow as it tends to regurgitate through the fissure in the lip. Pumping the breasts and feeding with a Breck feeder (Fig. 11) or a spoon is often resorted to. Every effort should be made to preserve the mother's milk, as it is important for the child to be kept as well-nourished as possible. All cases, of course, breathe largely through the mouth, which predisposes them to disease of the respiratory tract. Gastro-intestinal disturbances and bronchopneumonia have also in my experience been among the most common complications met with in the postoperative medical treatment.

Fig. 11. — The Breck feeder. (Kirley.)

Prognosis.—The disfigurement associated with a hare-lip is so marked that almost all parents, even the most ignorant, will give their consent to an operation. The risk of the infant's losing its life is not great, the mortality of the operation being only about 3 per cent. or less.

Treatment.—Long before the days of antiseptic surgery, hare-lip was a common condition upon which to operate; the dangers of the operation are hemorrhage, infection which is traumatic in character, and shock, the latter especially in a frail young infant. The time when the infant should be operated upon depends upon the general physical condition and vitality. Here, as in all other conditions in children, the vitality is in proportion to the weight, general physical development, and constitution. The age of the child is less important, as

regards the time of operation, than its physical condition. A vigorous child two months of age may be operated upon; a delicate infant of two months should, if possible, be built up physically, and perhaps not be operated upon until it is six months old. Before operating the child should be carefully examined for other congenital malformations, especially of the heart, which, if present, would naturally necessitate the postponement of operative measures. Nasal catarrh and bronchitis should be cured by appropriate treatment before operation, and surgeons should insist on the child's feeding and nutrition being looked after before and after operation by a skilled pediatrician. Secondary operations should be deferred for at least a few months. For details as regards the operation surgical treatises should be consulted.

FIG. 12.—Hare-lip.

Cleft Palate.—Etiology.—What has been said in regard to the etiology of hare-lip applies largely to cleft palate. Cleft palate may be single or double. The fissure may involve only the soft palate and uvula, and this is the most common form, it may involve more or less of the hard palate, or, in rare cases, the palate alone. It may or may not be associated with hare-lip.

Pathological Anatomy.—Deformity results from the failure of perfect fusion between the hard palate and the intermaxillary bone, as well as imperfect union of the vomer and the velum.

Symptoms.—The child has marked difficulty in both nursing and swallowing, as the milk may regurgitate through the nose. The infant, breathing, as it does, through the mouth, is exposed to the danger of infection of the nose, throat, and lungs. Speech is always impaired; the voice has a nasal tone, and many of the consonants are imperfectly articulated. Owing to the mechanical difficulty in feeding these

children, they are often small, ill-nourished, and delicate, and their nutrition and general care become a matter of the first importance. It is absolutely necessary that the nasal pharynx be kept clean, yet care must be taken never to exert more than the minimum amount of force, since these mucous membranes are easily injured mechanically. They are also especially liable to be affected with thrush, which may be a serious complication, particularly if the child is weak and frail. A weak alkaline and mildly antiseptic wash may be employed to keep the nasal pharynx clean, especially before and after feeding.

Prognosis.—The prognosis depends, as does that of hare-lip, on the physical condition of the child. The mortality from the operation is 3 per cent.

Treatment.—Many surgeons prefer to operate as soon after the thirteenth or fourteenth month as the child's physical condition will permit, and the operation should always be performed before the beginning of the third year. If a large fissure remains after the first attempt at closure, a second operation is indicated; but a small fissure can often be closed by local applications. It is claimed that, if possible, the repair of a cleft palate should be accomplished before the period when the child ordinarily begins to talk, and that, as a result of this, systematic exercises directed toward improvement of the speech are a valuable aid which should, moreover, be begun before bad habits of speech are formed. On the same principle, it is important that everything possible should be done to improve the general health and nutrition of the child, both before and after operation. Breast milk is, of course, the best food for these infants, and it may be necessary to feed them with a spoon, medicine dropper, or a Breck feeder. An admirable arrangement consists in attaching to the ordinary nursing nipple a flap which more or less perfectly fits the roof of the mouth and assists, partially at least, in closing the fissure.

During the period of residence in the hospital, both before and subsequent to operation, gavage is of advantage, since by this method a larger amount of food can often be introduced into the stomach. After leaving the hospital it is applicable only to those cases where a trained nurse is in attendance.

CONGENITAL MALFORMATIONS OF THE TONGUE.

Tongue-tie.—This condition consists in a shortening of the frenum of the tongue to such an extent as to render the protrusion of the tongue beyond the lips difficult, and it is also bound down to the floor of the mouth by the unnaturally tight and short frenum. Articulation may be more or less interfered with, and sucking may be imperfect. It is a common experience with all physicians to be consulted in regard to children two or three years of age who are supposed by their parents to be tongue-tied because they do not talk. In the large majority of such cases, the failure to talk is not in any way connected with the tongue, but is significant of some mental condition

or deaf mutism. The treatment of tongue-tie is simply the elevation of the tongue by passing the slit of a groove director over the frenum, and nicking the frenum slightly with a pair of blunt scissors. If the cut is made close to the gum the artery is easily avoided.

Macroglossia.—Macroglossia, giant tongue, or hypertrophy of the tongue, is usually a symptom of some general pathological condition such as cretinism, idiocy, mongolism, and acromegaly. In some patients, however, it may be due to a more or less advanced hemangioma or lymphangioma of the tongue, according to whether the cavities of the tongue show enlarged bloodvessel involvement or enlarged lymphatics. This condition is usually progressive, the tongue steadily growing larger until finally, owing to its great size, it protrudes more or less from the mouth, and plainly shows the marks made by the pressure of the teeth. It may become so large that swallowing and even respiration are interfered with. A few cases are on record where increase in size has been the result of hypertrophy of the muscular fibers.

Thyroid extract is, of course, the proper treatment in cases due to cretinism. In other conditions the size has been reduced by the thermocautery or excision. If the entire tongue is involved, portions of the growth may be removed by successive operations. On account of the danger of hemorrhage, the lingual arteries should be ligated before operation.

Ranula.—A ranula is situated beneath the tongue, and is caused by the duct of the sublingual glands or of the mucous glands becoming occluded. The secretions of these glands are consequently retained, and the cyst or ranula develops. As the cyst increases in size it causes a protrusion or swelling of the mucous membrane beneath the tongue, which is gradually lifted and pushed upward. The swelling presents a bluish appearance, is translucent, painless, and gives a feeling of fluctuation. It contains a thick, viscid, colorless fluid. The cure of the ranula is best accomplished by its total extirpation, although it may often be brought about by cutting out as large a portion as possible of the anterior wall, and touching up the interior of the sac with tincture of iodine or nitrate of silver, five grains to the ounce. Simple incision and evacuation of the sac is not to be recommended; such an incision, as a rule, rapidly heals and the sac refills.

CONGENITAL DISEASES OF THE NECK.

Fistulæ of the Neck.—*Etiology.*—The incomplete fusion of certain primitive or embryonic structures results in these malformations. Lateral fissures are the result of imperfect development in the branchial clefts, and a median fissure is associated with imperfect development of the median lobe of the thyroid. Heredity, alcoholism, and amniotic bands are operative here as causative agents, just as they are in hare-lip and cleft palate.

Symptoms.—At the middle or along the sides of the neck small fistulæ are occasionally found which exude, normally or under pressure,

a whitish liquid. The lateral fissure may end in a cul-de-sac, or open internally in the neighborhood of the tonsil. The external opening coincides with the anterior margin of the sternocleidomastoid muscle. The fistulæ in the middle line of the neck, if not blind, open under the tongue.

Treatment.—The treatment consists in the total extirpation of the fistulæ and the cyst, as experience has shown that any operation or treatment other than this is useless.

Congenital Cystic Lymphangioma of the Neck.—**Pathological Anatomy.**—This malformation is composed of dilated lymphatic vessels, which become cysts, and adhere closely to the neighboring skin, muscles, and bloodvessels, the tumor usually increasing rapidly in size.

Symptoms.—The tumor develops in the side of the neck, and is composed of many cysts. It tends to grow downward below the clavicle, and may even penetrate into the deep tissues of the larynx and esophagus. As the tumor becomes larger, difficulty in swallowing and oppression in breathing develop, which become more marked as the pressure from the enlarging mass increases.

Prognosis.—The prognosis is unfavorable as, unless the diagnosis is made early and the tumor completely removed, recurrence is likely.

Treatment.—Its complete extirpation is always rendered difficult on account of its close adhesion to all neighboring structures; but only radical extirpation will bring about a cure, and it is therefore most important that the diagnosis be made early, since, owing to the rapid growth of these tumors, the difficulties and dangers attending radical operations increase with any delay. If the operation be only partial or incomplete, it will be followed by relapse and the continued new growth of the mass. As there is no communication between the cysts, their formation being much like that of a bunch of grapes, puncture accomplishes little.

Congenital Torticollis.—**Etiology.**—Heredity is undoubtedly a factor, as are also intra-uterine inflammations and adhesions. A traumatic injury to the sternocleidomastoid muscle during delivery may be followed by slow interstitial inflammation in the muscle tissue, the contraction resulting therefrom producing the typical deformity.

Symptoms.—The position of the head is caused by the contraction of the affected sternocleidomastoid muscle. The head is rotated toward the unaffected side, and deflected toward the diseased side, and is held fixed in this position. The sternal portion of the muscle produces the rotation of the head, and the clavicular the deflection.

Diagnosis.—The muscle can be felt as a firm hard band. It should be remembered that diseases of the upper cervical vertebræ and occipital periostitis are capable of producing torticollis, and that the diagnosis of tuberculosis in this upper spinal region can be often corroborated by *x*-ray plates.

Treatment.—The treatment of congenital torticollis is operative; either open section or partial extirpation of the diseased muscle is the operation of choice. Open section of the muscles is to be preferred

to subcutaneous section as being more thorough and less apt to injure the large bloodvessels. The lower portion of the muscle, dividing as it does into two parts, is the place of election for operation. The spinal accessory nerve is in this region, and care must be taken to avoid injuring it. After operation the head is fixed by pads and bandages in a position of overextension. If any scoliosis has developed as a result of the torticollis, it must receive appropriate treatment. It seems almost unnecessary to urge the necessity of early operation in congenital torticollis. During the first few weeks of life a pad may be worn and daily massage employed, and if, at the age of three months, there is not distinct improvement an operation should be performed. Firm and fixed scoliosis has not occurred at this period, and the subsequent result is better and treatment is simpler if the operation is performed at this early age. Torticollis is often only a symptom, and in these cases it is necessary to treat the underlying cause as well as the torticollis.

CONGENITAL MALFORMATIONS OF THE ESOPHAGUS.

Congenital malformations of the esophagus include absence of the esophagus, in whole or in part, stenosis of varying degree, dilatation, a bending or twisting of the canal, and fistulæ communicating with the trachea.

Symptoms.—Vomiting is, of course, the cardinal symptom. If the stenosis is of such degree as to permit the slow trickling of fluids through the passage, a small amount of liquid nourishment may be ingested and retained; whereas, if a large amount is given, a portion of it is always vomited. If solid or semisolid food blocks the small aperture in the esophagus, the giving of such food may be followed by vomiting of almost all nourishment so long as the more solid material continues to occlude the small esophageal opening. Congenital obstruction is undoubtedly much more common than was formerly supposed, and cases of persistent vomiting in young children should be studied with this possible diagnosis in mind.

Treatment.—The treatment is most unsatisfactory. Stenosis of moderate degree has been successfully treated by dilatation; for the other conditions gastrostomy is necessary, after which an attempt can be made to feed the child through the artificial gastric fistula. If the infant's strength will permit, an effort may be subsequently made to overcome the congenital defect by surgical measures. As the operation, except in cases of moderate stenosis, usually has to be performed when the infant is only a few days old, the risk attending it is of course very great. The condition of cardiospasm may be congenital; it is not a malformation, and can usually be treated successfully; still it may in early life produce symptoms identical with those of organic stenosis, hence its existence should be borne in mind.

Meckel's Diverticulum.—Meckel's diverticulum is a persistence of the ductus omphalomesentericus.

Etiology.—This duct, which normally exists in the fetus and connects the intestine and the vitelline membrane, closes at about the end of the second month. The canal or duct arises from the lower portion of the small intestine, about twelve inches above the ileocecal valve. It may be a cul-de-sac a few inches long, or it may remain open as far as the umbilicus, and permit the passage of fecal matter. The mucous membrane of the duct may prolapse through the umbilicus, producing a small tumor. Instead of persisting as an open or closed duct, this congenital condition may exist as a cord extending from the lower ileum to the umbilicus, and a portion of the bowel may be caught and constricted by this cord, and intestinal obstruction and strangulation produced. It must be remembered that this condition is possible in child or adult life as well as in infancy. In those children in whom the duct remains open a discharge of mucus or feces will occur at the umbilicus. If the umbilical end of the duct is obliterated a cyst is formed at the umbilicus. This is reddish, globular, and usually contains a whitish secretion. Occasionally, if the lumen is sufficient, the intestine may be drawn into this cyst, and intestinal obstruction thus develop. A twisting of the pedicle of the duct may lead to intestinal perforation and resulting peritonitis; an intestinal stenosis from the prolapse of the ductus into the lumen of the intestines has been observed, and intussusception may be produced by inversion of the duct. Perforation of the diverticulum with resulting peritonitis may be induced by ascarides in its lumen, or fecal impaction, or any cause which may produce localized inflammation and necrosis. Adenomata may develop from the tissues of the duct, and present themselves as a tumor of the umbilicus.

Symptoms.—Symptoms of intestinal obstruction or strangulation are possible as a result of Meckel's diverticulum. They may be caused by a prolapse of the diverticulum into the bowel, or the band-like cord which may exist, instead of the more or less patulous duct, may cause a sufficiently tight constriction of a loop of intestine to produce strangulation.

Diagnosis.—The presence of a fecal discharge at the umbilicus is strongly suggestive of Meckel's diverticulum. In a fistula of the urachus, the direction of the fistula is toward the bladder, and the reaction of the discharge is acid. A fistula of the urachus is also indicated if any of the normal ingredients of urine can be demonstrated in the discharge. An alkaline reaction suggests a connection with the lower bowel. If any connection with the bladder exists, methylene blue injected into the fistula will later show itself in the urine. In fistula of the urachus prolapse of the mucous membrane may also cause a tumor at the umbilicus, but this is comparatively rare.

Prognosis.—It depends upon the patulous condition of the duct; the persistence of the duct as a cord or band; the presence of a tumor at the umbilicus and the contents of this tumor; and the healthy or diseased condition of the walls of the duct.

Treatment.—The removal of the diverticulum is the proper treatment. An incision made along the median line of the rectus permits its complete removal and the invagination of the stump. The complication of tumor at the umbilicus must, of course, be treated surgically.

HERNIA OF THE UMBILICAL CORD.

In the development of the fetus the intestines lie outside the abdominal walls, the closure of the abdomen being brought about by the fusion of two sides of the abdominal walls. In this developmental process the bowel appears in the umbilical cord, and is covered by peritoneum, Wharton's jelly, and amnion.

Symptoms.—The tumor is usually of small size, although occasionally it is quite large. It contains, perhaps, a loop of intestine, Meckel's diverticulum, or it may, if of large size, contain one or more abdominal organs, possibly the liver, spleen, pancreas, or kidney. The mass is situated at the umbilicus, and the umbilical cord is a part of, and extends beyond, the tumor.

Prognosis.—The prognosis is fatal unless the condition is of such a nature that it can be immediately relieved by operation. It is dangerous to ligate these tumors without first opening them and assuring one's self that the mass does not contain any coil of intestine. If the tumor is small, the method advised by Olshausen of making an incision in the healthy skin surrounding the tumor, detaching the amnion from the inner membrane in Wharton's jelly, reducing the hernia, and closing the skin over it has been practised with success. In this operation the peritoneum is not opened. Large tumors, if containing only intestine, may be operated upon, and at times successfully, by opening the hernial sac and returning the contents within the abdomen. If the tumor contains any large part of one or more of the abdominal organs, the operation is both a dangerous and difficult one, and infection of the peritoneum frequently results. Cases have been reported where the cautery has been used successfully to remove the middle lobe of the liver which has been found in the hernial sac.

UMBILICAL HERNIA.

Normal development causes the complete closure of the abdominal walls with the exception of that small portion at the navel which must remain open in order that the umbilical vessels be patulous. As the cord desiccates and is detached, this small opening has a normal tendency gradually to close, and, after the cord has fallen, the only use of the abdominal binder is to hold a small compress over this still unclosed umbilicus. As the child grows very rapidly at this period, the tissues fill up this unclosed portion, and it soon becomes obliterated. If the infant is premature, frail, or badly nourished, closure may not take place; this is more common in girl infants. Severe crying, coughing, or marked abdominal distention also has a tendency to

prevent closure, as does traction of the umbilical cord. The coverings of the bowel in the hernia are skin, transverse fascia, and peritoneum.

Symptoms.—This form of hernia rarely produces any symptoms. It may, however, gradually increase in size, especially if constipation, cough, and much abdominal distention are present, and is often much increased later in life by pregnancy.

Treatment.—Prophylaxis embraces the wearing of a snug, but not tight, abdominal binder, with a small retaining pad over the navel. This should be worn at least during the first two months, or longer if

Fig. 13.—Adhesive plaster applied for umbilical hernia.

necessary. As soon as the hernia is discovered, it should be returned within the abdominal cavity and retained by doubling in over the umbilicus two longitudinal folds of tissue, one from either side of the abdominal wall and on opposite sides of the umbilicus, and retaining these folds in place with two broad strips of zinc oxide adhesive plaster. If properly applied, these strips will remain in position for some days, and will produce very little irritation of the skin. They should extend from one side of the outer abdominal wall across to the other, that is, about half way around the child's body (Fig. 13), and this method

of treatment should be continued for two or three months, or longer if the hernia persists. It is much more liable to result in a cure than the application of the ordinary umbilical truss with a conical projection which fits into the opening and really tends to prevent its closure. A flat object, such as a coin, may be covered with zinc oxide plaster, and retained over the umbilical opening with adhesive strips. The skin must always be kept absolutely sweet and clean. It is extremely difficult—in fact, almost impossible—to retain in proper position any form of apparatus on a child who has reached the walking age. In older children many of these umbilical herniæ, if small, gradually tend to close as the child grows and develops, and it has rarely, in my experience, been necessary to resort to surgical treatment. However, if the hernia is large and especially if, in a girl, it tends to enlarge, a surgical operation is undoubtedly the proper treatment.

CONGENITAL DILATATION OF THE COLON.

Congenital dilatation of the colon, or Hirschsprung's disease, is a rare congenital affection.

Pathological Anatomy.—The colon is longer than normal, is convoluted, greatly dilated, and the muscular walls of the dilated portion are usually much hypertrophied. In some cases the transverse portion of the colon is affected, and in others the descending portion of the colon and the sigmoid flexure. The dilatation produces constipation, and the materials retained in the bowel undergo fermentation and decomposition which tend still further to increase the dilatation.

Symptoms.—The most marked symptom is constipation, which may be present at birth, or, if not, develops soon after, and is shortly followed by abdominal distention. The infant, and especially the older child, will often go for a number of days without a stool, this constipation being followed by a conservative diarrhea which partially clears out the bowel, the stool consisting of hard, lumpy masses of fecal matter and much mucus. Enemata are only fairly successful in bringing away the retained fecal mass, and large doses of laxatives usually become necessary. The infant, and later the child, is weak and emaciated, and the contour of the enormously distended abdomen often changes in a curious way while it is being examined, as a result of the gas moving from one portion of the bowel to another, and the coils of enlarged intestine can often be outlined through the thin abdominal walls. After the administration of purgatives, large masses of feces are passed with much flatus, and the abdominal distention is, to a slight extent, temporarily lessened. Intestinal toxemia of more or less marked degree is often present, and in most cases that come to operation or autopsy the mucosa of the bowel shows superficial ulceration. A stricture below the dilatation has not been observed. The child becomes anemic and cachectic, and in infants severe nervous symptoms are not uncommon. The exact size, shape, and location of the colon

can be absolutely mapped out by giving the child a bismuth meal, or injecting the colon with bismuth, and then taking *x*-ray plates.

Prognosis.—These infants are all under weight, and, owing to their poor physical condition, many of them die of inanition or secondary diseases. If the dilatation is slight, they may reach adult life, but the majority of the severe cases die in infancy. A number of cases are on record in which the dilated colon has been successfully removed by operation.

Treatment.—Medical treatment can, at least, alleviate the symptoms, and make the child more comfortable. The most important object to be accomplished is to build up as well as possible the child's general health. Fresh air, an easily digested and nourishing diet, selecting those foods that are best adapted to the child's digestion, will help to maintain bodily weight and vitality. Daily irrigation of the colon with two quarts of warm salt solution is a valuable aid in overcoming the constipation, and tends to cleanse, at least, the lower portion of the large bowel. A daily laxative, such as podophyllin or cascara, is of service, although it is advisable to change the laxative from time to time, as the child will otherwise require increasing doses. Abdominal massage, especially over the colon, will assist in promoting peristalsis. Give the child plenty of water to drink, and establish regular hours for him to go to the toilet. The habit of placing small children at a certain hour each day on the chamber is, in my experience, the most essential part of the treatment of constipation, no matter what its cause. If the above outlined medical treatment were instituted as soon as the first evidences of constipation were observed, it would probably be effective in a small proportion of cases. This is, however, rarely possible except among the most intelligent classes, and the failure of the child to gain in weight and strength, or its progressive loss in strength, often makes a surgical operation the only procedure.

Formerly an artificial anus was the usual operation, the opening being made in the bowel above the dilated portion. This puts largely at rest any portion of the colon which is abnormally dilated, and the physiological rest enables it to regain in part its normal vigor and tone. A second operation is then performed to close the artificial anus and reëstablish the normal passage of the feces per rectum. Recently the tendency has been to remove the dilated portion of the colon, and while it is a very serious and radical operation it has, nevertheless, been performed successfully in a few cases. Some surgeons divide the operation into two stages, in order to lessen shock. It is to be sincerely hoped that with advance in modern surgery this operation will soon be established on a firm basis.

ATRESIA OF THE BOWEL.

The principal etiological factors are peritonitis, volvulus, and syphilis. Fetal peritonitis manifests itself by an inflammatory exudation

resulting in adhesions formed between coils of intestine. Volvulus is the probable cause when atresia is found in several different portions of the bowel.

Location and Variety of Malformations.—The most common location is at the anus. The anal opening may be the only part that is defective, everything else being normal. The normal anal opening may be present, and nothing but a delicate septum interfere with the normal passage of feces. In other cases the location of the anal orifice may be simply represented by a dimple, the rectum ending in a cul-de-sac. This cul-de-sac may be situated very close to the normal anal opening, or it may be so far removed from this position that a long and, perhaps, difficult dissection may be necessary to establish an anal opening. The intervening space between the two points of atresia of the bowel may be replaced in either the entire or a portion of the distance by a fibrous cord.

In another form of malformation, the anal opening and adjacent portion of the rectum are present, but end in a cul-de-sac. The distal portion of the bowel may immediately join this cul-de-sac, being, perhaps, separated by only a delicate septum, or a considerable space may intervene between the two extremities of the bowel. The rectum may open internally into the bladder, or a fistulous opening may connect the rectum and bladder; in other cases, the rectum is connected with the urethra or with the vaginal vestibulum.

Symptoms.—The symptoms vary according to the location of the obstruction. If the fecal contents do not escape through some fistulous tract, the condition invariably produces constipation, followed by dilatation of the portion of the bowel immediately above the obstruction, and subsequent vomiting. If the obstruction is in the small intestine, especially if in the upper portion, vomiting is an early symptom, and is persistent. The portion of the bowel below the obstruction is emptied of meconium, and the subsequent stools contain nothing but mucus. These cases are conspicuous by the rapid loss of flesh, increasing asthenia, and early death. With the exception of the lower end of the rectum, the upper portion of the small intestine is the most common seat of intestinal atresia. If the obstruction is only partial, the symptoms are less pronounced, and the child's life, unless a surgical operation be performed, depends upon the amount of food that trickles through the constricted portion. The constipation in these cases is, of course, in proportion to the degree of stenosis, which limits the amount of food that passes on into the lower bowel. The portion of the bowel above the atresia is always distended and dilated, while that below is small in calibre, and generally poorly developed. If the urine at any time contains fecal matter, a communication between the rectum and the bladder will be naturally suspected.

Diagnosis.—An absence of the normal anal opening should be discovered during the first bathing and cleansing of the infant; otherwise, there is nothing at birth that would cause one to suspect the malformation. Vomiting soon after birth associated with constipation and

abdominal distention suggests an obstruction high up in the small bowel, while constipation followed by abdominal distention and vomiting suggests an obstruction low down in the bowel. In atresia, where the anal opening is present, and a small portion of the adjacent lower bowel also, a finger inserted in the rectum discloses the fact that the bowel ends in a cul-de-sac. If the examining finger discloses a mass which presses down against it on crying, the amount of tissue lying between the upper and lower portion of the bowel may probably be more or less accurately estimated.

The diagnosis may be confused by the fact that the obstruction is present at more than one point, and a bismuth meal and x-ray plates may be of assistance in diagnosing this condition. Possibly they may assist in locating the site of the lesion, particularly in those cases where a fistula allows some of the fecal matter to pass beyond the point of obstruction, or may aid in locating the several points of, possibly, partial obstruction, atresia of the bowel not being complete, but the condition being one of stenosis. Injections of bismuth per anum may also be of advantage in those cases where the lower portion of the large bowel, extending upward from a normal anal orifice, ends in a cul-de-sac.

Prognosis.—The infant at birth has, of course, little power to endure a severe surgical operation, and, except in those cases where the obstruction is situated near the anal region, the condition is usually fatal. If, however, the bowel contents empty externally by means of a fistula, the prognosis as to life is good.

Treatment.—With the exception, however, of such cases, all patients will require a surgical operation to effect a cure.

MALPOSITION OF THE BOWEL.

Malpositions of the bowel are now more often recognized and understood, owing to the taking of x-ray plates after bismuth meals, but one must not be too positive in regard to the permanent position of any particular portion of intestine after one x-ray examination. I have, in a number of cases, seen x-ray plates which showed portions of the bowel evidently much out of position as a result of an unnaturally long and loose mesentery, where, in a plate taken later, the bowel had practically returned to its normal position. A portion of intestine situated temporarily out of its normal position, may or may not be the cause of symptoms, and even if one is reasonably sure that certain symptoms are the result of the malposition, it is wiser in the majority of cases to avoid, if possible, surgical interference, in the hope that the bowel may return to a more normal position and the symptoms disappear. If the displaced portion of the bowel is fixed in its new position and produces no alarming symptoms, it should be carefully watched; if the symptoms persist and are of a sufficiently severe type to warrant it, surgical interference is indicated.

The bowel may be in malposition in a congenital umbilical hernia

which may contain more or less intestine; also in a diaphragmatic hernia which may contain coils pushed through the diaphragm up into the thorax.

EXSTROPHY OF THE BLADDER.

In these patients the bladder is situated outside of the abdominal wall, and the malformation is the result of arrested development which creates a fissure in the abdominal wall. It is by some believed to be the result of the bursting of the allantois, caused by an unusual amount of secretion before the lower aperture for its escape had been formed.

FIG. 14.—Exstrophy of the bladder and epispadias in a child five months old.

Pathological Anatomy.—The anterior part of the abdominal wall is absent, and there are often other congenital malformations, among which is an absence of union at the symphysis pubis. The fissure may extend to the urethra with epispadias, fissured scrotum, and undecended testes in the male, and open urethra and fissure of the labia and clitoris in the female.

Symptoms.—The mucous membrane protrudes through a fissure in the abdominal wall; it is of normal color, is folded into ridges, and is continuous with the skin of the abdomen. Any increase of intra-abdominal pressure causes a still further protrusion of the mass. The openings of the ureters are situated in the lower portion of the tumor. The urine dribbles continuously, and causes a most annoying irritation of the skin. This, associated with the continual odor of urine, makes all patients who have arrived at an age when they can appreciate their condition only too willing to submit to a surgical operation.

Prognosis.—Many of these children, if unoperated upon, die from infection of the bladder and kidneys. A number have been successfully operated upon both in child and young adult life.

Treatment.—The treatment is, of course, purely surgical; either the covering up of the defect in the anterior wall with a skin flap and the formation of a new urethra, or the implantation of the ureters into the intestinal tract, is the operation now most popular. Very favorable results have followed the transplantation of the ureters into the rectum; the patient develops a control of the urine which may last for hours during the day or night. The results are often so satisfactory that it is wise to give all cases the benefit of an operation.

Fig. 15.—Exstrophy of the bladder in a child seven months old.

UNDESCENDED TESTES; CRYPTORCHIDISM.

The testicles develop in the abdomen, and usually descend into the scrotum in the last few weeks of intra-uterine life. It is not uncommon, however, to find one or even both of them in the abdominal cavity in an infant otherwise normal and healthy and born at full term. Ordinarily we find that one testicle has descended into the scrotum while the other is still retained in the inguinal canal. As a rule, it descends in the first few weeks of life, its retention having produced no symptoms, and after its descent the inguinal canal usually closes normally. It may be retained, however, either within the abdominal cavity, or at the entrance, or in some portion of the inguinal canal for months or years; occasionally, possibly owing to its increased size and weight, it descends into the scrotum. This may occur at any time in the child's life up to the age of ten years. The longer it remains out of its normal position, the less likelihood is there of its descending into the scrotum. In the inguinal canal it is exposed to traumatism, and as puberty approaches it is apt to cause a nauseating pain; often, if out of its normal position, it does not develop properly, and an

7

undescended testicle is more liable to become diseased than one in normal position. If it remains in the canal, it predisposes to hernia.

Diagnosis.—The absence of the testicle in the scrotum can hardly escape the notice of the mother or nurse. By passing the little finger up the inguinal canal, the testicle can usually be felt either in the canal or at the internal opening of the abdomen.

Treatment.—In a young infant it is, in some cases, possible by gentle manipulation to push the testicle downward, perhaps into the scrotum; this may assist in restoring the organ to its normal position, and, if performed gently, does no harm and may, possibly, do good. If at the age of ten years the testicle has not descended, the recognized operation of the day is to transplant the organ into the scrotum, which is usually possible of accomplishment. If, however, in any particular case, this operation cannot be performed, it is wiser to return the organ into the abdominal cavity. If the testicle becomes diseased, and it is a well-known fact that in its unnatural position it is more likely to undergo certain degenerative changes, it should be immediately removed; but its removal should never be advised unless it has undergone some such change.

HYDROCELE.

Hydrocele, of which there are several forms, is a collection of fluid in the tunica vaginalis, and is a very common condition in the infant. It is caused by a failure of fusion of the pars vaginalis of the peritoneum, and in about 50 per cent. of newborn infants the processus vaginalis is still open.

Congenital Hydrocele.—In this variety the fluid occupies a position in the umbilical canal between the peritoneal cavity and the tunica vaginalis. The fluid will flow back into the abdominal cavity if the child is placed in the horizontal position and a moderate amount of taxis employed.

Hydrocele of the Tunica Vaginalis.—This is the most common form and is irreducible. The inguinal canal becomes closed off from the abdominal cavity, but still communicates with the tunica vaginalis. The tumor is oval, firm to the touch, translucent, and usually one-sided. Fluctuation can generally be obtained. Ordinarily the testicle cannot be found by manipulation.

Encysted Hydrocele of the Cord.—This consists of a small amount of fluid, usually about one or two drams, surrounding a portion of the cord. There is no communication with the tunica vaginalis testis, and the tumor is irreducible. The same condition when found in girls is called encysted hydrocele of the canal of Nuck, and requires the same treatment.

Diagnosis.—About the only condition with which hydrocele is apt to be confounded is an irreducible hernia. A hard, tense, irreducible tumor, which has been present for some time, which has not been

and cannot be reduced, which produces no symptoms, and is not painful on pressure, is a hydrocele.

Treatment.—The simplest, and often the only treatment, is to puncture the cyst and draw off the fluid, which is best accomplished with a small aspirating needle. If after three such withdrawals cure has not resulted, surgical measures should be resorted to. The best treatment for the reducible form of hydrocele is to return the fluid by means of gentle taxis into the abdominal cavity, and then apply a truss with the hope that pressure may not only prevent the return of the fluid, but also gradually produce obliteration of the canal. Injections of iodine into the hydrocele sac are not to be recommended; the subsequent inflammation is often very severe, and in those patients in whom the canal is still open, the procedure is attended by a certain amount of danger. Phimosis which causes tenesmus and consequent increase of intra-abdominal pressure should be operated upon. The surgical operation, of choice, is extirpation of the hydrocele sac.

Fig. 16.—Hydrocele in an infant aged six months.

OBLITERATION OF THE BILE DUCTS.

The common hepatic or cystic duct may be obliterated as the result of imperfect development. A narrowing of the lumen of one or more of these ducts results in inflammation of their lining membrane which finally stops entirely the flow of bile. Either the common duct alone may be affected, or the hepatic or cystic duct may be the one involved. Intra-uterine peritonitis is probably secondary to the atresia of the bile ducts. In about 10 per cent. of these cases there is a history of syphilis. The gall-bladder is small, often only rudimentary, although in atresia of the common duct it may be greatly distended. The liver is usually much enlarged and shows the changes due to chronic interstitial hepatitis. The spleen is enlarged, and the bile duct may be reduced to a fibrous cord.

Symptoms.—All the tissues and organs of the body are deeply jaundiced, and hemorrhages beneath the skin, with vomiting of blood and blood in the stools, occur in some cases. Jaundice is the most marked symptom; it develops usually within a few days after birth, and progressively becomes more pronounced. The urine is very high-colored owing to the presence of bile pigment. Shortly after birth small stools composed of meconium may be passed; later the stools are clay-colored or white, and examination shows them to contain no bile except in those cases where the blocking is limited to the cystic duct. The child may be well-nourished at birth, but soon loses flesh, and death from inanition occurs in the course of a few weeks to four months. Marked abdominal distention with toxemia, as evidenced by convulsions, is a not unusual symptom, and convulsions are a not uncommon cause of death in those infants who live but a few weeks.

Treatment.—This exerts no influence upon the symptoms or the course of the disease.

SPINA BIFIDA.

Spina bifida consists of a fissure in the spinal canal with a hernia of some portion of its contents, the portion that protrudes forming a tumor posteriorly. Fluid is always present in the tumor. It is one of the most common congenital malformations. The walls of the tumor, according to the intra-uterine period in which the defective development occurs, may contain a portion of one or of all the following structures: Spinal cord, nerves, meninges, and vertebral arches. The tumor, in rare instances, instead of appearing posteriorly through a fissure in the bony spine, may project anteriorly into the thorax or abdomen. These tumors resemble hernia of the brain in pathology and in mode of development. There are several varieties of spina bifida, although all present two features in common: a defect in the bony spinal column and a lesion in the spinal canal. All result from an error in development. The spinal canal closes at different places at varying stages in the development of the fetus, the lower lumbar and upper cervical being the last portions to unite, and these two locations are the places in which the deformity usually occurs. In all forms *x*-ray plates will show the bony defect.

Spinal Meningocele.—In this variety of spina bifida a tumor is found in the cervical or sacral region. It is translucent, globular, and pedunculated. As a rule, there is no disturbance of sensation or motion. The tumor contains only the spinal meninges and cerebrospinal fluid. The fluid is in the subarachnoid space. The opening into the spinal canal is small, and the skin over the tumor is firm and healthy. This form may become quite large, but spontaneous rupture is not very likely to occur owing to the healthy condition of the overlying skin. Patients with this form of tumor may live for years— in fact, well into adult life. It is the least dangerous form to operate upon, and the easiest to cure by an operation.

Myelomeningocele.—This is the most common form of spina bifida, especially in the lumbosacral region. The fluid is in the anterior arachnoid cavity. The tumor is not large, but has a large base, is soft, flat, elastic, and not pedunculated. Often it is covered only in part by skin; the central portion of the tumor being covered by a thin tissue, commonly showing ulceration. A depression in this central part marks the position of the attachment of the cord. The tumor is composed of the prolapsed spinal cord. Paralysis of the bladder, rectum and lower limbs, and deformities of the foot may accompany this form.

Myelocystocele.—This is the rarest form. It is found in the dorsal, lumbar, or sacral regions, and may be combined with abdominal fissures and club-foot. The tumor is round with a wide base, is elastic, transparent, and fluctuates. It may also be associated with hydrocephalus, and in this form there is a dilatation of the central canal of the cord. The wall around the sac is composed of medullary substance. If hydrocephalus is present the prognosis is less favorable, and pressure upon the anterior fontanelle causes an increase in the size of the tumor. There is usually no paralysis. The skin covering the tumor is very thin.

Spina Bifida Occulta.—This consists of a slight fissure or defect at the inferior end of the spinal canal; it is covered by normal skin. Here, as in the other forms, the x-rays will reveal the bony defect. A small tumor may show itself or none may exist, the spinal defect being indicated only by a slight depression or dimple in the overlying skin. A swelling in the sacral region associated with any neighboring trophic changes, or paralysis of the rectum, bladder, or lower extremities suggests a probable spina bifida occulta. This form is not incompatible with a long life (Fig. 17).

Symptoms.—A tumor is always present at birth, and is usually tense and fluctuating. It is either directly in the median line or slightly to the side of this line. The skin covering the tumor may be well nourished or atrophic. The usual location is in the lumbosacral region; it may attain a very large size if the skin covering is healthy. If, however, the skin covering is thin, rupture and early death generally result.

All the varieties of spina bifida contain fluid which may be, at least partially, returned to the spinal canal by gentle, continuous pressure; if the sac is relaxed and soft, any contents may be clearly and firmly compressed and possibly outlined. The tension within the sac is often increased by holding the child in the upright position, especially if it kicks and screams. If the tumor is translucent it may be largely reduced, and if no central mass is palpable it points to the absence of any prolapsed portion of the spinal cord. Untreated cases sooner or later develop infection of the skin covering the sac, with subsequent spinal meningitis and death. Ulceration of the skin may take place, with rupture of the sac, the rupture healing, but again breaking open, and finally death from infection results. Paralysis, as it depends upon the amount of injury done to the cord or the existence of the

cord in the tumor, is rarely present in spinal meningocele. In a cervical tumor paralysis is rare, being much more common in the lumbosacral forms. Paralysis, to a certain extent, depends upon the location of the tumor; if low down in the sacral region and only the cauda equina is involved, the bladder and rectum may escape, and the legs be only partially paralyzed. A very small spinal opening may occasionally be closed by inflammatory exudate, resulting in a spontaneous cure; this, however, occurs so rarely that it is not to be expected.

Fig. 17.—Spina bifida occulta.

Trophic changes, as shown by ulceration, are not uncommon. Motion and sensation may be normal or disturbed in the legs, and incontinence of urine or feces may be present. Other deformities of the body are not uncommon. A depression marks the centre of the myelocystocele, and the fissure in the entire vertebral column is usually palpable.

Diagnosis.—The different forms of spina bifida are difficult to diagnose but the location of the tumor is of some assistance. A menin-

gocele is apt to be translucent and pedunculated, and shows no evidence
of spinal fissure; it is important to differentiate this form on account
of the better results following operation. Myelomeningocele is apt
to be associated with paralysis, a depression exists in the centre of the
tumor, it has a broad base, and on palpation the bony fissure can usually
be distinctly felt. The tumor is rarely large, and is not apt to be
pedunculated. Myelocystocele is the form most apt to be asso-
ciated with hydrocephalus, and pressure upon the anterior fontanelle
increases the size of the tumor.

Prognosis.—The prognosis varies according to the character of the
contents of the tumor, and the integrity of its skin covering. In
spinal meningocele operation offers a reasonable hope of cure, provided
hydrocephalus does not exist. In other forms, if there is associated
paralysis or hydrocephalus, the prognosis is grave, although the
existence of paralysis does not necessarily contra-indicate operation.
Hydrocephalus may develop after a successful operation.

Treatment.—All tumors should be protected from pressure. The
skin covering them should be kept clean, and the tumor supported
by an air cushion or rubber ring. A healthy well-developed child
should be operated upon, but a wasted, atrophic infant is less able to
bear operation. If the tumor is well covered by healthy skin, and not
increasing rapidly in size, it is a good plan to wait until the child is
six months old, and is better able to withstand the shock of operation;
at the same time, in my experience I have found it much better to
operate at once if the infant and tumor are not receiving the best care
hygienically and dietetically. The usual operative procedure consists
of the excision of the sac, and a plastic operation to cover up the defect.
Recovery as to life is very common, but paralysis or trophic symptoms
may persist.

In estimating the possible benefits from operative treatment, the
results commonly depend upon the strength of the patient and the
condition of the sac. The removal of the entire growth by ligation
or by injections is not warranted. Paralysis does not necessarily
contraindicate operation, as, in a few reported cases, a cure of the
spina bifida has followed surgical interference, and the paralysis has
partially improved.

ATRESIA OF THE VAGINA, LABIA, AND URETHRA.

Atresia of the vagina is usually the result of an imperforate hymen
or a septum. It may not be discovered until the time for menstruation
arrives. Perforation or, if necessary, removal of a portion of the
septum, and packing the opening with gauze is usually all that is
necessary to effect a cure. If the condition is discovered in infancy,
the obstruction can ordinarily be easily overcome by the passage of a
probe and packing the opening with gauze. At this period the tissues
are only slightly vascular and the membrane extremely delicate;
later in life the septum becomes tougher and much more vascular.
The labia minora may be more or less firmly adherent. Their separa-

tion is, however, as a rule easily accomplished, and if a small piece of gauze is inserted between them to prevent subsequent adhesions a cure is quickly effected. Atresia of the urethra will, if complete, produce anuria. A small, blunt probe will often penetrate the thin membrane which, in the majority of cases, is the cause of the obstruction, and quickly and permanently relieve the condition. A contracted meatus may cause difficult and slow micturition with tenesmus. The meatus may be dilated, or it may be enlarged by a slight incision.

HYPOSPADIAS AND EPISPADIAS.

Hypospadias.—Hypospadias is caused by arrested development in the corpus spongiosum. In this developmental process the urethral groove becomes a canal, and if the fusion of the portions necessary to form it be imperfect, an opening is left at some point in its lumen. The most common seat for this deformity is at the base of the glans, the defect appearing in the lower portion of the urethra and the adjacent part of the corpus spongiosum. The arrest of development occurs at or before the fourth month of intra-uterine life. If the urethra opens at the base of the glans, it is called glandular hypospadias; if between the scrotum and the glans, it is called penile hypospadias; and if behind the scrotum perineal hypospadias. In the latter the scrotum, and even the perineum, may be fissured, and if the testes are in the abdominal cavity the sex of the infant may be temporarily in doubt. In many cases there is more or less difficulty in micturition; in others control of the bladder seems to be normal. The treatment is purely surgical. The operation is a delicate one, but in the hands of a skilful specialist the result is often quite satisfactory, and the patient's condition is usually much improved. The most suitable time for the operation is when the child is four or five years old.

Epispadias.—Epispadias is due to an arrst of development occurring in the same manner as in hypospadias. In this condition the urethra opens on the dorsum of the penis; there is a defect in the upper wall of the urethra, and the adjacent portion of the corpus cavernosum. Epispadias is a very rare condition; hypospadias is quite common. Exstrophy of the bladder may be associated with epispadias; persons with this condition are apt to be depressed mentally, and to suffer from incontinence of urine, the dribbling of which produces excoriation of the skin and causes a most disagreeable ammoniacal odor; infection of the urethra and cystitis are not uncommon complications. The treatment is, of course, purely surgical, although local applications will, to a certain extent, relieve the irritation caused by the dribbling of urine. The operation should be attempted only by a specialist, as it is a very delicate and difficult one; but, in view of the almost constant nervous and depressed condition caused by the continuous dribbling of urine, it is only proper to give the child an opportunity to be benefited by surgical treatment. The results of operation are fairly satisfactory, and the best time for it to be performed is when the child is six or seven years old.

DISEASES OF THE NEWBORN.

GENERAL PARALYSIS OF THE NEWBORN.

Prenatal Paralysis.—Etiology.—Prenatal paralysis includes all those cases in which the paralysis, or the cause of the paralysis, develops before the onset of labor. Consequently it comprises all cases of defective cerebral development, such as porencephalus, microcephalus, brain atrophy, congenital cysts, and all brain lesions of such a nature as to cause paralysis at birth, or some mental or physical defect that shows itself, perhaps, months or years after birth; also agenesis corticalis, in which one finds defective development of the cellular elements of the brain cortex more or less widely distributed, but especially marked in the pyramidal cells. The origin of the paralysis is always intra-uterine, the lesion is prenatal and occurs before labor begins.

If the mother while pregnant suffers from any severe systemic disease, has uremic convulsions, or sustains a severe blow or fall on the abdomen, this form of paralysis may be induced. Arrested development of the brain is undoubtedly responsible for some of the cases, and syphilis, insanity, alcoholism, and pyogenic infections in the parents are supposed to be more or less effective in a small percentage of the patients, while secondary degeneration in the lateral columns of the cord is noted in a considerable number.

Symptoms.—The symptoms are, of course, in proportion to the extent of the lesion. They may at birth be very mild, and only the slightest impairment of the mental power and gait be present; usually, however, the paralysis is marked at birth, is spastic in type, and associated with great mental deterioration, the patient being commonly a helpless and hopeless invalid.

Diplegia and paraplegia are usually present, although hemiplegia also may occur. The child may be microcephalic, or show evidences of cranial or facial asymmetry with arched palate and the stigmata of degeneration.

Prognosis.—This depends upon the extent of the brain lesion or the extent of the lack of brain development. Most cases are very little benefited by treatment. In the mildest cases something may often be accomplished by judicious feeding, good hygiene, massage, and electricity.

Treatment.—The infant should receive all possible care dietetically and hygienically. Its general nutrition should be kept at as high a level as possible, and every effort should be made under instructions from the mother or trained assistant to develop whatever brain capacity

the child may possess. Many patients are best cared for in institutions, where the results of treatment are often remarkably good. Slight gastro-intestinal disturbances may cause a marked temporary increase in the nervous symptoms, and it is, therefore, important to map out for these children such a diet as they can easily digest. To control convulsions, and possibly with the hope of preventing them, the bromides and iodides can be used. Iodides internally and mercury by inunction have been recommended.

Natal Paralysis.—Etiology.—Natal paralysis includes all those cases of paralysis in the newborn in which the lesion takes place either during labor or immediately after birth. Hemorrhage, meningeal in origin, is the cause in a great majority of the cases, and is usually induced by a tedious labor, a difficult forceps delivery, or a breech-presentation. The fact that birth palsies are most common in firstborn children suggests that labor is the cause. Meningoencephalitis, followed by degenerative changes in the cortical motor area or other portions of the brain, is among the conditions found. The meningeal hemorrhage that produces the paralysis may be a part of the bleeding in hemorrhagic disease of the newborn. The hemorrhage may occur in the brain substance, it may be at the base of the brain in the cerebellar region, or in the region of the occipital lobes of the cerebrum, or at the convexity. It may be profuse or slight, diffused or localized, and the blood may come from the pia, the cerebral veins, or a sinus. In stillborn children, hemorrhage is often found in the upper membranes of the cord. A few cases have been reported as the result of premature birth, and asphyxia is a recognized cause. Secondary sclerosis and atrophy may develop and involve large areas of the brain, followed by secondary degenerative changes in the cord, especially the lateral columns.

Symptoms.—Asphyxia and paralysis are, as a rule, present at birth, or appear within the first twelve hours after birth. Convulsive twitchings and paralysis may not, however, develop until several days after delivery. Bulging of the fontanelle and slow pulse are suggestive of cerebral hemorrhage, and nose bleed or blood in the pharynx are common symptoms. A slight lesion is often unnoticed at birth, and remains unobserved until the child is some months old, when its failure to take notice, to creep, to walk, to talk, and otherwise to develop normally forces itself upon the attention of the parents, and a physician is consulted. It may not walk until almost two years of age, and then the gait is very unsteady. Mental development is slow, and in early infancy acute attacks of weakness associated with cyanosis may occur. If not seen early by the physician, such cases are apt to be ascribed to convulsions occurring, perhaps, months after birth, the convulsions being really due to the injury at birth, and merely a symptom of the progressive changes following the birth lesion.

Athetoid movements, if present, will suggest the brain origin of these cases, and epilepsy as a later condition is very common. The convulsions often begin in the paralyzed limb and spread over the

entire body, and may be associated with opisthotonos. If the lesion is extensive convulsions are common, and paraplegia and diplegia develop early, with coma and possibly early death.

The symptoms may be merely those of a slight monoplegia or none at all may be noticed by the parents; later a physician is consulted because the child is backward. An examination of the child may now reveal strabismus, poor gait with slight spasticity and exaggerated knee-jerks, and backwardness in walking and talking. The history of the birth is often one of prolonged and difficult labor.

Where convulsions occur, they may be limited to the arm or face, or, after beginning in one portion, may involve the rest of the body. Slow and irregular respiration is suggestive of an intracranial hemorrhage, as are also a slow, weak pulse and increased reflexes.

These patients may show nystagmus and change in the pupils. Convulsions are especially apt to be associated with a cortical lesion. After the spastic condition of the arm or leg has lasted for a few days, hemiplegia or diplegia may appear. If the convulsions develop within the first few days of life, they quite commonly cease when the child is two weeks old, only, however, to reappear after an interval of one, two, or three months. During this intervening period the myelin sheaths of the pyramidal tracts are developed, and in consequence a communication between the brain and cord is establshed which results in a spastic hemiplegia and a return of the convulsions.

The fact should be noted that no matter what the character of the symptoms at the onset, spastic paralysis commonly develops, the paralysis being usually most marked and persistent in the arm. The extent of the paralysis may vary from complete loss of power to a loss so slight that it is manifested, perhaps, only after prolonged exertion. If at birth the child is drowsy or dull with irregular respiration, the hemorrhage is probably profuse, and the child is apt to die within the first twelve to thirty-six hours. If the child is born dead, the hemorrhage is usually extensive and is apt to be at the base of the brain. If the child is asphyxiated at birth, the prognosis is better if it can be made to breathe without much difficulty, and is able to nurse within the first few hours.

Diagnosis.—Mild cases affecting one limb may suggest Erb's palsy or an anterior poliomyelitis; the latter usually affects groups of muscles in a limb, but not an entire limb. Reflexes are absent; the limb is flaccid with marked wasting, the reaction of degeneration is present, but there is no mental impairment. Poliomyelitis comes on a considerable time after birth, and is usually preceded for from twenty-four to forty-eight hours by a more or less definite group of symptoms.

In natal paralysis convulsions are common, mental impairment is often noted, the reflexes are increased, and a spastic paralysis may be present. The paralysis is apt to be paraplegia, diplegia, or hemiplegia, but rarely monoplegia. Athetoid movements may be noticed.

Prognosis.—The prognosis depends upon the location and extent of the injury, and the degenerative changes that follow. It may vary

from the slightest loss of mental power with trivial paralysis to idiocy with extensive paralysis.

Treatment.—The skilful handling of difficult labor cases is important, and when to apply and when not to apply the forceps is often a difficult question to decide. As soon as the child is born, its head should be raised and kept elevated and a small ice-cap applied with the hope of at least preventing further hemorrhage. The bowels should be thoroughly moved, and the child kept absolutely quiet.

If convulsions are frequent and severe, bromide of soda, 5 grains, with chloral hydrate, $\frac{1}{2}$ grain, should be given every two or three hours by the mouth or rectum, and continued as long as the symptoms persist; or chloroform may be given cautiously by inhalation. If possible the general health of the child should be kept up, and especial attention be paid to the nutrition of the paralyzed part, every possible endeavor being made to prevent contractures. The best of food, abundance of fresh air, massage, and passive exercises are all of advantage.

Braces may be of assistance, and lengthening, cutting, and transplanting of tendons may be necessary. Short school hours should be the rule for these children; if nervous and highstrung, school attendance should be, at least temporarily, given up.

If the position of the clot can be accurately determined by localized convulsions or paralysis, a surgical operation should be performed for its removal, especially in those cases where, in spite of medical treatment, alarming symptoms continue. The difficulty, of course, is to locate the clot accurately. Medical literature now records a number of instances where the operation has been successfully accomplished. If a meningeal hemorrhage is diagnosed, and the anterior fontanelle is bulging, puncture of the fontanelle may be performed at once with the hope of drawing off some of the blood before it clots. A needle is introduced to a depth of one-sixth to one-fourth of an inch, as far from the longitudinal sinus as possible, and an attempt made to withdraw some of the effused blood. If any blood is secured, the procedure is of value, both in a curative sense and as a means of diagnosis.

BIRTH PALSIES.

Erb's Palsy (*Obstetric Paralysis*).—This form of palsy was first described by Duchenne, and later more carefully studied by Erb. The condition is due to an injury of the brachial plexus at birth. It may be caused by pressure from the application of the forceps, or during labor by a contracted pelvis. It is especially common in breech presentations, rarely occurring in a spontaneous delivery. It may be the result of traction in the axilla during delivery, or upon the shoulder, traction necessarily being employed in order to deliver the after-coming head; the injury may also be produced in bringing down the arms which are extended above the head. The lesions consist of a stretching and tearing of the nerves, and, to a certain extent, of the

surrounding tissues, which result in a localized area of inflammation, and possibly subsequent degeneration.

Symptoms.—The symptoms may be severe, or they may be very slight. The loss of power in the arm may be noticed immediately at birth, or, in mild cases, not for some weeks. The arm may hang limp and helpless without any power of motion at the shoulder. In old cases it is often noticed that the shoulder tends to drag lower than that on the healthy side. The nerves usually injured are the fifth and sixth cervical. According to Erb, a lesion at the spot where the sixth cervical passes between the scalenus muscles will produce the classical symptoms of Erb's palsy. The paralysis affects, wholly or in part, the deltoid, biceps, supinator longus and brevis, supra- and infraspinatus, and the brachialis anticus. The upper arm is rotated inward; the forearm is pronated, and the palm is turned more or less outward. Supination at the elbow is absent and motion at the wrist-joint is normal with the possible exception of more or less loss of extension. Flexion is always normal at the wrist. The forearm is not affected. The hands may be normal or show a slight loss in power of flexion.

Fig. 18.—Erb's palsy.

There may be disturbance of sensation on the outer surface of the arm in those portions supplied by the musculocutaneous nerves and the axillary nerve. The sensibility of the inner surface of the arm remains normal. If the child does not tend to recover, trophic changes develop, contraction of the affected muscles takes place, and the shoulder blades become prominent. The bones of the arm fail to grow normally, and subluxation of the shoulder which tends still further to interfere with motion may develop. A fracture of the clavicle or humerus, or a separation of the epiphysis of the upper end of the humerus, is a complication which is occasionally found at birth and renders the diagnosis and prognosis of Erb's palsy more difficult.

Diagnosis.—An old case of Erb's palsy, seen for the first time, may simulate anterior poliomyelitis. Erb's palsy develops at birth or soon after, and infantile paralysis always at a considerable period after birth. Subluxation of the shoulder is suggestive of Erb's palsy, and the group of muscles affected in Erb's palsy is not often found affected in infantile paralysis. Paralysis due to syphilis can be differentiated by the history and other evidences of syphilis. The inward rotation of the arm resulting from a separation of the upper epiphysis of the shoulder can be diagnosed by the x-rays.

Prognosis.—The fewer the muscles involved and the earlier the treatment is begun, the better the prognosis. Most cases recover entirely, although a few in spite of treatment show little or no improvement.

Treatment.—If the muscles respond to faradism rapid recovery often results, and the affected muscles should be treated with faradism every day, provided they respond; if they do not respond, galvanism should be employed. Daily massage will improve the nutrition of the parts, and tends to prevent the development of contractures. For those patients who have been treated faithfully and systematically by massage and electricity for some months without benefit, a plastic operation on the injured nerves and tendons is worthy of consideration; in a few cases, quite satisfactory results have followed the suturing of the involved nerve trunks.
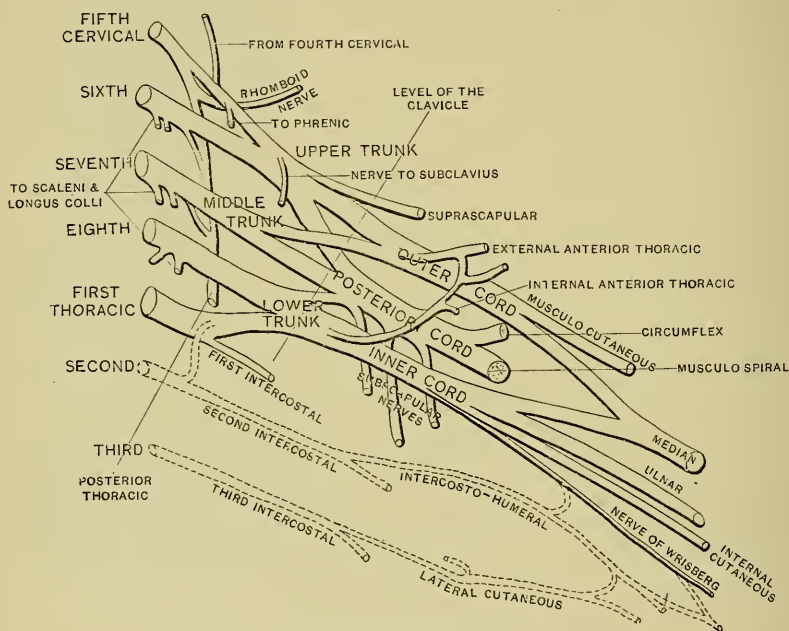
Fig. 19.—Plan of the brachial plexus. (Gerrish.)

Klumpke's Palsy.—In contradistinction to Erb's palsy, or the upper arm type, is the form described by Klumpke and called the lower arm type. The muscles involved in this form are those innervated by the seventh and eighth cervical and the first dorsal nerves. These muscles may be the only ones involved, or, in addition, those supplied by the fifth and sixth cervical nerves may also be affected.

Symptoms.—A single muscle, as the deltoid, may be affected, or all the muscles of the arm may be involved, and the entire arm and hand may be more or less completely paralyzed with loss of sensation. In certain cases, in addition to the muscles involved in

Erb's palsy, the subscapularis, rhomboideus, serratus and pectoralis major may be affected. The lesion is occasionally bilateral. Eye symptoms are present, due to the involvement of the sympathetic nerve from the first dorsal; the latter connects with the lower portion of the brachial plexus (Fig. 19).

The opening between the eyelids is narrow, and a contraction of the pupil is present which, however, reacts well to light and accommodation. The prognosis and treatment are the same as in Erb's palsy, as the lesions are identical, but different nerves are involved.

Facial Palsy.—The paralysis may be on one or both sides, is peripheral in origin, and may involve one or more of the branches of the nerve. It is usually seen in children delivered by forceps, when one of the blades has pressed firmly on the facial nerve, producing an injury to the nerve sheath and the nerve fibers. It may also follow delivery in cases of contracted pelvis. The prognosis is usually favorable, all traces of the paralysis disappearing, as a rule, in the first few months of life.

ALBUMINURIA AND URIC ACID INFARCTION.

Albumin in the form of nucleo-albumin is found in the urine of the majority of infants at birth, but never in large amounts. As a rule it persists for from one to ten days, although it may occasionally continue for a month.

During the first few days of life infants take only small amounts of liquids in the form of breast milk and water, but eliminate considerable fluid by the skin and breath which tends to lessen the amount of urine voided, and during this period of slight urinary secretion albuminuria and uric acid infarction are apt to appear. That these have some connection with the quantity of fluids ingested is shown by the fact that they are much less common in bottle-fed babies who invariably receive a larger amount of food during this period than those fed at the breast.

The urine voided immediately or soon after birth is normal, and the kidneys of stillborn infants rarely show uric acid infarction. These infarcts of uric acid are, however, often found in the kidneys of children a few days or weeks old, and consequently must develop after, and not before birth.

The same rule as to time is noticed in the microscopical findings. Normal at birth, in a day or two the urine becomes cloudy and contains hyaline and epithelial casts, leukocytes, and epithelium from the kidney. The casts are found much more commonly in breast-fed (40 per cent.) than in bottle-fed (10 per cent.) infants. The brick-dust stain on the diapers during the first few days is caused by urates. These urinary findings are all apparently due to uric acid infarcts in the kidneys, and are, as previously mentioned, largely dependent upon the amount of water and milk ingested during the first few days or weeks of life.

It is a question whether it is physiological for albuminuria and casts to be present in the urine of the newborn. The nucleo-albumin must be due to a disturbance of the kidney function; it cannot come from the blood since nucleo-albumin does not exist in the blood.

BONE INJURIES.

As a result of injury during a difficult labor, the bones of the infant may be more or less seriously injured. This is more commonly seen in the bones of the skull, where depressions and fractures may follow the use of the forceps, although the same conditions may be found when no instruments have been used. The parietal bones are the ones most often injured; but, unless the underlying membranes and brain tissues have been affected, the injury may, if not excessive, be productive of little or no harm, the bones tending to assume their normal relation as to shape with the gradual development of the head. If, however, a hemorrhage has occurred beneath the skull—inside the cranium—coma, convulsions, subsequent epilepsy, or death may result.

In breech presentations the lower jaw may be fractured by unnecessary traction. A fracture of the clavicle and humerus, or a separation of the epiphysis of the humerus, may result from the difficulty in bringing down the arm. The femur has been fractured by improper traction with the fingers, fillet, or blunt hook. In other cases paraplegia may follow an injury to the spine and cord occurring during labor.

The important point which such injuries emphasize is the necessity of a careful examination of all infants immediately after delivery, and this is especially to be enjoined after all difficult labors. The treatment of all such conditions is, of course, surgical.

ASPHYXIA OF THE NEWBORN.

A healthy normal child breathes deeply almost immediately after birth, cries loudly, kicks vigorously, and begins at once to inflate its lungs. The upper lobes expand first, and the posterior portion of the lower lobes last, the external portions expanding before the deeper internal parts. At least one or two days pass before the lungs fully expand, and in weak infants, or those born prematurely, it may be one or two weeks before expansion is complete. Owing to the changes which occur in the atelectatic portions of the lung, full expansion becomes more difficult as time progresses.

In asphyxia of the newborn the absorption of oxygen and the giving out of carbon dioxide are checked wholly or in part; and this condition, if severe, becomes extremely dangerous.

Etiology.—If the respiratory centre is stimulated before the child is born, attempts at breathing are induced, and asphyxia from inhalation of foreign matter is the result. The stimulus to breathe may be derived from the mother as the result of a fall in her blood-pressure due to hemorrhage, protracted labor, or to her death.

An abnormal form of uterine contraction, the so-called tetanus uteri, or any disease of the heart or lungs in the mother combined with poor circulation and poor oxygenation of her blood, will produce intrauterine asphyxia in the child. So will any condition which causes compression of the umbilical cord, as early detachment of the placenta or premature birth, the respiratory muscles being weak and the respiratory centre poorly developed; or any condition like cerebral hemorrhage, which produces abnormal intracranial pressure. As the carbon dioxide in the blood increases and the oxygen decreases, the respiratory centre loses more and more its power to respond to stimulation, until finally no effort at respiration is made, and it becomes paralyzed. It is, however, apparently more the lack of oxygen that paralyzes the respiratory centre than an increase of carbon dioxide.

Asphyxia Cyanotica.—A child suffering from intra-uterine asphyxia passes meconium, and invariably shows a weakness in the heart sounds; if the asphyxia is extreme the heart's action becomes unusually rapid, and the child is in danger of death, a condition which generally demands that labor be terminated as soon as possible.

In asphyxia cyanotica the child's color at birth varies from a bluish tint to a dark blue. It lies absolutely still, its face is swollen, its eyes are closed. The respirations are superficial, and the intervals between them may be much longer than normal. Mucous rales can often be heard, or felt by the hand on the thorax. The muscles preserve their firmness and tone, and the heart's action is strong but slow. A finger introduced into the pharynx to remove inspired material produces reflex choking. The respiratory movements can usually be increased with little difficulty by external applications, either hot or cold.

Asphyxia Pallida.—In this form the upper air passages are more or less completely blocked with mucus. The infant is very pale, its lips are blue, and the muscles are limp and relaxed. Reflex irritation is abolished, the heart is rapid and weak, and the extremities are cold. Respiration is practically suspended, although the child frequently gasps for breath. The temperature slowly falls, the heart's action becomes more feeble, and the child may sink and die. In milder cases, the respiration gradually becomes better established, the eyes open, and the child's movements grow stronger. The skin becomes warm and pink, the pulse stronger and regular; coughing and vomiting help to expel the mucus. The umbilical cord is soft, pale, and relaxed.

Pathological Anatomy.—The right heart is distended, the blood watery, the liver congested. Hemorrhages into the pia, pericardium, pleura, and liver occur, also hemorrhagic effusions into the serous cavities. There is marked congestion of the lungs, and the upper air passages and bronchi are filled with inspired matter. If the child at birth has made any efforts at respiration, and has, perhaps, lived for a short time, areas of more or less expanded lung tissue are found, which are light in color and elevated above the dark and tougher atelectatic portions. Inspired materials, such as mucus, blood,

8

amniotic fluid, and meconium are found in the upper air passages and large bronchi; sometimes also in the small bronchi.

Prophylaxis.—Prophylaxis includes the conduct of the labor in such a manner as to prevent, so far as possible, the causes which produce asphyxia, and also such care of the mother during pregnancy as may protect her from infections and injuries.

Diagnosis.—Asphyxia, either cyanotica or pallida, is evident as soon as the child is born. The clinical picture of either form is so clear that a diagnosis can be made immediately. The only question which may arise is, as to whether the asphyxia may be due to a cerebral hemorrhage instead of to some impediment in the placental circulation, or to material inspired into the upper air passages. Asphyxia due to brain hemorrhage is associated with a bulging fontanelle, convulsions, irregular respiration, slow pulse, and stupor, the respirations being sufficiently numerous to warrant some lessening of the asphyxia. Asphyxia not due to brain lesions is invariably relieved by any improvement in the respiratory rate, except, of course, in premature or very weak infants, where the respirations are fairly regular, the asphyxia being in these cases simply an index of the child's poorly developed nerve centres and muscles.

Prognosis.—If treatment is prompt and energetic, many of the apparently hopeless cases are saved. When the asphyxia is associated with any lesion of the central nervous system, mental defects or evidences of paralysis may develop. In asphyxia due to cerebral hemorrhage the prognosis, of course, depends upon the extent of the brain lesion.

Treatment.—Artificial respiration must be continued in every case until the child breathes with sufficient regularity to make one feel that this assistance is no longer required. It is difficult to say how long one should persist in artificial respiration in apparently hopeless or desperate cases. Many children have been saved by this method who, seemingly, had little chance of life, judging by their appearance immediately after birth. As long as a heart beat can be detected, it is possible for life to be saved.

The mucus and inspired materials that clog the upper air passages and bronchi must be removed as quickly as possible. The mouth and pharynx should be wiped out with gauze, and the deeper mucus removed by aspiration through a soft catheter. Alternate hot and cold douches are beneficial in stimulating respiration, or the child may be alternately plunged into hot, then cold water, always in asphyxia pallida beginning and ending with hot water. In this form, too, special efforts should be made to keep the infant's body warm, and the child should be carefully watched for from twenty-four to thirty-six hours to see that the asphyxia does not return. Inhalations of oxygen are of benefit in these cases, also small doses of whisky, 10 drops every three hours, with strychnin $\frac{1}{400}$ of a grain hypodermically, repeated in six hours.

If blood is allowed to escape from the umbilical cord a portion,

at least, of the carbon dioxide is removed; and, in a few reported cases, this blood has been replaced with good results by transfusing normal salt solution into the umbilical vein. Intratracheal insufflation of oxygen has been performed with apparent benefit. In a doubtful case of cerebral injury, lumbar puncture may help to clear up the diagnosis; if the fluid removed is clear it points to the absence of hemorrhage within the skull; if blood-tinged, it indicates some injury to the central nervous system, and the removal of the blood-tinged fluid may possibly assist in the treatment.

Atelectasis.—The lungs at birth are normally in a condition of atelectasis, but begin to expand at the very first breath; the upper portions being inflated first, and the lower parts last. It is not only necessary that the lungs be inflated as soon after birth as possible, but, once inflated, that they be kept expanded—a task not always easy of accomplishment in weak and puny infants. The longer the time after birth that the lungs remain atelectatic, the greater the changes that take place in them, and the more difficult it is for the infant to inflate the collapsed portions.

Etiology.—Atelectasis is often the result of the same causes as is asphyxia, *i. e.*, tedious labor, premature birth, inhalation of foreign materials before birth, and cerebral hemorrhages. It is frequently seen in frail and delicate infants, and in cases of hereditary syphilis.

Pathological Anatomy.—If a child with atelectasis lives but a day or two, only a small portion of the anterior borders of the upper lobe become expanded, and this portion may also be emphysematous. The posterior portion of the lower lobes is the most common seat of atelectasis. The unexpanded portion is brownish-red, very vascular, does not crepitate, and shows globular outlines on the surface and on section; it can very easily be inflated. Small hemorrhages are frequently found beneath the pleura. Both lungs are usually involved. The child may live several weeks, and yet be quite atelectatic; and, if the condition persists a long while, it is apt to be associated with pneumonia. The right heart is commonly dilated, the liver and spleen are enlarged and congested, and cerebral hemorrhage, either at the base or convexity, is often present, especially where there is a history of difficult labor. Lung conditions, such as aplasia, may be noted, or atelectasis may be associated with an enlarged thymus. Delicate thoracic walls and poorly developed respiratory muscles favor the non-expansion of the lung.

Symptoms.—In atelectasis the whole or a part of the lung remains in the fetal state. The condition is usually associated with asphyxia, and in relieving the asphyxia we generally assist in clearing up the atelectasis to a greater or less extent. Feeble and premature infants do not breathe deeply enough fully to expand their lungs, and portions remain atelectatic. The respiratory movements are weak and apt to be superficial, with irregular rhythm. In severe cases of atelectasis, the cyanosis is generally marked. As a rule, the history is that of a moderate asphyxia at birth, the infant, however, being revived without

great effort. The child is below the normal weight, and frail and delicate, perhaps premature. It fails to gain in strength or weight, the hands and feet are generally cold, and the temperature may be subnormal. The cry is feeble. From time to time, there are evidences of cyanosis which may be slight or marked; it may develop suddenly without apparent cause and be extreme, even ending fatally, convulsions often appearing before death.

It must be borne in mind that an atelectatic child need not necessarily have been asphyxiated at birth. The child may present no lung symptoms until the final attack of cyanosis develops. Many of the cases occur in puny delicate infants, or the prematurely born, with low vitality, and an improvement in the atelectasis is an indication of improvement in their general health. If the child gains in weight the atelectasis gradually disappears. Often an excess of carbon dioxide —either small or great, but persistent—is present in the blood, while the amount of oxygen is subnormal, thus causing paralysis of the respiratory centre. The infant is unnaturally quiet, often drowsy, the face and hands may be puffy, and the temperature perhaps subnormal. Breathing is irregular and slow, and on crying a few rales are heard at the back and base of the lungs. Attacks of cyanosis, mild or severe, may develop without apparent cause or warning, and a premature child several weeks of age may suddenly become cyanotic, and develop dangerous symptoms, and even death follow. In the most severe cases asphyxia is marked at birth, the child is only partially revived, lives a few days or hours, and dies with all the evidences of asphyxia, prostration, and coma.

Diagnosis.—Atelectasis is most common in the posterior portion of the lower lobe of the lung. Over this portion resonance is impaired, but, as the collapsed areas are surrounded by portions of inflated lung, the impairment is not marked, the respiratory murmur is feeble and harsher than normal, and on deep inspiration rales are usually heard. The cardiac sounds may be transmitted more clearly than is normal.

Prognosis.—This depends on the cause and the treatment. If the child improves, as shown by a gain in weight, strength, and vitality, the chances are good. If the cyanosis tends to recur, with loss of flesh and vitality, a subnormal temperature, and cold hands and feet, the prognosis is grave.

Treatment.—A frail and delicate infant, especially if drowsy and with irregular respirations, must be aroused five or six times a day. Its face may be washed with hot and cold water alternately, or may be sharply smacked with a handkerchief dipped in cold water, or the abdomen may be slapped in a similar manner. If possible, the infant must be made to cry and to take deeper, fuller breaths. The temperature can be maintained with hot water bottles, and the cyanosis combated with oxygen inhalations. Excessive handling is bad for babies; but a certain amount of it is beneficial, especially if the baby is frail and delicate. Pick it up, rub it, change its position

from one side to the other. If allowed to lie a long time in its crib, as is occasionally the case in asylums and hospitals, a feeble delicate child is apt to develop atelectasis, and certainly recovers from it with difficulty. The cold hands and feet should be protected with mitts and socks. Breast milk is by far the best food; the breast may be pumped out, and the milk fed to the infant with a dropper, a spoon, or a Breck feeder until the baby is strong enough to nurse. In order to keep up the mother's secretion of milk, another infant should, if possible, be put to the breast. For relief of the cyanosis artificial respiration, warm baths, whisky internally, and oxygen by inhalation are recommended.

MASTITIS.

There appears in all children two or three days after birth a swelling of the mammary glands, which increases gradually, is more marked in some infants than in others, and reaches its height at about the tenth day, then slowly subsides, and disappears in the third week. The condition is physiological, and produces no symptoms, so far as we can judge. According to J. Halban, it is caused by the presence in the fetal and infant blood of certain bodies that exist in the blood of pregnant women, and are carried by the placental circulation to the blood of the fetus. At birth these bodies are present in the blood of all children, boys and girls, and they reappear in girls at puberty, and in women during pregnancy. Halban claims that they are produced by placental and chorion secretions, and that the swelling which normally appears in the breasts of the newborn infant is due to the temporary existence in the infant's blood of these bodies, and that the same secretion in the blood of the mother stimulates the marked changes that occur in her mammary glands. The infant's breasts after birth show distended ducts, hemorrhages, leukocytes, and proliferated epithelium—products similar to those in the breasts of its mother. The baby's breasts are enlarged, more or less firm, and on gentle pressure a few drops of milky fluid can be expressed. This fluid, chemically examined, contains fat, milk sugar, protein, salts, and ash. Microscopically it shows milk globules, leukocytes, and colostrum corpuscles, and in composition it is practically the same as colostrum. Mastitis developing in this physiologically enlarged breast manifests itself by enlargement, with redness and tenderness. The swelling gradually increases, and unless checked may go on to abscess formation. Loss of sleep, crying, and restlessness, with vomiting and diarrhea, are the usual symptoms, and high fever is common.

Etiology.—Squeezing or pressing the breast to express the milk is probably the most common cause of mastitis, and a binder, if applied too tightly, may by its continuous pressure produce the same result. The tendency to inflammation is, of course, increased by the physiological activity and congestion normally present, particularly during the second week of life, when it is greatest. The skin of the newborn

is liable to infections through the slightest injury, hence organisms effect an easy entrance.

Bacteria are normally present in the milk ducts of infants, and any local condition which lowers the resistance of the epithelium permits the migration of these bacteria with resulting inflammation. Any abrasion, crack, or injury to the infant's nipple, such as might result from a too vigorous washing and first cleansing of the infant, can easily become the portal of entry for the invading bacteria.

A child's body should always be kept absolutely clean, and want of care, as a rule, is probably a common cause for the development of the condition. In the girl baby, suppuration will necessarily produce more or less destruction of normal glandular tissue, and this will interfere to a greater or less extent with the future normal development of the breasts.

Prognosis.—A mild attack of mastitis is not apt to make the infant ill; if, however, the inflammation spreads and becomes phlegmonous, as it may in a delicate and frail infant, the condition may be serious.

Treatment.—Prophylaxis comprises the protection of the breasts from pressure and trauma, and the observance of absolute cleanliness. It is never wise to attempt, even in the gentlest way, to squeeze out the milk normally present. The breasts should be carefully protected from unnecessary pressure and manipulation. If inflammation appears a pad of gauze, kept wet with alcohol one part to three parts of water, and covered with oiled silk, should be continuously applied to the inflamed area and kept in place by a loose, broad bandage.

The infant's food should be diluted by giving it a tablespoonful of water before each nursing, and its bowels moved by a dessertspoonful of castor oil. If pus forms, an incision should be made parallel with the direction of the milk ducts, and a suitable antiseptic dressing applied. The incision should be made near the periphery of the gland in order to cut as few milk ducts as possible. Treatment is usually successful, and the abscess heals in a few days. The suction apparatus of Bier may possibly be of service.

ICTERUS NEONATORUM.

Etiology.—This form of jaundice occurs in the newborn and is considered physiological; so far as is known at present it is unconnected with any pathological condition. Undoubtedly it is in some way associated with the liver, and, while there are many theories as to its origin, it is probably due to an active production of bile immediately after birth; this, added to the fact that the capillary bile ducts are filled with viscid bile before birth, produces distention of the capillaries in excess of their emptying capacity, and this excess of bile is absorbed by the blood. The disintegration of the maternal erythrocytes is also to a certain extent concerned in the production of the jaundice.

Pathological Anatomy.—The liver is found to be only slightly jaundiced in small areas. The kidneys and spleen are normal, and the

color of the stools is unaffected by the icterus. Macroscopically the urine does not show the presence of bile, although delicate tests may reveal bile pigment, bilirubin, and glycocholic acid. In a case of icterus neonatorum death must, of course, be caused by some fatal disease or condition unconnected with the icterus.

In such instances, according to Orth, bilirubin crystals are found in the kidneys, in the blood, fatty tissues, brain, and other organs. With the exception of the spleen and kidneys almost all of the organs and tissues are jaundiced, and this is especially marked in the serous membranes, the intima of the bloodvessels, and the exudates and transudates. The bile ducts are normal.

Symptoms.—Jaundice appears between the second and fifth days after birth, occasionally later. It is usually first seen on the face, and then spreads over the entire body. The hyperemia normally present in the skin may obscure the jaundice for the first twenty-four hours after its appearance, but pressure on the skin makes the icterus easily recognizable. The sclerotic coat of the eye is usually but not always yellow, and the mucous membrane of the mouth is also jaundiced, as is shown by the yellow tint which appears after firm pressure upon the buccal mucous membrane. The pulse rate is normal in the infant with icterus neonatorum, and this is also true of older children with jaundice, probably owing to the fact that only small amounts of the biliary acids are found in the bile of children; according to Jakubowitsch glycocholic acid is present but taurocholic is not. The icterus may be slight or very intense; it usually persists for from four to eight days, although it may last only for two days, or may continue for three weeks.

If, after two or three weeks, the jaundice shows a tendency to persist or to deepen, it is unlikely that the disease is merely icterus neonatorum, but it is probably a more serious form, the result of either sepsis or obliteration of the bile ducts. The condition is very common; if carefully examined over 50 per cent. of all children will show more or less evidence of it. It is especially common in premature children, and the smaller the birth weight the more intense is, as a rule, the jaundice. It is claimed that it occurs more frequently in the children of primipara than of multipara, and early ligation of the umbilical cord is said to lessen the liability of its occurrence. It is probably a physiological condition which, so far as is known, does not permanently harm the child, although it is claimed that children deeply jaundiced are especially apt to lose weight during the first few days of life, and their subsequent gain is slower. The urine is usually normal in appearance, but is said to contain increased amounts of urea and uric acid. The jaundice has no effect on the color of the stools.

Diagnosis.—Jaundice associated with sepsis does not appear until after the fifth or sixth day, it is accompanied by fever and loss of weight, and often by umbilical infection, pneumonia, meningitis, peritonitis, or epiphysitis. Cases with congenital malformations of the bile-ducts are usually, but not always, jaundiced at birth. The icterus steadily deepens, there is no bile in the stools, the urine is high-colored,

and the symptoms continuously increase in severity until death occurs. Hemorrhages under the skin and in the mucous membranes are common. In interstitial hepatitis, which may be syphilitic, the liver and spleen are enlarged, and the jaundice is usually more intense than in icterus neonatorum. Gall-stones are rare in children, but are more common in infancy than during child life. Still reports 15 cases in infants, in several of whom the jaundice was marked at or soon after birth, and gall-stones were found in the ducts. In catarrhal jaundice icterus of the conjunctiva is noted as the first symptom before there is any evidence of jaundice in the skin. The urine is invariably high-colored.

Prognosis.—This form of jaundice in itself is never dangerous, but a coexisting condition, such as premature birth or atelectasis, may of course influence the prognosis.

Treatment.—The disease requires no special treatment, and, if no complications arise, is self-limited. Care as to diet, fresh air, and general hygiene is important in all well-marked cases of jaundice in infants, owing to the fact that the child may be premature, and have a tendency to regain the initial loss in weight more slowly than those not affected with icterus.

ACUTE SEPTIC INFECTION OF THE NEWBORN.

This includes all acute infections in the newborn produced by bacteria. The infection may be local, as in the eye, mouth, umbilicus, and vagina, or bacteria may enter the circulation and produce a severe or fatal septicemia or pyemia.

Etiology.—Septic infection of the newborn was formerly common in large maternity hospitals, and to a less degree in private practice; but as a result of the general adoption of asepsis the number of cases has largely decreased. The newborn infant is especially liable to infection owing to the open wound at the umbilicus, and to the abrasions of the skin and mucous membranes which may happen at birth. Moreover, its power of resistance to infection is very slight, the inability to resist or cast off septic infections being probably due to the lessened antibodies and other protective substances in the blood, as well as to the comparatively undeveloped condition of the lymphatics and spleen.

Breast-fed babies are less liable to, and resist, infection better than bottle-fed babies, the vital principles of breast milk probably being one of the factors contributing to this result. Frail and premature infants, too, are not only more susceptible to septic infection but less able to resist its progress. The infection in the large majority of cases comes from without; the main portals of entry are the umbilicus, the skin and mucous membranes, and the respiratory and gastro-intestinal tracts. Many cases are umbilical in origin, even when there is apparently no local evidence of infection at the navel. Infection of the thrombi in the umbilical veins readily occurs owing to their proximity to the bacteria normally present and active in the necrotic

processes which result in the separation of the umbilical stump. If septic phlebitis follows, general sepsis may develop and involve one or many of the organs of the body; in these infants the liver is especially liable to infection because so much of the blood immediately passes through it. Septic infection may be conveyed through any abrasion of the skin or mucous membrane, or infected materials may be inhaled or swallowed either just before or after delivery. Sepsis may be produced by many different bacteria; most commonly by the staphylococcus pyogenes albus and aureus, the streptococcus, the pneumococcus, the colon bacillus, the bacterium lactis aërogenes, the bacillus enteridis, the bacillus pyocyaneus, or the proteus group, and, less often, by the meningococcus, the influenza bacillus, the gonococcus, and the bacillus of Friedländer. Modern aseptic treatment of the cord has, however, lessened the number of these cases, as the majority of infants are infected from external sources.

Bacteria are often air-borne, especially in hospitals. They are found in breast-milk when the breast itself may apparently be normal; bacteria may enter the milk ducts from without through the skin, or an abscess, ulcer, or fissure may exist. Judging clinically, this milk apparently seldom in any way injures the infant. Infection is less likely from human than from cow's milk; bacteria are present in both, but are, of course, less numerous and less apt to be pathogenic in human than in cow's milk.

The nurse may, perhaps, be the carrier of infection from the mother or from another child, or it may be carried by the physician by means of instruments, dressings, or unclean hands. Soiled clothing and dirty bathing water are not infrequent sources of infection through the injured or normal skin of the infant. The bathing of infants before the navel has healed apparently increases the chances of infection. Of 1420 infants tubbed during the period preceding the healing of the umbilicus, infection occurred in 18 per cent., whereas in 1692 infants not tubbed only 8 per cent. were infected.

The epithelial lining of the gastro-intestinal and respiratory tracts in the infant offers little resistance to the invasion of bacteria and, as has been previously stated, the baby's ability to produce antibodies or protective substances is slight. Infection may come from within and the newborn be septic at birth, the infection occurring through the placental circulation. The mucous membrane of the mouth is also often the seat of infection.

Pathological Anatomy.—In all severe cases hemorrhages and degenerative changes take place in the parenchyma of the heart, liver, and kidneys. Hemorrhages occur in almost all of the organs of the body, as well as in the skin, the mucous membrane, and in the membranes of the brain, especially the dura, but only occasionally in the brain substance. The latter is often edematous and congested. In the lungs bronchopneumonia and atelectasis are often found to exist, and septic emboli may produce infarcts or small abscesses; while degenerative changes, fatty in nature, are found in Buhl's disease.

The pleura often shows a purulent or serofibrinous exudate. Multiple abscesses may develop in the liver. The spleen may enlarge, and inflammation of the mucosa of the gastro-intestinal tract may exist. As a rule, the peritoneum is inflamed only in those cases where the infection has entered by the umbilicus. The kidneys are always involved, the lesion being usually either parenchymatous or fatty degeneration with necrosis of the renal epithelium and involvement of the kidney pelvis. Periostitis, osteomyelitis, and arthritis may be present. Umbilical phlebitis and arteritis commonly coexist with umbilical infection. Gastro-enteritis may also be found.

Symptoms.—The symptoms of septic infection in the newborn vary greatly, according to the location of the portal of entry, the severity of infection, the resistance offered by the infant, and whether the child was born prematurely or at full term. In a large majority of cases the symptoms are severe and the baby seriously ill, but they may be mild and localized; as, for example, an inflammation in the skin or joints, or a slight infection of the umbilicus. Inasmuch as the symptomatology varies so much in different infants, a better idea of the clinical aspect can be obtained by studying the symptoms separately than from individual cases.

Temperature.—The temperature is very variable; it may be quite high, only moderate, or even subnormal. No fixed temperature is typical. It may fluctuate between wide limits. The most common temperature chart is one that shows high fever, 103° to 105° F., with a drop each day nearly to or below normal. This continues for a few days, after which, with the rapidly increasing weakness of the child, it continues normal or subnormal with more or less irregular fluctuations.

The Skin.—The skin is decidedly jaundiced, especially in umbilical infection, the marked changes that occur in the blood being a factor in its production. Cyanosis may be noted in the lips, hands, and feet, and there may be swelling of the feet and pretibial edema.

Hemorrhages.—Hemorrhages are very common, and form a distressing and dangerous symptom. They may occur from the umbilicus, bowel, stomach, or any mucous surface, or may be noted as large or small purpuric rashes in the skin. Large areas of skin may undergo necrosis as a result of infection from without, while a deeper necrosis may produce extensive bed-sores.

The Mouth.—Inflammation, either deep or superficial, may appear in the mucous membrane of the mouth, and blood may ooze from cracks and fissures in the lips. O. Kneise claims that in 97.5 per cent. of a large number of infants examined bacteria were abundant in the mouth at birth. Staphylococci and streptococci were particularly common and virulent. The bacteria evidently entered the mouth either before or immediately after labor. This is probably a frequent cause of septic oral infection.

The Lungs.—The lungs are more or less involved in all cases. Bronchitis is almost invariably present, bronchopneumonia is a com-

mon complication, while pleurisy, and especially empyema, are not rare. The respiration is often rapid and superficial, and the respiratory symptoms may be the most prominent and most serious.

The Heart.—Pericarditis is quite common, and is usually secondary to inflammation of the pleura or the anterior mediastinum. Endocarditis is seen less frequently.

The Kidneys.—As a rule, the urine contains albumin, casts, leukocytes, bile pigment, and perhaps hemoglobin in solution.

The Gastro-intestinal Tract.—The gastro-intestinal tract is usually involved. Vomiting and diarrhea are common, and in some cases the sepsis produces the typical symptoms of gastro-enteritis. The vomitus may be green or of a brownish tint, or may contain blood. The stools are frequent, thin, greenish or brownish, or may be red or black from blood. The abdomen may be greatly distended. Intestinal paralysis may occur, and if associated with abdominal tenderness and pain usually points to peritonitis, the probability of which is increased in umbilical infection.

The Bones and Joints.—The bones and joints may show periostitis or osteomyelitis, the hips and shoulders being especially likely to be involved. If there is restricted motion, tenderness on pressure, local swelling, and pain on moving the joint, this diagnosis is warranted.

Meningitis.—Meningitis of the acute purulent type may exist. The exudate is usually extensive, and may be associated with meningeal hemorrhages or small multiple abscesses. Stupor, convulsions, paralysis, and bulging of the anterior fontanelle are common symptoms. Lumbar puncture will confirm the diagnosis. The child is often dull and drowsy, and may show irregular tremors or twitchings. If inflammation or hemorrhages exist in the central nervous system, a corresponding paralysis will be observed.

Diagnosis.—If there is local evidence of infection at the umbilicus, associated with irregular fever, jaundice, rapid wasting, involvement of the gastro-intestinal and respiratory tracts, or hemorrhages, the diagnosis can be made with certainty. But when the child presents no visible external portal of entry, and the symptoms resemble those of acute gastro-enteritis, pneumonia, or meningitis, it is often difficult to decide whether acute septic infection is present or not. The milder cases of gastro-enteritis running a slow course must be differentiated from the ordinary feeding case. Blood cultures assist in doubtful cases by demonstrating the presence of bacteria in the circulation. A negative culture, however, does not necessarily prove that the infant is not suffering from septic infection, as a considerable number of cases of septic infection show no bacteria in the blood during life.

Prognosis.—The mild cases recover; most of them, unfortunately, are of the severe type, and end in death. The prognosis depends upon the seat and severity of infection, the local or general involvement of the body in the septic invasion, the question whether the child was prematurely born or not, and its vitality.

Prophylaxis.—The newborn infant should be cared for under the most rigid laws of modern asepsis. The umbilicus should be treated as an open wound, and dressed accordingly. In hospitals and other institutions each infant should have its individual thermometer, basin, cotton, and its own mouth-wash prepared in a separate jar. The clothing should be changed often, and kept scrupulously clean. The breasts of the mother should be carefully cleansed just prior to nursing, and all feeding bottles should be regularly sterilized. Incubators for premature infants should be thoroughly disinfected after being occupied, and only those used that insure a constant supply of fresh air. A child should not be cared for by a nurse who is in attendance upon a septic mother.

Treatment.—The child's nutrition should be kept at as high a standard as possible. Breast milk is, of course, the best nourishment, and the mother's nipples should be carefully cleansed before and after each nursing. If the child refuses to nurse, it should be fed breast-milk with a spoon, medicine dropper, or Breck feeder. The child should be kept in a room where there is fresh, moving air. Whisky in 15 drop doses should be given every two hours. Infusion of digitalis, ℳxx, every four hours, or camphorated oil, ℳvj, given hypodermically three times a day, will be of service as a heart tonic. All symptoms of severe type should be treated as they arise. Any infectious condition of the mouth should receive early and careful appropriate local treatment. In all cases of septic infection the umbilicus, even if apparently healthy, should be washed daily with a weak antiseptic solution, and if there is pus in or around the umbilicus an incision should be made, the pus evacuated, and the abscess thoroughly washed out with 1 to 6 peroxide of hydrogen solution, or 1 to 2000 bichloride of mercury solution, and a sterile dressing applied. If an abscess forms at any other place, it should, if possible, be opened, and all lesions of the skin or mucous membrane should be promptly and carefully treated. Periostitis and osteomyelitis should receive early surgical treatment. Cases of meningitis should be treated according to the indications given under meningitis, the clinical diagnosis being confirmed by a lumbar puncture.

DISEASES OF THE UMBILICUS.

Disease of the umbilicus may be local or may become general by the spread of the infection through the umbilical veins. Normally the umbilical stump desiccates and drops off in about five or six days, although in frail and premature children this may not happen until several days later.

Omphalitis.—The first symptoms are usually noticed on or after the sixth day. An inflammation appears at the navel, and spreads more or less over the surrounding abdominal wall. The folds of the navel become swollen and edematous, and considerable pus usually forms. The abdominal veins become enlarged, lymphangitis may develop, and also cellulitis of the abdominal wall surrounding the

umbilicus which may end in abscess, gangrene, or infection of the umbilical vessels. In many instances the affection is purely local, and the case ends in recovery; if, however, the umbilical veins become infected, the condition is very dangerous.

Treatment.—As the cause is almost invariably an infection of the umbilicus at or after birth, aseptic treatment of the cord at birth and its subsequent care are absolutely essential in every case. This includes asepsis as regards the mother, nurse, and physician. Many physicians insist that the child be kept in a separate room from its mother during the first two weeks; this is, of course, always advisable if she is septic. If an umbilical abscess forms it should be freely opened and dressed aseptically. The child should be well nourished with breast milk, and given 15 drops of whisky every two hours.

Gangrene of the Cord.—Gangrene of the cord of the moist variety is not uncommon. The stump, wholly or in part, becomes moist, swollen, and dark, with a discharge of offensive odor. When the gangrenous mass sloughs, it leaves an unhealthy umbilical stump. Anything which tends to keep the cord moist favors the development of this condition. Under local aseptic treatment the disease usually terminates favorably. All oils, ointments, or wet umbilical dressings should be avoided.

Gangrene of the Umbilicus.—This is, fortunately, rather a rare condition, and is seldom seen except in feeble and premature infants. It may develop without previous local lesion, or may follow some of the more common forms of umbilical infection. It usually appears between the seventh and twentieth days. The gangrenous area involves the skin, adipose tissue, and, perhaps, the underlying muscles; it may even perforate into the abdominal cavity, and involve the wall of the intestine. Severe umbilical hemorrhage will indicate that the gangrene has perforated the umbilical vessels. A few cases end in recovery, the gangrenous tissue sloughing off, and granulations forming at the edges of the necrotic area. Most of the cases terminate fatally. The infant rapidly becomes exhausted and profoundly toxic, and dies in coma, greatly emaciated.

Treatment.—If possible, the gangrenous tissue should be removed with the cautery, the cauterization extending beyond the gangrenous area. If the condition is recognized early, this treatment offers some hope of cure. If, however, the child is premature, frail, or profoundly toxic, and the gangrene is extending, it is better to omit the use of the cautery, and apply hot, wet, antiseptic dressings locally.

Umbilical Hemorrhage.—The cord may have been imperfectly ligated, it may have fallen off too early, or the hemorrhage may result from necessary or unnecessary manipulation of the umbilicus. In all cases of hemorrhage due to the above causes the bleeding is slight, transient, and devoid of danger. Unless sepsis or some local disease of the navel is present a profuse spontaneous umbilical hemorrhage is rare, as the arterial blood-pressure falls very materially after birth as soon as the pulmonary circulation is established, and after

ligation of the cord the contraction of the walls of the arteries tends to check hemorrhage by the partial closure of the lumen of the vessels. In fact, the normal fall in the blood-pressure and the contractility of the arterial coats would, in the large majority of newborn infants, prevent a fatal umbilical hemorrhage, although in asphyxia, atelectasis, prematurity of birth, and congenital heart disease, a higher blood-pressure exists. Bleeding may take place from the umbilical arteries as they emerge from the body, but this is an uncommon form of umbilical hemorrhage, which is seen only after detachment of the cord; it occurs from the fourth to the fifteenth day.

In the large majority of cases of umbilical hemorrhage the bleeding takes place from the surface of the umbilicus, the blood simply oozing out from the small vessels in and around the navel; it may occur before or after the cord has fallen. The amount of blood lost is usually large, and the hemorrhage may be more or less continuous for days, often ending fatally; the mortality is over 75 per cent.

The cause in most cases is a septic infection, and umbilical hemorrhage is simply one of the varieties of hemorrhage associated with this condition. Sometimes hereditary syphilis causes the hemorrhage, and a careful inquiry should be made into the family history, especially as it is a well-recognized fact that syphilis causes definite changes in the bloodvessels. Umbilical hemorrhage as a symptom of hemophilia is extremely rare.

Treatment.—Bleeding from the cord may be controlled by a broad, firm ligature. Slight bleeding from the umbilicus may be checked by compresses moistened in adrenalin solution, 1 to 1000, or by a suture of the umbilicus. In some cases gelatin in four dram doses, carefully sterilized and injected under the skin, and repeated in eight hours if necessary, has been used with more or less success. The general treatment of septic infection should be carried out as indicated under that heading.

HEMORRHAGE IN THE NEWBORN.

Hemorrhagic Disease of the Newborn.—In the newborn hemorrhage is usually a symptom of infection. It may be hetero-infection or auto-infection, although the former is much more common than the latter. While sepsis is accepted as being the most common cause of hemorrhage, some cases are undoubtedly due to syphilis. In both sepsis and syphilis hemorrhage is usually but one of a number of symptoms, and the bleeding is generally not profuse. The more closely, however, these cases of hemorrhage are studied, the more evident it becomes that sepsis is, as a rule, the underlying cause. This applies also to those cases where hemorrhage may be the main, or is, in fact, the only symptom. To some extent at least, the bleeding usually depends upon some abnormal conditions present in the substances associated with blood coagulation. Under this heading spontaneous hemorrhages alone are considered. A few only of the hemorrhages that occur in the first few days of life are due to hemophilia.

Hemophilia is very much more common in boys than in girls, the proportion being as thirteen to one, whereas hemorrhage in the new-born is about as common in the female as in the male, and the study of a large number of histories of hemophiliacs does not show that they were subject to hemorrhage in the first few days of life. The bleeding rarely continues longer than a few days or weeks, and the strong and healthy child is as liable to hemorrhage as the delicate or premature. The disease resembles an acute infectious process in that it runs a self-limited course and ends in either death or complete recovery. The hemorrhages are especially apt to occur from mucous membranes, as the mouth, umbilicus, or gastro-intestinal tract; less commonly in the meninges of the brain, abdominal cavity, pleuræ, lungs, thymus gland, and suprarenals; it may also be subcutaneous. While it is usually spontaneous, it may also follow a slight bruise, or may occur over areas of pressure, as the occiput, the back, and the sacrum. More cases occur in maternity hospitals than in private practice. The disease ordinarily runs its course in from one to four days, occasionally in seven to nine. The hemorrhage usually appears between the second and seventh days, rarely after the fifteenth day, and is often the first sign of the disease. The amount of blood lost is usually not great, but a slow oozing persists, and from the onset of the bleeding the infant is markedly prostrated. A moderate fever is quite common, although the temperature may be normal or subnormal. At autopsy no definite pathological changes are found to account for the hemorrhage, but gastric and duodenal ulcers have been noted in a few cases. Blood cultures have, as yet, thrown no light upon the etiology. That the disease is due to different forms of infection is, however, the general belief, although in the first week or two of life the marked changes occurring in the blood, the weakness of the walls of the bloodvessels, and the change from intra- to extra-uterine circulation are factors that certainly predispose the infant to hemorrhage. If the bleeding is from the nose alone, hereditary syphilis should be suspected.

The *prognosis* is poor, the mortality being about 75 per cent.

Treatment.—To accomplish anything treatment should be prompt. In hemorrhage from the stomach or bowel a 5 or 10 per cent. solution of gelatin may be given in large amounts by the mouth. Only well-sterilized gelatin should be used; the English gelatin is the safest, as the commercial product often contains the tetanus bacillus. It may also be used subcutaneously, 4 drams (15 c.c.) of a 10 per cent. solution being the usual dose. Adrenalin, 1 to 1000 solution, is valuable if applied locally on gauze, and for gastro-intestinal hemorrhage adrenalin by the mouth in 2-grain doses may be given every three hours.

Transfusion of blood is a valuable remedy, and should, when possible, be resorted to in all severe cases. In the hands of the experienced surgeon it is not as difficult an operation as many would lead us to suppose, and recent improvement in technic has made it more easy of accomplishment. Next to transfusion, injections of sterile normal human blood serum offer the greatest hope of saving the infant's life. The blood should be withdrawn and kept under absolutely

aseptic conditions; 5½ drams (20 c.c.) of the serum should be injected subcutaneously every six to eight hours as long as the hemorrhage persists. Improvement in the child's general condition is usually noted immediately after the transfusion or serum administration, and the bleeding is often checked in twenty-four to forty-eight hours.

Normal horse serum may be used if transfusion of human blood serum is impracticable. It should be injected subcutaneously in doses of 2½ drams (10 c.c.), and repeated every eight to twelve hours if no improvement is apparent. If the infant has suffered from a profuse hemorrhage, transfusion of blood or saline solution is the best treatment. Whole blood injections are simple and easy of accomplishment. Four drams (15 c.c.) of blood are withdrawn by a syringe from a vein in the donor's forearm, and immediately injected subcutaneously into the infant; later, blood serum or transfusion can be employed, or the whole blood injections can be repeated every eight to twelve hours. Diphtheria antitoxin, owing to its being so readily obtained, may be used instead of human or horse serum. Lactate of calcium, in 5-grain doses every four hours, is of benefit in some cases, while breast-milk and also whisky, 20 drops every two hours, tend to increase the child's resistance.

Acute Fatty Degeneration of the Newborn (Buhl's Disease).—The symptoms in this condition are associated with fatty changes in the liver, kidneys, and heart, and hemorrhages are often found in these organs at autopsy. The liver and spleen are enlarged, and jaundice is a common symptom. The child is born asphyxiated, and is with difficulty made to breathe. Hemorrhages occur from the umbilicus, the blood oozing from the surface of the navel. There are also hemorrhages from the stomach, bowel, and, in some cases, from the mouth, conjunctiva, and nose. Profuse hemorrhages beneath the skin are common, and there may be considerable edema. The temperature is normal. The disease is rare, causes profound depression, runs a rapid course, and usually terminates fatally in a week or two. It is undoubtedly due to septic infection, the portal of entry in the majority of cases probably being through the umbilicus.

Epidemic Hemoglobinuria (Winckel's Disease).—This affection occurs in the first few days of life, its special symptoms being cyanosis, jaundice, and hemoglobinuria.

Etiology.—It is undoubtedly due to infection, and the cases reported by Winckel occurred as an epidemic in an institution; the infection was probably conveyed through bathing or drinking water. At autopsy the colon bacillus and streptococcus have been found in the blood and internal organs in a few cases.

Pathological Anatomy.—While all the evidence points to septic infection of the newborn, still the umbilicus and the umbilical vessels are normal. The spleen is swollen and contains much blood pigment. The kidneys are enlarged, the tubules filled with hemoglobin crystals. Minute hemorrhages are seen in almost all of the organs, while larger amounts of blood may be found in the stomach, bowel, liver, and pleura.

The mesenteric glands and Peyer's patches are swollen, and there is fatty degeneration of the liver and other internal organs.

Symptoms.—At birth the infant is apparently healthy and normally developed. Usually about the fourth day it is noticed that the baby is extremely fretful and more or less cyanotic; the symptoms rapidly progress, the child becoming profoundly prostrated, with rapid pulse and respiration. Jaundice of a severe type appears, the skin becoming deep yellow or bronze, the cyanosis more marked, especially on the body and legs, while the temperature may be either normal or slightly elevated. The infant loses strength rapidly. Diarrhea or vomiting may develop, urination is frequent, and the urine voided very dark, containing blood cells, hemoglobin, renal epithelium, granular casts, and a small amount of albumin. Before death the child often becomes comatose, and may die in convulsions. The typical urinary findings will confirm the diagnosis. The disease is fatal in the severe form, and, as it is undoubtedly the result of an infection, the treatment is the same as that recommended for septic infection.

Melena Neonatorum.—The name, melena, is applied to those cases in which bleeding occurs either from the bowel, the stomach, or from both. It is, of course, well understood that hemorrhage from the stomach or bowel may occur in various diseases, and may be a symptom of many different pathological conditions; but, as hemorrhage is often the only dangerous symptom that presents itself, and one which, if not controlled, may cause the death of the infant, its consideration is important. Melena is not the name of a definite specific disease, but, as the term is generally used, it describes a symptom-complex; therefore, it is well, perhaps, to retain it. The more, however, one studies the conditions associated with hemorrhage of the newborn, the more thoroughly one is convinced that it is due to sepsis. The study of the coagulation of the blood in these hemorrhagic cases assists somewhat in explaining the persistence, at least, of the bleeding. The elements that are normally present in the blood and which produce coagulation may not be present in normal amounts or there may be a normal amount of some of these elements and a deficiency in others. Whipple has recently demonstrated the interesting fact that old fibrin has been found in the alveoli of the lungs, but that no fibrin of fresh formation exists, which proves that the elements necessary for coagulation were present at birth and that they disappeared in the first few days following birth. The reason why transfusion of blood and subcutaneous injections of blood are beneficial in these cases is quite clear when we consider that transfused blood or injected serum may supply the elements needed to restore coagulation.

Melena includes only those cases in which the source of the bleeding is in the gastro-intestinal tract. Blood from the nose, lips, or mouth may be swallowed by the infant and later vomited. A bleeding fissure in the mother's nipple may result in the infant's swallowing blood during nursing, and this may subsequently be vomited or appear as blood in the stools; this condition is not spoken of as melena.

9

Hemophilia is rarely the cause of hemorrhage during the first few days of life; as proof of this is the fact that the cases of melena that recover show no tendency to subsequent bleeding.

In the newborn gastro-intestinal hemorrhages may occur in many conditions, as already stated in the consideration of sepsis, the hemorrhagic diseases of the newborn, Buhl's disease, and Winckel's disease. But all of these affections give rise to other decided symptoms besides the hemorrhages; therefore, they can be differentiated, at least clinically, from melena.

Etiology.—Sepsis is the most common cause of hemorrhage in the newborn, and, while the hemorrhage of sepsis is not usually a single hemorrhage unconnected with other symptoms, still this undoubtedly does occur, and sepsis is now accepted as the usual cause of melena. Many observers regard syphilis as a cause, since in this disease the intima of the small vessels and capillaries becomes thickened, cell infiltration and subsequent connective-tissue formation produce a narrowing of the lumen and cause venous stasis and hemorrhage. In the newborn, syphilis of the liver, as well as abdominal growths and congenital heart disease, may also produce hemorrhage from the stomach or bowel. In a few cases a gastric or duodenal ulcer has been found at autopsy, and less often an ulcer in the esophagus. A thrombus of the umbilical vein or of the ductus arteriosus Botalli may possibly explain the presence of the ulcers; this results in emboli of the pancreatic, duodenal, or gastric artery, followed by necrosis and ulcer.

Some cases of melena are caused by abdominal pressure and injury during labor; and, as gastro-intestinal hemorrhage may be associated with cerebral hemorrhage, a bulging fontanelle, slow pulse, and asphyxia, associated with gastro-intestinal hemorrhage, would indicate a possible connection between the brain and the melena. If melena is noticed within two or three hours after birth it suggests a birth injury, and it is probably due to some other cause than sepsis, especially if there are no other evidences of sepsis before or during labor. In many cases of melena, however, the child is at birth apparently normal and in perfect health. In melena the bleeding usually begins in the first four days of life, often on the first or second day. In some cases no definite pathological condition can be found; in others a simple congestion without ulceration or small hemorrhagic areas with erosions of the gastro-intestinal mucous membrane are seen at autopsy. Blood cultures or a Wassermann may assist in deciding as to the septic or syphilitic origin of the melena.

Prognosis.—This depends upon the cause of the melena, and upon the amount of blood lost. In some cases the hemorrhage is small, the bleeding does not recur, and the child is not seriously ill. In others the hemorrhage is more profuse, and the child becomes pallid, prostrated, and is in shock. In the most severe cases the infant passes rapidly into collapse and dies. The mildest symptoms may be followed by the most alarming hemorrhage, so that one is always fearful of the outcome until the child has entirely recovered.

Treatment.—The best treatment is transfusion of blood repeated in twenty-four hours if necessary; next to transfusion is the injection subcutaneously of human blood serum, 4 drams (15 c.c.), which may be repeated every eight or twelve hours as long as the symptoms persist. Horse serum may be used if it can be procured more readily than human serum, or diphtheria antitoxin, containing, as it does, horse serum, may be employed. The two latter may be used in doses of 3 drams (11 c.c.), and repeated every eight or twelve hours if necessary. Sterile gelatin in 10 per cent. solution, injected subcutaneously in 4 dram (15 c.c.) doses and repeated every twelve hours, is of value in this disease and it may also be given by the mouth in 5 per cent. solution every three hours in as large amounts as the child can take and retain without producing nausea and vomiting. If the child is extremely weak, with cold extremities, whisky, 20 drops every two hours, should be given, and external heat applied.

Erysipelas.—Erysipelas is due to the entrance into the system of septic material. The infection usually takes place through the umbilicus, although it may enter through a scratch or abrasion in any portion of the body, especially in the genital region, scalp, or face. While erysipelas in the newborn was formerly not uncommon, the modern practice of aseptic obstetrics has reduced the number of cases very greatly.

Etiology.—It is not as yet definitely settled what the specific organism of erysipelas is. Many observers believe that streptococci positively produce the disease, and there is considerable experimental proof to support this view. The infection may be brought to the child by the nurse, the physician, unclean dressings or soiled clothing. Erysipelas is more liable to develop in the infant if the mother is septic, the infection being conveyed to the child by the infected lochia either through infected dressings, clothing, or the soiled hands of an attendant.

Symptoms.—The infant offers only a slight resistance to the infection, consequently the disease shows a tendency to spread and frequently extends more or less rapidly over a wide superficial area. In early life it may also involve not only the skin but also the subcutaneous tissues. It usually appears first from the sixth to the tenth day after birth, and, if at the umbilicus, the redness and swelling quickly involve the neighboring portions of the abdomen and spread to the pubic region and down one or, perhaps, both thighs. If it begins at the corner of the eye or in the region of the ear, or in the scalp, it quickly spreads over the entire face, and commonly extends also to the neck and upper chest. The involved area is red, edematous and hot. The systemic symptoms are marked. There is usually high fever, 102° to 105° F., although the temperature often shows wide fluctuations, and it may be normal or subnormal. Rapid pulse, marked prostration, and often loss of appetite, with vomiting and diarrhea and, possibly, convulsions are common symptoms. Wasting may be rapid, although if the child takes and retains the breast, and there is no diarrhea, it

may not be especially noticeable. Hemorrhages from the navel, intestine or beneath the skin may occur, and jaundice may be a marked symptom. Bronchopneumonia is, in erysipelas, as in so many of the dangerous illnesses of infants, a not unusual complication. Septic arteritis, phlebitis, and peritonitis are quite often associated with an erysipelas which originates at the umbilicus, and sloughing of the subcutaneous tissues is much more common in infants than in older children or adults. Septic infection of the lungs, cerebral meninges, and pericardium are frequently found at autopsy. The liver, kidneys, and spleen are less often involved.

Prognosis.—Erysipelas in the newborn, especially if it begins at the umbilicus, is a dangerous disease and often ends fatally, and if internal organs are involved the prognosis is unfavorable. The less severe cases which involve the scalp, face, and chest are dangerous but not necessarily fatal. In children of one year or older the prognosis is much more favorable, although even in the older children, in my experience, the disease shows a much greater tendency to spread than in the adult and is apt to run a much longer course.

Treatment.—As erysipelas in the newborn is the result of septic infection prophylactic treatment should embrace the aseptic care of all lesions of the skin or mucous membrane occurring at birth. The umbilicus especially should be treated according to modern aseptic methods. Dry dressings are the only ones that should be used. At the first sign of infection, strict antiseptic methods are necessary. Daily washing of the infected part with 1 to 1000 bichloride solution and the application of gauze kept wet with this solution are of assistance in limiting the spread of the infection. Prophylaxis also includes the absolute separation of the child from a septic mother. It is of the first importance to sustain the child's strength and for this purpose breast milk is by far the most useful food. Whisky, 10 drops every two hours, may be given to combat the general depression.

Local applications to the affected area tend to relieve somewhat the intense burning and pain. Ichthyol, 10 to 15 per cent. in lanolin, is one of the best local applications. It partially relieves the pain and may possibly have some influence upon the erysipelatous inflammation. Gauze dressing kept wet with normal salt solution and applied over the inflamed portion lessens the intense burning of the skin. Unguentum Credé rubbed into the healthy skin daily is recommended by many careful observers. Antistreptococcic serum, 4 drams (15 c.c.), administered every twenty-four hours, is a remedy that has been largely used in the treatment of this condition, and, while the results are not always favorable, still there is no question but that it is well worthy of a trial in severe cases. Vaccine treatment vies with antistreptococcic serum as the best method to employ. Unfortunately erysipelas of the newborn is usually of such a virulent type that often no plan of treatment can bring about a cure, but, personally, I believe that all cases should have the benefit of either the vaccine or antistreptococcic serum. One must not expect the rapid cures that have so often been

reported as following this treatment in adults, but if used daily it is of assistance in the erysipelas of the newborn although of much greater benefit in erysipelas of older children.

TETANUS (TRISMUS NEONATORUM).

This is an acute infectious disease due to the entrance into the body, through a wound, of the tetanus bacillus.

Etiology.—The infection usually occurs through the umbilical wound, although it may enter through the umbilical vessels or any wound or abrasion in the child's body. It is essentially a filth-born disease and is usually found where the surroundings of the child are unclean. It may be carried to the open umbilical wound by soiled hands and dressings, or by the dust-laden air. The bacillus is usually found in the top-soil of garden earth, but not at a greater depth than one foot, and in the excrement of animals, especially that of the horse. Tetanus is more common in tropical countries, and the colored race is particularly susceptible. Since the cause of the disease has become known the number of cases has greatly diminished.

The bacilli of tetanus increase rapidly in numbers at the point of infection, but remain for a short period of time only, being evidently short-lived. A few only of the bacilli are absorbed; the toxins produced, however, enter the lymph channels and combine with the motor cells, in the anterior cornua of the cord and medulla, increasing greatly the excitability and irritability of these motor cells, so that the slightest external irritation is sufficient to produce tetanic spasms. There is no involvement of the muscles of the peripheral nerves, as is shown by the fact that the tetanic spasms cease if the nerve trunks are divided. So far as is known the disease produces no definite lesions in the internal organs of the body.

Symptoms.—The first symptoms usually appear from the fifth to the twelfth day after birth; rarely after three weeks. The special symptoms are—continued contraction of the voluntary muscles, and the development at irregular intervals of tetanic spasms of greater or less severity. The first symptom noticed is the inability of the child to nurse; this is due to the fact that the child cannot open its mouth —it already has trismus or lockjaw. The spasm spreads to the face producing a characteristic expression—the so-called risus sardonicus. The forehead is drawn into deep wrinkles, the eyes are tightly shut, the mouth is closed and puckered. The muscles of the neck and back are the next involved. The head is drawn backward, the abdominal muscles become fixed, the arms are flexed at the elbows, the legs are stiff and extended. The elbows and knees can, with difficulty, be either flexed or extended, and opisthotonos develops. The tonic spasms increase in number and severity and the slightest reflex is sufficient to produce the spasm. The merest touch, the effort of nursing, or a current of air may bring on an attack. As the disease progresses the periods of relaxation occur less often and are of shorter

duration. The arms remain more or less continuously stiff and flexed, the hands tightly closed, the legs stiff, the jaws closely locked, and the child swallows with difficulty or not at all. Owing to a spasm of the laryngeal muscle the respiration may be noisy and dyspnea and asphyxia may be more or less marked. The contraction of the muscles and the spasm may relax during sleep. The pulse is accelerated, albumin is usually present in the urine, and there may be other signs of septic infection. The navel may appear normal, although very commonly it presents an unhealthy and ulcerated appearance, accompanied by a thick, purulent discharge. In the severe cases the temperature is usually high, 104° to 106° F. The fatal cases usually run a rapid course, death occurring commonly in from six to forty-eight hours after the onset of the symptoms. Recovery may occur, however, even in the worst cases. In the milder cases the temperature is lower, 100° to 102° F., and the spasm is limited to the jaws, face, and neck. The attacks gradually diminish in frequency and severity, and the case may go on to recovery. In these cases the spasm relaxes to a greater or less degree, the child gradually breathes and swallows better, and the intervals between the spasms increase in length. In those infants that recover the symptoms last, as a rule, from a few days to three weeks. The child is left emaciated by the disease, and following the spasm is invariably weak and exhausted.

Diagnosis.—The diagnosis is usually made without any great difficulty. The development of trismus as an early sign, and the continued contractions, well marked in the severe cases and present in a less degree in the mild cases, with the occurrence of tetanic spasms at irregular intervals, are very suggestive of the disease. Tonic convulsions may occur with meningitis, and congenital spastic paraplegia may follow cerebral disease, but lockjaw is not present and slight external causes do not provoke a tetanic spasm.

Prognosis.—This depends upon the severity of the case, the time at which the first symptoms develop, and the duration of the symptoms. The more severe the symptoms and the earlier they appear the graver the prognosis; the later the symptoms develop and the longer they persist the better the prognosis. Sporadic tetanus is less apt to be fatal than epidemic tetanus. Modern treatment is helpful in reducing the mortality; although probably 75 per cent. of the cases end fatally.

Treatment.—As the majority of cases occur from umbilical infection the aseptic care of the cord is of the utmost importance. This includes the aseptic care of the umbilicus until the wound at the navel has entirely healed. In those regions where epidemics of tetanus have occured the antiseptic treatment of the cord and its subsequent care are essential as a means of prophylaxis. As soon as the first suspicious symptoms of tetanus appear, tetanus antitoxin should be administered. In order to be of much benefit the antitoxin should be used early, as the toxins of tetanus combine with the motor cells in the cord and medulla, and after combination are only slightly influenced by the antitoxin administered. The toxins still circulating in the blood can,

however, be neutralized. The antitoxin apparently does no harm even if administered in large doses, and consequently should be used freely. It may be administered by lumbar puncture, intravenously or subcutaneously. Ten cubic centimeters of spinal fluid should be withdrawn by lumbar puncture, and 10 c.c. of tetanus antitoxin injected every twelve or twenty-four hours as long as the severe symptoms persist; if the symptoms become less marked the injections may be given every second or third day. If not injected into the spinal canal the antitoxin may be given either intravenously or subcutaneously in the same doses as recommended by lumbar puncture. Bromide of soda in 5-grain doses combined with chloral, 1 grain, may also be given every hour either by the mouth or rectum as long as the violent symptoms persist. Small doses are useless and large ones essential in assisting to control the symptoms. If, owing to the trismus, the child is unable to swallow it may be fed breast milk with a tube passed through the nose. Absolute rest and quiet should be insisted upon, and, as the tetanic spasms are often produced by very slight causes, all external sources of irritation should be removed. While the treatment of tetanus with antitoxin has not given good results, still, in such a dangerous disease, it is well worthy of trial, and, if possible, should be administered in every case.

SCLEREMA.

In this disease the skin and subcutaneous tissues become firm and hard. The induration of the tissues usually appears first in the legs below the knees and gradually extends upward, involving a portion or practically all of the body. The genitals, soles of the feet, and palms of the hands are not involved.

Etiology.—The etiology is still in doubt. The composition of the fat of the newborn, containing as it does about 20 per cent. less of oleic acid as compared with the adult, and a greater amount of stearic and palmitic acids, is believed by some to predispose the infant to the disease. Others believe that it is an infectious disease, and, while its infectious origin has not been proven, personally I believe that it will be shown to belong to this class. Streptococci have been found at autopsy. Still others believe it is associated with certain changes in the nervous system. Prematurity and unhygienic surroundings are predisposing causes, and it is more common in institutions than in private practice. In atrophic infants a few months old, sclerema may follow cholera infantum or an enteritis with frequent watery stools.

Pathological Anatomy.—The skin and subcutaneous tissues are dry and hard. The superficial fat is firm and dry. Atelectasis is often present, and enteritis is not uncommon in children who have lived for some days. Hemorrhages into the lung and myocardium may occur.

Symptoms.—It is an uncommon disease, and may occur in the first few days of life, or, in weak and premature infants, as late as the third or fourth month. The most common locations are the calves of

the legs, mons veneris, cheeks, thighs, and lower portion of the trunk, more particularly those parts of the body where fat is abundant. As a rule the disease lasts in the severe cases only a few days. The temperature falls soon after birth and never returns to normal. A temperature of 95° F. is quite common; it may fall as low as 85° F., and cases have been reported where it has fallen as low as 72° F. The baby feels cold, and its skin is hard and atrophied. The child loses in weight, its legs become stiff, and it lies absolutely quiet in bed. The mucous membranes are dry, the sleep is restless, the urine is scanty, and there is constipation. The amount of nourishment taken is small, the respirations are superficial and slow, often below 18. The pulse is slow, falling to 60 or even lower in some cases, and is difficult to find. The lips and finger tips may be cyanotic, and there may be more or less associated edema. The skin may be of a blue or reddish tint, and a serous fluid exudes if the skin is slightly incised or punctured. The child utters a sharp cry; this is more often noticed in older children. Jaundice may be present, albumin in the urine is not uncommon, and, less often, granular casts and red blood cells are found, and occasionally, sugar. The younger the child the more serious the prognosis and the more rapid the course of the disease. In the fatal cases the asthenia becomes progressively greater, and the infant sinks into coma and dies. In the cases that recover the hardness of the skin and subcutaneous tissues gradually lessens, the normal color returns, and the infant slowly gains in vitality and strength. Effusions have been found in the pleura and peritoneum, and pneumonia may develop as a complication.

Diagnosis.—Hardness of the skin, subnormal temperature, and stiffness of the body are characteristic.

Prognosis.—The prognosis is bad in the newborn; probably about 35 per cent. die. The more vigorous the child the better is the prospect of recovery.

Treatment.—The best results are obtained by combating the low bodily temperature. This is best accomplished by placing the child in an incubator, or by keeping it surrounded with hot water bags, or the heat may be supplied by an electric warmer. Hypodermoclysis of normal salt solution, or the introduction of salt solution per rectum according to the Murphy method, is a treatment that has apparently been beneficial in some cases. The child should be nourished by breast milk. If too weak to nurse the breast milk may be fed to the infant in small quantities at regular intervals.

SCLERO-EDEMA.

This is not a rare disease. It is most common in small, weak, and premature infants or in twins. A history of syphilis may exist. It has been found associated with congenital heart disease and with nephritis. It is most likely to appear in winter and in cold climates, in institutions and hospitals, and where the surroundings and hygienic

conditions are unfavorable. It usually develops between the second and fifth days of life, rarely after the fifteenth day.

Etiology.—Poor circulation and feeble respiration are factors present in practically all cases. If an infant so affected is exposed to cold it increases the liability to develop this condition. It is claimed by some that sclero-edema is the result of an infection.

Pathological Anatomy.—An exudate is present in the skin and underlying fatty and muscular tissues. Venous congestion is found in the organs of the abdomen particularly, congestion of the pulmonary tissues with minute hemorrhages is common, and the myocardium may show fatty changes. The microscopic examination of the skin shows no specific lesion; only a swelling and dilatation of the lymphatics and minute vessels can be demonstrated.

Symptoms.—It is noticed almost immediately after birth that the infant is restless and nurses poorly. The edema usually appears first on the dorsum of the foot, and in many cases is soon afterward seen in the cheeks and inferior portion of the abdomen. The affected tissues are swollen and hard and pit on pressure. The skin and subcutaneous tissues are much more tense than is usually found in edema, and the muscles are also more or less involved, although to a less degree. The affected part may be a reddish pink, blue, or mottled color, and the superficial tissues may be lifted up from the underlying structures. The skin feels cold, and if the swelling is extreme the child's body is stiff and almost motionless. The infant, owing to edema, does not lose weight. The temperature is subnormal in all cases. In mild cases it may average 95° F., and in severe ones it may fall to 90° F., or even 85° F. The pulse is weak and the respiration unnaturally slow. The urine is diminished in amount but albumin is not usually present. Swelling of the scrotum and penis generally occurs and a large part of the body may be involved. In the worst cases the child gradually becomes weaker and passes into a stupor which ends in death in from three to six days. In the milder cases the temperature gradually returns to normal, the child's respiration and circulation improve, the edema slowly disappears, and the patient recovers, the duration of the disease being eight to fourteen days. Pneumonia may appear as a complication.

Diagnosis.—In well-marked cases the diagnosis is not difficult. In sclerema the affected parts do not pit on pressure, they are much firmer than in sclero-edema, and the scrotum and penis are not involved. In the edema of nephritis the tissues are much softer and more pliable.

Prognosis.—In the mild cases the prognosis is favorable but is bad in severe cases, especially if heart disease, pneumonia, or nephritis coexist.

Treatment.—Prophylaxis includes the prevention of unnecessary chilling of the body in weak and premature infants, the application of external heat in all such cases, and insistence upon breast-feeding. The most important part of the treatment is the application of external

heat. This is best accomplished by placing the child in an incubator, or it may be surrounded with hot water bags, and its entire body wrapped in cotton kept in place with woolen bandages. Hot baths are also of assistance and a thorough massaging of the child with 10 per cent. of iodide of ammonium in glycerine has been recommended as having served a useful purpose. Oxygen inhalations are of benefit, and breast feeding is absolutely essential. If unable to nurse, the infant must be fed with a spoon, medicine dropper, or by gavage.

ACHONDROPLASIA (CHONDRODYSTROPHY—MICROLELIA).

In this disease the arms and legs at birth are short in proportion to the trunk, which is of about the normal size. If the children affected survive the first few years of life, they often live on to advanced age, and become robust and muscular, but are dwarfs.

Etiology.—The disease affects both sexes equally, but is usually transmitted by males, and has been known to exist in three successive generations of males. The children of parents who have achondroplasia may inherit the disease or may be absolutely normal. The changes in the affected bones probably occur in the first few months of fetal life. There are no known predisposing influences, the true cause being entirely a matter of conjecture and theory.

It has been suggested that a perversion of the internal secretion which affects the development of the epiphyseal cartilages may be the cause; that the changes may, possibly, be toxic in nature; may be due to an infection; or may be associated with certain changes in the placenta. Summers and Wallace have recently described a cretinistic variety of achondroplasia in which certain changes were found in the thyroid gland; but in these cases there were cretinistic symptoms that are not present in typical cases of achondroplasia.

Pathological Anatomy.—In this disease the bony system of the infant at birth shows deformities which are the result of interference with the normal deposit of bone cells in the fetal cartilage. No changes, or but slight ones, are found in those bones in which ossification develops intramembranously—as in the flat bones of the skull—nor in those which have a tendency to remain cartilaginous *in utero;* but changes are especially noticeable in those bones in which ossification of the cartilage normally takes place in fetal life.

The bones of the arms and legs are the ones most markedly deformed. The tribasilar bone in the floor of the skull often partly ossifies during fetal life, and prevents normal development of the *base of the skull,* consequently the cranium is expanded above and an unusual prominence of the parietal and frontal bones results. The faulty nutrition of bone cells is especially marked in the long bones—bowing and shortening them—and is the result of interference with endochondral ossification which is most marked at the extremities of the long bones.

The pathological changes are found only in endochondral ossification. Bone formation from the periosteum continues, and the shafts

of the long bones are formed largely from this periosteal growth. The epiphyses are more or less enlarged. The long bones fail to increase in length owing to dystrophy of the epiphyseal cartilages during intra-uterine life; in some cases a fibrous ingrowth of the periosteum between the epiphysis and the diaphysis is present and tends to check the increase in length.

The thyroid gland and all other organs are apparently normal. The chest is smaller than normal, and the ribs are badly formed. The bones of the limbs, the ossa innominata, the ribs, and the basa occipital are especially affected.

FIG. 20.—Achondroplasia in a child aged two years.

Symptoms.—The infant may be stillborn or die in the first few weeks of life. The short arms and legs, with the trunk much longer in proportion than the extremities, produce an unusual type of dwarf, the ordinary height being from 40 to 48 inches. The upper arms and the thighs are proportionately shorter than the forearms and legs. The head is large, with prominent forehead and saddle-shaped nose, which constitute a characteristic type of face. The features are large and massive.

The maximum circumference of the head is above the normal, and may suggest hydrocephalus, although hydrocephalus and chondro-dystrophy have nothing in common. The normal spinal curve in the lumbar region is much exaggerated, the scapulæ are short. The abdomen is large and prominent, the buttocks unnaturally thick and heavy, the genital organs are normal.

During early child life the muscular development is usually poor, and the child is late in walking. The anterior fontanelle often remains open until the end of the second or third year, or even later. As the child grows older, the muscles of the arms, legs, and body often become unusually well developed, and the resemblance to cretinism is more or less marked. The condition is, however, not of the same nature as cretinism, and is entirely uninfluenced by thyroid treatment. The hands are thick, the fingers short and nearly of equal length, and the fingers are spread apart by an usual development at the second joint, which produces the so-called "trident hand."

Mentally the children affected are usually rather subnormal, but their mental powers are not markedly defective, and they may even be normal. As a rule, however, they develop slowly, are late in talking, and behind the normal child in acquiring knowledge. The deformity in the sacral region and pelvis caused by defective ossification of the innominate bones may make normal labor difficult and necessitate the use of forceps, or, possibly, the performance of Cesarean section.

Diagnosis.—The condition has been confounded with hydrocephalus, rickets, and cretinism; but careful examination will invariably result in a correct diagnosis. X-ray plates will show the short and curved bones of the arms and legs, and will aid in the recognition of the disease.

Prognosis.—The general nutrition of the infant is affected, as is evident from the poor ligamentous and muscular development. Many of the children die during birth, or, owing to poor development, soon afterward; others, as has been stated, may live to extreme old age. They marry and have children, and their offspring may either show achondroplasia or be absolutely normal.

PLATE I

Achondroplasia.

Expanded diaphyseal ends and epiphyses with very short shafts. The arms and legs are much shorter in proportion than the trunk. The upper arms and the thighs are proportionately shorter than the forearms and the legs below the knees.

CHAPTER VIII.

INFANT FEEDING.

Cow's Milk.—The infant may be fed on cow's, goat's or ass's milk, but as the milk of the cow is almost always obtainable, and that of the ass and goat is often difficult to procure, cow's milk is, for practical purposes, the one which is universally adopted in the United States.

Our first thoughts in regard to cow's milk should be of the cows and the farm. The herd should be healthy and free from any taint of tuberculosis. They should be properly fed both in and outside of the barn, and care should be taken to see that the pasture fields do not contain weeds and rank growths. The cows should be carefully groomed, given an abundance of fresh air and fresh water, and the manure should be removed from the barn as frequently as possible.

The milkers should be clean, and, if milking is done by hand, the milkers should wash their hands just before milking, and the milk be received into the pail in such a manner as to avoid any contamination from the cow's udder The first few streams of milk which flow from each teat should be thrown away, as this contains the major portion of the bacteria.

The milk should be cooled to 45° F. within an hour after milking, and kept at or below this temperature until it reaches the consumer.

Much could be written about the production and care of milk, but space does not permit. It is, however, the plain duty of every physician to familiarize himself with the production of milk as seen in a model, modern dairy, and to urge upon all those with whom he comes in contact professionally the importance of a good and unvarying milk supply. All milk used in infant feeding should be fresh and clean, should contain no pathogenic organisms or preservatives, and its chemical composition should vary from day to day as little as possible. Unless the percentages of fat, protein, and sugar remain fairly constant, it will be impossible to give to the infant, whose bottles are prepared from this milk, the same milk mixture each day.

If the herd is properly cared for on a modern farm, there is less variation in the mixed milk of the herd than is noted in the milk of a single cow. For this reason it is wiser to use mixed milk from a herd of cows rather than the milk of a single cow. The milk from one cow is often, however, used with the very best results in feeding one or more infants, but one should be certain that the cow receives every requisite care and attention.

The chemical composition of the milk will vary according to the breed. Holstein cows produce a large amount of milk with an average composition of fat 3 per cent., sugar 4 per cent., proteins 2.8 per cent.

Jersey cows usually give less milk, but the fat will often average 5 per cent., sugar from 4.5 per cent. to 5 per cent., proteins 3.65 per cent.

The milk from a herd of healthy, but common, varieties of cows is to be preferred to that of high bred or fancy stock, simply because the high bred animal is often delicate and susceptible, therefore more sensitive to its surroundings, and more apt to become temporarily sick or diseased than are the hardier herds.

The average composition of the milk of such a herd is as follows:

Fat .	3.5	to	4.0 per cent.
Sugar .	4.0	to	4.5 "
Proteins .	3.5	to	4.0 "
Ash .	0.7	to	0.8 "
Water .	88.3	to	86.7 "
	100	to 100	"

The importance of securing a clean, raw milk, of definite chemical composition, free from pathogenic organisms, containing not more than 10,000 bacteria per cubic centimeter, and no preservatives, is readily understood and appreciated.

It is, of course, apparent that to produce such milk entails much additional expense to the dairy owner, and, of course, an extra high price to the consumer. Dairies capable of producing such a product, which is commonly called "certified milk," are now established near almost every large centre of population, and, in fact, in many small communities the additional price which certified milk brings to the producer has been a sufficient stimulus to make an energetic dairyman develop a model farm.

In some large cities, as Philadelphia, a number of such model dairy farms have been established, and the resulting competition keeps the standard high, and the public is, consequently, all the better protected. In order to maintain the standard of certified milk, it is customary to have samples bought in the open market, and examined by competent chemists and bacteriologists who are selected by a local Milk Commission to whom they make their report.

Bacteria in Milk.—Milk is one of the best culture media for bacteria, although their growth depends largely on the temperature. Milk kept at 45° F. will show very little bacterial growth; at ordinary summer heat, 70° to 90° F., the growth is rapid. All milk contains a certain number of bacteria, and a low bacterial count, such as 10,000 per cubic centimeter, is the usual standard for certified milk.

There are, as a rule, fewer bacteria in milk bottled at the farm than in milk transported in large cans from the dairies, and sold to the consumer from the cans. A milk that does not contain more than 50,000 bacteria in winter and 100,000 in summer per cubic centimeter is generally considered good milk.

Cream usually contains a much higher bacterial count than milk. In gravity cream, especially, the bacterial count is high, as, apparently, most of the bacteria are carried by the fat globules to the upper cream

layers. Centrifugal cream contains less dirt and bacteria than gravity cream. The number of bacteria within certain limits is, however, not a matter of vital importance, provided that no pathogenic bacteria are present.

If, however, the bacterial count is only 10,000 or 20,000 per cubic centimeter it points to the fact that the conditions under which the milk is produced are exceptionally good; consequently it indicates less probability of pathogenic organisms being present. The cleanliness of the cows, the barn, and the milker, the fact that no dry fodder is fed to the cows before milking, and no sweeping permitted—both of which produce dust—and the careful wiping with a wet cloth of the cow's body, especially its udder, just before milking, are important aids in the reduction of the bacterial count.

The sterilization of all milk containers, the straining of the milk through sterilized cloths, and its rapid cooling, with absolute cleanliness of the milkers, are also factors which assist in reducing the bacterial count. The custom of bottling milk at the dairy is an excellent one, and that of serving all milk to the houses of consumers in bottles most important.

If the cow's udder is diseased, pyogenic organisms may be found in the milk, or the germs of tuberculosis, anthrax, or foot or mouth disease may enter the milk directly from the cow. The menace to public health when cows are infected by these diseases, and the danger of transmitting them by means of their milk, as well as the danger of infecting other cows, are now quite generally appreciated, and State laws are being made more and more stringent.

Of the pathogenic bacteria found in milk the most important is the typhoid bacillus, which, being water-borne, may gain access to the milk in many ways, especially through an infected milker, or by using infected water for cleaning cans and diluting the milk. Diphtheria and scarlet fever may be spread by milk, commonly through some one who has the disease, and is employed in the dairy.

The putrefactive bacteria in milk act on the proteins and may form toxins which, being taken by the infant with its milk, may cause dangerous symptoms; or, again, certain putrefactive bacteria may develop rapidly in the intestinal tract of the infant, especially if the child has intestinal indigestion, and often produce severe or even dangerous symptoms.

The Souring of Milk.—This is caused by the lactic acid-producing group, and the rapidity of their growth is increased by the milk sugar. At the stage of souring they form 90 per cent. of all bacteria present.

The Preservation of Milk.—Milk, when it reaches the consumer, is usually a mixed product, a part of it twelve hours old, the other part twenty-four hours old. This milk is usually the day's supply for the household, so that, by the time it is used, some of it is forty-eight hours old. If the milk comes from a first-class dairy, where absolute cleanliness is insisted upon, and the milk is, immediately after milking, removed from the milking barn, and rapidly cooled to 45° or 50° F.,

and kept at this temperature until it reaches the consumer, it ought to be fresh and sweet. When consumed this milk should not have a bacterial count above that which is considered safe.

Now, as a matter of practical every-day experience, we know that the major portion of the milk sold is not produced in these model dairies, and it is difficult to enforce strictly those regulations that require the milk to be kept cool until it reaches the consumer, although much has been done by law to insure the keeping of milk at a low temperature during transportation.

The problem is, how is this ordinary market milk to be preserved? It can be said without hesitation that it is most unwise to add to the milk any chemical preservative whatsoever. The use of formaldehyde, salicylic acid, or any other preparation for this purpose should be forbidden by law. Given a milk which we believe to be unfit to feed to an infant or child, we can by the application of heat largely destroy the bacteria present, and by keeping it as cool as possible after this heating tend to prevent the subsequent growth of bacteria and, also, its becoming sour. In other words, we may sterilize or pasteurize such milk.

Fig. 21.—Arnold steam sterilizer. (Abbott.)

Sterilization. — By sterilization we mean the heating of milk to the boiling point, 212° F. or 100° C. This is most easily accomplished by the use of an Arnold steam sterilizer. The milk should be kept at 212° F. for twenty to thirty minutes, and then rapidly cooled by placing the bottles in the rack under cool running water, and then setting them on ice. The rapid cooling is important, as it prevents the separation of the fat globules.

Among the very poor and ignorant, it is impossible to have the milk kept on ice after it has been sterilized for the usual twenty or thirty minutes. In such cases it is easier to sterilize the milk for one hour, as such milk, if kept in well stoppered bottles, will not turn sour, and will show no increase in the growth of bacteria for twenty-four hours or longer. If an Arnold sterilizer is not available, the bottles of milk may be placed upright in a vessel containing warm water, and this water may be boiled for twenty to thirty minutes.

Among the poorer classes the day's supply of milk may be placed in a large corked jar, the jar placed in water, and the water boiled for the requisite number of minutes. Sterilization in this manner will destroy all pathogenic bacteria, and practically all of the other bacteria, but it will not destroy the spores, and these spores may subsequently develop into bacteria.

When the milk supply is poor the need of sterilization is self-evident. Not only does it destroy the bacteria, but it provides for the children of the poor a milk that is safe to be used in hot weather, and requires very little intelligence or time for its preparation or subsequent care. Even for sterilization, however, as clean a milk as possible should be procured, since the spore-bearing bacteria are not destroyed; they may, when they develop, act upon the protein of the milk and not upon the milk sugar. Such milk may be capable of producing severe toxic symptoms, and yet be considered safe, since it may not be sour. Sterilization tends largely to prevent the appearance of casein curds in the stools; but, if already present, it is one of the best methods for getting rid of them.

Sterilization makes it possible to feed cow's milk to a large number of children among the poor who would otherwise be made ill by the milk, consequently are fed on poor substitutes, such as barley water, albumin water, condensed milk, and proprietary infant foods.

Yet this process is advisable only when the milk supply is unsatisfactory, as it produces changes within it that are undesirable, and all its protective properties, such as ferments, alexins, antitoxins, and agglutins are destroyed. The taste of the milk is altered, and on this account some infants will not take it readily. It is also more constipating; but, as sterilized milk is usually given in summer, this is of little consequence. The milk sugar is partly changed into caramel, the lecithin and nuclein are decomposed, the organic forms of phosphorus are reduced, the lactalbumin is, at least, partially coagulated, the emulsion of the fat is altered, the action of rennet upon the casein is affected, and oxygen, nitrogen, and carbonic acid gases are expelled. In a considerable proportion of the cases scurvy has been shown to have developed in children fed on sterilized milk.

Pasteurization.—In order to avoid the disadvantages of sterilization, and at the same time obtain all the benefits which it affords, a temperature was sought which would be below the boiling point, 212° F., and yet would destroy pathogenic and other bacteria. It has been positively demonstrated that pasteurization, or the heating of milk to 150° to 157° F. (65° to 69° C.), will accomplish this. The milk is kept at this temperature for twenty to thirty minutes. It is then cooled rapidly by placing the bottles in cold running water, and they are then immediately placed on ice or in a refrigerator, so that the temperature will not rise above 45° F.

Pasteurization produces no important chemical or biological change in the milk, and its taste is very little changed. The pathogenic and other bacteria in the pasteurized milk are destroyed, but the spores are not. The lactic acid-producing bacteria, which cause the souring of milk, and are present in considerable numbers in fresh milk, are also destroyed, consequently the tendency of pasteurized milk to sour is lessened. The presence of the lactic acid group also tends to prevent the development of other bacteria.

In pasteurized milk the lactic acid bacteria are destroyed, but the

10

development of the spores is not checked or hindered by either the pasteurization or lactic acid, and the development of these spores is liable to proceed unchecked. It is important, therefore, to use pasteurized milk within twenty-four hours, as the destruction of the lactic acid group prevents the milk from turning sour, and also favors the development of spore-bearing bacteria.

The putrefactive group that affect the protein may develop rapidly, yet the fact that the milk does not turn sour may give one the impression that it is safe for the infant.

The best method of pasteurizing milk is by the use of the Freeman pasteurizer. This is uncomplicated, reliable, and not expensive, and the use of a thermometer insures accuracy in the temperature to which the milk is raised. During the hot summer months pasteurization is an additional protection to the infant, but it should be done in the home of the child; since if milk is pasteurized at the farm, twenty-four hours before it reaches the consumer, the dangers to the infant may be increased rather than lessened.
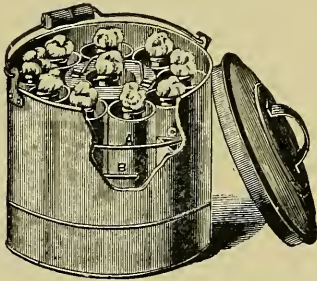
Fig. 22.—Freeman pasteurizer.
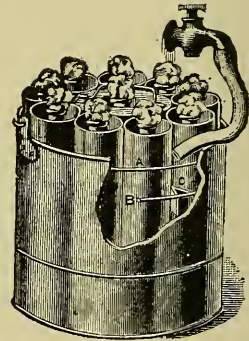
Fig. 23.—Freeman pasteurizer. Receptacle raised for cooling.

Heating does not destroy the toxins that may be present in the milk, but it does destroy the bacteria, and checks their subsequent growth. Therefore, it is not safe to assume that either pasteurization or sterilization compensates for uncleanliness, or can make an unclean and toxic milk safe food for an infant.

Concerning the Feeding of Sterilized, Pasteurized, or Raw Milk.—The only reason for sterilizing or pasteurizing milk is because it is feared that, if fed to the infant raw, it will make the child ill. In deciding then, in a particular case, whether the milk should be heated or not, and, if heated, whether it should be pasteurized or sterilized, the question depends primarily on certain factors—the freshness of the milk and the number of bacteria it contains when it reaches the consumer, also whether or not it has been kept at 45° to 50° F., and the probability of its contamination after it reaches the consumer.

Another important factor is whether the season is summer or winter. Among the poor, especially in cities, the milk they can afford to buy

is always twenty-four hours old, contains usually a large number of bacteria, and is not kept at a low temperature—45° to 50° F.—after they receive it. Moreover, if their surroundings are uncleanly, the risk of contaminating such milk is very great.

Under such circumstances, it is safer for the infant if the milk is sterilized for one hour, and this can be accomplished by boiling the milk in any suitable vessel, preferably a double boiler, pouring it into a clean quart jar, corking it tightly, and keeping it in the coolest place available. Such milk will keep for at least twenty-four hours, often longer, and is, under the circumstances, safer than when pasteurized.

When infants are fed for any considerable period on sterilized milk it is always wise to give them a small amount of fresh orange juice every day or two, as it is a well known clinical fact that many cases of scurvy develop in infants who are fed on sterilized milk.

In the feeding of infants in hot weather all milk that is not " certified " should be pasteurized except when the supply of milk is produced close to the consumer and under exceptionally sanitary circumstances, is cooled immediately after milking, and receives proper care after it reaches the consumer.

It is, in my opinion, much safer in hot weather to pasteurize all milk concerning which there is, in the mind of the physician, the slightest doubt. The heating of the milk to 157° F. practically makes no change in its chemical or biological composition, and children digest it well, and show a satisfactory gain in weight and development. It must be remembered, however, that pasteurized milk should not be kept longer than twenty-four hours, and must always be kept cool. It is true that at a temperature of 70° C. a portion of the lactalbumin and lactoglobulin is coagulated, but the nutritive value of such change is apparently very slight.

The sterilization or pasteurization of milk destroys the pathogenic bacteria of tuberculosis, typhoid fever, and diphtheria, also the streptococcus, staphylococcus, and bacillus coli communis. It is also of assistance in the treatment of the gastro-intestinal infections of infants and children. It does not, however, lessen in any degree the necessity of properly modifying the milk to adapt it to the age and digestive capabilities of the infant.

When May an Infant be Fed Raw Modified Milk?—Personally, I feed many infants on raw modified milk in summer as well as in winter. If a " certified milk " is accessible, and one is sure that this certified milk is only twenty-four hours old, or even less, and has been kept at 45° to 50° F. or lower since it left the dairy; if the consumer can give this milk the best care as to home modification, icing, and freedom from contamination, then I see no reason why the infant should not be fed on raw certified modified milk. Among the intellligent and well-to-do the importance of a clean, fresh milk with low bacterial count is quite generally appreciated; and, if the mother is properly instructed by the physician, the results of such feedings are very satisfactory. If, however, there is any doubt that the various steps in the preparation

and care of this milk will not be properly carried out, then it is much safer, even with certified milk, to pasteurize it, particularly in summer.

Peptonization.—Peptonized milk is of service when an infant is unable to digest sufficient milk to make it gain in weight. It must not be understood that every child that does not gain in weight should be fed on peptonized milk; but by its use a larger amount of food may often be given, and the digestion and absorption of this food be rendered less difficult for the stomach and intestines.

The object of peptonizing is partially or completely to digest the protein before feeding, consequently to give the child a higher protein percentage than would be possible without its use. One may use the peptonizing tubes on the market, which contain five grains of the extract of pancreatin and fifteen grains of bicarbonate of soda, or may order this amount put up in waxed paper powders.

If one of the powders is dissolved in four ounces of cold water, and this added to one pint of fresh milk which is placed in water at a temperature of 110° F., peptonization begins at once, and may be continued as long as desired for each individual child. If the milk is removed after twelve minutes, and immediately placed on ice, further peptonization is checked, and the milk is unaltered in taste. If kept in the warm water for twenty (20) minutes, it usually becomes so bitter that many infants will not take it readily.

Peptonization does not interfere in any way with the modification of the milk, or make it undesirable or unnecessary to use modified milk. The amount of peptonizing powder added is regulated by the amount of milk used at each feeding. Since peptonization takes place only in an alkaline medium, the bicarbonate of soda must always be added. This milk is useful in premature and delicate infants as an aid to digestion, also in convalescence from acute and chronic gastro-intestinal diseases, when it is desirable to give the infant milk, and one more or less doubts its ability to digest it.

It is often, in my experience, possible to give the infant so fed a larger amount of milk if it is partially peptonized, then if there is a gain in weight the amount of powder can be gradually reduced until, in the course of a few weeks, it may be entirely omitted. The fact that the digestive organs of an infant will develop and acquire the power perfectly to digest cow's milk, if the infant is healthy and properly fed, is well known, and this fact is often used as an argument against peptonization, which reduces the normal work of digestion; but peptonization is advised only in those cases where the digestion of the child is temporarily poor, and as the child improves it is gradually discontinued.

Completely to peptonize modified milk requires that the bottles be kept in water at a temperature of 110° F. for one hour. This milk may be used in feeding by gavage those infants who can not, or will not, take enough food properly to nourish them.

In giving nutrient enemata to infants, skimmed milk should be used. A linen or silk catheter, full male size, should be well oiled and gently

passed about 7 or 8 inches up the rectum, and the milk, at a temperature of 100° F., be allowed to flow gently into the bowel. About the same amount of food should be given by rectum as would be suitable for a bottle feeding; this amount, of course, depending on the age of the infant.

The child should lie on the left side with the buttocks elevated, and, if possible, the enemata should be given without causing any fright or nervousness. They should not be repeated oftener than every six or eight hours, and while they are being given the lower bowel should be irrigated once daily with normal salt solution. As a rule, this method of feeding cannot be continued more than a week or ten days, as the rectum is apt to become irritable and the enema is quickly expelled.

Diluents—*Water*.—In order to reduce the 4 per cent. protein in cow's milk to a percentage that the infant is able to digest, it is necessary to add to the milk a diluent, and the most common diluent is water. The amount of water can be regulated so as to produce any desired reduction in the protein. The protein of cow's milk is not only greatly in excess, as compared with breast milk, but the protein in cow's milk has a tendency to form large curds in the infant's stomach, and these are more difficult to digest than the finer flocculi produced by the protein in human milk.

Cereal Diluents.—Cereal decoctions act mechanically, and break the coagulable proteins up into finer particles than is usual when plain water is added. Any of the cereal decoctions, as prepared on page 157, may be used, and by reference to this table one may see exactly the amount of starch that is to be added. If the infant is young, dextrinized gruels are preferable, as the functions of the salivary glands and pancreas, which are only partially established in the young infant, are thus compensated for.

The preparation of dextrinized gruel is very simple. A teaspoonful of liquid diastase, or five grains of taka diastase added to a quart of any of the cereal gruels after they have been prepared, converts the raw starch into soluble carbohydrates. The cereal decoction should be stirred after the diastase is added, and as the starch becomes dextrinized the gruel becomes thinner. The diastase is added after the gruel has cooled sufficiently to be tasted. After it becomes thin by the addition of the diastase, it should be strained, and then kept cold.

One of the best preparations of diastase to use for dextrinizing gruels is called cereo. Half a teaspoonful of cereo is sufficient for one pint of gruel. In the infant of six months or older the starch digestive function is sufficiently developed to render dextrinization unnecessary. The most common form of cereal diluent is barley water, and, if there is any vomiting or diarrhea, it is the one to be preferred. Oatmeal water, being slightly laxative, is to be selected if there is constipation. The digestion of protein is rendered less difficult by the addition of cereal diluents, probably owing to the finer protein curds which they form.

Alkaline Diluents.—The alkaline diluents commonly used are lime-water and bicarbonate of soda water. It is a matter of clinical experience that the addition of either of these alkalies is of service in infant feeding, and their beneficial effect is due to the fact that they tend to retard the action of the rennet ferment in the stomach on the casein. Any lactic acid which may be present in the milk is also neutralized by the alkali added, and, by their action in partly neutralizing the normal acids of the stomach, they lessen the tendency of the casein to form in large curds.

Lime-water is the alkaline diluent commonly employed, and is usually added in the proportion of 5 per cent.—one ounce of lime-water to every twenty ounces of food mixture. The effect of the lime-water is in direct proportion to the amount of cow's milk in the food. If the percentage of milk is large, the effect will be much less than if, as in the feeding of young infants, the percentage of milk is small.

Acting, as it does, on the protein to prevent the clotting action of rennet in the stomach, the formation of clotted paracasein is delayed, and the masses formed are, therefore, smaller. It is evident that, if large amounts of lime-water are added to very weak milk mixtures, the clotting of the milk in the stomach is largely prevented, and the milk will pass into the intestine but little affected by the acids of the stomach.

Next to lime-water, bicarbonate of soda is the alkali most commonly added to the diluent, usually in the proportion of one grain to each ounce of the food mixture. If, however, it is added in the proportion of two grains to each ounce, it tends, as do larger amounts of lime-water, to prevent the clotting of the casein by delaying acid formation in the stomach.

CONDENSED MILK.

Condensed milk consists of cow's milk which has been first sterilized, and then evaporated in a vacuum. Cane sugar is then added, and the preparation is hermetically sealed in tin cans, or sold fresh and unsweetened.

Condensed milk contains, even when diluted with six parts of water, only a little over 1 per cent. of fat and protein, and more than 7 per cent. of sugar. In feeding young infants it is often diluted twelve or fourteen times with water, so that all of these percentages are reduced to one-half of the above. Now if, for any particular reason, it is advisable to feed an infant on these low percentages, condensed milk may be used with advantage.

It is, of course, at once apparent that condensed milk, being so weak in fats and proteins, is poor nourishment for an infant. The child, if fed continuously upon it, owing to its ability to digest sugar, may gain in weight and become quite fat, but its tissues are soft and flabby, it cuts its teeth late, its bones fail to develop properly, it is late in creeping and walking, and becomes rachitic and anemic.

The great objection to condensed milk is that (unless one bears in

mind the necessity of a proper amount of fat and proteins in the diet and the dangers of a deficiency of these food materials) because the baby looks fat and well it may be allowed to remain too long on the condensed milk. Under certain circumstances, as a temporary food, condensed milk is valuable.

Among the poor, especially in large cities where it is impossible for them to procure good milk, or when, owing to ignorance or carelessness they cannot prepare good cow's milk properly or keep it after its preparation, condensed milk is often the safest and best food, especially during the hot summer months. It is cheap, easily prepared, each feeding is made ready at the time of feeding, so that the bottle and nipple can be cleaned each time, and it is sterile.

It is also often very satisfactory for young infants during a few days when travelling, or when the supply of fresh cow's milk is uncertain or unsatisfactory, and it is of use sometimes for young infants who are suffering from acute indigestion, especially, in my experience, when they have been fed excessive amounts of fats, and are suffering from acute fat indigestion. Such babies usually take it greedily, owing to its sweet taste, and it is often retained when modified cow's milk is vomited.

As the infant's digestion improves, the proportions should be gradually reduced from twelve parts of water and one part of condensed milk to six parts of water and one part of condensed milk; cream should then be added, a teaspoonful to each feeding, and this gradually increased to a tablespoonful at each feeding, and then the infant is placed upon weak modified milk mixtures.

THE FOOD MATERIALS USED IN INFANT FEEDING.

An infant may be fed in one of three ways: By its own mother, which, if she has a suitable supply of breast milk, is to be preferred; by a wet nurse, which, if certain precautions are taken as regards her health and her ability to secrete a normal amount of breast milk of a certain quality, is undoubtedly the next best method; or the child may be raised on the bottle.

The raising of a child on the bottle is at all times a responsibility which is not to be assumed by the physician unless he has devoted much time and thought to the study of infant feeding. If the child is placed in the hands of a skilled pediatrist immediately after birth, or as soon as it is decided that it requires food other than breast milk, the difficulties encountered are often slight and the disturbances of the gastro-intestinal tract are usually mild.

If, however, it is poorly nourished and has had more or less severe gastro-intestinal disturbances for weeks or months, its subsequent feeding on the bottle becomes one of the most difficult tasks of the pediatrist; and while the final result is almost invariably favorable, one must expect to encounter, during the period of feeding, relapses and setbacks which, being looked for, will not discourage the physician who has thoroughly studied and understands his subject.

It must be well understood at the outset that infant feeding is a difficult problem and that it is a very broad and important subject. It includes the feeding of the infant in sickness and in health. It is intimately connected with the problems of etiology and diagnosis, and is linked with many other associated factors, as heredity, environment and fresh air. The food which is provided by each mammal for its young is particularly suited to develop its digestive organs as well as the rest of its body, and is always animal and never vegetable.

During this early nursing period of child life, it seems, therefore, normal for the food to be in animal and not vegetable form. It is also highly important that the digestive powers and possibilities of the infant be carefully studied and understood, and only such food given as the child is able to digest. It is important to remember that while cow's milk is usually the best artificial food for an infant, it was intended for the stomach of the calf and not for the stomach of the infant. It is, therefore, safe to give this cow's milk only after the power of the infant to digest it is thoroughly understood.

Infant feeding is one of the most important departments of pediatrics. The problem is not alone to provide suitable food in the proper proportions to sufficiently nourish the infant, but it is important that all the different tissues of this rapidly growing organism be appropriately nourished.

The infant's increase in weight and length is so unusual as compared with any other period of life, that the problem of its feeding differs radically from what one meets with in adult life. The adult requires sufficient food to keep his body in a state of equilibrium. The young child must have food not only to nourish his body, but an extra amount to produce the very remarkable increase in growth that occurs at this period.

Now if it were simply a question of giving the infant a large amount of milk, the matter would be a very simple one. Unfortunately, instead of being such a simple task as this, the problem is often a complex one, and the difficulties are much increased when, instead of being fed on the breast, the infant is being raised on the bottle. The gastro-intestinal apparatus of the infant is extremely delicate, complicated, and sensitive. It is easily disturbed by influences that are often difficult to appreciate and guard against, and if any disturbance of digestion is allowed to continue for more than a short period, it often complicates, to an excessive degree, the question of its food.

The study of infant metabolism clearly shows that there is a continuous using up of the body tissues, and that to replace these tissues and in addition to cause an increase in their growth, requires that the child be fed not only a certain amount of food which it can digest, but also a food ration so well balanced that the different requirements of the various tissues may be so amply met that the baby will develop along well established and normal lines.

To produce gain in weight by a food that does not possess the proper

proportions of the various food elements must always be considered as a temporary expedient, perhaps useful and necessary for the time being, but never to be persisted in long enough to produce anemia, malnutrition or rickets. Unfortunately, the ill-effects of such feeding may not be apparent for weeks or months, and are easily overlooked, especially if the child is gaining in weight. Many of the foods of this character possess a high percentage of carbohydrates and often a low proportion of fat and protein.

If there is one factor in infant feeding that is more important than another, it is the appreciation of the fact that the regular nourishment of the infant must contain certain food elements in more or less definite proportions. These proportions are not absolutely fixed percentages, but they can vary only within certain broad limits, and it is most unwise either greatly to reduce one or markedly increase another food element, unless there be, temporarily, some very good and sufficient reasons.

There are certain laws of physiology which make it imperative that a definite amount of food be given the infant, and, further than this, the food must be composed of fairly accurate amounts of the different food ingredients. If the principles that underlie infant feeding are well understood and adhered to, many of the more common errors of feeding will be avoided.

The child's body is composed of protein, fat, water and mineral salts, and is constantly undergoing a process of waste. This loss must be replaced by the food elements, and the principles of nutrition that underlie the replacing in the child's body of this continuous using up of its own body cells, are the first facts of infant feeding which the student and physician must appreciate. It is important, therefore, that we understand the part that the different food elements perform in the replacing of the loss of the body tissues. These food elements are water, protein, fat, carbohydrates, and mineral salts.

Water.—The infant's body is composed of about 68 per cent. of water, and it is, therefore, only natural that in building up this infant's body, or, in fact the body of any young mammal, a large percentage of the nourishment taken should consist of water. The various food elements are dissolved or suspended in this water and the food is consequently always in a liquid form. The water also serves the important function of eliminating the waste products of the infant's economy, and it is estimated that to meet the various demands of the infant, five times as much water is required, in proportion to its weight, as is needed by the adult. The common practice of giving a child water to drink between its feedings, especially in summer, is, therefore, a good one.

During illness, when the amount of fluids lost by the body is great, or when toxemia exists, the administration of large quantities of water is most important. In fact, water, being present in such a very large proportion in both human and cow's milk—86 to 87 per cent.—is evidently the most important single food ingredient, and if it can be

given in fairly large quantities will assist materially in maintaining body weight, especially in gastro-intestinal disease.

The absence of the other food elements for more than a few days is, however, a great disadvantage to the body tissue, and results in their rapid wasting. The water carries the nourishment, by the various blood and lymphatic vessels, to every organ and tissue in the body, and by carrying off waste products through the skin, bowels, kidneys and breath, prevents the development of auto-intoxication.

Protein.—Protein is an important element in the food, and is that portion of the diet which replaces the nitrogenous waste of the tissues. It is also that element in the diet that is capable of building up new cells in the various organs and tissues, and as the growth of the infant's body is unusually rapid a proper amount of protein is absolutely essential if this growth is to be maintained.

In order that the protein may be utilized for its purpose of cell-building, it is necessary that the fats and carbohydrates be given in sufficient amount to produce all the heat and energy that the infant's body may require. If this requisite amount of carbohydrates and fats is not given, an excessive amount of protein is necessary to produce the bodily heat required.

By supplying this fat and carbohydrate in the diet, not only is the normal animal heat of the body preserved, but they make it unnecessary for the protein to furnish this energy and heat, and the protein is then capable of performing its normal function of cell upbuilding.

There is, however, a decided difference between the protein of human milk and cow's milk. Human milk contains 1.5 per cent. protein, of which lactalbumin forms from one-half to two-thirds of the total protein. Cow's milk contains 3.5 per cent. protein, of which casein forms two-thirds of the total protein. It is at once evident that not only is the protein present in much larger amounts in cow's milk than in human milk, but the proportion of casein is also much greater.

It has been shown by Chapin that each mammal furnishes an animal food which is peculiarly adapted to the digestive organs of its young, therefore we can not doubt that the lactalbumin in mother's milk is easier for the infant to digest than is the casein of cow's milk. It is, however, equally true that we have formerly exaggerated the difficulties that infants have in digesting casein, and that it is not nearly so hard for the infant to digest it as we formerly supposed.

In fact, it is not difficult for the infant stomach to digest cow's casein, if it is not thrown down in the stomach in large clots. If this large clot formation can be prevented, cow casein is digested by the infant with very little difficulty. This can be accomplished by adding to the cow's milk, before it is given to the infant, an alkali, as lime water or bicarbonate of soda, or an acid, as dilute hydrochloric acid. Either the acid or the alkali will combine with the casein and, since rennet can act only in a slightly acid medium, will prevent the casein from forming large clots.

Fats.—In the healthy breast-fed or bottle-fed infant, fat is always found in the stools, and its presence renders the stools less hard and dry. The total amount of fat in the food of the infant at the breast is not, therefore, utilized entirely for its food value. A certain amount of fat cleavage begins in the stomach, and fatty acids and soluble soaps are produced in the intestinal canal.

The bile affords a medium for the solution of free fatty acids, and, to a certain extent at least, fats are transformed into soluble forms before being absorbed. The rapidly growing infant requires a proportionately larger amount of fat than does the older child or adult. This fat, which in human and cow's milk is present in the proportion of 4 per cent., has a very complex composition.

Moreover, fat metabolism is a complicated process which is not yet perfectly understood. There is a decided difference in the composition of human milk and cow's milk, and cow's milk is more difficult for the infant to digest than human milk. Cow's milk contains a smaller percentage of oleic acid and a larger percentage of volatile fatty acids than does woman's milk.

The caloric values of the fat, protein and carbohydrates, according to Rubner, are as follows:

    1 gram of protein yields 4.1 calories.
    1 gram of fat yields 9.3 calories.
    1 gram of carbohydrate yields 4.1 calories.

It is thus evident that fats are the chief source of animal heat, since their caloric value is double that of either proteins or carbohydrates. Although we must appreciate the fact that while fat is always present in the nursing infant's stools, only a portion of the fat in the food is utilized in metabolism. Fats do not build up the cells of the body as does the protein, but the fats and carbohydrates, by supplying the animal heat and energy to the body, save nitrogenous waste and allow the protein metabolism to be used for the growth and repair of the cells of the various organs and tissues.

Fat deposits are found beneath the skin over practically the entire body surface and have an important function in increasing the body weight. They are also an evidence of health; a healthy baby is always a moderately fat baby. This fat supply is a valuable source of heat and energy during an illness when the supply of fat ingested may be much below normal.

The nervous system, which shows such remarkable growth during infancy and which is composed largely of fat, requires a large amount of fat for its normal development. This is clearly shown in the impaired development of the nervous system if either the proportion of fat in the food is too small, or the child is suffering from prolonged fat indigestion. Fat also plays an important part in bony development, and rickets is often the result of fat starvation. Malnutrition and anemia are common conditions due to either too little fat in the food, or to poor fat digestion.

The importance of fat in the diet of the infant may, however, lead

to its being given in excessive amounts. It should never exceed 4 per cent. in a milk mixture, and should always be reduced below the normal or usual amount for the age of the infant in acute gastro-intestinal diseases and febrile conditions; as infants with malnutrition are very apt to suffer from fat indigestion, the amount of fat in the food must be cautiously increased.

Carbohydrates.—The carbohydrates in the form of milk sugar are present in human milk in the proportion of 7 per cent., and as milk sugar in cow's milk in the proportion of 4 per cent. The percentage of milk sugar in both human and cow's milk does not vary to any appreciable amount during the period of lactation. Moreover, a chemical analysis of the sugar contained in human and cow's milk fails to show any differences that are of importance, and, further than this, the sugar in the milk of all mammals is this same milk sugar.

In the feeding of cow's milk to infants, the regulation of the sugar content is brought about by simply adding the required amount of milk sugar to the cow's milk mixture. Milk sugar is less liable to undergo fermentation in the infant's stomach than is cane sugar or maltose. All three varieties—cane sugar, maltose and milk sugar— easily undergo fermentation in the intestines.

It is a matter of clinical experience, however, that the milk sugar of cow's milk is especially liable to fermentative changes in the intestines. This is supposed to be due to the fact that the milk sugar contained in the whey protein of cow's milk is rendered less easy of digestion and assimilation; hence its greater tendency to fermentation.

The carbohydrates represent the largest proportion of solids in human milk, and whether the infant be given carbohydrates in the form of milk sugar, maltose, or cane sugar, the percentage is usually the same, unless it is reduced because the infant has sugar indigestion. All three forms of sugar are changed into glucose by the digestive fluids.

The newborn infant has very little ability to digest starch, and starch should not, as a rule, be added to the diet until the infant is six months old. After this age, the starch-digesting ability of the infant rapidly increases, and many bottle-fed infants are given starch in their food, after the eighth or ninth month. If one decides to give the baby some starch, it is absolutely necessary that the percentage of carbohydrates added to the food by this starch be exactly known. The addition of the starch adds no fat to the food and only a very minute amount of protein.

The amount of starch in the different gruels varies according to the cereal employed, and whether the whole grain, cracked grain, or flour is used. Cereo gruel flour can now be purchased, and, by adding a certain amount of this flour to a fixed proportion of water, definite amounts of starch can be added to the infant's food.

It cannot be too strongly emphasized that while starch is often advantageously added to the infant's diet, it should be added with the same accuracy as in adding to the fat or protein in the mixture,

and as breast milk contains 7 per cent. carbohydrates, starch should not be added to the cow's milk mixture in such amount as to raise the carbohydrates above this 7 per cent.

By the use of the standardized flours, it is possible to know the exact percentage of the proteins and carbohydrates added, and, knowing the exact percentage of the added gruel, one can easily calculate the amount of protein and carbohydrates which the gruel adds to the milk mixture:

PERCENTAGE OF PROTEINS AND CARBOHYDRATES OBTAINED BY THE USE OF STANDARDIZED FLOURS.

	Oat and barley.		Legume.		Wheat.	
	Pro-teins.	Carbo-hydrates	Pro-teins.	Carbo-hydrates	Pro-teins.	Carbo-hydrates
1 level tablespoonful to 1 quart water	0.12	0.60	0.19	0.53	0.10	0.62
2 level tablespoonfuls to 1 quart water	0.24	1.20	0.39	1.06	0.20	1.25
3 level tablespoonfuls to 1 quart water	0.36	1.80	0.58	1.59	0.30	1.88
1 level coverful (1 ounce) to 1 quart water	0.48	2.40	0.78	2.12	0.40	2.50
2 level coverfuls (2 ounces) to 1 quart water	0.96	4.80	1.56	4.24	0.80	5.00
3 level coverfuls (3 ounces) to 1 quart water	1.44	7.20	2.34	6.36	1.20	7.50
4 level coverfuls (4 ounces) to 1 quart water	1.92	9.60	3.12	8.48	1.60	10.00

The carbohydrates are easy of digestion and for this reason are popular as infant foods. They are, however, as previously mentioned, liable to undergo fermentation, and hence carbohydrate indigestion is not uncommon. Infants fed on carbohydrates often increase rapidly in weight, but the flesh is soft and flabby and the infant is deficient in vitality and strength, and may be rachitic.

The carbohydrates have an equal heat-producing power with the proteins. They are, to a certain extent, converted into fats, and so have a tendency to add to the infant's weight. They can not build up the cells of the body as does the protein, but by helping to maintain the body heat, storing up fat and supplying energy to the body cells, they are of great assistance to the protein.

"The ordinary soluble carbohydrates are utilized to a high degree by the animal organism" (Hawk); and if the amount given is not above the power of the individual to assimilate, about 97 per cent. is utilized. The amount of cellulose utilized by the human digestive apparatus is "too small for it to play a role of importance in the diet of a normal individual" (Swartz). There is probably no formation of glycogen or sugar from ingested cellulose.

Mineral Salts.—The rapid growth of all the tissues and organs of the infant, especially of the bones, renders the ingestion and absorption of the mineral salts of great importance.

Potassium, sodium, calcium, magnesium, phosphorus, and a trace

of iron are the most important mineral salts found in milk. These salts, with the exception of iron, are present in sufficient amounts in both human and cow's milk to supply to the infant all that its body requires for normal growth and development. They are not, however, as a rule, found in sufficient quantities in the various foods often substituted for human or cow's milk.

The deficiency of iron in the milk is compensated by the reserve store of iron which is found in the body of the infant, especially in the liver and spleen. The adult, in proportion to its weight, has only about one-third as much iron in the liver and other organs of the body as has the infant. The diet of the infant after the first year contains much more iron, and the iron requirements of the older child are accordingly supplied by the food as the quantity stored in the body has been largely utilized.

According to the analyses of Harrington and Kennicutt, the mineral constituents in human milk are:

Calcium phosphate	23.87
Calcium silicate	1.27
Calcium sulphate	2.25
Calcium carbonate	2.85
Magnesium carbonate	3.77
Potassium carbonate	23.47
Potassium sulphate	8.33
Potassium chloride	12.05
Sodium chloride	21.77
Iron oxide alumina	0.37
	100.00

Calcium phosphate is present in considerable quantity and is especially necessary for the formation of the skeleton. The potassium salts are also present in proportionately large amount, and are especially utilized in the muscular tissue and red blood cells. The percentage of chloride of sodium is also large. The salts are an absolutely necessary part of the infant's food. They are essential for the performance of the numerous functions of cell activity and for the proper development of the nervous system.

It is important, therefore, that the mineral salts be given in such form as is easily appropriated and used by the infant. This is best accomplished by feeding the child either human or cow's milk. Mineral salts are present in definite amounts as important elements of all the fluids of the body, and are necessary for the proper performance of circulation, digestion, absorption, secretion, and excretion.

Breast-feeding.—The best food for an infant is the milk from its mother's breasts. It is assumed, of course, that the mother is reasonably strong and in fairly good health. If she willingly undertakes the duty of nursing her baby, it is of advantage. She should be relieved as far as possible from all unnecessary nervous strain and should use moderate care in her diet, exercise, and sleep. These precautions will all tend to increase the quantity and improve the quality of her milk and make it more likely that she will successfully nurse her baby.

It is, therefore, necessary to inform the young mother who hopes and expects to nurse her child that it is important for her to do everything within reason to preserve her physical health and nervous energy, as otherwise she may unintentionally interfere greatly with her ability to nurse. After an experience of thirty years in the practice of pediatrics; I am persuaded that if the physician is an enthusiast on breast feeding, he will almost always be able to convince the mother of the importance and necessity of nursing her baby.

Breast-feeding must not be made too hard a task, and, occasionally, it may be wise to provide a bottle feeding once a day, for even a very young nursing infant. This allows the mother, on any day that she may desire it, a few consecutive hours for amusement or social pleasure.

Mammary Gland.—The secretion of milk from the mammary gland, if the gland is healthy and the mother not disturbed by disease, worry or overwork, will almost invariably be a milk well adapted to the requirements of the growth and development of the baby.

Its delicate mechanism, as yet only imperfectly understood, is very easily disturbed by illness, pregnancy, menstruation, worry, excessive fatigue, errors in diet, and many other causes, and the milk secreted may undergo many and various changes which may temporarily or permanently lessen its value as a food for the infant.

The mammary gland is a self-regulating apparatus elaborating a smaller or a greater supply according to the demands of the infant. It is not only an organ of secretion but also an organ of excretion. It is important to appreciate that certain drugs may be in part excreted by breast milk, and that during the colostrum period the milk differs distinctly from the milk secreted later.

The child should be put to the breast as soon as possible, usually within three hours, after the mother has recovered from the strain and exhaustion of her labor. This early nursing accomplishes several important objects. It draws out the nipples, promotes uterine contractions in the mother, and tends to hasten the formation of milk in the breasts.

Moreover, there is always in the infant an initial loss in weight, which means a corresponding loss in vitality, and this is at least lessened by giving the child a reasonable amount of nourishment during the first three days. During the first twenty-four hours the child should be applied four times to the breast; during the second and third twenty-four hours, six times; and after this every two hours from 6 A.M. to 10 P.M.

It is true that during the first two days the child receives very little milk, and this colostrum milk differs greatly from the milk secreted later; but I certainly believe that this milk must be of nutritive and protective value, otherwise it would not always be present. If, during this preliminary feeding, the child is extremely restless and cries as if from hunger or thirst, sterile water may be given in two dram feedings three or four times a day.

Frequency of Breast-feeding.—The extremely rapid growth of the infant and the necessity of food to compensate for repair of waste due to its great metabolic activities necessitate the giving to the infant of more food proportionately than to the older child or adult. The infant requires 110 calories every twenty-four hours during the first four months of its life per kilo of body weight; 100 calories during the second four months per kilo; and during the third four months 90 calories per kilo of body weight.

During the night a healthy infant should never be awakened, but, if awake, it may be nursed twice at night during the first six weeks, between the age of six weeks and three months once or twice each night, and between three and six months the child should not receive more than one breast feeding between 10 p.m. and 6 a.m. If it awakes oftener, it should be given a bottle of water.

A normal, healthy infant—if awakened every two hours from 6 a.m. to 10 p.m. during the first six weeks; every two and a half hours from the age of six weeks to three months; and after this, every three hours—receives during the day a large amount of nourishment, and being awakened so often its sleep is more or less disturbed. It is only natural that this baby will require during the night—10 p.m. to 6 a.m.—very little food and a good many hours of sleep.

This regularity of feeding is of great practical importance; it secures regular hours of sleep for the mother, baby and the baby's nurse, and the infant, as a rule, quickly adapts itself to this routine and nurses willingly, sleeps well, and is quiet and happy. If the infant so fed is kept in the fresh air, not picked up when it cries, not rocked or fussed with, it rarely, unless sick, should be a source of great care or anxiety.

Unfortunately, this systematic life, as regards feeding, bathing, sleep, fresh air and exercise, is often deliberately broken. The baby becomes nervous and fretful and cries because, having become accustomed to petting and rocking, it misses this attention. Such a baby may easily disturb the peace and routine of an entire household.

The infant should be allowed to nurse from fifteen to twenty minutes at each feeding, the time being equally divided between both breasts. Some infants, especially if robust and nursing vigorously, and there is a plentiful supply of breast milk, will satisfy their hunger in less time than this. An infant may, however, nurse too quickly from the breast just as it may nurse too quickly from a bottle, and it is advisable, in such cases, to interrupt the nursing every two or three minutes for a period of one minute.

As soon as the infant ceases to nurse, it should be removed from the breast, unless it is evident that it has taken only a small amount of milk—too small an amount properly to nourish it. In such cases, the nipple should be moved gently in the child's mouth, which may stimulate it to nurse. The child should never, however, be allowed to lie with the nipple in its mouth, and not nursing, for more than one or two minutes.

After removing the infant from the breast, the nipples should be

cleansed with water or boracic acid solution. Alcohol and water, equal parts, may be applied if there is any tendency to inflammation; if any fissures are present, a 2 per cent. nitrate of silver solution should be painted over the cracks, and the baby should nurse through a nipple shield.

If the breast becomes inflamed, nursing should be discontinued, and an ice-bag or a number of thicknesses of gauze wet in a saturated solution of magnesium sulphate should be applied over the inflamed surface. The breast should be well bandaged, the mother kept at rest and given a laxative. If the breast becomes filled with milk, a sufficient amount may be pumped out to relieve the tension and pain.

Human Milk.—Colostrum appears in the breasts at about the fourth month of pregnancy, and a few drops can usually be expressed from the breasts at this early period. The amount of colostrum gradually increases as pregnancy advances, and persists until the flow of milk is established, which usually occurs about three or four days after the baby is born.

Colostrum is yellow in color due to the colostrum corpuscles, less sweet than human milk, more distinctly alkaline in reaction, and the specific gravity is higher—1.036 to 1.040. It forms large coagula upon heating and may even coagulate upon standing. It contains a high percentage of protein and 3.34 per cent. of fat. The fat globules vary in size, and colostrum corpuscles are present.

These colostrum corpuscles consist of large cells filled with fat globules of varying size and containing a large nucleus, and are four or five times as large as the milk globules.

The composition of colostrum, according to Camerer and Söldner is:

Water	86.70
Proteids	3.07
Fat	3.34
Milk sugar	5.27
Ash	0.40

The colostrum corpuscles gradually disappear from the milk, and usually are not found after the twelfth or thirteenth day following delivery.

Advantages of Breast-feeding.—It is impossible to place too much emphasis upon the statement that breast milk is the best food for an infant. It is a well established fact that the milk of each mammal is especially adapted not only to supply the demands of nutrition for the growing body of its young, but also to develop the digestive apparatus. If, then, an infant is nursed by its mother, its digestive apparatus develops normally, and gradually builds itself up to the point where it can digest other foods than milk.

Unfortunately, among the wealthier classes, many mothers are unable to nurse their babies for the entire period that is usually considered advisable—ten or twelve months—and it is also not uncommon to find among the working class women who are so poor that they have

11

to leave their babies at home or in a Day Nursery, and go out to work; or, but this is less common—those who are unable to nurse their babies because of poor food, overwork, and bad hygienic surroundings.

However, the large majority of American women are between these two extremes, and many of them are able, and almost all of them can be persuaded to try, to nurse their babies. A very brief study of the chapter on Infant Mortality will show that almost all the deaths occur in bottle-fed babies. During the famous siege of Paris, the mortality among infants actually decreased because the women, in spite of all the privations and hardships which they were compelled to undergo, had to remain at home and nurse their babies.

It is a mistake to wean an infant simply because it is not gaining in weight a sufficient number of ounces each week, or because it vomits occasionally, has abdominal pain, or the stools are not normal in color or consistency. It is possibly true that the breast milk this child is receiving is not an ideal food, but the correct treatment is not to wean the baby but to go carefully over all the facts connected with its feeding; see if the mother is worried by household or other cares, regulate her food, sleep and exercise; give her, if she requires them, appropriate tonics.

Investigate the daily life of the infant with the same care—is it being fed regularly; are its bathing, clothing, sleep, and outdoor life all that is to be desired; can any medicine be given that might assist its digestion? Our efforts then would be directed not only to improving the quality and increasing the quantity of the milk by building up the mother, but also to place the child under those conditions which would tend to improve its digestion.

If it still failed to gain and the quantity of the milk was deficient, a small supply of properly prepared cow's milk should be given after each breast-feeding. This method of giving after each breast-feeding just enough properly prepared cow's milk to compensate for the deficiency of the breast milk, is a plan that is applicable to a very large number of breast-fed infants.

If the supply of breast milk, as is frequently the case, gradually becomes less, and it is found that at the proper hour for nursing the breast is practically empty, an entire bottle feeding may be given once or oftener during the day, and the number of partial breast feedings be reduced. It is, however, well recognized that the nursing of the infant tends to keep up the secretion of milk, and one is therefore reluctant to omit entirely any more breast feedings than are necessary.

Many infants are weaned because a chemical analysis of the mother's milk shows that it does not conform to the accepted standard of breast milk. While there is a standard for breast milk, it is also true that this standard may vary normally between very wide limits, that different specimens taken from the same mother may also vary, and that the milk nursed by an infant may vary from the milk drawn from the same breast with a pump.

It is also true that the analysis varies according to whether it is the

fore, middle or end of a nursing. Moreover, it is expensive to have an analysis of breast milk made, and repeated analyses of the same milk may be necessary before its average composition is determined. While I am a firm believer in the necessity and importance of breast milk analysis, I still wish to enter a protest against weaning a baby simply because the analysis shows a milk that does not conform to the accepted standard.

It is surely unwise to wean in such cases until every effort has been made to improve the quality and increase the quantity of the milk; we must remember that a poor breast milk is often better than a prepared bottle, and that breast milk contains substances that are of great importance to the infant, and of great protective value.

There are rare cases when the breast milk should not be given, because, in spite of all that one can do, the baby does not gain in weight and does have constant indigestion; but in my experience, they are very rare indeed.

It may well be asked "When and how should a baby be weaned?" Having decided to wean the baby, it is always safer to begin giving the cow's milk mixture much weaker than one would ordinarily employ in a healthy baby of the same age who had been fed continuously on the bottle. If the cow's milk mixture is started at about one-half the strength usually given to an infant of the age of the one being weaned, it may be gradually and cautiously increased if it is evident that the infant can digest it.

If an infant is doing well on the breast, it is not wise to wean it during the hot summer months as the change from breast to bottle is quite liable to be associated with gastro-intestinal disturbances if the child is in a locality where the weather is very warm. Infants who are taken to a climate that is cool in summer would not come under this rule.

It is safer to wean slowly than suddenly. At first one bottle-feeding each day may be given; if this agrees with the child, in two or three days a second bottle-feeding may be given. By this plan the breast-feeding may be gradually withdrawn and the bottle completely substituted.

It is advisable to wean the baby if the mother has any disease which she may transmit to her child, such as tuberculosis or typhoid fever; or if the mother has any disease which the strain of nursing might aggravate, as acute pneumonia, tuberculosis or nephritis. Pregnancy in the nursing mother requires the weaning of the infant, and weaning may be done gradually if desired. If, however, the prepared milk given the baby does not cause any intestinal disturbance, no longer than one week should be consumed in the complete weaning.

If the illness of the mother is of a minor character, and of such a nature that it will not be transmitted to the child, nursing at the breast may be continued for two or three feedings a day and a weak milk mixture given for the other feedings; but if the illness, while of a mild and transient character, is associated with severe symptoms, it is

often safer to discontinue nursing entirely for a few days, give the child a weak, modified milk mixture, and pump the breasts several times a day to retain the milk. Menstruation may cause temporary gastro-intestinal disturbance in the infant and perhaps make weaning necessary for several days.

A wet-nurse who is syphilitic should not be allowed to nurse an infant, and an infant who has syphilis should not be allowed to nurse from a wet-nurse. In the first instance, the wet-nurse may infect the child, and in the second case, the child may infect the wet-nurse. If, however, an infant is suffering with inherited syphilis, it may, of course, be nursed by its own mother.

Temporary weaning from one breast is necessary if the mother is suffering from a mastitis of that breast, and, if the inflammation has been severe, nursing must be resumed cautiously.

Weaning depends, in the large majority of cases, upon the ability of the mother to nurse her baby. It is a mistake to keep an infant entirely on the breast, if the amount of milk secreted is insufficient for the nutritional demands of the infant. Many women of the higher classes are unable to nurse their infants entirely for more than a few months, and partial weaning, when the milk begins to fail, is necessary.

Much can often be done to improve the quality and quantity of breast milk by building up the general health of the mother, giving her an abundance of milk, meat and eggs, with plenty of sleep and avoidance of all worry.

In other cases, when the baby is doing fairly well, rather than make a change, nursing is prolonged until long after the infant is a year old. Almost all infants so fed become anemic, fail to develop normally, and rarely show a continuous and normal gain in weight. If a child is doing well on the breast, it is wise to begin giving it one bottle feeding a day when it is nine months old. The one bottle of prepared cow's milk is of advantage, in that it gradually trains the digestive organs of the infant to digest cow's milk; it also makes the weaning less abrupt.

The amount of food being received by the infant at the breast is often a matter of importance as regards weaning, nutrition, and gastro-intestinal disorders. Assuming that an ounce of breast milk weighs one ounce, the infant may be weighed before and after feeding, and the difference in weight represents the number of ounces nursed. From a number of such records in my private practice, where the infants were in good health and gaining well, the following table is compiled:

7 days old	. . . .	10 to 16 ounces
2 weeks old	. . . .	14 to 20 "
2 to 4 weeks old	. . . .	18 to 24 "
4 to 8 weeks old	. . . .	22 to 29 "
2 to 3 months old	. . . .	25 to 32 "
3 to 4 months old	. . . .	26 to 35 "
4 to 6 months old	. . . .	27 to 38 "
6 to 9 months old	. . . .	30 to 41 "

These amounts represent, of course, only averages; the larger the child, the greater the gastric capacity and the more food it will require. The average composition of breast milk according to Rotch is:

	Woman's milk directly from the breast.	Cow's milk freshly milked.
Reaction	Amphoteric (more alkaline than acid)	Amphoteric (more acid than alkaline).
Water	87 to 88 per cent.	86 to 87 per cent.
Mineral matter	0.2 per cent.	0.7 per cent.
Total solids	12 to 13 per cent.	13 to 14 per cent.
Fats.	4 per cent. (relatively poor in volatile glycerides)	4 per cent. (relatively rich in volatile glycerides).
Milk sugar	7 per cent.	4.75 per cent.
Proteins	1.5 per cent.	3.5 per cent.
Caseinogen	$\frac{1}{3}$ to $\frac{1}{2}$ of the total proteins	2.66 per cent.
Whey proteins	$\frac{1}{2}$ to $\frac{2}{3}$ of the total proteins	0.84 per cent.
Coagulable proteins	Small proportionately	Large proportionately.
Coagulation of proteins by acids and salts	With greater difficulty; curds small and flocculent	With less difficulty; curds large and tenacious.
Coagulation of proteins by rennet	Does not coagulate readily	Coagulates readily.
Action of gastric juice	Proteins precipitated but easily dissolved in excess of the gastric juice	Proteins precipitated but dissolved less readily.

The following table from Rotch shows the composition of normal, poor, over-rich and bad milk:

	Normal milk (healthy life as to exercise and food).	Poor milk (starvation).	Over-rich milk (rich feeding; lack of exercise).	Bad milk (pregnancy, disease, etc.).
Fat	4.00	1.10	5.10	0.80
Sugar	7.00	4.00	7.50	5.00
Proteins	1.50	2.50	3.50	4.50
Mineral matter	0.15	0.09	0.20	0.09
Total solids	12.65	7.69	16.30	10.39
Water	87.35	92.31	83.70	89.61
	100.00	100.00	100.00	100.00

Wet-nurse.—If a mother is unable to nurse her baby, and in those rather rare cases where she cannot be persuaded to nurse her infant, the employment of a wet nurse offers the best, simplest and safest method of feeding the baby.

There are, however, certain objectionable features connected with wet-nursing that it is well to appreciate at the outset. The class of women who are usually available is generally undesirable. The baby is often illegitimate, and the nurse worried by her misfortune and annoyed by the father of her child and her so-called friends. The bringing of such an individual into a household is not usually desirable. A woman of a quiet and phlegmatic temperament is generally to be preferred, as nervousness and excitement on her part will often cause the breast milk temporarily to disagree with the infant.

It is not necessary that the age of the nurse's child should correspond to that of the child she intends to nurse. It is desirable that her baby be as young as possible if it is proposed to have her continue nursing for any considerable number of months. If her own child is seven months old or more, the probability of her being able to nurse for more than three or four months is not great. She should be between twenty and thirty years of age, and a chemical analysis of her milk is of advantage as a fair index of its quality.

The only advantage in her being a multipara is that we have the history of a previous successful ability to nurse for a definite number of months. She should be free from syphilis and tuberculosis, have an even temper, and live as normal a life as possible as regards sleep, fresh air, and exercise.

Her diet should consist of an abundance, but not an excess, of plain food, with a liberal supply of cow's milk, about one quart a day. She should avoid drinking much tea or coffee and all alcoholic drinks should be forbidden. A wet-nurse who must have ale or beer to stimulate her secretion of milk had better be dispensed with.

The best test is to see and examine carefully her own baby. If it is strong, robust and well developed, one may be reasonably sure of her milk being of good quality and quantity. In Philadelphia, it is usually possible, by applying at several agencies, to secure almost immediately a wet-nurse with a negative Wassermann.

In private practice among the class of patients who are capable of paying for the services of a wet-nurse, it is almost always possible to have one's directions, as to bottle feeding and general care and management of the infant, carried out in compliance with the orders of the attending physician. This is the reason why wet-nursing has never been popular in the United States; still, there are a certain number of infants who, as a result of malnutrition or disease, will usually do better on wet-nursing than on the bottle.

Among this class may be mentioned especially premature or delicate infants. It is of great help in such cases to be able to secure for them a plentiful supply of good breast milk. Young infants, convalescing from an acute illness, especially of the gastro-intestinal tract, usually do well if given to a wet-nurse; or a young infant who has been badly fed, and has failed to gain in weight for some weeks, will usually gain rapidly if given a sufficient supply of breast milk. In Philadelphia I have seen the mortality in a certain foundling asylum very greatly reduced by boarding out the infants to women who would partly breast-feed and partly bottle-feed them. The feeding and care the infant receives is watched systematically by a competent social worker, and a member of a board of women managers.

Alexins.—There are present in human milk substances called alexins, which possess bactericidal and globulicidal properties. They are probably partly formed in the breasts and partly derived from the blood. They, undoubtedly, tend to partially protect the breast-fed

infant from infection of the gastro-intestinal tract. Breast milk also contains other protective substances—agglutinins and antitoxins—and still other antibodies, if the mother is immune, may be present in her milk.

PERCENTAGE FEEDING.

The most satisfactory and accurate way of determining the composition of the infant's food is to mix it according to percentages. It is not, however, a method of feeding, nor does it determine the ingredients or the amount of the various food elements which are suitable in the individual case. These factors depend upon the age, size, and health of the infant, and upon the digestive ability of its gastro-intestinal tract.

The problem is how to change or modify the percentages of cow's milk so as to make it resemble human milk. The first question which naturally suggests itself is, why one should feed a baby on different percentages at different ages when the percentages of breast milk do not change to any appreciable degree during the entire period of breast feeding.

The answer is that each mammal furnishes a milk that is especially adapted to the growth and development of the digestive organs and body of its young. The milk of the cow is therefore adapted to the growth and development of the digestive organs and body of the calf, and is *not adapted to the infant.*

The young infant can digest cow's milk only in weak mixtures, and as it grows older and stronger can digest larger quantities and stronger mixtures. Cow's milk can never be transformed into human milk; but we can modify it so as to make it possible for an infant to live and grow and develop upon it.

Percentage feeding offers a method of calculating in ounces and drams the composition of a given formula when that formula is written in percentages; or, if written in ounces and drams, one can easily calculate the percentage of fat, proteins, and sugar that it contains.

The accuracy of such a mixture, however, depends primarily upon the constant composition of the milk and cream used. In the laboratory there is very little variation in the composition of such a formula; but, in the home, modification of the milk is necessarily not so accurate. The errors which occur, however, are in the actual percentages, the proportions of the various food elements being little altered, consequently most infants are able to compensate for these errors, and the slight variations in the composition of the food are not noticeable. Fats, carbohydrates, and proteins are the elements that especially concern us in the modification of cow's milk, the salts being of less practical importance, hence the percentage formula states only what percentage of these three elements should be used.

Modification of Milk.—Modifying milk is the process of diluting it and adding to it in such manner that the fats, proteins, and carbo-

hydrates are combined in the proper proportions to make them assimilable and properly to nourish the child. Because of the difference in the composition of cow's milk and human milk, modification is always necessary when cow's milk is fed to the young infant; for, while the percentage of fat is nearly the same, the percentage of sugar is lower in cow's milk, and the percentage of proteins higher.

The old method of infant feeding, which was simple dilution while decreasing the amount of proteins to normal, caused such a decrease in the amount of fat and carbohydrates that it did not meet the indications of modified milk. In order to make the mixture prepared from cow's milk correspond to the general relation of fat, sugar, and proteins in human milk, it is necessary to use cream in the feeding mixture so that the dilution which reduces the amount of proteins will not cause too great a diminution in the amount of fats; for when cream forms there is an unequal division of fats in the upper and lower contents of a receptacle, but the proportion of sugar and proteins is practically equal. Thus, simply stated, the modification of milk consists in the dilution of cream with water and the addition of milk sugar.

The protein in cow's milk is 4 per cent.; in human milk 1½ per cent.; and to reduce the 4 per cent. in cow's milk to the required percentage in human milk, it is necessary to add a diluent, and the one commonly used is water. This addition of water, however, also reduces the percentage of fat; in fact, usually reduces it to a point lower than the percentage desired in the modified milk mixture. Now, to increase this percentage of fat it is necessary to add to our mixture something that contains a relatively high percentage of fat and a low percentage of protein, therefore we add cream. Cream, containing as it does a high percentage of fat, can easily be reduced by the addition of water so as to contain any desired percentage. But, as this addition of water also reduces the percentage of protein in the cream, it is often necessary to add to the mixture an ingredient that contains a low percentage of fat and a high percentage of protein. This is well supplied by fat-free milk, which contains 4 per cent. of protein and practically no fat. Cow's milk contains 4 per cent. of milk sugar, human milk 7 per cent., and our addition of water still further reduces the sugar percentage. To bring the sugar percentage in our modified milk up to the desired amount, we simply add milk sugar. The modification of milk is thus briefly as follows:

We add water to cow's milk to dilute its 4 per cent. of protein; we add cream which is rich in fat to bring up the fat to the percentage desired; and we add milk sugar to raise the percentage of sugar to that required by the infant, usually 6 or 7 per cent. Modified milk mixtures may be made with cream, whole milk, water, and milk sugar; or, instead of the whole milk, fat-free milk may be used. The latter method is, I believe, the better for home modification, therefore, will be the one followed. It is easy to understand, and the modifications are quickly made at home.

In order that the modification of milk at home may, as nearly as possible, approach the exactness of the laboratory, it is necessary that the family be supplied with milk and cream of definite percentages. This milk or cream may be procured in Philadelphia from those dairies recommended by the Milk Commission of the Philadelphia Pediatric Society. All bottles and nipples should be carefully sterilized. The arms and forearms of the person selected as modifier should be thoroughly cleansed and she should have the following for her modifications: milk and cream of known percentages, sterile water, lime-water, milk sugar, a cream dipper, milk sugar measures holding $3\frac{3}{8}$ drams, an 8 ounce graduate, a large spoon sterilized. Gravity cream is often used, and can be obtained as follows: if the milk is allowed to stand in the ordinary quart jar for eight hours, the top 4 ounces represent a 20 per cent. fat cream, the top 6 ounces a 16 per cent. fat cream, and the top 8 ounces, a 12 per cent. fat cream. Gravity cream contains more bacteria than does separator cream, but the centrifuge, it is claimed by some, does injury to the emulsion of the fat. Personally, I have obtained equally good results from both creams.

It must be remembered that accuracy is the keynote to a home modification, and it is best that all directions to the mother or nurse be carefully written out in ounces of cream, milk, water, lime-water, and measures of sugar of milk. The physician should learn to think in percentages, and, having decided upon the percentages to be used, be guided by the child's age, development, weight, digestion, and, if indigestion be present, also influenced by the fact whether it is the fat, protein, or sugar which is the cause of the indigestion. After taking these factors into consideration, he should decide upon the exact percentage of fat, sugar and protein to be used, and, by reference to his pocket memoranda, transfer this into ounces for the benefit of the mother or nurse.

A convenient and easily understood form of home modification is as follows: from a quart of milk, which has been bottled eight hours, remove the top 8 ounces; count this as 12 per cent. fat cream. Count as fat-free milk, the lowest 8 ounces of the quart. Using this 12 per cent. fat cream and the fat-free milk, the following percentages can be obtained, covering fairly well the different combinations of fat, protein and sugar desired. One quart of milk is enough by this method until the baby is about three months old.

FIRST WEEK.

Fat	2.00
Sugar	5.00
Proteins	0.75

12 per cent. cream. Fat-free milk.

Cream	$3\frac{1}{4}$ ounces
Milk	$1\frac{1}{2}$ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2 measures

SECOND WEEK.

Fat	2.00
Sugar	6.00
Protein	1.00
Cream	3¼ ounces
Milk	2½ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2½ measures

THIRD WEEK.

Fat	2.50
Sugar	6.00
Proteins	1.00
Cream	4¼ ounces
Milk	1¾ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2½ measures

FOUR TO SIX WEEKS.

Fat	3.00
Sugar	6.50
Proteins	1.50
Cream	5 ounces
Milk	3½ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2¼ measures

SIX TO TWELVE WEEKS.

Fat	3.50
Sugar	6.50
Proteins	1.50
Cream	5¾ ounces
Milk	3¼ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2¼ measures

THREE TO FOUR MONTHS.

Fat	4.00
Sugar	7.00
Proteins	1.50
Cream	6¾ ounces
Milk	2¼ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2½ measures

FOUR TO EIGHT MONTHS.

Fat	4.00
Sugar	7.00
Proteins	2.00
Cream	6¾ ounces
Milk	4¾ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2¼ measures

EIGHT TO NINE MONTHS.

Fat	4.00
Sugar	7.00
Proteins	2.50
Cream	6¾ ounces
Milk	7½ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	2 measures

NINE TO TEN MONTHS.

Fat	4.00
Sugar	7.00
Proteins	3.00
Cream	6¾ ounces
Milk	10½ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	1½ measures

TEN TO TWELVE MONTHS.

Fat	4.00
Sugar	5.00
Proteins	3.50
Cream	6¾ ounces
Milk	11¾ ounces
Lime-water	1 ounce
Water	q. s. 20 ounces
Milk sugar	½ measure

AFTER TWELVE MONTHS.

Unmodified cow's milk.

In order to obtain certain low protein percentages with certain fat percentages, it is necessary, instead of removing the top eight ounces and using a 12 per cent. fat cream, to remove the top six ounces for a 16 per cent. fat cream, or the top four ounces for a 20 per cent. fat cream. The following table designed by Dr. Maynard Ladd, makes this a calculation of a few moments only.

No.	20-ounce mixtures. Percentage of				Ounces of cream				Ounces fat-free milk used with creams of				Ounces lime-water.	Milk sugar, measures.
	Fat.	Sugar.	Proteins.	Alkalies.	10 per cent.	12 per cent.	16 per cent.	20 per cent.	10 per cent.	12 per cent.	16 per cent.	20 per cent.		
1	1.50	4.50	0.25	5	..	..	..	1½	..	..	..	..	1	2
2	1.50	4.50	0.50	5	3	2½	2	1½	..	½	1	1½	1	2
3	2.00	5.00	0.25	5	..	..	..	2	..	..	..	..	1	2¼
4	2.00	5.00	0.50	5	..	3¼	2½	2	..	..	½	1	1	2¼
5	2.00	5.00	0.75	5	4	3¼	2½	2	¾	1½	2¼	2¾	1	2
6	2.00	5.50	1.00	5	4	3¼	2½	2	1¾	2½	3¼	3¾	1	2¼
7	2.50	5.00	0.50	5	..	..	3¼	2½	..	..	..	¾	1	2¼
8	2.50	5.50	0.75	5	..	4¼	3¼	2½	..	¼	1¼	2	1	2¼
9	2.50	6.00	1.00	5	..	4¼	3¼	2½	1	1¾	2¾	2½	1	2½
10	3.00	6.00	0.50	5	..	..	3¾	3	..	..	..	¾	1	2½
11	3.00	6.00	0.75	5	..	5	3¾	3	..	..	1¼	2	1	2½
12	3.00	6.00	1.00	5	6	5	3¾	3	..	1	2¼	3	1	2¼
13	3.00	6.00	1.25	5	6	5	3¾	3	1¼	2¼	3¼	4¼	1	2¼
14	3.00	6.50	1.50	5	6	5	3¾	3	2½	3½	4¾	5½	1	2¼
15	3.00	6.50	2.00	5	6	5	3¾	3	5½	6½	7¾	8½	1	2
16	3.50	6.00	0.50	5	..	..	..	3½	..	..	..	..	1	2½
17	3.50	6.00	0.75	5	..	..	4½	3½	..	..	..	1	1	2½
18	3.50	6.50	1.00	5	..	5¾	4½	3½	..	..	1¼	2¼	1	2½
19	3.50	6.50	1.25	5	7	5¾	4½	3½	½	1¾	3	4	1	2½
20	3.50	6.50	1.50	5	7	5¾	4½	3½	2	3¼	4½	5½	1	2¼
21	4.00	6.00	0.60	5	..	..	..	4	..	..	..	..	1	2½
22	4.00	6.00	0.75	5	..	..	5	4	..	..	..	1	1	2½
23	4.00	7.00	1.00	5	..	..	5	4	..	..	1	2	1	2¾
24	4.00	7.00	1.25	5	..	6¾	5	4	..	¾	2½	3½	1	2½
25	4.00	7.00	1.50	5	8	6¾	5	4	1	2½	4	5	1	2½
26	4.00	7.00	2.00	5	8	6¾	5	4	3½	4¾	6½	7½	1	2¼
27	4.00	7.00	2.50	5	8	6¾	5	4	6¼	7½	9¼	10¼	1	2
28	4.00	7.00	3.00	5	8	6¾	5	4	9¼	10½	12¼	13¼	1	1½
29	4.00	6.00	3.00	5	8	6¾	5	4	9¼	10½	12¼	13¼	1	1
30	4.00	5.50	3.00	5	8	6¾	5	4	9¼	10½	12¼	13¼	1	¾

This, however, will only result in making the percentages of the different food elements the same as in human milk; for, no matter how cow's milk is modified, it will still differ from human milk because the composition of the fat is different, the ferments are not the same, and the specific serum reaction remains unchanged.

The method devised by Rotch is, perhaps, the most popular one for infant feeding. It is based upon the principle that we cannot feed all infants on the same mixture, and that each infant should be considered as a distinct problem in constructing a formula. The mixtures are made from cream, skimmed milk, milk sugar, and lime-water, and the diluent generally used is water. The constituents of the milk can be rearranged by this method so that any desired formula can be produced, and such mixtures are usually prescribed in percentage form; that is, fats, 2.5 per cent.; carbohydrates, 5 per cent.; proteins, 0.75 per cent.

If a given formula does not agree with an infant, it is easy to reduce or increase any of the food elements by decreasing or increasing the percentages in the mixture. The best method is for the physician to think in percentages. After deciding just what percentage of fats, protein, and sugar the infant or child may require, he should write a prescription for these percentages, the number of feedings necessary in twenty-four hours, the amount in ounces and drams of each feeding, the degree of alkalinity, and the degree of heating which the feedings may require. This is sent to a laboratory, and each day the bottles of milk are prepared according to his prescription, and are left packed in ice at the home of the patient.

If a milk laboratory is not located near the residence of the child, or if the expense of laboratory feeding is too great, the milk may be modified at home. This home modification is not difficult. The physician must write out his directions carefully and in detail, and any one of ordinary intelligence is capable of carrying them out. In fact, home modification has practically largely displaced laboratory feeding. A family that can afford to feed a child by the laboratory method usually has a child's nurse who is quite competent to do home modification.

Calculation of Percentages.—Given a milk or cream containing a standard amount of the various food elements, the calculation of a percentage formula is easily made by employing the following method:

$$\frac{\text{Percentage desired} \times \text{quantity desired}}{\text{Divided by the standard per cent.}}$$

By means of this formula the amount of fat and proteins is determined; for example, 20 ounces of a 3 per cent. fat and 2 per cent. protein mixture is desired, for which a 12 per cent. fat cream is used. In order to determine the proper amount of this 12 per cent. cream to use, the desired percentage (3) is multiplied by the desired number of ounces (20) and divided by the standard percentage (12) which gives us 5 ounces of cream.

$$\frac{\text{Standard per cent. 3} \times \text{desired quantity 20}}{\text{Standard per cent. 12}} = 5$$

In order to determine the proper amount of milk or cream which this 20-ounce mixture must contain to give it a 2 per cent. protein content, the desired percentage (2) must be multiplied by the number of ounces desired (20), and this divided by the standard percentage ($3\frac{1}{2}$), which gives approximately $11\frac{1}{2}$ ounces as the result.

Inasmuch as the cream added to give the desired percentage of fat to the mixture does not contain enough proteins to furnish the desired percentage of proteins, the difference is made up by adding skimmed milk which raises the protein content without influencing the fat content. This particular formula requires only 5 ounces of cream to furnish the necessary percentage of fats, whereas $11\frac{1}{2}$ ounces of milk or cream are necessary to provide the proper amount of proteins; therefore, the difference between 5 ounces and $11\frac{1}{2}$ ounces, or $6\frac{1}{2}$ ounces, of fat-free milk must be added.

The amount of sugar in percentage formulas is determined by estimating the amount of sugar needed in the mixture, subtracting the amount of sugar in the milk and cream utilized to supply the desired amount of fats and proteins for that particular mixture, and supplying the difference by adding milk sugar. Thus, if 6 per cent. carbohydrates is desired in the above formula, 6 per cent. of 20 ounces, or 1.2 ounces, will be required.

Five ounces of cream are used to furnish the fats and proteins, and this contains 4 per cent. carbohydrates or 0.2 ounce; therefore 0.2 ounce, or the amount of carbohydrates already contained in the formula, is subtracted from 1.2 ounces, the total amount of carbohydrates required, leaving 1 ounce of carbohydrates to be supplied.

Five per cent. of lime water is usually added to these mixtures, and then enough of boiled water to make 20 ounces. This formula would read as follows:

℞—12 per cent. fat cream	5 ounces
Fat-free milk	none
Sugar of milk	1 ounce
Lime-water	1 "
Boiled water	q. s. ad. 20 ounces

Amount of Cream in Milk.—Cream is technically any milk which contains over 4 per cent. of fat. When milk is allowed to stand, the cream which forms at the top of the receptacle varies in its fat content, that at the top being much richer, and the cream below it thinner at different depths, until skimmed milk or fat-free milk is reached. The following diagram shows approximately the percentages of cream to be found at different levels in a quart bottle of a 4 per cent. fat milk which has been allowed to stand for six to twelve hours (Fig. 25).

Albumin Milk: Eiweissmilch: Protein Milk.—Albumin milk is a preparation containing a high percentage of protein (3 per cent.), very little sugar (1.5 per cent.), and 2.5 per cent. of fat. It has been developed on the theory that sugar is the main cause of intestinal fermentation, and that fermentation of the sugar depends on the

concentration of the whey and the relative proportions of casein and sugar in the mixtures. A diminution of the salts is effected by dilution of the whey.

Albumin milk is claimed by Finkelstein and Meyer to be beneficial in all disturbances of nutrition which are accompanied by diarrhea, and may also be given to the well infant from birth. Albumin milk is prepared by heating one quart of whole milk to 100° F., and adding 4 teaspoonfuls of essence of pepsin, which is thoroughly stirred in, and the mixture is then allowed to stand at 100° F. until a curd has formed. The whey is then separated from the curd by straining through a linen cloth, and is discarded, and the curd is pressed through a fine sieve two or three times by means of a wooden mallet or spoon, one pint of water being added during this process.
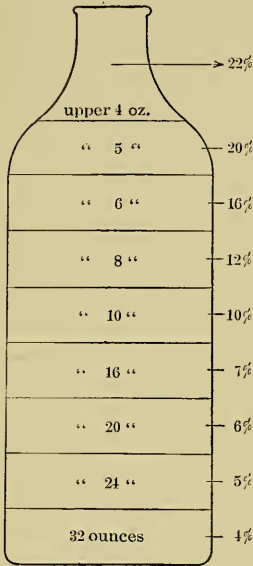
The precipitate should now be very finely divided, and the mixture should look like milk. One pint of buttermilk is now added. Buttermilk contains very little sugar of milk, and its lactic acid content is of benefit. One quart of albumin milk contains about 380 calories, so that it is necessary to increase the caloric value of this food by giving some additional nourishment as soon as improvement is noted.

Fig. 24.

It can readily be seen that the preparation of albumin milk may prove quite a difficult task in the home, and this to a great extent limits its general application. But the principles of albumin milk feeding may be carried out in the home by the addition of powdered casein and paracasein to ordinary milk mixtures.

Buttermilk.—Buttermilk has long been used in the feeding of infants, and good results are usually obtained, because it contains a low percentage of sugar, 3 to 4 per cent., a low fat content, $\frac{1}{2}$ to 1 per cent., and a high protein content, 2 to 3 per cent.; this is the old-fashioned buttermilk which was made from sweet milk as a by-product in the manufacture of butter.

It can be made at home by thoroughly mixing a quart of fresh milk, a pint of water, a pinch of salt, and one lactic acid bacilli tablet, and allowing this mixture to stand in a covered receptacle at room temperature for from eighteen to twenty-four hours, when it may be placed on ice until used. But since it can be procured from any milk dealer, it is needless to make it at home except in localities where it is unobtainable.

Buttermilk contains from 0.5 to 0.7 per cent. of lactic acid, and the lactic acid bacilli are alive and active in it, unless they have been

destroyed by heating. It contains more whey protein than fresh milk, and the casein it contains is very finely divided, so that it is most advantageous in those cases which require a low fat and high protein content in easily assimilable form.

Buttermilk is very rarely given plain, however, but is usually mixed with wheat flour and cane sugar. To each quart of buttermilk two tablespoonfuls of cane sugar and a tablespoonful of wheat flour are added, and this mixture is boiled for two or three minutes while constantly stirred, after which the desired feedings are bottled and placed on ice until needed for use.

The action of the lactic acid bacilli is, of course, lost in this mixture, since these organisms are killed by the boiling.

Commercial buttermilk is now made on a large scale, and is a stabile product. Much time and thought have been devoted to its production, and, as made by the Abbott Dairy, of Philadelphia, it is a product which has a constant food value, and can be used with confidence in those cases where buttermilk is indicated. Their process is as follows:

From the best whole milk, 2 per cent. of the fat is removed, leaving a milk that contains fat 2 per cent., sugar 4 per cent., protein 3.5 to 4 per cent. This milk is heated to 180° F., and kept at this temperature for thirty minutes. The milk is then rapidly cooled, and when it has been reduced to 76° F. a starter is added, and during the following twelve hours the milk is kept, as nearly as possible, at 75° to 76° F. It is then churned for thirty minutes, being cooled during the process of churning. The milk is now bottled, the temperature of the milk when bottled being reduced to 34° F. It is then placed in the refrigerator and sold to the consumer the next day.

The starter is made by adding lactic acid and Bulgarian bacilli to sterilized skimmed milk. The total solids of this buttermilk are 10 per cent., containing 2 per cent. fat, 4.25 per cent. sugar, 3 per cent. protein, and 0.75 per cent. of lactic acid.

Whey.—Whey is an opalescent liquid which remains after the coagulation of casein. Its composition is as follows:

Protein	0.8 per cent.
Fat	0.2 "
Sugar	4.7 "
Lactic acid	0.3 "
Salts	0.6 "

The solid portion of the whey of cow's milk is composed of lactalbumin, lactprotein, and extractives. Whey is made by adding two teaspoonfuls of essence of pepsin or liquid rennet to a pint of lukewarm skimmed milk, and stirring it just enough to mix it thoroughly. It is then allowed to stand until the milk separates into a solid and liquid portion, when the curd is broken up, and the whole preparation is strained through several thicknesses of cheesecloth. The whey passes through the cloth, but it contains the rennin of the rennet so that, before it can be mixed with skimmed milk, whole milk, or cream, the rennin must be destroyed by heating the whey to 150° F., and then allowing it to cool,

Whey is given to infants either combined with a cream mixture or alone. When combined with certain percentages of cream, we have a mixture in which the proteins are largely lactalbumin and lactoglobulin, thus resembling the protein content of human milk. Whey, alone, is an excellent preparation for infants suffering with gastro-intestinal disturbances when it is desirable to suspend milk temporarily; it is used quite often in infant feeding.

Proprietary Foods.—Patent, or proprietary, infant foods may be divided into milk modifiers and milk foods, those in the first group being used in combination with milk, those of the second being intended as a perfect food which does not require the addition of anything but water. These foods may be subdivided according to their ingredients as follows:

1. Dried milk, to which completely malted cereals have been added. Horlick's Malted Milk and Allenbury's Food are examples of this group.

2. Dried milk in combination with partially malted cereals, as combined in Nestlé's Food and Carnrick's Food.

3. Completely malted cereals which contain the carbohydrates in soluble form and which are practically a desiccated malted extract. They contain a very small amount of protein. Mellin's Food is a good example of this class.

4. Partially malted cereals, such as Benger's Food, which contains a pancreatic ferment that causes the conversion of starch; and Allenbury's Food, No. 3, Moore's Food, and the Savory Foods. Certain preparations in this class, such as Loeflund's Malt Soup, are not only digestible, but when combined with cow's milk may prove quite nourishing for a limited time.

5. This group includes the cereal foods in which there has been little or no conversion of starch, and includes Robinson's Patent Barley, Ridge's Food, Neave's Food, and Imperial Granum.

The following table, furnished by Morse and Talbot, shows the composition of the most widely known infant foods on the market:

	Fat.	Sugar.	Protein.	Starch.	Ash.
Condensed milk:					
Eagle Brand	9.61	54.94	8.01	..	1.78
St. Charles Brand	8.70	10.95	8.80	..	1.40
Ramogen	16.50	34.65	7.00	..	1.50
Mammala	12.12	55.34	24.35	..	4.93
Horlick's Malted Milk	8.78	67.95	16.35	..	3.86
Mellin's Food	0.16	79.57	10.35	..	4.30
Mead's Dextro-Maltose	..	93.00	..	..	2.00
Laibose	17.00	55.00	18.00	..	4.00
Allenbury's Food No. 1	18.60	66.55	10.66	..	3.95
Allenbury's Food No. 2	15.88	70.90	9.90	..	3.71
Allenbury's Food No. 3	1.05	25.11	10.23	60.01	0.60
Eskay's Albuminized Food	3.52	55.82	6.70	29.90	0.99
Nestlé's Food	5.50	58.93	14.34	15.39	2.03
Ridge's Food	0.26	7.80	12.50	73.67	0.61
Benger's Food	0.92	3.34	12.12	77.02	0.97
Imperial Granum	1.04	1.80	14.00	73.54	0.39
Wheat Flour	1.00	..	11.40	76.10	0.50

It is readily apparent from the preceding analysis of these foods that no individual preparation contains the proper amount or proportion of food elements to sustain the life of an infant and provide for its growth for any great length of time. Most of them show a deficiency in fats and an excess of carbohydrates which prohibit their continued use, inasmuch as the proportion of fat in an infant's food is a most important consideration.

The proteins in these preparations are either heated and dried proteins of cow's milk, which are most indigestible, or vegetable proteins which are unsuitable for the infant. It is not uncommon to hear of infants who have apparently thriven on one particular food; but these cases do not prove that patent foods are better than modified milk; on the contrary, they should be regarded as occasional instances in which the particular food given has fortunately contained the various food elements in proper proportion and combination for the individual case.

The milk modifiers, such as Eskay's Food, are, perhaps, least objectionable, since they provide starch in a form easy of digestion, and the food is added to fresh cow's milk.

Patent foods are used principally by the careful practitioner at the weaning period; when there is milk idiosyncrasy; or as a substitute for milk during an acute illness; but their routine use in infant feeding is to be condemned, not only on account of the faulty composition of these preparations, but because of the careless prescribing it leads to in treating infants. Another great objection is the cost of the food, which far exceeds that of modified milk.

Meat Preparations.—Meat preparations have no place in the feeding of a normal infant, but they are important additions to the diet of the sick child when cow's milk must be temporarily withdrawn. Raw meat juice is the most popular preparation, and is made by adding an equal quantity of cold water to finely ground raw beef, and allowing it to stand for half an hour, after which the juice is squeezed out through muslin.

Meat juice, thus made, contains approximately 5 per cent. proteins, 3 per cent. extractive matter, and 0.7 per cent. salts, and may be given alone or combined with whey, cream, barley-water, or even with Mellin's or some other prepared food. Meat juice is especially valuable in the treatment of scurvy, since it possesses distinct antiscorbutic properties.

Proprietary meat preparations occasionally give good results, but are not nearly as suitable for the infant as the fresh juice, and have no antiscorbutic effect.

Meat broths, made from mutton, beef, and veal, also chicken broth, are all useful when milk feedings are temporarily contraindicated on account of gastro-intestinal disturbances. Several prepared beef juices are on the market, the most popular of which are Valentine's, Brand's, Armour's, Wyeth's and Burgoynes's, also the preparations known as Puro and Bovinine.

12

Malt Soup.—Malt soup is a proprietary food containing wheat flour, malt, and milk, upon which an infant can be fed for several months if the child's stomach tolerates it. It is indicated particularly in rachitis and in dyspeptic infants who have suffered greatly from malnutrition.

To make malt soup, 4 ounces of wheat flour should be mixed thoroughly with a quart of milk, and strained through gauze. To this is added a quart of warm water containing 6 ounces of thick malt and 30 grains of potassium carbonate in solution. For young infants one-half of this quantity can be made up, and more water and less milk may be used. As the infant grows older, and the gastro-intestinal tract becomes stronger, more milk should be added to the soup, but the amount of wheat and flour need not be changed.

ARTIFICIAL FEEDING.

Artificial infant feeding is the substitution of any other form of nourishment for breast milk, which is the normal food of infants. There are but few food elements, however, and the baby's food must contain some of them, perhaps all of them, but should not contain any other elements. These food elements are fats, carbohydrates, proteins, and salts, and they must be given in correct quantity and proportion. The infant must also be given sufficient food to make it thrive and gain weight, and this amount can be determined by calculating the caloric value of the food given.

Since the milk from the mother's breast for a period of twelve to fourteen months shows no variation in composition, it would seem as though the artificial food substituted for breast milk should be made to resemble human milk in composition, and that it would need but little change; but this is not the case, for no food has been discovered which is even approximately a substitute for breast milk.

A modification of cow's milk that will make its composition resemble human milk is the most satisfactory substitute yet discovered, although it is neither as nutritious nor digestible as human milk, is not sterile, and contains none of the biological constituents. Mare's milk resembles human milk more closely than does cow's milk; but it is rarely used because of the difficulty in obtaining it. Goat's milk bears a slightly closer resemblance to human milk than does cow's milk, but this, too, is rather difficult to obtain, and must also be modified, therefore its use in infant feeding is not popular.

Modified cow's milk is consequently the best practicable substitute for human milk. Proprietary foods and condensed milk are rarely used by the physician for any considerable time, while other preparations, such as dextrinized gruels, malt soup, albumin-water, meat preparations, albumin milk, barley-water, and rice-water are used principally as substitutes when milk is temporarily suspended in the course of diseases of the gastro-intestinal tract.

Variety of Fats, Carbohydrates, and Proteins.—**Fats.**—The character of the fats in a feeding mixture can not be changed, but the emulsion

can be made more complete by homogenization, which reduces the fat droplets to a very small size. Olive oil, which is another form of fat, may be introduced into the infant's food by homogenization.

Carbohydrates.—*Milk Sugar.*—Lactose is the most suitable form of sugar to insure the growth of the infant, because it is more completely absorbed than other disaccharides, and it favors the growth of the normal intestinal flora, thus aiding digestion. Moreover, few of the other organisms in the intestinal tract thrive upon it, so that to a certain degree it protects the tract from the development of undesirable bacteria.

Maltose is too expensive to be used alone in infant feeding, and is usually combined with dextrines. On ingestion it is immediately split up into dextrose, and has, therefore, more of a laxative effect than the dextrines, and is less favorable to the growth of normal intestinal flora. Dextrin-maltose is valuable in cases where intestinal disturbances are due to fermentation of milk sugar.

Cane Sugar.—This is the least desirable of the various sugars because it undergoes alcoholic fermentation instead of lactic acid fermentation, and does not promote the growth of normal intestinal flora. There are no indications for using cane sugar in infant feeding.

Starch.—Starch should not be given to infants under four months of age, and it is inadvisable to allow any large quantity of starchy food until after the first year. On the other hand, some starch should be included in the food after the sixth month, but must be given in definite amounts, the same as fats and proteins. Starch is especially indicated in cases of sugar fermentation and intolerance to sugar. It is usually given as a cereal cooked in water, such as oatmeal, or as rice-water, or barley-water, or in the form of gruel. An excessive amount of starch may cause constipation and marked disturbance of digestion and nutrition.

Proteins.—The protein of cow's milk is less easy of digestion than that of human milk on account of the greater amount of casein which it contains, resulting in the formation of large, tough curds. Whey protein is not coagulable by rennin, therefore is the best form of protein to give the infant, since it does not form curds.

CALORIC FEEDING.

This is a method of providing nourishment for the infant by giving it a food which contains the number of calories suitable for the individual case; but the caloric requirements of an infant are influenced by so many different factors that the caloric method of feeding is not practicable. It offers, however, a method of determining whether a child is overfed or underfed, and can be employed when an infant suffers from persistent gastro-intestinal disturbances, or whenever a baby fails to thrive.

The large calorie, which is the amount of heat necessary to raise one kilogram of water 1 c.c., is the one used in infant feeding. The

normal infant requires from 100 to 120 such calories per kilogram of its body weight during the first six months in order to thrive. From its sixth month until it is one year old it needs 100 calories, and 90 calories during the second year.

The number of calories required by an infant in order that it may gain weight depends also upon its state of health. Fat babies never need more than 90 calories per kilogram, and the fatter they are the less calories they need in order to gain; while thin babies may require from 140 to 160 calories per kilogram in order to gain, and the thinner the baby the more calories necessary.

Babies that have been underfed, or are recovering from some debilitating illness, also require temporarily more calories than healthy normal infants, and the active child needs more than the quiet one; so more food must be given the infant which cries most of the day and is restless at night than the baby that sleeps nearly all of both day and night.

The caloric value of modified milk mixtures may be quickly determined by means of the formula recommended by Fraley. The letters "F," "P," and "S" represent the respective percentages of fats, proteins, and sugar, and the letter "Q" the total quantity of food. This formula follows:

$$2F + P + S \times 1\tfrac{1}{4}Q = \text{Calories.}$$

The caloric value of a 20-ounce mixture containing 3 per cent. fat, 1 per cent. protein, and 6 per cent. sugar can be calculated as follows:

$$2 \times F (3) = 6 + P (1) = 7 + S (6) = 13 \times 1\tfrac{1}{4}Q (25) = 325 \text{ calories.}$$

HOME MODIFICATION OF MILK.

Milk can be prepared at home with such accuracy that most infants will thrive upon it, and percentage formulas are easily obtained. The excuse is sometimes made that modification of milk is too difficult a procedure to be attempted at home, but I have not found this to be the case.

The actual quantity of the various food elements to be used must be carefully calculated, however, instead of merely writing percentages on a prescription blank, and the mother must be told just how much cream, skimmed milk, water, lime-water, and sugar of milk to use. She must also be instructed how to secure a certain percentage of fat cream from the top of a quart bottle of milk. A 4 per cent. fat milk will, upon standing, yield 12 per cent. of fat in the upper 8 ounces of a quart bottle, 20 per cent. of fat in the upper 5 ounces, and 16 per cent. of fat in the upper 6 ounces; and with cream of these varying degrees of fat content most milk mixtures can be made. A cream dipper should be used to obtain the cream, and the fat-free, or skimmed, milk should be gotten from the very bottom of the bottle.

In measuring the milk sugar a rounded tablespoonful is considered to weigh half an ounce. Therefore, in prescribing a modified milk mixture to be made at home, the physician not only states the exact

percentage of the various food elements to be used, but estimates the quantity of each ingredient to be put in the mixture, and writes his directions accordingly. Such a prescription follows:

Cream (12 per cent. fat)	5 ounces
Fat-free milk	3 ounces
Sugar of milk	2 tablespoonfuls
Lime-water	1 ounce
Water (boiled) enough to make	20 ounces

Three ounces of this may be given every three hours. Any percentage of cream may be used, but, for practical purposes, as well as economy, it is generally advisable to make our home modification with

12 per cent. cream
Fat-free milk
Milk sugar
Lime-water
Water

With these ingredients almost any desired percentages may be obtained.

LABORATORY METHOD OF MODIFICATION OF MILK.

The physician who has the advantage of proximity to a milk laboratory in which milk formulas are prepared must first decide what percentage of fats, carbohydrates, and proteins will be suited to the infant's case, and how much he wishes to give the infant in twenty-four hours. The amount to be given at each feeding should be determined, and the caloric value of such a mixture also calculated, in order that the infant may be neither overfed nor underfed.

The amount of protein in a mixture for the newborn infant must be extremely small, and very little fats should be given. The carbohydrates are reduced to 5 per cent. for the first few weeks, after which 6, and later 7 per cent. are prescribed, and this latter percentage remains practically stationary throughout infancy, although the percentage of fats and proteins is increased at various intervals. In addition to determining the percentage of these various food elements, the physician must decide what kind of sugar is to be used, also whether or not part of the protein shall be in the form of whey protein.

He must also decide whether or not an alkali shall be added, and whether the milk shall be given raw, pasteurized, or boiled. Having decided all these points, it is only necessary to write a prescription on the blank furnished by most laboratories, stating what the composition of the formula is to be, and how often it is to be given.

There is no doubt that the milk laboratory prepares the milk mixture more accurately than does the mother, but the expense attached is too great for poor people. A table, giving approximately the composition and percentages of milk mixtures, with the number of feedings at different ages, is here given. But the physician must remember that no two infants can be fed alike, and that these figures are only approximately correct for the normal healthy infant. A form of prescription blank to be used in conjunction with laboratory modification is here appended.

R— Date....... For.......
Fats

Carbohydrates . . . { lactose.
maltose.
sucrose.
dextrose.
starch.

Dextrinize
Proteins . . . { whey.
casein.
Peptonize
Sodium citrate . . { per cent. of milk and cream.
per cent. of total mixture.
Sodium bicarbonate . { per cent. of milk and cream.
per cent. of total mixture.
Lime-water . . . { per cent. of milk and cream.
per cent. of total mixture.
Lactic acid bacilli
Heat at   ° F.
Number of feedings
Amount at each feeding   oz.

                                                                    M.D.

Fat.	Sugar.	Protein.	Amount at each feeding.		Number of feedings.	Night feedings	Day intervals	Total quantity, 24 hours.		Calorie value.	Caloric requirement.
			Ounces.	Grams.				Ounces.	Grams.		
First week to fourth week:											
1.0	5.0	.25	1½	45	10	2	2	15	450	138.6	330
1.0	6.0	.50	1½	45	10	2	2	15	450		
1.25	7.0	.75	2	60	8	2	2	16	480	to	to
1.5	7.0	1.0	2½	75	8	2	2½	20	600		
2.0	7.0	1.0	3	90	8	2	2½	24	720	370	370
Second month to fourth month:											
2.0	7.0	1.0	3½	105	7	1	3	24½	735	377	370
2.25	7.0	1.0	4	120	7	1	3	28	840		
2.5	7.0	1.25	4½	135	7	1	3	31½	945	to	to
3.0	7.0	1.25	5	150	7	1	3	35	1050		
3.0	7.0	1.5	5	150	7	1	3	35	1050	658	520
Fifth month:										606	520
3.5	7.0	1.5	5	150	6	0	3	30	900	to	to
Sixth month:											
3.5	7.0	1.75	6	180	6	0	3	36	1080	738	576
Seventh month:										799	576
3.5	7.0	2.0	6	180	6	0	3	36	1080	to	to
Eighth month:											
4.0	7.0	2.25	7½	225	6	0	3	45	1350	1017	640
Ninth month to twelfth month:											
4.5	7.0	2.5	8	240	5	0	4	40	1200	952	560
4.0	6.0	3.0	8½	255	5	0	4	42½	1275	to	to
4.0	4.5	3.5	9	270	5	0	4	45	1350	945	640

Feeding premature infants.

Thirtieth week:			
1.0	3.0	0.25	Heat thirty minutes at 155° F., 24 feedings of 2 drams each.
Thirty-second week:			
1.0	4.0	0.50	Heat thirty minutes at 155° F., 24 feedings of 2 drams each.
Thirty-fourth week:			
15.0	45.0	0.75	Heat thirty minutes at 155° F., 16 feedings of 5 drams each.

These formulas should all contain 5 per cent. of lime-water.

FEEDING AFTER THE WEANING PERIOD.

From the ninth to the twelfth month the food should consist of whole milk diluted with one-seventh its bulk of water. In addition cereals, such as Imperial Granum, oatmeal, and cream of wheat may be given once daily, and orange juice and beef juice three times a week.

From the twelfth to the eighteenth month the child is fed four or five times daily, and may be allowed soft-boiled eggs, plain crackers, bread and butter, milk custards; clear beef, mutton, or chicken soup; prune juice, baked apple; baked potato. When all of the teeth have appeared, and the food can be properly masticated, lamb chop, white meat of chicken, and underdone beafsteak may be given, also green vegetables, such as beans, spinach, asparagus, potatoes, and peas.

After the fifth or sixth year three or four meals are sufficient, and the child may take well-cooked vegetable soups, meat, fish, poultry, fresh vegetables, ripe fruits, puddings, and ice cream once or twice a week.

CARE OF BOTTLES AND NIPPLES.

After use, all bottles and nipples should be scrubbed with very hot water and a brush, and put into cool, sterile water containing a little sodium bicarbonate. The bottles should be boiled for ten minutes just before the day's milk supply is prepared.

The nipple should be of rubber, and thin enough to be easily turned inside out when cleansed. The opening should be only large enough to allow the milk to trickle out drop by drop when the bottle is inverted. Any complicated nipple or any tubing which extends into or from the nursing bottle is difficult to clean and positively harmful.

It is usually unnecessary to cleanse the mouth of a child either before or after feeding, provided the oral mucous membrane is normal, the mother's nipple healthy, and that, if bottle-fed, the rubber nipple is properly cared for. If, however, the mother's nipple is sore, eroded, or cracked, and a nipple shield is not used, the child's mouth should be gently cleansed with plain sterile water just before feeding.

CHAPTER IX.

NORMAL DIGESTION.

The newborn infant is not a perfectly developed individual, but is still in the stage of development. This is true as regards both its digestive organs and their functions. Yet these organs are called upon not only to sustain life by repairing tissue waste, as in the adult, but also to provide sufficient material for promoting growth and proper development.

When we consider that during the first year of life the body weight must approximately be trebled, we understand why the delicate, immature digestive apparatus of an infant is so easily upset, and the importance of proper nutrition during this period becomes evident. In order to avoid overtaxing this immature function, Nature has provided in mother's milk all the necessary food elements in proper proportion and in assimilable form. She also furnishes lipase, diastase, and, in all probability, a protein-digesting ferment, as well as certain substances which no doubt act as protectives against diseases of the intestinal tract.

The Oral Cavity.—The absence of teeth, the peculiar fat polster in the cheeks, which is seen even in marasmic babies, and other anatomical conditions plainly show that the oral cavity is at first intended for the reception of liquid food only, its mechanism being purely suctional. The child grasps the nipple between the tongue and the hard palate, and by a downward movement of the lower jaw, aided by the contraction of the muscles in the floor of the mouth and later on by deep inspirations, creates a partial vacuum, and thus the milk is made to flow from the galactiferous ducts into the nursling's mouth.

Sucking is, therefore, only possible when the nose is free for respiration and the palate and the floor of the mouth are perfectly intact. Hare-lip, cleft-palate, nasal obstructions, and adenoids greatly interfere with nursing and may even make it impossible. The mouth is merely the natural entrance to the digestive organs, and at birth does not aid digestion by salivary secretion; although the salivary glands are present and apparently well-developed, their secretory function is not yet fully established, consequently the mucous membrane of the mouth is rather dry, the small amount of salivary amylase or ptyalin contained in the saliva clearly indicating that the latter is not ready to be utilized for starch digestion.

After the third or fourth month—toward the beginning of dentition—the salivary secretion increases markedly in quantity and its characteristic quality is intensified. Even then it probably plays but an insignificant role in digestion, because very little of it is swallowed,

the greater part flowing out of the mouth unused (drooling). Toward the latter part of the first year the amylolytic function is fully established and is comparatively as active as in later life in the partial conversion of insoluble starch into soluble starch—erythrodextrin, achroödextrin, and maltose.

Dry food stimulates the secretion of saliva, which is usually alkaline or neutral, while food containing much water excites but little flow. Salivary amylase or ptyalin, the active enzyme of saliva, the digestive power of which is limited to starchy food, acts in a neutral medium, or in a slightly acid solution. It continues to act in the stomach until the gastric contents have been acidified, which occurs ordinarily in from one-half to one hour after a meal. It may be worth mentioning in this connection that the reaction of the oral cavity has been found to be slightly acid in 95 per cent. of infants whose mouths had not been thoroughly cleansed after feeding.

Dentition in the normal infant is of interest chiefly on account of the importance which parents are ever ready to ascribe to it as the cause of convulsions and gastro-intestinal disturbances. The eruption of the temporary teeth is a physiological process. The first tooth usually appears during the sixth month, but variations in time from the fifth to the eighth month are not abnormal. The remaining teeth appear in groups at more or less regular intervals in the following order: lower incisors, upper incisors, canines, and molars. At the age of two and a half years all of the teeth should have been cut. Teething is therefore an almost continuous process for about eighteen to twenty-four months. This fact alone shows the fallacy of attributing to its influence diarrhea and other diseases which are merely coincidental. Forcheimer says, "Dentition produces teeth, nothing more."

On the other hand, dentition may be painful, especially in the case of the narrow palatal arches so frequently seen in children suffering from adenoids or other nasal obstructions. Sometimes it takes a tooth days and weeks actually to penetrate the mucous membrane. A dry, hot mouth, a peculiar irritability, fretfulness, and other slight disturbances of health seem to occur so often during dentition that one cannot doubt a certain causal relationship between it and the other conditions; and it can be readily understood that infants with inherited neurotic and spasmophilic tendencies may show even more severe symptoms of disturbance of their unstable equilibrium.

The *sense of taste* is well developed at birth, and probably that of *smell* also.

There is little to be said about the *esophagus* except that its epithelium is soft and delicate, and that the glands are usually lacking.

Gastric Digestion.—Gastric digestion has been said to be insignificant in infants, the stomach serving merely as a reservoir for the milk, which is passed into the intestines at convenient intervals, and in such quantities as the duodenum can best take care of. While it is true that the organ does not play so important a role in the digestion

of infants as in that of adults, and that its mechanical and chemical processes are only preliminary to intestinal digestion, it has been convincingly demonstrated by Pawlow and his school that the functions of all the different parts of the digestive apparatus are interrelated and dependent upon each other to a great extent.

A description of a single physiologic function must, therefore, be more or less incomplete unless due consideration be given at the same time to all of the other supplementary or complementary processes involved. The process of digestion in infants, on the whole, is by no means as yet clearly understood, in spite of the most painstaking and elaborate investigations.

The majority of observations and conclusions are based either on lavage of the infant's stomach, limited necessarily to short periods, or on animal experimentations. The latter are of comparatively little use, because animals suffer little from digestive disorders, their digestive organs being much more fully developed at birth, and they can assimilate adult food at a much earlier period of their lives than do infants.

Moreover, it must be borne in mind that even apparently normal infants differ individually as to their digestive capabilities, at least as much as adults, and that in different infants the secretory functions are not equally developed in the formative stage of the first year of life.

GASTRIC DIGESTION (ANATOMICAL AND PHYSIOLOGICAL PECULIARITIES).

Considering the ease with which infants vomit, the position and form of the stomach are matters of interest. Its position is more vertical than oblique, the cardiac end is well fixed, and is slightly to the left of the tenth dorsal vertebra; the more freely movable pylorus is located at a point half way between the ensiform cartilage and the umbilicus, and in front or even slightly to the left of the spinal column. In form it stands midway between the tubular type of fetal life and the pouch-like adult organ.

At birth the stomach is rather small, its fundus and muscles being only slightly developed, but it grows rapidly, except when insufficient quantities of food are given for a prolonged period. The peculiarities of the adult shape and position appear in early childhood, and it is noteworthy that the pyloric opening is proportionately much wider during the first year than it is in later life.

The problem of gastric capacity has not been definitely solved by any system of measurement. This is probably no loss, from a clinical point of view, for, as a rule, an infant instinctively ceases to feed before its stomach is mathematically full. The capacity is said to be greater in the artificially fed baby than in the breast-fed infant (we think not necessarily so when properly managed), and to increase rapidly during the first three months, slowly in the fourth, practically not at

all during the next two months, and then again increasing until the adult size is attained, as is shown in the following table:

At birth	1 ounce
At four weeks	$2\frac{1}{2}$ ounces
At eight weeks	$3\frac{1}{4}$ "
At three months	4 "
At four months	5 "
At five months	$5\frac{1}{4}$ "
At six months	$5\frac{3}{4}$ "
At seven months	$6\frac{1}{4}$ "
At eight months	7 "
At nine months	$7\frac{1}{2}$ "
At ten months	$7\frac{3}{4}$ "
At eleven months	$8\frac{1}{4}$ "
At twelve months	9 "

For practical purposes it is sufficient to remember these figures:

At birth	1–2 ounces
At three months	4 "
At six months	6 "
At twelve months	9 "

The stomach is a muscular organ. Its motor function is chiefly exerted in mixing the food and expelling it into the duodenum in proper quantities and at convenient intervals. According to Cannon, the pyloric mechanism seems to be controlled by hydrochloric acid. A certain acidity of the stomach contents in the pyloric region, due to its admixture with free hydrochloric acid, causes the pylorus to open, while an acid reaction on the duodenal side causes it to close and to remain closed until the acid is neutralized. A high fat percentage retards the passage of food through the pylorus.

Pawlow has demonstrated that the quantity and the properties of the digestive secretions vary with the character of the food to be digested; that is, the secretions produced by different kinds of food vary in amount, in acidity, and in their digestive action. Quite apart from psychical stimulation, it seems that certain foods when taken into the stomach cause a secretion of the gastric juice owing to the secretagogues they contain. Meat extracts, soups, water and meat juices are especially active; milk, and egg albumen, less so.[1]

The gastric secretion begins about five to seven minutes after the ingestion of food, and increases rapidly, reaching its maximum of digestive power in about two hours, after which the flow rapidly decreases. The development of the glands in the mucous membrane of the stomach is rarely equally advanced in infants of the same age. Every baby is a law unto itself in this respect, as well as in respect to its digestive and other assimilative powers; in other words, there exist individual differences in the development of the gastric, the glandular, and digestive functions of the infant which to some extent explain the different findings of various investigators.

[1] The secretion produced by bread, though less in quantity than by meat, possesses a greater digestive action.

The acid reaction of the stomach contents in the normal infant is due to hydrochloric acid, the presence of which can be easily proved and its quantity determined. Lactic acid, probably produced by the fermentation of carbohydrates (milk sugar), has not been definitely shown to be a normal constituent, and indeed some authorities consider its presence in the stomach as pathological.

Of course, there is a possibility of other acids being formed in the stomach, for example, by the action of bacteria on food; but, according to our present knowledge, the hydrochloric acid alone is of real physiologic importance. The ingested milk excites the secretion of hydrochloric acid,[1] which then combines with the proteins of the food.

This occurs in the healthy breast-fed infant in one and a quarter to two hours after nursing, in the baby fed on diluted cow's milk it takes not less than two to two and a half hours, while with whole milk the process is still longer delayed. Free hydrochloric acid undoubtedly exerts an antiseptic, and, if sufficiently concentrated, even a bactericidal power.[2]

Although not of great importance this power is sufficient to add one more to the many advantages of breast-feeding. Inasmuch as mother's milk requires less acid for combination, one-half to one-third of that of cow's milk, the amount of free hydrochloric acid normally present is sufficient as a bactericidal and, as is sometimes claimed, also a detoxicating agent.

The extent to which *pepsin* combined with hydrochloric acid assists in protein digestion depends upon the consistency and the character of the food, and the length of time it is retained in the infant's stomach. Although some of the protein is, no doubt, acted upon and partially peptonized, the bulk of it in all probability passes almost unchanged in the chyme into the duodenum together with primary and secondary proteoses and even some peptone.[3]

As all gastric processes are merely preliminary to, and important for, subsequent digestion, so the true value of peptic action lies in its combination with trypsin. This enzyme is said to split proteins which have been submitted to the action of pepsin and hydrochloric acid in a way different from that which takes place with food not so prepared. The fact that a low temperature retards the action of pepsin shows the importance of giving food at a proper temperature, preferably from 100° to 105° F.

Wherever proteolytic enzymes may be found in the body there is evidence of a milk-curdling function; therefore the analogy has been drawn that curdling in the stomach is said to represent an action of pepsin itself, and is not due to a specific ferment. However, the consensus of opinion attributes the coagulation of milk in the infant's

[1] A weak infusion of tea and albumin water produces little secretion of hydrochloric acid.

[2] Free HCl 0.132 to 0.158 inhibits growth of typhoid bacilli; free HCl 0.185 kills growth of typhoid bacilli; free HCl 0.08 inhibits growth of cholera bacilli; free HCl 0.10 kills growth of cholera bacilli.

[3] The latter may, however, result from the action of bacteria in the stomach.

stomach to a ferment variously called rennet, lab-ferment, chymosin, rennins or pexin.[1] It is supposed to be present in the glands as pro-rennin, which under the influence of acids is converted into rennin. This acts upon the casein of milk, producing soluble paracasein, which in its turn reacts with the soluble calcium phosphates of milk. As a result the insoluble salt, calcium-paracasein, is precipitated in the form of curds, which differ considerably in the breast-fed and in the artificially-fed baby.

Undiluted cow's milk is said to curdle in rather compact masses containing much fat entangled in their meshes, and making it less digestible; in fact, large coagula are still to be found in the infant's stomach half an hour after feeding. Barley, oatmeal, rice, or arrow-root-water added to the food are said to cause curds with finer flocculi and thus to facilitate digestion.

Mother's milk, on the contrary, coagulates quite imperfectly and in fine flakes, enclosing only a little fat. Half an hour after nursing, the gastric contents of a breast-fed baby are seen to be a homogeneous mass which can be more easily acted upon by the digestive juices than the dense coagula of cow's milk. As regards the coagulation time, mother's milk is almost neutral, and coagulates only after its alkalinity has been sufficiently reduced; this requires about one-half to three-quarters of an hour, while cow's milk, being acid from the beginning, curdles much earlier.

Rennet coagulation does not invariably take place, nor is it always complete, but is influenced by the composition, the dilution, and the temperature of the milk, as well as by the presence in the stomach of clots from previous feedings. Curdling is hastened by warmth, and occurs most promptly at 106° to 108° F. with the addition of an inert foreign matter, such as starch, etc., and by the presence of acids, as well as of certain acid salts. It is retarded when the amount of soluble calcium salts is relatively decreased, as in diluted or heated milk, and when alkalies or alkaline salts, such as sodium chloride or lime water, are added to the milk.

No coagulation at all takes place with rennet when the soluble calcium salts are completely removed, and since boiling the milk decreases their amount considerably it must also materially affect the curdling process. The action of rennin is limited to coagulation. The digestion of the curd is carried on to a small extent by the combined action of pepsin and hydrochloric acid, and is practically completed by trypsin with the formation of proteoses, peptones, peptids and amino-acids as in other proteins.

The formation of curds seems to be the effort of Nature to throw part of the burden of protein digestion upon the stomach by delaying the passage of the food into the duodenum. This function of digestion should therefore be encouraged, as it probably influences the muscular development of the organ to a considerable degree.

---

[1] Casein is also precipitated from milk by an excess of acid as is seen in sour milk (lactic acid.)

The duration of gastric digestion varies with the age and the development of the child, as well as with the quantity and the quality of the food taken. At one month the stomach of a healthy breast-fed infant ought to be empty in from one to one and a half hours after nursing, and in about two hours up to the eight month; cow's milk requires one-half to one hour longer.

During the first months the gastric contents pass with comparative rapidity into the duodenum; but, as the stomach increases in size and larger meals are taken, the food remains longer within it. All fluids except alcohol, which is only partially absorbed, begin to leave the stomach very soon after a meal, and a considerable amount passes the pylorus during the first half hour. Proteins in various stages of digestion follow, while the fats are ejected last of all. The higher the proportion of fats, the longer is the food retained in the stomach and the smaller is the amount of gastric juice secreted.

Absorption in the Stomach.—Absorption does not take place readily in the stomach, for at least 80 per cent. of it occurs in the small intestine; it may, however, be somewhat increased by the ingestion of alcohol and certain condiments. Water, when ingested alone, is practically not taken up at all, but quickly passes on into the intestines.

Investigations concerning soluble salts are still incomplete, but it seems certain that they are not absorbed, at least not to any extent, until they reach a certain definite concentration. Peptones, peptids, and amino bodies are present only in minute amounts, but they can be and are absorbed, especially when in highly concentrated solutions. Fats are liquefied, partly emulsified, and perhaps to a small extent split up, but their intestinal digestion only is of importance. A small proportion of sugar is, no doubt, also absorbed by the gastric mucous membrane.

In summarizing, we may say that very little absorption takes place from the stomach directly: a certain proportion of sugar and of salts and a small amount of nitrogenous material are absorbed, but practically no water and no fat.

ANATOMICAL AND PHYSIOLOGICAL PECULIARITIES OF THE INTESTINES.

In infants the intestinal tract, which develops from a mere loop loosely attached to the posterior abdominal wall, and which is still in the formative stage after birth, is found to be less fixed than in adult life. Its elastic tissue is only slightly developed, the musculature is thinner and weaker, and there is relatively less difference between the diameters of the large and the small intestines. These peculiarities explain the ease with which the bowels become distended, and the pronounced tendency to constipation, colic, and, last and most important, to intussusception.

The intestinal villi are well developed; Peyer's patches are found

very early, while Brunner's glands are said to be less abundant than in adults. The small intestine is, on an average, about nine feet long at birth, and this length increases during the first two months by about four feet, making it relatively longer than in the adult. The length, however, may vary greatly, depending partly upon the quantity and the kind of food taken. This is illustrated by the fact that among the poorer classes in Russia, who of necessity live almost exclusively upon vegetables, the intestines are found to be considerably longer than among other people.

At birth the length of the large intestine averages about one foot, ten inches, which is about the height of the body. The sigmoid flexure is situated higher than in the adult, only one or two of its loops curving down into the pelvis; it is usually half the length of the large intestine. The colon, however, grows more rapidly, so that at the end of the fourth month it has about reached adult proportions, its relatively broad mesentery allowing full displacement. The cecum also occupies a higher and more median position than in the adult and is very mobile. The ascending colon is very short, and practically free, as a relatively large part of it is invested with peritoneum. The descending colon often has no mesentery; the length of the appendix is variable, and its position most uncertain.

The size of the pancreas at birth is still a matter of discussion. It shows no special anatomical features; its secretion is alkaline in reaction, and is stimulated as acids come into contact with the duodenal mucous membrane. It begins to flow very soon after food has entered the stomach, and reaches its maximum in from two to four hours. The amylolytic enzyme (pancreatic amylase) acts similarly to ptyalin (salivary amylase), but both its amount and its activity are said to be limited at first; its function seems fairly well established in the second half of the first year, and in an infant of six months it can safely be called upon for the digestion of moderate quantities of starch.

In the early months of life the power of pancreatic lipase (steapsin) seems to be only slight, but it increases gradually, and reaches its full development toward the end of the first year. This ferment does not act rapidly unless aided by bile, but it emulsifies, saponifies, and finally splits the fats into glycerin and fatty acids, thus fitting them for absorption by the intestinal epithelium. By the action of the same enzyme, they are probably recombined to form neutral fats which are used in intermediate metabolism. A milk-curdling ferment has been described.

Trypsin like pepsin causes hydrolytic cleavage of proteins, but its effects are more rapid and more powerful, and it requires an alkaline medium. The actual products formed depend to some extent upon the length of time and the special conditions under which trypsin acts; i. e., proteins are said to be more rapidly and more completely broken up when they have been previously acted upon by pepsin.

Trypsin attacks the protein molecules and apparently breaks them up into their end-products—amino-acids. Such peptones, proteoses or peptids as may have escaped its final action are acted upon by the erepsin of the succus entericus before reaching the blood.

Thus the proteins are almost completely disposed of under normal conditions and the nitrogenous substances which are found in the feces of a normal infant are principally mucus, bacterial residues of secretions, and epithelial cells.

In disease of the intestine the pancreatic juice seems to lose its proteolytic and some of its fat-digesting properties, while its diastatic function is said to be less disturbed.

The liver is proportionately large in infancy and childhood, being about one-eighteenth of the entire body weight as compared with the adult proportion of one-thirtieth; its lower border can normally be palpated one-half to one inch below the costal margin in the mammary line. This relatively large size and weight indicate that it performs a very important function and that it is well adapted to the great metabolic activity of this period of life.

It may not be out of place to revert to the fact that all material coming from the intestines by way of the blood must pass through the great hepatic filter. While it has been definitely proven that the glycogenic function, the formation of urea from ammonia, and the secretion of bile exist in the newborn babe, it seems that the hepatic efficiency for destroying poisons is not thoroughly established in early infancy.

The secretory function of the liver is developed as early as the third month of intra-uterine life, when both bile salts and pigments find their way into the intestine. At birth they are evidently in the meconium. The bile, relatively abundant in infants, presents no essential difference in its action from that of the adult. Some writers believe that its composition is deficient in organic salts, and also that it contains a smaller percentage of cholestrol, lecithin, glycocholic and taurocholic acids.

Bile salts are believed to have the property of aiding in the emulsification and the cleavage of fats. The small proportion of bile salts present during infancy accounts for the feeble antiseptic power of the bile, and for the consequent fermentation of the intestinal contents, as well as for the incomplete absorption of very fatty foods. Perhaps the resulting fatty acids irritate the intestinal mucous membrane and thus disturb digestion.

Succus Entericus.—The secretions from the follicles of Lieberkühn and from Brunner's glands are alkaline, and are stimulated by the ingested food, which is then subjected to their chemical action. The following enzymes have been isolated from the mucous membrane of the walls of the small intestine:

1. Enterokinase, which converts trypsinogen into trypsin.
2. Erepsin, which digests products of protein digestion that may

have escaped the action of trypsin; *e. g.*, proteoses, peptones and peptids; it also digests casein of milk.

3. Inverting ferments. These are as follows: Maltase, which converts maltose into dextrose. Invertase or sucrase, which converts cane sugar into dextrose and levulose. Lactase, which converts milk sugar into dextrose and galactose.

The intestinal canal of the newborn infant does not differ so much anatomically as it does functionally from that of later life. The gastric contents, after having been propelled through the pylorus into the duodenum, are mixed with, and subjected to the combined action of, the bile and the pancreatic and intestinal juices. It is in the small intestine that the most profound changes occur, and the products of digestion are mainly absorbed. By the time the food arrives at the ileocecal valve these processes are practically complete.

Absorption in the Intestines.—Chyme, or the stomach contents passed into the infant's duodenum, is chiefly composed of watery elements containing sugars, salts, etc., in solution. The proteins are partly unchanged (casein coagula), and partly appear in the form of acid metaprotein, proteoses, and peptones. Fats pass into the intestines suspended in fluids or entrapped in the meshes of casein curds. The acidity of the chyme, due chiefly to HCl, is neutralized by the pancreatic and intestinal juices and the bile.

About 80 per cent. of all absorption takes place in the small intestine, which accounts for the fact that disturbances in this part of the digestive tract, with their concomitant diarrhea and frequent watery passages, are followed by extremely rapid wasting and exhaustion. Proteins are supposed to be absorbed in the form of amino-acids, and in healthy breast-fed infants this absorption takes place almost entirely in the upper part of the small intestine; only traces of protein are found in the lower ileum. The reaction of the upper part of the gut is slightly acid. In artificially fed babies the digestion and the solution of casein are less complete in the duodenum, and the reaction is distinctly acid.

Pancreatic lipase (steapsin), assisted by bile, splits fats into fatty acids and glycerol and also saponifies and emulsifies them. In these forms they are readily taken up by the intestinal villi, but become converted into neutral fats during their passage through the mucous membrane. Fats are not completely digested and absorbed in the intestines of infants, considerable being excreted in the feces as neutral fats and fatty acids.

The sugars are taken up as dextrose, which is the most readily absorbed monosaccharide. Galactose and levulose must undergo a further process of inversion to dextrose before they can be taken up. It would thus appear that maltose, which splits into two molecules of dextrose, may be absorbed more readily than either lactose or saccharose. There is still a difference of opinion as to whether pancreatic amylase (amylopsin) is active in early infancy; later on it can and does convert starch into maltose, as shown on next page.

13

```
                                    Starch
                                       |
                                 Soluble starch
                                       |
                  ┌────────────────────┴────────────────────┐
            Erythrodextrin         ·                      Maltose
                  |
        ┌─────────┴─────────┐
   α-Achroödextrin       Maltose
        |
   ┌────┴────────────┐
β-Achroödextrin    Maltose
   |
┌──┴────────────┐
γ-Achroödextrin   Maltose
   |
   └────────────┘
        |
     Maltose
```
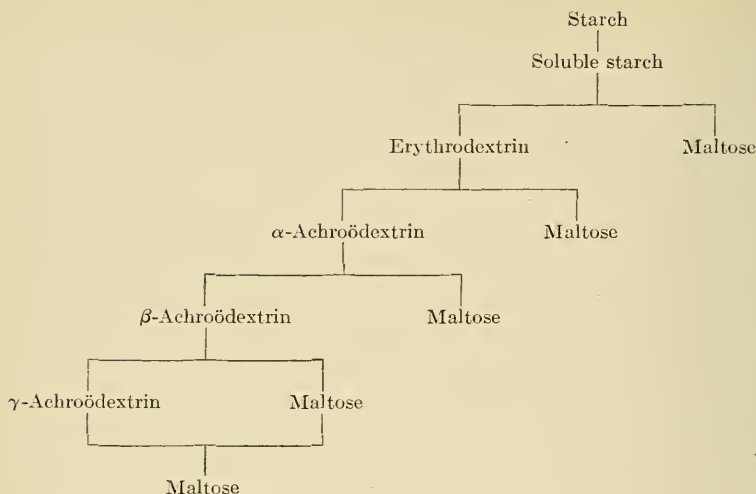
In the large intestine little else but water is absorbed, so that an infection of the colon alone produces comparatively little wasting. Fat absorption is slight, while sugars, salts, and peptones may be absorbed with moderate facility; therefore, in rectal feeding the food should be thoroughly predigested.

The stools of infants and of children are a delicate index of the state of the digestive functions, and a careful examination of the feces in conjunction with other clinical observations will give us a fair idea as to whether the food is being digested and assimilated. In order to detect abnormal conditions it is necessary to be thoroughly familiar with the characteristics of normal evacuations. Normal, of course, does not mean uniform; it is well known that even in a perfectly healthy infant the stools may at times vary in color and consistency, and may even contain coarse white particles. This is not astonishing when we consider that even such an ideal food as mother's milk is not always uniform in composition, especially in its percentage of fat.

During the first two or three days after birth the infant has about four to six passages of semisolid, dark, brownish-green meconium.[1] These may continue for a week, but they usually begin to change in character on the third or fourth day, and soon assume the usual appearance of the healthy feces of the breast-fed infant. These discharges contain mucus, fats, epithelial débris, and a small portion of albuminous matter, their normal amount varying between two and three ounces per day. They are golden-yellow in color, owing to the presence of bilirubin, are homogeneous, of butter-like consistency, with an acid reaction, and a slightly acid odor, probably due to lactic and fatty acids. The small whitish curd particles already referred to—the

---

[1] Meconium is composed of intestinal mucus, bile, vernix caseosa, epithelial cells, hairs, fat globules, and cholesterol crystals.

so-called milk granules of Uffelmann—are not albumin, but fat crystals and zoöglea of bacteria.

Normal milk feces contain about 85 per cent. of water and 15 per cent. of solids, mostly fats. Hydrogen and carbon dioxide are the only gas constituents; H_2S and marsh gas are never present. Protein is almost entirely absent, the minute quantities which may have escaped solution and digestion being transformed by the intestinal bacteria into indol, skatol, phenol, and ammonia.[1] Fats occur in the form of fatty acids, soaps, and neutral fats, and form from 10 to 20 per cent. of the dry residue of milk feces. Sugar is absent, but its derivative, lactic acid, may be encountered in small amounts, while starch may appear unchanged in the stools. The inorganic salts are chiefly represented by the calcium salts, and the biliary elements by hydrobilirubin, unchanged bilirubin, and cholesterol.

In addition to these the feces contain much mucus, immense numbers of bacteria, and various products of bacterial germination. The stools number from three to six per diem during the first month, afterward decreasing to from one to three, and later on to one daily movement. The stools of the bottle-fed baby are larger in bulk and contain less water than those of the breast-fed infant; they may also be more frequent, as many as four or even six being compatible with health as long as their consistency and color remain normal. The number of stools is merely an indication of the amount of intestinal residue, and not of disease.

In a baby fed on cow's milk that has been perfectly digested, the feces may closely resemble those of the breast-fed child, but usually they are firmer, paler, and putty-like, with a neutral or alkaline reaction, and slightly offensive odor. Infants fed on malted or farinaceous foods have more or less dry and broken-up movements, yellowish-brown in color, and slightly alkaline, with a malt-like odor.

The stools are never fully formed until a mixed diet is given. The peculiar character of milk stools then disappears, the feces become darker and emit the adult odor, but are softer than the latter. The reaction of the fecal discharges is still a matter of discussion. As a rule, it is acid in the breast-fed baby, while in the bottle-fed baby it is said to be feebly alkaline or neutral, provided the cow's milk is well digested; but if the fat percentage of the milk is too high or if more carbohydrates are given than can be digested, the reaction becomes acid.

Bacteria.—Experiments and investigations concerning the bacteria which inhabit the intestines are as yet incomplete, and probably will be so for some years to come. After all, what would be gained if we could really distinguish and study each variety separately? Considering that every species existing in the intestines is influenced in its development and its function by other varieties and groups actually present, or temporarily predominating, it seems more important for

---

[1] The nitrogen content in these feces is derived chiefly from the intestinal secretion and from the bodies of bacteria.

practical purposes to study the chemical changes produced in intestinal secretions and in the ingested food by the combined action of all the microörganisms living in the intestines.

What changes are known to be bacterial in origin? In the normal breast-fed infant the intestinal flora are almost constant. To begin with, bacteria are absent from the gastro-intestinal tract at birth, but twenty-four hours later they have effected an entrance through the mouth and rectum. When breast milk is exclusively used the principal organisms present are: Bacillus aërogenes, Bacillus coli communis, and Bacillus bifidus, although as many as 19 different kinds have been isolated from the stools of healthy nurslings.

Their number and distribution vary considerably in different parts of the gut, relatively few being found in the small intestine, while they flourish in the cecum and the colon. In the healthy baby they serve a useful purpose by aiding in the digestion of the food elements.[1] Their most important role, however, seems to consist in producing the acid fermentation which prevails in the intestines of the breast-fed infant. This lessens the development of other harmful microbes,[2] and reduces their action considerably.

As carbohydrates favor the development of some of these normally-present, acid-producing bacteria, the putrefactive processes can be modified, and to some extent controlled, by means of a diet rich in carbohydrates and relatively poor in proteins. Aside from the beneficial effect of partial starvation induced by such a diet, it in a measure explains the value of carbohydrate food in the early treatment of intestinal indigestion and also the temporary success which sometimes follows the use of condensed milk or similar foods.

The great danger of nutritional disorders in infancy and childhood hardly needs emphasis. By lowering the power of resistance to disease, they directly or indirectly cause more deaths in this early period of life than all other illnesses combined. Disturbances of nutrition do not involve merely the processes of digestion and absorption in the gastro-intestinal canal, but also the very important functions of assimilation and disassimilation, known as intermediary metabolism.

A derangement of these processes not only hinders the normal function, but also retards the development of the immature body. The proper appreciation of this fact will enable us to realize the great importance of this subject, and will lead to the selection of the method of feeding most suited to the individual case. As the process of metabolism in older children progressively becomes like that of the adult we may confine our attention to the differences observed in the infant.

To begin with, it must be acknowledged that the number of ascertained facts is small and that their clinical significance often seems

---

[1] Experiments have demonstrated that newborn animals, fed with sterilized food, remained decidedly backward; some of them could not even be kept alive.

[2] For instance, putrefactive bacteria, which thrive on the proteins of boiled milk,

obscure. What then are the abnormalities known to occur under pathological conditions within the digestive tract? As to the study of the changes in the *gastric secretions*, the stomach tube has considerably facilitated research in this direction. In almost all of the constitutional diseases of infancy, gastro-intestinal as well as febrile, the secretion of hydrochloric acid is greatly diminished, so much so that in many cases it is not present at all in the free state. This entails more or less complete loss of the antiseptic action of the normally present free hydrochloric acid.

On the other hand, as regards therapeutics, it clearly demonstrates the necessity of longer intervals between feedings for the sick child, especially when cow's milk is taken, since the latter requires about 30 per cent. more acid than does mother's milk. Hyperchlorhydria is found in rare conditions, such as congenital pyloric stenosis. This, however, is due to overproduction as well as to accumulation, favored by delayed evacuation of the gastric contents. Since the salivary amylase (ptyalin) and the gastric ferments, rennin and pepsin, are all secreted in sufficient quantity in sick children, it is evident that the administration of these enzymes by mouth would not prove very beneficial in these nutritional disorders.

As for the other secretions, the detoxicating action of the liver in the infant has not been definitely proven, but there is good reason for accepting the statement that in the course of certain diseases the digestive power of the pancreatic secretions, and especially that due to trypsin and pancreatic lipase (steapsin) may be diminished. We know that the power of digesting starch is slight during the first few months of life; but as yet we know very little concerning the changes in the activity of the intestinal glands during illness, although duodenal catheterization will no doubt aid us considerably in the future in gaining a clearer insight into this part of the body economy.

In basing our conclusions upon these few facts, we scarcely feel justified in declaring that disorders of nutrition are identical with a disturbance in the digestion or the absorption of the various foodstuffs within the intestinal canal. On the contrary, it is well known that, even by very sick children, nitrogen and milk sugar are very well taken up; the milk sugar is absorbed unchanged only in exceptional cases, and then appears as such in the urine.

As regards fats, there is no doubt that during illness a considerable amount passes unutilized through the intestinal canal to be excreted with the feces, either in the form of neutral fats—true fat diarrhea, which is rare—or as soap-stools, *i. e.*, combined with alkalies. Free fatty acids too may be found predominating in the fecal discharges, but in none of these eventualities has the loss of fat been demonstrated to be so large as seriously to injure nutrition.

The few data which we possess concerning the excretion of mineral salts in the feces show that these bear no direct relationship to any definite disease. Normally, fermentation predominates in the intestines of the breast-fed infant, while putrefaction is present to a limited

extent in the bottle-fed baby. In certain pathological conditions, however, putrefaction occurs with both types of feeding, its presence being confirmed by the offensive odor of the feces and the appearance of indican in the urine.[1]

Raw milk does not putrefy readily; indeed, its milk sugar may under favorable circumstances even prevent putrefaction; it is, therefore, probable that the intestinal secretions themselves, increased by certain disturbances, furnish the material for putrefaction, especially in the bottle-fed baby. This process is also aided by the diminution or the complete absence of free HCl, which normally acts as an antiseptic.

Whether or not the products of putrefaction can directly injure the organism is not definitely known. Since assimilation and retention of the necessary food-elements are essential for growth, the question arises, to what extent do pathological conditions influence these normal functions? Again we must confess our limitations as to any knowledge on the subject. There is no doubt that a certain amount of nitrogen is retained under practically all conditions, even when a sick infant loses in weight, and it necessarily follows that at such periods other foodstuffs, especially fats, must pass through the body unutilized. Very often the fluctuations in the weight of the infant are so marked within short periods of time that these variations can only be explained by a pathological change in the watery content of the body. This is not surprising when we consider that more than 60 per cent. of the total food assimilated consists of water.

Phosphates originating from milk are usually well retained, even by a sick baby, while chlorides are apt to vary with alterations in the water content of the whole organism, or *vice versa*. An abnormal loss of lime may be a causal factor in defective ossification, as seen in rickets, and may, perhaps, play a role in the etiology of tetany, an affection in which the brain has been found to be exceptionally deficient in lime salts.

The processes of disassimilation in the sick infant presumably differ from those in a healthy baby. Excessive oxidation is supposed to interfere with normal metabolism in atrophic conditions, where an infant with an excellent appetite loses weight instead of gaining. Certain disturbances during infancy, presumably intestinal in origin, but more often during childhood, lead to the excretion of acetone bodies through the lungs instead of in the urine, as in adult life. The concomitant cyclic vomiting and fever, so frequent in older children, are often associated with the presence of considerable acetone in the urine.

Keller, of Breslau, has demonstrated some interesting and practical facts for the diet of sick babies concerning the metabolism of mineral salts. It has been noted that the urine of infants suffering from certain nutritional disorders often contains a relatively large amount

[1] Indican is absent in the urine of healthy breast-fed babies, but a small amount is present in that of healthy bottle-fed babies.

of ammonia. Keller and his school claim that an excess of fat in the infant's[1] food combines with alkalies in the intestines and forms soaps which are excreted in the feces. The alkalies required for the neutralization of the inorganic acid end-products of metabolism are thus prevented from reaching their destination, and fulfilling their purpose, and Nature provides the necessary substitute in the form of ammonia which later appears in the urine.

While it has not yet been definitely proven, it seems probable that an excessive loss of alkalies may lead to actual acidosis, and the injurious consequences of overfeeding with one food constituent are at once apparent. The same investigators have by analogy, basing their conclusions upon their clinical observations, described similar food injuries occasioned by diminished tolerance of starch, sugar, proteins, and of whey salts.

Czerny and Keller claim that the ingestion of fats, carbohydrates, proteins, and whey salts "beyond the point of tolerance" produces distinct symptoms resulting from food injuries, which concern not only the digestion and absorption, but also the intermediary metabolism of the infant. This symptom-complex is of great clinical value, inasmuch as its recognition may obviate the prolonged starvation so frequent in chronic and acute indigestion. The mere withdrawal of the offending constituent from the food is sufficient to inaugurate improvement, and with proper care will insure a final cure.

It must, however, be admitted that these symptom-groups are not always clearly defined. For instance, in the same individual there may be shown different degrees of tolerance for more than one food element. Likewise, in a given case, carbohydrates may be tolerated only when the food contains a low percentage of fats, or fats may be metabolized when the carbohydrates are cut down. Proteins do not seem to aggravate the intolerance for either fats or carbohydrates, but protein intolerance is aggravated by a higher percentage of either fat or carbohydrates in the food.

The objection has been made that these subdivisions are too schematic. Possibly they are; but they have the great advantage of being practical and simple; for a correct diagnosis at the same time quite definitely indicates the proper remedy. At all events, they seem to be a step forward in emancipating us from the one-sided bacteriological viewpoint, and, though only a stepping-stone to the final solution of the complicated problem of infant feeding, they are worthy of consideration.

Fat Indigestion.—The proper amount of fat in an infant's diet is necessary for the production of teeth, for the regulation of the bowels, and for the nourishing and building up of the bony and nervous systems. Moreover, there exists an intimate relationship between fat metabolism and the resistance of the child to infection. It follows, therefore, that a baby may suffer more or less from malnutrition,

---

[1] This differs in different individuals according to their digestive ability, etc.

anemia, lack of development, and decreased power of resistance to disease (rickets) either because of too little fat in the food or because of its inability to digest and assimilate the fat. Fat indigestion is very often due to overfeeding with rich milk or cream mixtures; oils— olive oil and cod liver oil—curiously enough, seem to create less trouble.

In addition to the ordinary manifestations of indigestion, an excess of fat in the food causes a more or less distinct group of symptoms, such as malnutrition, coated tongue, fetid breath, and gastric disturbances, especially sour vomiting soon after feeding. The child looks pale, its complexion is muddy, and it often suffers from eczema and intertrigo of the buttocks.

Constipation is the rule but this may alternate with diarrhea. The stools sometimes have a shiny, oily look (fat diarrhea) with a butyric odor. More often they are small, fragmentary, hard, dry, crumbly, and grayish-white in color; they then contain a large amount of insoluble salts, and do not soften even under the influence of a water enema. Microscopic examination of the feces reveals an excess of neutral fats, fatty acids, and soaps. The loose stools contain small soft curds, somewhat resembling scrambled egg.

Colic is a frequent symptom, but it is usually caused by overfeeding or by undigested proteins. The urine is rather irritating, and smells strongly of ammonia. The more serious fat injuries, however, are uncommon except when the food contains an excess of both fats and sugars. The fat of mother's milk contains less fatty acids,[1] and is therefore more easily digested. This in a measure explains why an infant can assimilate mother's milk with its 4 per cent. of fat, while it may fail to digest the 2 per cent. fat in cow's milk, and also why the latter with its excess of acids may be readily converted into diacetic acid and acetone.

Carbohydrates.—Sugars, being the most easily digested elements in the infant's artificial food, are liable to be increased at the expense of the fats and the proteins. Like fats, they serve as fuel, furnishing heat and supplying energy to the cells. While they may be partially converted into fats, and temporarily replace these, a prolonged sugar diet will lead to serious disturbances.

Most physicians, aware of the excess of carbohydrates in many proprietary foods which are so often advertised as substitutes for milk, advise against their use except in certain cases where, with the proper admixture of milk, they serve a useful purpose. By changing the intestinal flora they are apt to create fermentation,[2] which, within limits, seems necessary for the proper functionating of the intestinal processes in infants. We say "within limits," because the products of excessive acid fermentation may irritate the mucous membrane, and thus cause purgation.

---

[1] Glycerides of butyric, caproic, caprylic, and myristic acids.

[2] This fermentation cannot always be produced by starch alone, but sometimes requires the addition of sugar.

Cereals, such as barley, rice, arrow-root, etc., are useful even before the child is able to digest them perfectly, since they mechanically facilitate protein digestion by preventing the formation of very coarse casein curds. Some infants, it is true, can take a great deal of carbo-hydrate food with apparent impunity. They often look well and sturdy, and, although rather fat, they have a good color and tolerably firm flesh. The stools are yellow or brown, not green, and are often formed, and have an acid reaction.

Except for a certain amount of meteorism, there seems to be noth-ing wrong with the baby, but should it be attacked by disease it shows very little power of resistance, thus confirming the every-day experience of the temporary character of the apparently good results of artificial feeding and its probable ultimate failure.

As a rule, however, food containing an excess of carbohydrates soon leads to intolerance, indigestion, or to sugar intoxication, as indicated by loss of weight, flabby musculature, colic, nervousness, irritability, urticaria, and even fever.[1] The stools are numerous, watery, non-putrid, light green in color, and irritating to the skin of the buttocks on account of their acidity; they contain no curds and are passed with flatus. Gas formation producing tympanites, vomiting, and regurgitation of sour material is a common symptom.

In more severe cases where the intolerance has been aggravated by prolonged dietetic errors, lactose, and frequently acetone and diacetic acid, may be found in the urine. These babies become thin, delicate, anemic; the tongue is coated, the appetite is poor or capricious, and they suffer from constipation, abdominal distention, and severe constitutional disturbance. With such patients sugar intoxication is commonly associated with an inability to metabolize whey salts.

Treatment consists in eliminating the latter as well as the sugars from the diet temporarily, and when the symptoms have subsided, a different sugar in proper proportion should be cautiously added; maltose and dextrin are preferable, because they are not apt to produce fermentation, while milk sugar is prone to set up fever and diarrhea.

Protein Indigestion.—While indigestion of proteins, especially the casein of cow's milk, does not seem to be so common as has generally been considered, its existence cannot be altogether denied. It certainly does occur, especially with foods having a high fat content, and is accompanied by the ordinary symptoms of intestinal disturbance, such as diarrhea, constipation, fever, colic, and sometimes even severe nervous manifestations.

The stools in these cases are foul-smelling, alkaline, and occasion-ally contain curds, which, unlike those of fat indigestion, are usually hard, tough, and not friable. They are frequently oblong, rounded or bean-shaped, and vary in size from that of a lentil to a lima bean, while in their consistency and appearance they resemble hard American

---

[1] Sugar fever is similar to salt fever. In the body of infants, sugar seems to act much as an inorganic salt; being loosely combined and probably stored in the subcutaneous tissues, it favors the retention of water in the system.

cheese. The curds can easily be picked out of the napkin with a pin, washed and shaken about in water without being broken, and when dropped into water they sink to the botton.

It is said that these casein curds disappear from the stools if a diet of boiled and diluted milk exclusively is given, and that they occur only in infants fed on pasteurized or raw milk with a low fat percentage. The proteins alone supply the body with nitrogen, and no other food element can take their place, consequently the feeding of proper amount of proteins is of primary importance during early infancy—the time of rapid development.

If, for therapeutic purposes, it becomes necessary to reduce the amount of proteins temporarily, they should be raised to the normal proportion as soon as possible in order to prevent anemia, loss of muscular tone and power, and circulatory disturbances. A food containing too little of both proteins and fats predisposes to constipation and probably to rachitis. Whey proteins are very easily digested and rarely cause disturbance; any trouble they may set up is due to their whey salts.

For many years attempts have been made to classify nutritional disturbances, but the results have been unsatisfactory, the grouping becoming more and more complicated and confusing instead of more simple. A mere clinical classification which takes account only of symptoms such as vomiting, constipation, colic, diarrhea, loss of weight, wasting, complications of dentition, etc., is, of course, not sufficient.

Pathological study of the subject has cleared up much that was obscure, but the clinical course often differs widely in different individuals in whom the anatomical and pathological changes are similar. In other, not exceptional, cases, the trivial morbid changes demonstrable postmortem bear no proportion at all to the serious symptoms observed during the course of the disease. Moreover, a classification based on a particular part of the digestive tract is not admissible, because of the intimate functional relationship of the various portions which may be involved at the same time or in rapid succession.

Since Pasteur's discovery of the bacterial uncleanliness of food, bacteriologists have given us much information concerning the normal and the pathological intestinal flora as well as specific infections in infantile intestinal disorders. But these brilliant investigations have so far yielded relatively few practical results; at any rate, they have not enabled us to differentiate the various clinical pictures of nutritional derangement, nor, according to general statistics, does the mortality appear to have been materially diminished by either pasteurization or sterilization of milk.

Physiological chemistry teaches that disturbances of nutrition comprise not merely local pathological processes in the gastro-intestinal tract, but represent conditions which affect the whole organism in that most vital function, intermediary metabolism. But, so long as the more intricate functions of normal nutrition are shrouded in

mystery, we certainly cannot hope to solve the etiological problem of its derangements, especially since idiosyncrasies, individual peculiarities, and special environment, such as heat, infection, and hospitalism, must be taken into consideration. Are these nutritional disorders due to defective digestion, faulty absorption, disturbed metabolism and assimilation, or to intoxication and decomposition?

A serious and very important problem is the question of food anaphylaxis. It seems that this phenomenon even occurs in very young children so that they are abnormally sensitized toward the proteins of milk, for example, or a little later to those of the various foodstuffs. The recognition of this condition is still rather difficult, but the introduction of these substances in the diet will result in so-called anaphylactic shock, inducing colic, nausea and vomiting, diarrhea, and even fever, as well as various skin manifestations.

It is only when scientific investigation along the lines of pathology, bacteriology, and metabolism goes hand in hand with mature clinical observation that we can hope to make any progress toward a solution of these questions. Czerny and Keller propose the following classification, which is based not only upon scientific data, but is clinically of the greatest value to the physician in deciding upon the treatment of the case:

1. Nutritional disturbances arising from alimentation.
2. Nutritional disturbances arising from infection.
3. Nutritional disturbances arising from congenital anomalies.

"Proper nutrition makes for the perfection of the coming generation, improper nutrition mars it." How, then, is a baby to be fed in order to insure proper growth and development if it cannot have the advantage of the natural ideal food? The feeding problem in itself, full of difficulties, is often still further increased by a diversity of individual circumstances and idiosyncrasies which must enter into a consideration of the question of establishing an equilibrium between the quantity and the quality of the food and the infant's capacity to digest and assimilate it.

In the perfectly normal infant the amount and the composition of the food may vary to a considerable degree without producing nutritional disturbances. This is owing to a great tolerance for the different food elements, and explains the common experience that a normal baby may thrive on a milk formula or on food which would cause serious trouble in another less robust infant. It is true that every infant is a law unto itself; nevertheless, there are certain rules for the guidance of the physician who prefers scientific methods to empiricism.

Proper food must make possible a proper functioning of the intestinal mucous membrane, its glands and its adnexa. It should incite a sufficient peristalsis, and favor the growth of normal bacteria. It should be sufficient in amount to maintain the body heat, and so assimilable as to favor the building up of the body cells. Improper food, failing in one or more of these requirements, produces morbid changes in the gastro-intestinal canal that lead to derangement of

the intermediary metabolism, followed by disturbance of the general nutrition and, consequently, by a lessened power of resistance to disease. It must be emphasized that nutrition and nutritional disturbances are distinctly different in infants and children.

The foods should contain fats, proteins, and carbohydrates in a fairly definite percentage, and in such proportions as will best meet the nutritional demands of the baby. If, for any reason, it is necessary to reduce the fats and proteins markedly they should be raised to normal as soon as possible. No other food element can take the place of nitrogen or proteins. Carbohydrates may temporarily replace the fats, but in time the lack of fat will produce serious nutritional disturbances. Too great reduction of proteins, fats, and salts may result in rickets, scurvy, anemia, and other forms of malnutrition.

In artificially fed infants uncomplicated cases of under-nourishment resulting from simple lack of food are rarely seen. Usually they are referable to disordered absorption or assimilation, resulting in chronic indigestion, and originally brought about by improper feeding. Underfeeding may occur in the breast-fed child if the mother's milk is too weak in composition or too small in quantity or when some deformity, such as inverted nipples, etc., interferes with nursing.

Hare-lip, cleft-palate, large adenoid vegetations, or debility may often prevent the nursling from getting a sufficient amount of milk, but this trouble is easily recognized and remedied. The infant fails to gain or even loses weight; it sleeps well, is not restless, and its whining cry is neither frequent nor prolonged; in the absence of gastro-intestinal symptoms, the scanty stools and urine and a subnormal temperature will clinch the diagnosis.

Holt has drawn attention to the fact that in the early days of the disorder there is a rise of temperature to 104° or 105° F. (inanition fever), which, however, soon declines if plenty of food is given. Infants can be made ill with a perfectly wholesome food if it be given in excessive quantity. Overfeeding may mean a superabundance of all ingredients or of a single one. It sometimes causes severe and persistent indigestion when the infant is fed at irregular or too short intervals, or is given too much at a single feeding.

The baby suffers from colic announced by a sharp piercing cry, which often begins at a definite time after feeding and is continued until eructation, or vomiting, or the passage of flatus apparently brings relief. During the attack the extremities are rather cold, the thighs are flexed upon the abdomen, and the latter is rigid, evidently due to a circulatory disturbance in the splanchnic area (especially of the intestinal walls). Spitting frequently heralds the trouble, followed in some cases by vomiting. Generally the infant is cross, fretful, runs a slight temperature, and sleeps restlessly. Not infrequently the baby suffers from facial eczema and seborrhea of the scalp (often limited to the anterior fontanelle), though other areas of the skin may look fresh and pink and show normal elasticity.

These disturbances are seen in artificially fed as well as breast-fed babies; in the latter a high fat percentage is said to be the etiological factor. The condition is serious, principally on account of difficulties that may arise during and after weaning. With the artificially fed baby nutritional disturbances and their serious consequences most commonly result from too large quantities, or too strong modifications of milk or too frequent feedings.

Too much even of a properly modified milk mixture, given at one time or at too short intervals, will often retard weight development; therefore, the mere fact that a baby does not gain but even loses weight is no indication for increasing its amount of food. If other causes, such as adenoids, stomatitis, syphilis, and tuberculosis can be excluded from the etiology, a decline in the weight curve, in spite of a plentiful supply of proper food, strongly indicates disturbed nutrition. The digestive tract needs rest. If this is prevented by too frequent feedings, the stomach is never emptied completely; less HCl is secreted, and is therefore not present in a free state, which in turn acts unfavorably on the secretion of the pancreas.

Ambitious mothers should therefore be warned against the disastrous consequences of excessive feeding; not only does it cause gastrointestinal disturbances and a predisposition to intestinal infection, but it may so upset the equilibrium of the metabolic processes that it may take weeks or even months to restore the same. The food should be increased, not merely to make the infant weigh more, or because its restlessness and crying are wrongly interpreted as a sign of hunger, but only when steadily declining weight is accompanied by a small amount of feces and urine, while the intestines are apparently healthy.

Disturbance of Balance.—A relatively unobjectionable, pure milk, given at regular intervals and adapted in quantity and in composition to the requirements of the average infant, does not always insure good results. While most children thrive upon it, others, after a few weeks of comparatively rapid gain, remain stationary or nearly so in weight and in physical development. Increasing the quantity or the strength of the food does not improve matters; it may even lead to an appreciable decrease in weight.

Curiously enough the baby hardly seems to be ill; the pulse, respiration, and the temperature, as well as the urine show no serious disturbance, but the child is restless, peevish, and pale, though apparently not in pain. The stools are often fairly regular, but in fully developed cases they are rather dry, light in color, and offensive in odor. They leave no stain on the diaper, and their relation to litmus is alkaline. Evidently, the food is not being assimilated.

Proteins and carbohydrates do not seem to be the cause of the trouble, because a change to a food rich in both but poor in fat, given in small quantities at first, usually remedies the trouble. The disturbance is due to the fat of the cow's milk, only a small percentage of which is tolerated, and whenever this limit is exceeded the trouble

reappears. We emphasize "the fat of cow's milk," on account of the remarkable fact that in these cases breast milk is well borne in spite of its high fat content.

Dyspepsia.—In artificially fed infants, even under the most favorable circumstances, the digestive organs must perform an increased amount of work, and overfeeding, of course, adds to this burden. When combined with an alteration in the osmotic conditions in the intestine, it can be readily understood that it might cause a certain exhaustion of the digestive and the absorptive capacity. Naturally, intermediary metabolism in turn is affected, and the degree of derangement finds expression in the progressively severe symptoms observed in dyspepsia, intoxication, and decomposition.

The first of these—dyspepsia—often quickly follows a neglected condition resulting in loss of weight, but the symptoms vary greatly. It represents a very extreme disturbance of nutrition, the term implying not merely non-digestion, but rather a disturbed digestion through the action of the food or its derivatives. If the digestive processes themselves are involved, dyspepsia is not a primary affection, but the result of a general condition.

Thin, delicate infants, especially those under three months old, are more susceptible, but even the robust are not immune if serious dietetic errors are made. Diarrhea, vomiting, and gas formation dominate the clinical picture. The stools are watery, green, and frequent, four to six being usually passed within twenty-four hours; they contain small curds, consisting, not of undigested proteins, but of fats and fat soaps. Vomiting, apparently the result of gastric irritation, may occur immediately after feeding or later. The temperature exceeds by one or two degrees the daily variation in healthy infants. The weight drops distinctly during the first few days, then either remains stationary, or declines slowly.

Symptoms of abnormal fermentation in the gastro-intestinal tract—eructations, foamy fecal discharges passed with flatus, colic, and distention of the abdomen—are marked the higher the percentage of carbohydrates, especially of sugar, in the food; as, for example, in condensed milk. The urine is negative as to albumin, sugar, and casts, but contains an increased amount of ammonia. The little patient does not appear seriously ill, the heart, the lungs, and the kidneys seem unaffected, but it is peevish, restless, and often cries with pain.

Here, again, fat is at fault, as to all appearances it lowers the tolerance for sugars, though the latter (in the order of their injuriousness, milk sugar, cane sugar, and maltose) are the principal cause of the disorder. Sugars and fats probably cause the formation of acids which, by withdrawing the alkalies from the system, may produce a relative acidosis, as evidenced by the increased ammonia coefficient in the urine. If the condition is recognized in time, and either good breast milk is given or the fats and sugars are reduced to a minimum, improvement will most likely set in. The chief danger lies in the fact that

the disorder may go on to intoxication and decomposition, which would be serious for the weakened child.

Alimentary Intoxication.—Alimentary intoxication is a severe disturbance of intermediary metabolism with evidences of relative acidosis, a condition resembling diabetes or uremia, which follows dyspepsia when the injurious food is continued. It is characterized by its sudden onset (there is also a lingering form), diarrhea, vomiting, high fever, sugar in the urine, leukocytosis, and eventually collapse. On account of its most prominent symptoms, the disease was formerly thought to be bacterial in origin, and was classified as acute gastroenteritis, or as cholera infantum.

It is true, bacterial infection can scarcely be disregarded as an etiologic factor, but is it the chief one? The experience that most of these cases, if seen the first day, respond within twenty-four to forty-eight hours to starvation treatment certainly does not favor this assumption; but, on the contrary, points to the food as the primary cause. Indeed Finkelstein and Meyer have proved that an excess of milk sugar can produce the whole symptom-complex of this intoxication, especially if the food has a high fat percentage. That the disorder is not only intestinal but is also connected with metabolism is shown by the presence of sugar in the urine.

There is a possibility that milk decomposed by bacteria before or after ingestion may irritate the intestine, and after its absorption derange the metabolism. So far, however, the toxic action of these products has not been definitely demonstrated, and we have reason to believe the primary cause to be a dietetic one, and that the decomposition of the food and the presence of pathogenic microörganisms, as well as the depressing effect of heat are not unimportant additional factors.

Previous alimentary disorders, a lowered resistance from any cause, and artificial feeding in itself, create a tendency to the disturbance. The onset is usually sudden, the temperature rises steadily to 104° or 105° F., or it may fluctuate, and in the case of collapse it falls suddenly. Vomiting and diarrhea occur early, though the latter is not always severe—four or five stools daily—but when they reach ten to forty, the sunken fontanelle, the pale skin, and the dry mucous membranes are symptoms that cannot be mistaken.

In the milder cases the baby looks sick, the face is pale, expressionless, and sleepy-looking, while in the more serious ones the child is apathetic, and never smiles. The half-opened eyes, together with a pinched expression, a bluish-gray color of the skin, and coma make the prognosis doubtful. The more severe the diarrhea, the more decided is the loss in weight. After the initial loss (sometimes as much as half a pound to two pounds a day) the weight remains stationary or declines slowly. The respirations deepen, their rhythm is accelerated, and the infant gasps for air.

On examining the chest signs of hypostatic pneumonia are often found. On account of the great loss of fluid no anemia is demon-

strable, but leukocytosis seems always to be present. The disturbed circulation is manifested by the pallor of the skin, cerebral symptoms, hypostatic pneumonia, and meteorism, the latter resulting from splanchnic congestion. Glycosuria is a constant and early symptom. Albumin and casts are found in the urine, but the absence of leukocytes and epithelial cells excludes inflammation of the kidneys.

Decomposition represents the complete failure of the digestion, which progressively affects the resources of the organism, and finally leads to its complete breakdown. The condition usually follows a neglected digestive disorder, repeated attacks of dyspepsia, or a toxic condition, and is characterized by nervous irritability, emaciation, irregular respiration, subnormal temperature, and a slow pulse; the urinary findings are normal. The malady is aggravated by taking food, and leads rapidly to decomposition of the body. In this advanced stage the infant's organism seems to have lost all power of assimilating even minute quantities of food. To all appearances, a true reversal of nutrition has taken place, and the child has no chance for its life.

Even in mild cases improvement is tardy. The general state of the patient undergoes a radical change, it is nervous, sleeps badly, cries for hours at a time, and can only be quieted by the bottle, which is taken eagerly; it seems always hungry and thirsty, yet continually loses flesh. The weight falls with the progress of the disease, at first perhaps only an ounce per day, but the decline is more rapid as decomposition advances. The dry, inelastic skin soon hangs in loose folds over the bones, the eyes become large and hollow, the lips pale, and the skin assumes a grayish-blue hue, giving the child an unsightly, ape-like appearance—the horror of the pediatrician.

The bowel movements vary in character. They may resemble the normal or they may be slimy, loose and offensive, or may show all the characteristics of soap-stools. The urine, rather large in amount, is negative except for indican; sugar and albumin are found only in the last stages. The vital forces seem to be slowly ebbing away, sometimes interrupted by a short but passing toxic excitation. The pulse becomes small, frequent, and gradually falls from 110 to 80, 70, or even 60. Deep breathing, sighing respirations, finally assuming the Cheyne-Stokes type, and a frequently subnormal temperature, possibly now and then interrupted by a sudden rise to be followed by a sharp decline, complete the pitiful picture. The gradually increasing relaxation may end in collapse, or death may occur from terminal pneumonia, otitis, etc.

Every physician knows the picture of atrophy clinically, but the theoretical explanation is modern, and merits attention. The decomposition affects first the fat, only a small amount of which is tolerated, and only for a short time. A limited percentage of carbohydrates can be borne with relative impunity, especially if the fats are cut out. Casein and albumin also seem to favor the decomposition.

No other problem of infant feeding shows more clearly the tremendous advantage of human milk, which is the only salvation for very

young infants. When fed upon it mild cases of decomposition improve rapidly, in spite of the fats and carbohydrates, and even severe ones have a good chance of recovery under careful management.

Feces.—Examination of the feces is no more important than the observation of the child's weight, the skin, the turgor, and its activity; but, in the majority of cases, the first evidence of nutritional disturbance is an increased number of bowel movements. First, their looseness, then their change in color, later curds, perhaps the presence of blood and an excess of fats or of mucus (pus is not often visible) attract our attention.

These changes are significant of a diseased condition, but not necessarily of any special disease. Almost all of the diseases of infancy may be accompanied by diarrhea; this holds true even in certain cases of syphilis and tuberculosis. Although the most severe form of diarrhea is seen in intestinal intoxication, on the other hand, intoxication may cause only a slight diarrhea.

In studying the feces then, as to their color, consistency, composition, odor and bulk, or in comparing them with the normal, we must not forget that from time to time even in the healthy infant the movements may be more or less watery, or may contain coarse white flakes, without any appreciable functional disturbance in the gastrointestinal tract. As mentioned before, small whitish particles in the feces of breast-fed infants are not always casein but often are the so-called milk-granules of Uffelmann. For the characteristics of normal stools we refer to the description on pages 194 and 273.

The amount of the stool is increased in all disorders of nutrition except in starvation which is merely the result of lack of food. In acute illness the more numerous the movements and the larger the total bulk, the greater the resulting exhaustion. In some chronic affections the quantity of the feces discharged may be very large, indicating a marked diminution of the child's absorptive powers. In this way the baby is starved in spite of its consuming a sufficient amount of digestible food.

After the first few weeks of life the healthy infant has one to three passages per day; if artificially fed, even five are not pathological, other characteristics being normal. Their number is slightly increased in affections of the upper intestine, while in inflammatory processes of the colon as many as twenty are not uncommon.

The reaction of the feces is said to be slightly acid in the breast-fed and neutral or alkaline in the bottle-fed baby. Acidity can be demonstrated by litmus in cases of fat indigestion (due to fatty acids) and of carbohydrate intolerance (due to acetic and lactic acids); alkalinity, however, may be shown where putrefaction predominates, as in protein indigestion. The reaction of the intestinal contents differs in different parts of the intestines, and doubtless depends to some extent on the kind of food taken. It probably largely determines the variety of bacteria flourishing in the gastro-intestinal canal, although the contrary is claimed—that the microörganisms present determine the reaction.

14

The color of the stools in morbid conditions varies so greatly from the normal that it is impossible to describe all the variations. The stools, normally a golden-yellow in the breast-fed, and a lighter yellow in bottle-fed babies, become brownish when cereals are added to the food. When the solids are much reduced, as in acute and copious diarrheas, the discharges may lose all color and look like serum, or like water containing white flakes of lymph (the so-called rice-water stools).

Clay-colored stools are abnormal, but do not necessarily indicate a serious condition. They are often due to a diminished amount of bile (as from biliary obstruction) or to an excess of undigested fat. Ashen-colored stools, however, are not always referable to the absence of bile or bile salts. A marked decrease in the hydrobilirubin content, a reduction product of bilirubin from which the normal feces derive their color, produces urobilinogen, a colorless substance normally found in the dry whitish stool of fat constipation. Bilirubin when oxidized changes into biliverdin, which is supposed to be the cause of the so-called green stools so frequently seen in infantile diarrhea.

Formerly this green color was supposed to be due to the action of bacteria, and for a time this assumption seemed to be confirmed by the appearance of green stools in institutional bottle-fed infants. Sometimes the stools are yellow when passed, but acquire a greenish tinge on the surface after exposure to the air. Practically all shades of green are observed, owing to the mixture of green, yellow, white, and brown. The colored stools in themselves are not significant of any particular disease, but merely indicate an abnormal condition.

Calomel medication often produces green stools, but this color disappears as soon as the drug is excreted, and probably indicates an excess of biliverdin which is not reabsorbed because of the hurried bowel movement. An admixture of blood makes the feces look red when they come from the lower part of the bowels and reddish-black or tarry from lesions higher up. Iron, bismuth, and manganese taken as medicine all make the feces more or less black, the shade depending, of course, upon the size of the doses and the intervals between them.

The odor of the defecations is caused by gases which form under the influence of bacteria in the digestive tract. In infants fed entirely on breast milk or properly modified cow's milk, the odor is usually slight, but it becomes stronger when other articles are added to the food; it is supposed to be sour or pungent in acid fermentation (favored by carbohydrates), and foul when putrefactive processes predominate. The latter are probably not always due to protein indigestion, but often to decomposition of intestinal mucus or pus which is often present in intestinal irritation. In fat indigestion the fecal discharges smell rancid and sour.

Although these statements cannot be offered as scientifically proved facts, they may be helpful in diagnosis and treatment, since they indicate the cutting down of carbohydrates in excessive fermentation and the reduction of the percentage of proteins in putrefaction.

The consistency of the feces depends chiefly upon the water and the fat contents. Normal discharges of infants fed on milk are free from lumps and are of a butter-like consistency—semisolid. They gradually become formed when other kinds of food are taken and their passage through the large intestine is delayed. A liquid state is just as abnormal as a too solid one. Copious watery diarrhea tends to dehydrate and to demineralize the system and, therefore, is dangerous, aside from the fact that absorption must suffer considerably if the food is hurried through the gut. Thin and watery stools are met with in typhoid fever, in gastro-enteritis, in rectal stricture, and often after hydragogue cathartic medication.

Composition.—In addition to the substances ingested as food, the fecal discharges contain bile, mucus, epithelial débris, residues of secretions, and many bacteria; in diseased conditions, blood, membranes, and pus may also be found. A certain amount of mucus is normally present in the feces, but as it is mixed it is not very apparent. It may be found in excess where there has been prolonged irritation by hard scybala as well as in other digestive disturbances; but it may be much increased in purely functional conditions, therefore is not always indicative of intestinal inflammation. It is discharged either in little balls (resulting from peristalsis) from the small intestine, or in stringy fragments from the colon, and may make up as much as one-third of the fecal mass.

Excessive fat in the food may cause either large, hard, dry, crumbly stools, which do not even soften when a soapsuds enema is given, or loose, greasy movements which look like oil. Often such feces are sour-smelling, yellow, greenish-yellow, or even green in color, and have the curdled appearance of scrambled eggs. Sometimes, however, large, gray, putty-like movements of a peculiar ammoniacal odor are passed. Very little, if any, protein remnants of the food are found in the feces of breast-fed babies, while the stools of bottle-fed babies may contain a little more.

Occasionally undigested fat and balls of mucus are mistaken for casein curds; the latter are large, smooth, white or grayish, bean-like bodies, and frequently occur in otherwise normal stools. They may mean nothing but a simple non-digestion of the casein; in fact, they are said to disappear when boiled milk is given and to reappear on feeding raw milk.

Blood is not infrequently seen in the stools in severe intestinal conditions, probably more from congestion than ulceration. Hard fecal masses are often streaked with bright red blood which comes from a small tear in the anal mucous membrane, or occasionally from small bleeding polypi. Small, frequent stools of blood and mucus only, passed with straining, point strongly to intussusception. In melena neonatorum the stools are tarry black. Pus may be seen in rare cases of communicating ischiorectal abscess, gonorrheal proctitis, or impacted foreign bodies.

CHAPTER X.

FRESH AIR IN THE TREATMENT OF DISEASE.

ONE must always study all the factors leading up to and, possibly, causing disease, and before deciding the positive influence of one factor, eliminate wholly or in part other etiological influences.

In considering, therefore, the rôle that fresh air plays, "controls" should be employed, as far as possible, for the comparison of a series of cases of the same type of disease living under similar conditions should enable one to draw fairly accurate conclusions.

My first work in fresh air treatment began some fourteen years ago in the children's wards of the Philadelphia Hospital. The wards were large, the milk fairly good, enough nurses were on duty to keep the children fed according to my directions, they were bathed regularly and kept clean; but in the wards where the very young infants, mostly foundlings, were placed, the results were very unsatisfactory. Much depended upon the physical condition of the infant on admission. A frail infant, perhaps premature and under normal weight, would gain for a few weeks at best, then remain stationary in weight, finally gradually lose weight, begin to have diarrhea and die. Robust infants on admission often did well for three months. The same symptoms after this period began to develop as in the infants admitted in a condition of malnutrition and, in spite of my best efforts, many of these robust children died.

Infants of the same type in my private practice were almost without exception doing well. Convinced that neither the food, nursing, nor general care of these hospital infants was at fault, and that the so-called hospitalism was nothing but lack of fresh air, and lack of outdoor air, I ordered these children, in the month of January, placed for two hours each day on the fire-escapes.

The cribs were simply moved out upon the fire-escapes, and towels pinned over the top of both ends of the crib, as wind shields. The infant mortality began to lessen immediately, and I began to see some hope for my infant hospital patients. In the following two or three years, during my service in January, February and March of each year, I had the children, for a number of hours each day, unless it was raining or snowing, carried down to the large open space facing the hospital buildings, and kept in small hammocks. These infants were always bundled up in blankets, their heads well covered, and their eyes, nose and mouth covered with a gauze veil. They did remarkably well; so well, in fact, that instead of my infants dying most of them began to gain in weight and health, and the deaths were almost entirely in infants under three months of age, whose condition was distinctly bad upon admission to the hospital.

During the past eleven years the infants have been placed in the new modern and up-to-date building of the Philadelphia Hospital. The wards are large, the air space ample, the milk the very best; porches surround the hospital on two sides, the infants practically have an abundance of fresh air day and night, and they do as well as could be hoped for. I am no longer a pessimist when in the infants' ward, but an optimist. In the new Jefferson Hospital, where I am on duty the entire year, the children have an ideal ward on the eighth floor, large windows on three sides with a large roof garden adjoining, the latter fitted up with every convenience, such as hammocks, shade, wind shields, etc. The roof garden is used all the year round, winter and summer, and the results are most encouraging.

Of all the factors which have contributed to the reduction of this infant mortality, fresh air has, in my opinion, been the one of prime importance.

During the last nine years in my service in the Philadelphia Hospital, I have treated all my severe cases of broncho- and lobar pneumonia in children of all ages by the fresh air method. These infants and children, as soon as taken ill, are removed from the general ward and placed in a special room provided for such cases. The room holds six cribs comfortably, and rarely, during my service in the last three years, has a bed been vacant. Many of these cases are secondary bronchopneumonias. A large percentage of the children, in fact most of them, are hospital children, but the results have been so different from those following the methods of years ago, that I have lost much of my dread of pneumonia as a hospital disease. True, these cases have good nursing, good care and every possible attention, but still they are cases of pneumonia in hospital practice and they do remarkably well.

In another similar room in the same hospital, during the same period of time, I have been treating my typhoids. Occasionally, I have a typhoid under two years of age, most of them are over three, and the average age about six years. The typhoid cases come from the same class of patients as the pneumonias, and they do so much better in the fresh air that nothing could persuade me to return to the old method of treatment, similar in every way to the present, but *minus* the fresh air. The rooms where the typhoids and pneumonias are kept have large windows on two sides, and an open door on a third side; these are kept open day and night; a distinct effort is made to keep the hands and feet of these children warm by gloves, stockings and hot-water bags, but the rooms are always cold, except for a few minutes every two or three hours, when the windows are closed and all the children carefully examined as to cleanliness, etc.

The few cases of tuberculosis are kept in a special portion of one of the porches, living practically in the open air. Arrangements are just being made for a special pavilion for these cases. In the Jefferson Hospital the pneumonia and typhoid cases are treated by the fresh air method, and do infinitely better than formerly, when the fresh air treatment was not employed.

In private practice for eleven years I have treated all infants and children, sick and well, with fresh air; in fact, on my first visit to the child, I try to instill into the mother's mind the principle that fresh air differs from impure air as much as fresh milk from impure milk. Once gain her consent, and the treatment is an assured fact. I am more than surprised to see the willingness with which most mothers, no matter what their social status may be, enter into the treatment. The physician must be enthusiastic; the mother is, in my experience, readily convinced.

During both the winters and the summers of the past eleven years, I have kept numerous infants out of doors all day long, except bringing them in occasionally to the house for the purpose of removing soiled clothing, and I have never in all that time seen a single injurious symptom result, and I am positive it has been the means of saving many lives. Rickets, scrofulosis, gastro-intestinal disease, any and all conditions are benefited by the treatment. Measles, in hospital cases, I treat in separate rooms, each room opening upon a common porch, surrounded by glass. During the first few days the children are kept in the cool, darkened rooms, then moved to cribs on the porch; the windows on the porch are opened more or less, insuring an abundance of fresh air. The influence of climate associated with rest in the treatment of nephritis and cardiac disease is now so well recognized that comment is unnecessary.

It is an interesting question, which experience alone can decide, as to what arrangement will, in the future, be made to control the *degree* of temperature in which these children are kept when in the fresh air. In hospital practice it has been my custom to keep infants under three months for a few days in the cool air of the ward before putting them outdoors—the temperature of the ward usually being near 60° F. in the cooler months. In private practice, where the temperature can be more absolutely controlled for each individual case, I gradually but rapidly lower the temperature of the room to 60° F., then, dressing the child exactly as if it were to be taken outdoors, gradually lower the temperature of the room for a few hours each day until it approximates the degree of temperature in the fresh outside air. Indifferent temperature feels neither warm nor cold (Wachenheim), and is most restful. In children, indifferent temperature is above 75° F. in summer in summer clothing, and above 65° F. in winter in winter clothing, and varies with age and vitality. Temperatures above indifferent are not sedative, but cause a continuous stimulation that is harmful if prolonged, ending in exhaustion. The same author also says, "Young children stand severe cold badly." This is not my experience, if by "severe cold" is meant the ordinary winter weather in Philadelphia. True it is that these children are bundled up from head to foot, lie in a comfortable baby coach, and have thick gloves, stockings, veil and perhaps a hot-water bag, but they do breathe the cool air, and they all do well. Two or three rainy or snowy days will convince any one; the children are kept indoors in a warm room and they fuss and cry; out of doors they are quiet.

Humidity has an influence by checking or increasing the evaporation from the body, and further studies along this line will be of value.

The distance above sea level exerts a certain distinct influence upon the skin, kidneys and blood, and induces metabolic changes of importance. The influence of a few months of camp life on growing boys is appreciated by every one. "Camp life" is fresh-air treatment.

When it is possible to select the kind of fresh air desired, certain broad lines may be followed. Increase of heat production, and, consequently, an augmented metabolism, are rendered necessary by cold, dry air of high altitudes. This is proven by the larger amount of carbonic acid gas given off by the lungs. It also, as a rule, increases the red blood cells during the first few weeks of treatment. High altitudes are good for children with incipient tuberculosis or an inherited tendency to tuberculosis.

Fresh seashore air is of decided benefit in infants and children convalescing from severe illnesses, especially gastro-intestinal in type. It is of distinct benefit also in the so-called strumous type.

Fresh country air is better than fresh city air. Rural districts are better than urban; but density of population, such as one sees often in large cities, does not necessarily imply lack of fresh air. The number of people living in a given area may be very large, but if they live in comfortable houses, keep the windows open, and live under the best hygienic conditions, fresh air can be secured in abundance, and infants and children do well. If the same number of people living in the same area do not have an abundance of fresh air, the infants and children do badly. Density of population may have much or little to do with fresh air.

Statistics prove conclusively that in *all* countries where the mothers work in industrial plants, necessitating their absence from home a large portion of the day, the infants and children show a much higher mortality rate, owing to the fact of their being kept indoors, than is found among the children of the same class of people living under exactly similar conditions *except* that the mothers live at home and have time to keep their children in the fresh air.

In Berlin, 1903, Newman investigated 2701 infant deaths. Where the families were in one-room dwellings he found 1792 deaths; in two-room dwellings, 754 deaths; in three-room dwellings, 122 deaths; in larger dwellings, 43 deaths. Can anything prove more conclusively than this the power fresh air has to preserve life, or the rapidity with which bad or impure air can cause death? Unfortunately for the infant and young child, the ignorance of many mothers, the superstitions and traditions of others, and the carelessness of a few, are the greatest barriers to the keeping of children in the fresh air.

During the past few years much has been written upon the importance of fresh air for very young children, and the subject of fresh air as an aid in the treatment of disease is not of recent date. In the History of the Medical Society of the State of New York, as published in the *New York State Journal of Medicine*, it is shown

that in the early part of the nineteenth century the dangers of dust-laden air were recognized; the influence that certain occupations exerted upon the etiology of tuberculosis was appreciated, and even at that date "cold air" was used in the treatment of typhus fever.

In one of these essays upon "The Influence of Trades, Professions and Occupations in the United States on the Production of Disease," the author shows clearly how the crowding together of children in the tenement districts produced gastro-intestinal disease and death, proving that at this distant period the virtue of fresh air was appreciated.

In 1850 to 1860, Dr. Clark treated a very large number of cases of typhus fever in Bellevue Hospital by the fresh air method. The windows were removed; in winter stoves were placed before the open spaces to insure a slight heating of the air, but the patients were given the fresh-air treatment, as we understand it in the fullest sense today. The results were vastly superior, the death-rate very markedly lower than the mortality among the same class of patients in the same hospital at the same period in the hands of the other members of the staff where fresh air was not used. It is a well-known fact that in times of war patients treated in the fresh air of tents always do better than those confined in hospitals.

The phenomena of child life have often occupied the attention of psychologists, and new theories for children are formed every day by educators. Theoretically, they are making the superchild, soon to be the father of the superman. We, however, should be quite content if parents could be taught to appreciate the advantages accruing to the child from correct feeding, combined with fresh air, and the influence they exert upon the mental and physical development of the growing child.

Everyone should be made to understand how important it is for the very young to be taught how to stand, lie down, and sit properly, and that deep breathing is the proper and only sure way to secure full lung expansion.

Let us all join hands and preach fresh air; vote for open squares, endorse roof gardens, have adenoids and tonsils removed, and, if we are willing to endorse and work for the fresh air treatment with the same zeal and enthusiasm as that with which we have worked for fresh and pure milk, our results will be as great a success as has been secured by our milk enthusiasts.

DENTITION.

THE views held several decades ago, concerning the part which dentition plays as a causative factor, either direct or indirect, in the diseases of children, have undergone a radical change, and much found in the earlier literature must today be rejected. Many of the writers of that period considered the eruption of the milk-teeth to be influential in producing nearly all of the diseases which attack children. The claim was made that at least 25 per cent. of deaths occurring during the first two years of childhood were due to the cutting of the temporary teeth.
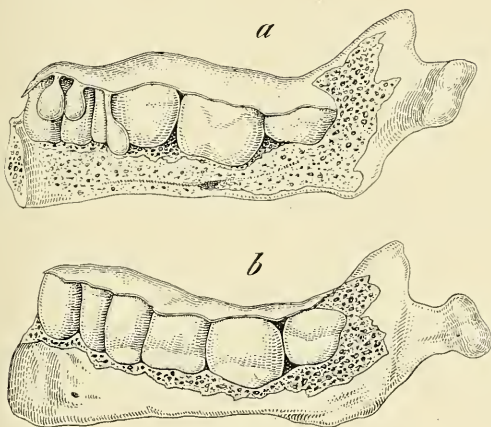
FIG. 25.—Inferior maxillary bone of a child one day old; portion of bone removed in order to show the dental sacs. *a*, left half of lower jaw; shows the sacs of the milk-teeth, of the first permanent molar, and of the permanent incisor and canine teeth; *b*, shows the first permanent molar, and the sacs of the milk-teeth.

Old-time theories and superstitions concerning teething still exist among the laity, both the well-to-do and the poor, and the physician today is often summoned, perhaps too late, to attend children seriously ill with gastro-intestinal disorders, or suffering from dangerous nervous affections, such as convulsions, the parents believing that the illness, being due to teething, is of comparatively little importance. It is obviously the duty of every physician to correct this popular but erroneous belief in the widespread influence of dentition as a direct cause of disease; for, until this is accomplished, hundreds of children will annually die from neglected diseases—diarrheal disorders especially—many parents believing that mild, or even moderately severe, diarrhea during the period of teething is distinctly beneficial.

As opposed to the former belief that all infantile disease was due to teething, it is interesting to note that some of the most advanced clinicians of today emphatically deny that dentition can, in itself, set up any distinct morbid condition. Teething, they claim, is simply a physiological process that is normal in the human body, and therefore should not be regarded as a pathologic factor.

Many of these physicians admit that during the period of dentition there exists a predisposition to illness, and I am quite in accord with this view. After careful investigations, all recent observers agree
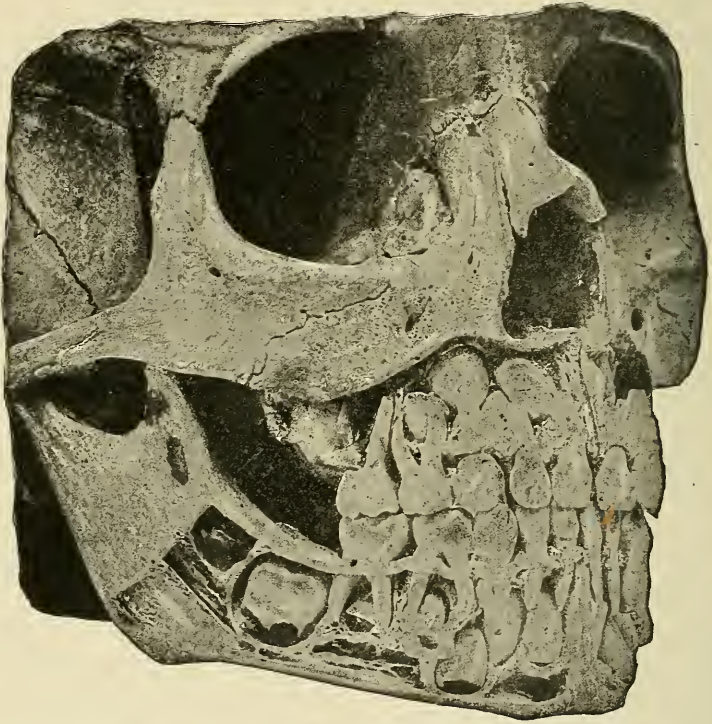
Fig. 26.—The milk-teeth in a child aged about four years. The permanent teeth are seen in their alveoli. (Cryer.)

in ascribing fewer and fewer symptoms to dentition; and, from the standpoint of both the safety of the patient and the reputation of the physician, it is well to examine the child's whole body carefully before making a diagnosis of "teething." This is especially important when the symptoms are not local, but remote, as it is often difficult, especially when no distinct cause other than teething is apparent, to give to dentition its proper causative significance.

Even if the gums are tense and swollen, and no other adequate cause can be detected, it is not wise to be too hasty in ascribing to dentition a positive causative influence; since such cases, after a few

days, often clearly show their pulmonary, cerebral, or other origin, the process of dentition being merely coincident, or, at the most, it intensifies the symptoms and does not produce them.

Dentition occurs in that period of life when physical development is most rapid and functional activity highest. The nervous system is in a preparatory condition; the follicular apparatus of the intestines is rapidly developing, and almost all of the organs and tissues of the body are undergoing change. During infancy and early childhood the mucous membranes are especially vulnerable to disease; hence the frequency of stomatitis and other digestive and diarrheal disorders.

If we bear in mind that, during this early period of dentition, the child is weaned, and is commonly given food more or less unsuited to its digestion, the development of diarrhea in its various forms seems obvious. In considering the frequency with which dentition causes or complicates disease, the attending circumstances must be carefully weighed, and cool judgment is necessary.

The effects of teething are governed largely by the child's physical condition. If healthy, children often pass through this critical period without any symptoms referable to the eruption of the teeth. If they are weak, rachitic, or suffering from malnutrition, inherited syphilis, scrofula, or a neurotic temperament, either inherited or acquired, the period of teething is one of distinct danger, and is often accompanied by pyrexia, with heat, swelling, and redness of the gums, stomatitis, and gastro-intestinal disorders.

In delicate children dentition may, and often does, aggravate the symptoms of any existing disease, and to a certain extent this is true also of breast-fed healthy infants. Where there is a profound constitutional disease, either inherited or acquired, the disorders which appear during teething may be traced to weakened vitality produced by disease, and not to the teething *per se;* for example, children who are constitutionally weak, and who, on the slightest febrile irritation produced by the erupting teeth, are rendered unable to digest the food best suited to their nourishment when well, the fever having temporarily weakened their digestive powers.

As early as the seventh week of intra-uterine life, the beginning formation of the milk-teeth can be detected. Fig. 26 represents the dental sacs exposed, as they exist in the lower jaw of a child at birth. Fig. 27 depicts a part of both jaws of a child about four years old, and shows the relation of the temporary and permanent teeth.

The formation of the hard part of the tooth begins quite early, thin caps or shells of dentin being found on all milk-teeth at the end of the fourth month of fetal life, when the coating of enamel begins to be deposited. It is, therefore, evident, as pointed out by Forcheimer, that the process of teething is in great part accomplished before the child is born.

The calcification of the fangs, and the increasing size of the tooth, forces the crown toward the mouth, and perforation of the gum

follows. Many observers claim that other factors are concerned in the eruption of the milk-teeth, but the elongation of the fang is certainly the chief one. For details as to the development of the teeth, and theories concerning the forces that are operative in the advance of the crown toward the gum, the reader is referred to works on dental surgery.

The usual time and order of eruption are as follows: The two lower central incisors appear at the age of six or seven months; the upper central incisors between the seventh and ninth. During the tenth month the upper lateral incisors usually erupt, and the lower lateral incisors between the eleventh and twelfth. Well-developed breast-fed infants usually have cut all of the incisors at the age of one year. The four anterior molars appear between the twelfth and sixteenth months; the canines from the sixteenth to the twentieth, and the entire set of milk-teeth, twenty in all, should have erupted by the time the child is two and a half years old.

These figures represent, however, only average periods, and a delay of a month or more in the appearance of the first teeth, or even of several months in the appearance of the later ones, may happen in the best-developed and most healthy children. The teeth are usually cut in groups or pairs, a considerable period intervening between the eruption of each group, and the teeth of the lower jaw, as a rule, preceding those of the upper by a few weeks.

This normal order of eruption often shows variations, and the upper incisors may appear first, especially when dentition is delayed. The central incisors may follow, instead of preceding the lateral; the posterior molars may precede the canines, or the anterior molars may even be the first to erupt. Rickets is commonly found in cases where the teeth appear irregularly.

Absorption of the milk-teeth begins at the extremity of the fangs, and progresses toward the crown, the teeth disappearing in much the same order as they appear. The fangs being absorbed, the crown becomes detached either by the advancing permanent tooth or by the accidental pressure to which the slightly attached tooth is necessarily subjected.

Dentition may be abnormal either in being premature or delayed; when premature, it is, in my experience, usually easy. Dentition beginning at five months is not uncommon, and, in rare cases, the first tooth may appear at sixteen or even fourteen weeks. Henoch mentions two cases of premature dentition at six weeks and thirteen days respectively; but such cases are medical curiosities.

Occasionally one tooth, and even two teeth have been noticed at birth. These natal teeth are usually the incisors of the lower jaw, and if poorly formed, consisting largely of crown with little or no fang, as is generally the case, they should be removed; for, if allowed to remain, they are likely to injure the mother's nipple and the frenum of the child's tongue. In a case which recently came under my notice, such an injury to the mother's breast necessitated the removal of two

lower natal incisors. Natal teeth, if fairly well formed and firmly attached, should certainly not be interfered with, unless they cause distinct disturbance or become loose.

Delayed dentition is, however, quite common, and indicates that the physical condition is below par. In this connection it is interesting to note that acute disease, if not followed by marked and persistent constitutional symptoms, has little or no effect upon the time when the teeth erupt. Artificially fed children, however, usually cut their teeth later than the breast-fed, and a delay of one, or even two months, is in such children not unusual. Cases in which dentition is delayed until the twelfth or sixteenth month are not uncommon, and Jacobi reports the case of a boy who had cut no teeth when two years and ten months old.

Imperfections in the enamel and early decay are quite common in cases of delayed dentition, and are due to the fact that, the constitution being below par, the enamel or dentin is imperfectly formed. This imperfect formation of enamel and dentin and early decay are regarded by Mr. Hutchinson as in some cases due to infantile stomatitis. Retarded dentition is usually, but not necessarily, connected with rachitis; and, while delayed dentition points strongly to rickets, it must be borne in mind that the two conditions are not always associated.

Children in whom dentition is delayed should undergo a thorough physical examination to ascertain whether they have any acquired or inherited disease. The diet, hygiene, clothing, bathing, and general care of the infant should be carefully looked into. For internal treatment, the two remedies that best promote dentition are cod liver oil and iron. All local measures are useless.

Any influence which rickets may have upon the time of eruption depends upon the age at which distinct rachitic symptoms develop. If the disease does not manifest itself until the end of the first year, the earliest teeth may appear at the normal period, and a long interval, during which dentition is arrested, may follow the symptoms of rickets. Teeth that appear during the rachitic period are usually deficient in enamel, and decay early.

When dentition is delayed, many of the symptoms usually attributed to teething are caused by the associated condition, *i. e.*, rachitis, with its inevitable malnutrition. In this connection Finlayson says: "The diagnosis of teething diverts the mind of every one concerned from the vital points of food, air, and hygiene; the processes leading up to rickets are largely under control, even in the cases of those who are comparatively poor."

Occasionally the full number of milk-teeth does not appear. This may be owing to a family peculiarity, since the influence of heredity can often be traced in the history, or it may be the consequence of destruction of the tooth-germ. Absence of one or more of the milk-teeth apparently has no effect upon the number or appearance of the permanent teeth, notwithstanding the fact that teeth more often fail

to appear in the second than in the first dentition, an excess in the
number of milk-teeth being more common than a deficiency. An
extra incisor is probably the most frequent form of this abnormality.
Cases of multiple dentition, *i. e.*, the appearance of more than two
sets of teeth, have been reported.

Irregularity in the position of the teeth is more common in the
permanent than in the temporary set, but is usually limited to a slight
overlapping of the incisors or a twisting of the canines on their axes.
This malposition is mainly due to a disproportion between the size
of the jaw and the space required for the developing teeth, the jaw
usually being undersized. It is claimed that rickets favors this lack
of development of the maxillary bones.

By almost continuous pressure upon the upper incisors, thumb-
sucking may cause a characteristic deformity. Vigilance on the part
of the mother or nurse, and, if necessary, binding the hand in a splint,
or wrapping the thumb in a small bandage saturated with some harm-
less but intensely bitter preparation, will usually correct this injurious
habit.

Lip-sucking is another, but less common, cause of depression of
the lower incisors. It consists in pressing the upper incisors against
the lower lip and thus forcing the lower lip against the lower
incisors.

The teeth may be poorly formed, in that either their structure
or their actual shape is defective. Deficiency in lime-salts and imper-
fections in the enamel, as shown by pigment spots, furrows, or pittings,
are powerful predisposing causes to early decay, which can be averted
only by cleanliness and watchful care.

Hutchinson's teeth are characterized by deformity of the upper
central incisors, the cutting edges of which are notched and crescentic
in shape, owing to changes in the early formation which are due to
inherited syphilis. This notched appearance is positively significant
of inherited syphilis only when found in the permanent teeth; in the
milk-teeth it often has no special significance. In inherited syphilis
both sets of teeth may be affected similarly.

Teeth of poor shape, often showing deep fissures, offer favorable
sites for the retention of secretions which may rapidly become the
breeding-ground of countless microörganisms, and thus predispose
to caries. Imperfections in the structure of the milk-teeth usually
indicate lowered vitality or a diseased state of the system during
intra-uterine or early infantile life.

As regards the role of dentition in producing definite symptoms or
conditions, it has already been pointed out that the first duty of the
physician is to examine the child thoroughly in order to determine
whether the symptoms may not be due to some cause other than
teething. The family and the personal history of the little·patient
should always be elicited, the latter including such facts as normal or
difficult delivery, the method of feeding, the age at which dentition
began, and the history of all preceding diseases.

Restlessness, peevishness, disturbed sleep, and moderate fever are symptoms commonly met with in children during the teething period. It will often be impossible, even after the most careful physical examination, to account for their occurrence unless we accept dentition as a cause; but the fact that these symptoms are much more common in weak and sickly infants than in the healthy, and that they do not accompany the eruption of every tooth or group of teeth, confirms the belief that dentition may occasionally produce such symptoms in a healthy child, and very frequently causes them in those whose powers of resistance are weakened by acquired disease or hereditary taint.

Drooling, which is said to precede teething, and to result from reflex stimulation of the salivary glands, is, in my opinion, mainly due to the normal establishment of the secretion of these glands, and merely betokens a stage in the development of the digestive system of which the salivary glands constitute an important part.

Biting of the fingers or of any hard substance that can be carried to the mouth is common during dentition, but it is due to the fact that an infant naturally carries everything to its mouth rather than to pain or tenderness of the gum. It expresses mild uneasiness or itching of the gums, not actual pain, since neither pain nor tenderness would be relieved by rubbing or biting on hard substances. A healthy child is, when awake, almost always in active motion, and the movements of the muscles of mastication may be, and probably are, often but a part of its general muscular activity.

In a well-developed, healthy baby the gums are of a pale pink color, and change little in appearance except that they become distinctly elevated as the tooth approaches the surface. Occasionally there is considerable redness as well as heat, and in such cases incision is followed by a slight oozing of dark-colored blood. It seems quite improbable that, in this portion of the gum which lies immediately over the advancing tooth, there can be any severe pain, since it is usually paler in color than the surrounding similar tissue, or, at most, is only occasionally slightly congested. It is much more reasonable to believe that if, as is true, the sensitive pulp and bony fang are absorbed without pain, the small portion of the gum overlying the advancing crown will be similarly disposed of.

It is claimed that dentition is a common cause of gingivitis and catarrhal stomatitis; but these conditions are rarely, if ever, found in healthy, breast-fed infants if the oral mucous membrane has been kept clean. When, in bottle-fed babies, the care of the bottles and nipples and of the mouth of the child, as is pointed out in the chapter on Infant Feeding (page 183), has been neglected, gingivitis and stomatitis are of common occurrence. They are due, however, not to dentition, but to carelessness on the part of the attendants. The irritation occasionally produced by the crown of the milk-tooth at the time of its detachment may, however, be sufficient, if strict cleanliness is not observed, to produce stomatitis or even local ulceration.

On slight exposure the bronchial mucous membrane of children

is easily excited to a mild degree of inflammation, and it is a well-known fact that between the ages of one and three years bronchial and pulmonary diseases are extremely common. Personally, I have never been able to trace any connection between these diseases and dentition.

The skin of a child is so delicate and so easily irritated that an explanation for various skin affections has naturally been sought in dentition, and its etiologic influence sturdily maintained. Lack of cleanliness, unsuitable food, digestive disturbances, improperly given baths, and too tight clothing, also inherited and acquired disease, are all powerful factors in causing skin diseases in children. When these causes are carefully considered there can be no reason for ascribing cutaneous disease to dentition.

Enlargement of the submaxillary lymphatic glands is generally due either to some local cause in the mouth which is probably the result of lack of cleanliness, or to one of the forms of stomatitis. In babies who suffer from scrofula, teething may occasionally be an exciting factor; but in healthy little ones it rarely causes any marked glandular enlargement.

In children of strumous diathesis, corneal ulceration is occasionally met with, especially during the eruption of the upper canines, or when the canines become carious. I have never known it to result from dentition in a healthy child. Conjunctivitis is said to be one of the many results of teething; but careful examination will usually demonstrate its dependence upon some other cause; and, as with corneal ulcer, I have never seen it in a healthy child when it could be traced to dentition.

The most common, and also the most dangerous, fallacy in regard to teething is the belief that during this period of tooth-cutting diarrhea is not only harmless, but even beneficial. It is claimed that the secretory activity of the glandular system of the intestinal tract is greatly stimulated, and normal peristaltic action greatly increased, by reflex irritation transmitted through the sympathetic nervous system from the gums to the vagus. It has also been maintained that diarrhea is often excited by the large quantities of saliva swallowed, this secretion being stimulated by the irritation of the approaching teeth.

Digestive and diarrheal disorders owe their origin (as is pointed out elsewhere in this work) to entirely different causes, and I am convinced that diarrhea is never produced in a healthy child by teething alone. When a child suffers with diarrhea, the slight irritation occasionally present from teething may, by increasing the fever and restlessness, aggravate the existing gastro-intestinal disturbance, but is not sufficient to originate this.

Teething has been regarded by many as a not uncommon cause of acute purulent otitis media. Such a conclusion must, however, be based upon the supposition that considerable inflammation, suppuration, or dental caries is present. If either one of these conditions exists it may cause inflammation of the ear; but, as one rarely finds

more than a slight redness or blueness of the gums, it appears most unlikely that dentition can play any important role in the causation of ear disease. In the past, inflammation in the external auditory canal has been ascribed to teething, but it is questionable whether reflex dental irritation is, in itself, ever sufficient to induce this condition.

The opinion that dentition is the most common cause of infantile eclampsia has never been disputed until recent years. There is no doubt that a considerable number of all cases of convulsions in children are reflex in origin, and that extremely slight causes may, in neurotic children, bring on eclampsia. It is, however, very doubtful whether dentition *per se* ever causes convulsions in a well-developed, healthy child. Certainly no such case has ever come to my notice.

It has been claimed, too, by prominent clinicians, that dentition may, in peculiarly nervous children, predispose to reflex disturbances and be the cause of convulsions, but recent investigations show that this must be rarely, if ever, the case. Careful study will usually disclose some more potent causative factor.

Caries of the temporary teeth may set in soon after their eruption, and is most commonly observed in the poorly nourished and bottle-fed, or those whose oral cavity is not kept scrupulously clean.

Decay proceeds more rapidly in the temporary than in the permanent teeth, often causing severe pain, especially when liquids, either hot or cold, are taken into the mouth. Temporary relief is usually afforded by plugging the cavity with a small piece of cotton saturated with chloroform, laudanum, or oil of cloves. All cavities should be filled with an alloy or gutta-percha in order to relieve the pain and preserve the teeth until the time when Nature intends them to be absorbed.

Much can be done by cleanliness to preserve the temporary teeth from caries, and during early infancy they should be cleansed every morning and evening with a soft piece of linen or muslin wet in sterilized water. After the teeth have pierced the gum, a soft tooth-brush should be used. All tartar formation should be at once removed from the teeth by gently rubbing them with a tooth-pick of soft wood dipped in powdered pumice stone.

If we consider teething as a process that rarely produces marked symptoms in healthy children, and a factor that, in delicate or sickly infants, may and occasionally does indirectly aggravate symptoms resulting from the child's condition or some disease, we cannot consistently believe in any treatment for dentition *per se*. Proper diet, bathing, clothing, sleep, and hygiene for the child, and the prompt treatment of any existing disease or morbid condition, constitute the treatment of dentition.

If we believe in the far-reaching effects of dentition as a cause of disease, lancing of the gums is naturally a remedial measure of great importance. If, on the other hand, we do *not* believe in this influence of dentition in producing disease, lancing is uncalled for, usually does

15

no good, and may, by diverting the attention of the physician away from the actual cause of the symptoms, do much harm.

It rarely, if ever, hastens the eruption of the tooth through the gum; indeed, through the resulting scar, it probably retards the onward progress of the tooth. Occasionally by relieving the congestion in the gum overlying the advancing tooth it may be of temporary service, owing to the slight loss of blood which follows. The physician who carefully examines his patients has little use for the gum lancet.

RICKETS.

RICKETS, or rachitis, is a constitutional disease of early childhood caused by disordered nutrition. It is characterized by developmental changes in the bones and cartilages which result in typical deformities, also by changes in the muscles, ligaments, nervous system, and many other parts of the body.

Etiology.—Rickets is essentially a disease of infancy, therefore most commonly seen in children under two years of age. In the majority of cases it appears after the sixth month, although it may be congenital, and it never occurs after the skeletal development is complete. The children of the poor in large cities are especially prone to the disease, children of the well-to-do being rarely affected, and then only lightly, although if these children are fed on proprietary foods, which usually contain an excess of carbohydrates and a deficient amount of fat and proteins, they may, and often do, show slight or marked rachitic changes. Rickets is most uncommon in the country.

Lack of hygiene is one of the principal causes of the disease, and in congenital rickets it is difficult to estimate the hereditary influence when both mother and child have been subjected to the same unhygienic surroundings and lack of proper nutrition. The health of the father is not believed to exert any influence upon the occurrence of rachitis except in cases of paternal syphilis, tuberculosis, or chronic alcoholism. The important predisposing factors which act upon the mother during pregnancy are alcoholism, tuberculosis, syphilis, scanty nourishment, lactation, and close indoor confinement.

Faulty nutrition is by far the most potent factor in the production of rickets, the disease being especially common in bottle-fed infants. Nurslings, as a rule, rarely show more than the milder manifestations of the affection, unless lactation is unduly prolonged to eighteen months or two years, or the mother again becomes pregnant, or is attacked by some chronic wasting disease which impoverishes her milk. The diet of artificially fed babies who are rachitic is usually too rich in carbohydrates, but poor in fats and proteins; and the impoverished milk of many of the women in the tenement districts, if analyzed, would be found to have a low protein and fat content.

Proprietary foods for babies, as a rule, contain an excess of carbohydrates, but are deficient in fats and proteins, and rachitis is not uncommon in those children who during infancy were fed on one or another of these preparations, or exclusively on condensed milk.

It appears, on the whole, that rachitis is more severe and develops much more readily when there is a lack of fats and proteins than when

there is a deficiency in fats alone; also that if, in addition to these errors in diet, there is an excess of carbohydrates, rickets is even more likely to result. In support of these theories that certain food changes are responsible for its development, may be cited in the following observations:

Bland Sutton, experimenting with lion whelps at the London Zoölogical Gardens, showed that if they were weaned early and fed entirely on raw meat they soon became rachitic, and that the rachitic changes were most marked when an excess of carbohydrates was given. If, however, a diet of powdered bones, cod liver oil, and milk was substituted, they quickly recovered. Chossat, in 1842, demonstrated by animal experimentation that rachitic changes appeared in the bones when lime was excluded from the diet. Heitzmann claims that if lactic acid is introduced into the food of young animals rickets will develop.

But, even though lactic acid forms a soluble salt when it unites with the calcium in the bones, thus eliminating lime from the system, this does not explain the various other abnormalities in the bones which are observed in rickets. Young animals fed upon a strictly vegetable diet also become rachitic, which fact further emphasizes the need of sufficient fat for the growing osseous system.

In all the foregoing experiments, whenever rachitic symptoms and signs developed, these could be made to disappear by so altering the diet that the fats and proteins were increased and the carbohydrates diminished without making any change in the living conditions; which seems to show how much more important is diet than hygiene in the etiology of rachitis. Sex has no influence on its occurrence, since boys and girls are alike subject to the disease.

The geographical distribution of the disease has been quite definitely worked out; and, although rickets occurs everywhere, it is most common in the temperate zones, the majority of cases being observed in large cities. It is more prevalent in Great Britain, Germany, Russia, and Italy than in the United States, is rare in the tropics, and infrequent in Iceland, Greenland, Denmark, and Norway.

Nationality and race are also important factors in the occurrence of rachitis, since the great majority of cases in the United States are seen in negroes and Italians. Practically all negro children exhibit some signs of rickets, and it is thought that the change of climate from a southern to a northern latitude may be largely responsible for this; since, on the whole, the diet of the negro does not differ materially from that of other races in the large cities, and rickets is uncommon in Africa.

Illnesses, such as chronic gastro-intestinal disorders, may precede rickets, and syphilitic children may show rachitic changes; but, aside from these influences which so impair the nutrition that the child cannot assimilate sufficient fats and proteins for the needs of the body, associated diseases have but little bearing upon the production of rachitis in children,

Most cases of rickets are seen during the winter months, which is thought to be due to close confinement and lack of exercise at this season of the year, especially among the poorer classes. Findlay demonstrated in animals that lack of exercise alone will cause rickets, even when plenty of fresh air is provided.

The chief cause of rachitis is now universally recognized to be faulty diet; and, although the precise way in which rachitic changes are brought about is not clearly understood, there is little doubt that the assimilation of calcium salts is in some manner interfered with. Mircoli attributes rickets to the action of ordinary pyogenic bacteria upon the bones and nervous system.

Pathology.—In the normal child the bones grow longitudinally by the production of bone in the cartilage between the diaphysis and the epiphysis, and their thickness is increased by the production of bone by the inner layer of the periosteum. In rachitis, however, owing either to chronic inflammation or simply to disorders of nutrition, this process of growth is altered and becomes abnormal, inasmuch as an excess of cartilage is produced, but very little mineral salts are deposited, which completely arrests ossification.

The cells produced by the inner layer of the periosteum also fail to ossify, and there is frequently abnormal absorption of the medullary layers of bone within the canal. These conditions tend to produce a much softer and more vascular bony structure than normal, which, owing to the lack of ossification, is very flexible and weak. The bones of these children sometimes contain twice as much animal as mineral matter, and ossification is so irregular that areas of bone may be found scattered throughout the cartilage and the bony parts may be infiltrated by islands of cartilage.

Deformity is most marked in the long bones, because of lack of ossification in the external layers of the shaft and increased bone absorption in the medullary cavity, which makes the bony shaft very thin. The increased proliferation of cells at the epiphysis results in a most constant and characteristic change in the form of the bones, namely, enlargement of the ends of the long bones, especially noticeable at the wrists and ankles, which may be half as large again as in normal children. Other characteristic enlargements occur at the costal ends of the ribs.

In addition to these bony changes, pigeon-breast and scoliosis are common. The abnormal shape of the softened bones is brought about, for the most part, by the weight of the body while the child is in different attitudes, and is most marked if it is allowed to walk, or even to sit up unsupported, too soon. In some instances the bones break instead of bending, but the fracture is usually of the green-stick variety.

The flat bones of the head and pelvis exhibit changes somewhat like those found in other bones of the body. The external surfaces are soft, porous, and extremely vascular. At the centres of ossification are produced large bosses which are soft and spongy. In other areas

of the flat bones there may be but a thin membrane in place of bony
structure. When longitudinal section of a large bone is made, the
epiphyseal junction may be outlined by its bluish color; it is very
vascular, softer than normal, and reveals attempts at calcification
nearest the head of the bone. The amount of cartilage at the end of
each long bone is four or five times greater than normal, and the
centres of ossification are larger and more vascular than normal.

On observing a cross-section of the shaft of a long bone, the inner
layers are found to be quite firm, and most of the decalcification is
in the external layers of the shaft. The medullary cavity appears to
be more porous than normal, and more vascular. If a section is made
through a boss on one of the flat bones, it is found to be soft, spongy,
and highly vascular, and the ordinary arrangement of the outer and
inner tables of the skull, separated by the intervening diploe, is lost.

Aside from the changes in the osseous tissues, there are also certain
pathological changes in the viscera. Among these enlargement of
the spleen is, perhaps, the most common, and is the result of hyper-
plasia of the splenic pulp and follicles. In addition to an increase in the
connective tissue within the spleen, the capsule is also thickened by a
fibrous perisplenitis.

When a section of the spleen is examined microscopically, atrophic
changes can be detected in the Malpighian bodies, the arterial blood-
vessel walls show thickening, and the whole organ looks pale and
anemic. In more than 50 per cent. of children with rickets an enlarged
spleen can be demonstrated; in exceptional cases it may be more than
double the normal size. Enlargement of the liver is less frequently
observed, not being so marked as splenic enlargement, with which it
may or may not be associated. It is also due to simple hyperplasia
of the connective tissue.

There are hyperplastic changes in the lymph nodes of the body
which render them distinctly palpable; but they do not become as
large as in syphilis and tuberculosis. The muscles of these children
show a lack of tonicity, and imperfect and defective striation due to
lack of nutrition and disuse.

The microscope reveals in the sectioned muscle excessive nuclei,
a distortion of the longitudinal striæ, and thin imperfect fibers. When
deformity of the chest is extreme, the lungs are often furrowed by the
pressure of the deformed ribs; the pressure also causes partial or
complete atelectasis in these areas, and a compensatory emphysematous
condition in the adjacent lung tissue. Acute and chronic bronchitis
and prolonged bronchopneumonia are also common findings in the
respiratory tract, owing to the general debility from malnutrition.

The mucosa of the stomach and intestines usually gives evidence of
more or less chronic catarrhal changes, and a mild degree of dilatation
not uncommonly results from the atonic condition of the gastric and
intestinal musculature. Mild hydrocephalus is sometimes observed
in rachitic children; but the exact relation between these two con-
ditions is not clear. Hyperplasia of the brain has also been described

in connection with rickets, but its relation and significance are as yet undetermined and obscure.

Histology.—Upon microscopical examination of the end of a long bone we see, next to the hyaline cartilage, a layer of proliferating cartilage cells and a highly vascular matrix in disordered arrangement. Beyond this are columns of hypertrophied cartilage cells in regular order, then a zone of calcification, finally a zone of ossification.

Rachitic changes are most prominent in the layer of proliferating cartilage cells and in the columnar area, the proliferating layer being thickest in fetal rickets, and the layer of columnar arrangement thickest in extra-uterine life. Because of its excessive thickness, the epiphysis is compressed and bulges laterally, causing the typical rachitic deformity. The increase in cartilage cells and in vascularity, which is most marked in the columnar layer, causes the characteristic changes in these two zones, while the area of ossification shows deficient calcification, and the cartilaginous areas jutting into it make the outline of the zone of ossified cells broad and irregular, instead of narrow and sharply defined.

The periosteum is greatly thickened, much more vascular than normal, and strips readily from the bone, revealing beneath it the excessive cell proliferation which sometimes causes such great thickening of the bone that in places the medullary cavity is impinged upon. The changes in the flat bones are practically the same as those in the long bones, and consist chiefly of increased cell production, increased vascularity, and imperfect calcification.

They are even more pronounced in the spongy bones; and, in addition to these changes, the spongy bones contain large medullary spaces filled with bloodvessels and cellular connective tissue, and occupying the spaces of the eroded bony trabeculæ.

Termination.—The rachitic process usually terminates in from three months to a year, when ossification sets in, as is evident from the appearance of lime salts in the lamellæ of the osteoid tissue; in some places there is a direct transformation of cartilage into bone. At the same time excessive cell proliferation at the epiphysis and on the inner surface of the periosteum ceases, and the amount of absorption in the medullary cavity decreases, the bones becoming less vascular.

Calcification is accompanied by contraction and condensation of the spongy bony structure, so that, on complete recovery, the bones are often harder and denser than normal. This shrinkage in the spongy bone structure decidedly reduces the degree of deformity from the enlarged and prominent epiphyses of the long bones. The bosses on the skull become smaller, and the beading of the ribs imperceptible, until, finally, all traces of these rachitic changes tend to disappear.

The curvatures and deformities of the spine, pelvis, and lower extremities due to the weight of the thorax are also lessened to some degree; but when these deformities have been marked and ossification has occurred, permanent signs of rachitis usually remain.

Symptoms.—The onset of rickets is very gradual. In some cases a moderate degree of gastro-intestinal catarrh precedes the actual manifestations. Constipation is a common feature, owing to the atonic state of the intestinal muscles; but, in some instances, there may be alternating attacks of diarrhea as a result of chronic irritation of the colon by the hard, dry stools. The appetite is often unimpaired, and may even be excessive. Vomiting is uncommon.

These infants are restless and irritable during the day and sleep poorly at night, tossing about in bed and waking at frequent intervals. There may be slight fever at the onset of rickets, but any high elevation of temperature during the course of the disease should cause the physician to suspect some intercurrent infection. Excessive perspiration about the head and neck, especially at night, is one of the earliest and most characteristic symptoms, and is an invariable feature of the disease, often continuing for months. This symptom is seldom overlooked, since the pillow upon which the rachitic child sleeps is always wet, while the rest of the bedclothing is dry. Head rolling is observed in many cases, and these children have a habit of grinding their teeth, which symptom, however, is not peculiar to rickets alone.

Chronic catarrhal rhinitis and pharyngitis, attacks of acute and chronic bronchitis, and acute colds are common in rachitic children. Some observers believe that, in addition to the influence of their lowered vitality which would predispose them to such affections of the upper respiratory tract, these children also acquire acute colds and bronchitis from sleeping constantly on a wet pillow and lying uncovered in bed, the bed-covers being kicked off in their restlessness.

During the day rachitic children seem languid and indisposed to move about, or to be picked up and fondled. This has been attributed to tenderness throughout the body, which is sometimes quite marked over bony·surfaces and certain groups of muscles.

The general appearance of rachitic children differs somewhat; but, as a rule, they are pale and look anemic. Although they frequently appear to be well nourished, their flesh is fat and flabby, and their muscular tissue has but little tonicity. In exceptional cases of rickets, the child's complexion may be very good and the general health apparently unimpaired; in still another class of cases the rachitic child may be thin and wasted, presenting the typical picture of marasmus.

The blood shows no characteristic changes aside from simple anemia. The hemoglobin content is low, ranging from 40 to 60 per cent.; the number of red cells varies but little from normal. The total number of leukocytes is increased, but there is no notable increase of any particular variety of white cells.

The urine presents no characteristic changes which would suggest rickets, but the calcium salts may be decreased. Heitzmann claims that there is an excess of lactic acid and phosphates in the urine, and some observers are inclined to believe that these phosphates are being excreted in abnormally large quantities instead of being utilized in bone formation.

The mucous membranes of the body are pale and undernourished, consequently they readily become inflamed from trivial causes, such as slight indiscretions in diet, and moderate changes in the atmosphere or climate. This inflammation is apt to pursue a chronic and protracted course rather than an acute one.

Although there are no lesions of the nervous system, yet neurotic symptoms are quite common and numerous in rachitis. The view is held that these manifestations are simply a result of the impoverished condition of the nervous system, in common with all other tissues of the body. Rachitic children are all neurotic, and the normal stability of the nervous system is retarded until a later age than in the healthy child. Baldness at the occiput is often observed in rachitic infants, and is due in large measure to the constant tossing of the head to and fro upon the pillow.

Muscular spasms are common, and may take the form of laryngismus stridulus, nystagmus, tetany, or convulsions. There is also increased susceptibility and response to reflex irritation. Laryngospasm and tetany are rarely seen in other than rachitic children, and rickets is one of the most common predisposing causes of convulsions. Nervous manifestations are most marked in young babies, and the liability to, and severity of, the symptoms seem to depend more upon the age of the child than upon the severity of the rachitic process. As a result, convulsive seizures are most common before the second year, and are often excited by gastro-intestinal disturbances, by reflex irritability, or by the action of toxins absorbed from the alimentary tract.

Dentition in rachitic children is usually delayed, and is accompanied by gastro-intestinal disturbances, which in large part are due to the lowered vitality of the alimentary mucosa; they sometimes prove quite serious. The first teeth may be cut at any time from the sixth to the eighteenth month, and, although dentition is late, when the teeth do erupt, they are well developed and do not decay readily. This is in sharp contrast to dentition in syphilitic infants, in whom the teeth appear very early, but are poorly developed, and quickly decay.

Early Physical Signs of Rickets.—The earliest and most typical sign of rickets is the rachitic rosary, which is formed by the enlarged and widened epiphyses of the ribs at the costochondral junction. The nodules, or beads, thus formed are most prominent at the fifth and sixth ribs, and it is here that they may be first detected. They can practically always be felt by the examining fingers, and in exceptional cases are distinctly visible, attaining the size of small marbles. When there is marked thoracic deformity, beading is often observed on the posterior surface of the anterior chest wall, due to the green-stick fractures which take place near the posterior angles of the ribs. The ribs in these cases join the cartilage at an angle, instead of end to end, and there is partial or complete dislocation of the bony rib backward, so that frequently when posterior beading exists there is no beading on the external surface of the chest.

The rachitic rosary can rarely be detected before the third month,

and tends to diminish in size under proper treatment or when the
disease terminates spontaneously, so that there is scarcely a trace of
these prominences after the fifth year, and they are not perceptible
in adults.

Craniotabes, which is an early rachitic sign, is a softening of the
cranial bones and the formation of thin spots from pressure within
the skull as well as from external pressure. It is on the posterior
portion of the parietal bones and on the occipital bone that most of
these thin areas are found, for this part of the skull is most frequently
subjected to pressure when the child is lying down. They are most

Fig. 27.—Rachitis.

numerous about the lamboidal suture, seldom appearing on the frontal
bones in the region of the coronary suture. They are sometimes an
inch in diameter, and several may be found on the skull at one time.
To detect them, light pressure should be made upon the skull in
a direction away from the sutures. When an area is pressed upon,
a parchment-like crackling sensation is transmitted to the fingers.
Craniotabes rarely appears in infants who develop rickets after the
sixth month, and it is much more marked in children who suffer from
both congenital syphilis and rickets than in those who have congenital
syphilis alone.

Rachitic Deformities.—The head of a rachitic child appears to be larger than normal; but this is often due to the diminished size of the facial bones, and to the disproportion between the head and the rest of the body. In severe cases there may be an actual increase in the circumference of the head which is due to abnormally thick cranial bones, to cranial bosses, or to hyperostoses. These hyperostoses cause a prominence of the frontal and parietal eminences which results in the typical square broad forehead. When the bosses are numerous they sometimes produce furrows along the line of the coronal, sagittal, and frontal sutures, thus forming the *hot-cross-bun* type of skull.

The occiput is flattened by pressure, the crown of the skull is flat rather than vaulted, and these flattened surfaces together with the square broad forehead give the skull a cuboid rather than a globular appearance. The anterior fontanelle is very late in closing, and may remain open until the second or third year.

A faint systolic murmur may sometimes be heard if the ear is placed directly over the anterior fontanelle; but this feature is of no diagnostic importance, since it is found in other conditions in which the fontanelle has failed to close. The closure of the other fontanelles is also delayed, and the sutures of the skull may not completely unite until after the first or second year.

The superficial veins of the scalp are enlarged, prominent, and distinctly visible. The hair is scant, its growth being retarded, and it is worn away from the back and sides of the head by friction and sweating. The upper jaw of the rachitic infant is unusually long and narrow, while the anterior portion of the lower jaw is broader and higher than normal, and curves rather sharply at the site of the canine teeth, which gives it a square and angular appearance.

The Chest.—In addition to the characteristic deformity known as the rachitic rosary, the ribs present certain curvatures due to atmospheric pressure, one of which is at the junction of the dorsal and lateral portions of the thorax, and the other is located anteriorly where the ribs curve toward the sternum. A lateral flattening is thus produced, which extends from the second rib to the hypochondrium along the line of the costochondral articulations. This flattening lessens the transverse diameter of the chest and causes the sternum to bulge, thus forming the so-called *pigeon-* or *chicken-breast.*

The anteroposterior diameter of the chest is increased. The thorax is narrowed at the clavicles, but flares outward below, giving rise to the so-called *funnel chest.* This widening of the costal angle at the tip of the sternum is due to the pressure of the liver and spleen. It is most marked on the right side owing to the presence of the liver. There is also formed at the upper level of the liver, stomach and spleen a transverse furrow, known as the rachitic girdle, or *Harrison's furrow*, which is produced by eversion of the lower thorax. It is more apparent on inspiration, since this portion of the chest wall does not retract.

These deformities of the chest are rarely severe in uncomplicated cases of rickets, but are exaggerated when the respiration is obstructed

by enlarged tonsils and adenoids, also in children with chronic bronchitis. In some rachitic children the sternum is depressed instead of prominent.

In mild cases of rickets the spine is normal; but in many well-developed rachitic children there is kyphosis in the dorsolumbar region, which extends from the middorsal vertebræ to the sacrum. This is due not only to the rachitic process in the bodies of the vertebræ, but is also induced by the laxness of the vertebral ligaments. It is most marked when the child is held in the arms, but can be made to disappear if the child is suspended by the feet or laid upon its belly with the legs extended. This sign is an important point in the differentiation of rickets from Pott's disease.

Lateral curvature is rare, but when a child under three years of age develops this condition it is almost always of rachitic origin. Lordosis is also uncommon, but may appear in association with deformity of the pelvis. Spinal curvatures and kyphoses of rachitic origin which arise during infancy show a tendency to spontaneous correction when the supporting ligaments and muscles become normal; but when these deformities appear after the third year, accompanied by changes in the pelvis, they very often persist throughout life.

The Pelvis.—The most common pelvic deformity is shortening of the anteroposterior diameter and flattening of the pelvis. A thickening of the iliac crests also occurs, and many minor changes take place which give the pelvis an irregular or crumpled appearance.

The Extremities.—Deformities of the extremities are usually symmetrical, and are more marked in the lower than in the upper limbs because the legs bear the weight of the trunk during the stage in which the bones are abnormally soft. The clavicle escapes deformity in all except the most severe cases, but it may be rendered quite prominent by either a green-stick fracture or an increase in the convexity of the inner third. The humerus is rarely deformed unless the infant is allowed to crawl about too early, when a forward and outward curvature often develops. The epiphyses are both enlarged, but not as prominent as the enlarged epiphyses of the radius and ulna, the distal ends of these two bones of the forearm being very conspicuous, and producing that characteristic deformity—the broad wrist.

The shafts of the radius and ulna may also be curved outward in children who use the arms as well as the legs for locomotion, and among such children green-stick fractures of these bones are not uncommon. Sometimes the radius and ulna become twisted upon their longitudinal axes, the radius developing a spiral bend which causes pronation of the hands. There are no characteristic deformities of the hands; but, in rare cases, there are rachitic enlargements of the ends of the metacarpal bones and phalanges which resemble syphilitic dactylitis.

The lower extremities are usually deformed, even in light cases. The femur is bent outward and forward by the weight of the child in the sitting posture, for these infants rarely attempt to walk or stand

because of pain. Coxa vara may also be produced by the weight of the trunk. The tibia and fibula usually curve outward and anteriorly, which produces bow-legs, but may in some cases curve inward, so that knock-knees result. These are the most common deformities of the lower extremities.

Knock-knees are most frequently seen in girls, and they are believed to be caused by an enlargement of the inner condyles of the femurs. Bow-legs, in light cases, are due to epiphyseal enlargement, and may totally disappear when ossification takes place and the enlarged epiphyses shrink and become smaller. In severe cases, bow-legs are the result of an outward curvature which is usually associated with a more marked anterior curvature, so that the bones are bent forward and outward.

Enlargement of the lower epiphysis of the tibia is quite common, and results in the abnormally large ankle so often seen in rachitic children; but marked enlargement of the upper epiphyses of the tibia and fibula is quite rare and found only in severe cases of rickets. In rachitic children fractures of the long bones are usually of the green-stick variety, and may be caused by trivial injuries.

Permanent deformity often results from subperiosteal fractures. The bones of the feet are rarely affected, and rachitic flat-foot is quite uncommon. The short stature of rachitic children is due to arrested growth of the long bones, and is usually exaggerated by the fact that the child is bow-legged.

The Ligaments.—The ligaments of the rachitic child are relaxed and weakened, thus giving but little support to the joints; this in large measure accounts for spinal curvatures and other deformities, such as knock-knees, overextension of the knee-joint, occasionally flat-foot, and abnormal laxity of all the joints of the body.

The Muscles.—In rachitis the muscles are affected in direct proportion to the damage done the bones. They are hypertonic, undeveloped, and exceedingly flabby. Muscular weakness may be so extreme as to make us suspect paralysis. These children are very late in sitting upright, standing and walking, because of this lack of muscular tone, and the weakened abdominal musculature is largely responsible for the protuberant abdomen of the rachitic child.

Diagnosis.—In well-developed cases the diagnosis of rickets is quite easy. Even in its milder form, and early in the course of the disease, such symptoms as excessive sweating of the head and neck, constipation, restlessness at night, enlarged fontanelles, craniotabes, and delayed dentition, are sufficient to warrant a diagnosis of rickets. It is this symptom-complex, however, upon which the diagnosis must be made, since most of these symptoms, appearing alone, might be caused by other diseases. For example, craniotabes occurs in syphilis; but the other bony changes in syphilis are found in the shafts of the bones, which may even be necrotic, while in rickets the epiphyses show the most marked changes, and necrosis never takes place. For this reason the x-rays may be of great value in the differential diag-

nosis between syphilis and rickets, and in doubtful cases a Wassermann test should also be made.

The rachitic rosary, bowed legs, and epiphyseal enlargements are the pathognomonic signs in well-established cases which make a rachitic child conspicuous; but, occasionally, cretinism and achondroplasia are mistaken for rickets, or *vice versa*. Cretinism can be differentiated by the marked mental deficiency, facial expression, macroglossia, and the striking disproportion between the height and the age of the cretin. Achondroplasia is also distinguished by the disproportion between the length of the trunk and of the extremities, which is much greater than in rickets, also by the greater softness of the bones, which causes, as a rule, more striking deformities.

The large head of the rachitic child may suggest hydrocephalus; but it is cuboid in shape, while the hydrocephalic head is globular. Other symptoms and signs of hydrocephalus, such as softening of the cranial bones, separating of the fontanelles, and irritability of the nervous system, also appear in rickets; but the changes in, and deformities of, the long bones so typical of rickets are never seen in hydrocephalus, and the rachitic head does not undergo the rapidly progressive distention observed in the hydrocephalic child.

Scurvy may be differentiated from rickets by the acuteness of the symptoms and such characteristic signs as spongy gums, ecchymoses of the skin, and the tendency to hemorrhage from the mucous membranes. The extremities in rickets are not nearly as tender as in scurvy, and there is practically no pain. Rachitic scoliosis may resemble caries of the spine; but, besides the fact that other signs of rickets are also to be found, the spine is flexible in early rickets, and the curvature may be made to disappear (as stated in the discussion of the symptoms) by suspending the child by its feet or laying it down upon its belly with the legs extended, while in caries of the vertebræ the curvature of the spine is fixed.

Rachitic coxa vara may be differentiated from congenital dislocation of the hip by *x*-ray examination. Rachitic pseudoparalysis which results from weakness of the muscles and ligaments, may be mistaken for cerebral palsy or infantile paralysis; but in neither of these organic nervous diseases are there any bony deformities such as accompany rickets. In cerebral paralysis the reflexes are exaggerated and the muscles rigid; in infantile paralysis the reflexes are absent and the muscles flaccid; while in rickets the reflexes are normal, and the muscles simply exhibit a general weakness due to malnutrition and disuse.

The paralyses caused by acute affections of the brain and spinal cord, such as polioencephalitis and myelitis, are characterized by a sudden onset, and the paralysis is usually confined to certain groups of muscles which show electrical changes; whereas in rickets all the muscles of the body are weakened, but there are no symptoms of paralysis nor of electrical changes. Postdiphtheritic paralysis may be differentiated by the absence of knee-jerks, the various reflexes of the body being unaffected in rickets.

Course and Prognosis.—In this affection the prognosis is very favorable as regards life, for rickets is never fatal when uncomplicated, and even neglected cases exhibit a tendency to spontaneous recovery eventually, although great deformity may remain. If rickets is recognized early, and properly treated, perfect recovery without deformity is the usual outcome. The prognosis as to deformity depends, however, not only upon treatment, but also to a certain extent upon the severity of the disease in each particular case.

While the rachitic child is in no danger of dying from rickets, yet it is predisposed to intercurrent acute infections because of its lowered power of resistance, and these diseases may cause death because of the child's want of vitality. Diseases of the respiratory tract, in particular, are usually serious in rachitic children on account of the abnormal shape of the thorax, while disturbances of the gastro-intestinal tract are stubborn, owing to the poor state of the alimentary mucosa.

The acute symptoms of rickets last, as a rule, for nine to twelve months, recovery being gradual. Improvement is shown by a return of muscular power, a lessening of the nervous symptoms, and diminution of the sweating of the head. Later the bony changes and consequent deformities become less prominent.

Prophylaxis.—The prophylaxis of rickets consists in so regulating the diet of every pregnant woman that she is sure to take sufficient nourishment during the period of gestation. Strict attention should also be paid to the hygienic and living conditions of the expectant mother. The same care should be given to the diet and hygiene of every nursing mother; for if a healthy mother who has plenty of good milk feeds her infant at regular intervals and gives it nothing but breast milk, rickets will not develop during the normal period of lactation.

Too frequent feeding should be avoided; once every two hours is ample during the first six weeks, after that every two and a half hours, and later once every three hours is enough. No preparation of patent baby food or condensed milk should be given to supplement breast-feeding; too much of barley-water is also harmful; but if the mother's milk is deficient in fats and proteins, or in quantity, a formula of cow's milk, properly prepared, should be given in addition to the breast milk.

Sometimes the quality of the mother's milk is improved by giving her tonics, such as the elixir of glycerophosphates, or the compound syrup of hypophosphites, in dram doses after meals during the latter months of pregnancy and during lactation.

An infant should never be weaned until it is at least nine months old if the mother has any milk, and if there are no contraindications to nursing. On the other hand, no child should be permitted to nurse after the fourteenth month, at latest. When it becomes necessary to wean a baby before it is old enough to take undiluted cow's milk, it should be placed upon a feeding mixture of diluted cow's milk, modified

according to its age, weight, and digestive capability. In this way it is supplied with the proper amount of fats, proteins, and carbo-hydrates.

Unfortunately, many mothers use proprietary infant foods for such children; either because they are more easily prepared, or may seem to be cheaper, or because some one has recommended such and such a food, or the mothers have seen them advertised. The con-tinuous use of these patent foods or of condensed milk as a regular diet should be condemned and strictly prohibited, which will largely prevent gastro-intestinal disturbances and any likelihood of rickets. In addition to modified cow's milk, after the sixth month it is a good plan to give artificially fed babies one-half to one ounce of beef juice two or three times a week, and a dram or two of orange juice daily.

Next in importance to diet in the prevention of rickets is attention to the hygiene and environment of babies and children. If the sur-roundings are clean and sanitary, if the child is given a daily bath, if its bowels are opened daily, and if it gets moderate exercise and plenty of fresh air, much is done to lessen the susceptibility to rickets. The foregoing prophylactic treatment is most essential in the children of a family in which there is a predisposition to rickets, and should be rigidly carried out.

Treatment.—The active treatment of rickets consists chiefly in regulation of the diet, the hygienic welfare of the rachitic child being of secondary importance. The treatment of deformities due to rickets needs consideration, but this is usually a problem for the orthopedist. In the majority of cases, active rachitic processes are really in progress only up to the eighteenth month; therefore it is important that rickets be recognized and treated before this time, if deformities are to be prevented. Treatment instituted after the second year has but little effect, as at this age rachitic changes have already taken place.

Dietetic Treatment.—When a breast-fed baby becomes rachitic, the diet of the mother should be increased both in quantity and quality to insure breast milk for the infant which will contain an ample pro-portion of fat and proteins. If the milk can not be made richer because the mother is not well, either a wet nurse should be secured or the breast feedings supplemented by several bottle feedings of a properly prepared formula. The infant should not be taken from the breast, however, unless the mother's milk can not be sufficiently improved to make it agree with the baby, or the mother again becomes pregnant.

If we find that a rachitic infant is being nursed beyond the normal period of lactation, it should be promptly weaned, and given food suitable to its age and digestive power. Besides increasing the diet of the nursing mother whose baby has rickets, her surroundings and personal hygiene should receive attention, she should take moderate exercise, plenty of rest, and never allow herself to become fatigued. In this manner her milk may be so improved both in quantity and quality that she can properly nourish the child.

Rickets in the artificially fed child should be carefully investigated and the diet corrected. If patent foods or condensed milk mixtures are being given, these should be stopped immediately, If a formula of cow's milk is being used, this should be adjusted, and the feedings prepared on a percentage basis that will insure a sufficiency of fat and proteins and no excess of carbohydrates.

If there are evidences of gastric or intestinal indigestion, such as anorexia, vomiting, and diarrhea, the bowels should first be swept out with one to three drams of castor oil, and a formula given, at first very weak, the strength of which can be gradually increased as the digestion improves.

The rachitic infant usually comes under observation when it is between twelve and eighteen months old, and at this age its diet should be composed of one quart of milk daily with at least an ounce of cream (or some other substance rich in animal fat, such as butter), also the yolk of an egg, and bread. Later, if the digestion is good, a few cereals, such as rice, cream of wheat, or barley may be given together with fresh vegetables, such as spinach, peas, beans, or asparagus, and a small amount of chicken, fish or mutton.

This diet is very liberal, and not difficult to follow, the chief object being to give an ample supply of fat and to limit the carbohydrates. Fresh milk is, perhaps, the most valuable article of food for the rachitic child, therefore it should always be taken plentifully at frequent intervals throughout the day. Fresh fruit juices and beef juice are very beneficial, and the craving for salt which these children often exhibit is an indication of their need for it, which should be satisfied.

Cod-liver oil is to be regarded as a form of nourishment for rachitic children, and should be given in every case unless it causes gastro-intestinal disturbances, in which circumstance it should not be used during extremely hot weather. Children under one year of age may be given one or two drams, divided in three doses, during the day; older children may take one dram three times daily, or even more than this if the stomach tolerates it, and it can be assimilated. Olive oil is, perhaps, not as valuable as cod-liver oil, for these children seem to require animal fats; but it may be given in the same dosage, or even in larger quantities, since it is not so harmful to the digestion.

Hygienic Treatment.—Rachitic children should be kept out of doors as much as possible, and the rooms in which they sleep should get plenty of sunlight and fresh air. The homes of these children should be clean, sanitary, and afford plenty of ventilation; but, unfortunately, such living conditions are hard to procure, since cases of rickets usually develop in the large cities; therefore it is wise to send such children to the seashore or country. The seashore is, perhaps, preferable because of its stimulating effect on the appetite and metabolism.

Care of the skin should not be overlooked, and a daily bath at the temperature of the body should be given. Inunctions of olive oil are not advisable because they interfere with the secretory function

16

of the skin. Cold baths are harmful during infancy; but a brine bath made by adding one ounce of sea salt to two gallons of water is very beneficial, given each morning, and followed by a brisk massage or rub. Exercise is absolutely essential to improve the muscular tonicity, and should be encouraged in so far as it does not increase deformities; for instance, no attempt at walking should be permitted while the bones are soft, as the legs will become bowed by the weight of the trunk.

Drug Treatment.—It is difficult to estimate the value of drugs in the treatment of rickets inasmuch as there is a tendency to recovery under proper dietetic and hygienic care. There are no specifics for the cure of the disease, hence tonics and preparations for special symptoms meet all the indications. Owing to the nature of the rachitic process in the bones, it is advisable to give hypophosphites of lime and soda, in combination with cod-liver oil, each teaspoonful of the oil containing one-half to one grain each of sodium hypophosphite and of calcium hypophosphite. Lime should always be given, preferably in the form of the glycerophosphates or the lactophosphates, dose one to five grains, three times a day. The elixir of glycerophosphates has proven of value in rickets because of its tonic effect, and may be given in 10- to 30-drop doses.

There is great diversity of opinion and much discussion as to the value of phosphorus in rickets; but it should always be administered, since experiments have proven that the giving of phosphorus to young animals in suitable dosage is followed by an increased activity of the processes in the epiphyseal ossification zone. The one possible objection to it is that it may set up gastro-intestinal disturbances; but, if the digestion is not already deranged, it may be given in $\frac{1}{250}$ to $\frac{1}{100}$ of a grain doses, three times a day, either in the form of the official oil, which may be combined with olive or cod-liver oil, or as Thompson's solution, which contains $\frac{1}{20}$ of a grain of phosphorus to the dram.

It is claimed by some clinicians that better results are obtained when phosphorus is combined with cod liver oil. Other authorities maintain that the effect of the phosphorus is negligible, and that the benefit derived from this combination is due solely to the cod-liver oil. Phosphorus is certainly of little or no value in the later stages of rickets, the most favorable time for its administration being early in the disease. It is especially beneficial in the cases accompanied by craniotabes.

Owing to the anemia present in rachitic children, iron is always indicated, and may be administered in several forms. The syrup of ferrous iodide is well borne by most children in 5- to 10-grain doses after each meal, or the hypophosphite of iron may be given in 1- to 5-grain doses, three times a day. Tincture of ferric chloride should be administered only to those children who show no gastro-intestinal disturbance, and then cautiously in 1- to 3-drop doses, but the saccharated carbonate of iron is very easily borne by the stomach, and may be taken in 1- to 3-grain doses.

Fowler's solution, 1 to 3 drops, is occasionally administered for its tonic effect, and the dose may be gradually increased. Glandular extracts have been employed with good results in some cases, but their use in rickets is not universal. Thyroid extract, in $\frac{1}{2}$-grain doses, may be given twice or three times daily with one of the iron preparations, preferably the saccharated carbonate. The pituitary gland has also been used.

Of the drugs indicated for special symptoms, atropine sulphate in $\frac{1}{800}$ of a grain dose, three times a day, may be recommended for the relief of the profuse sweating of the head. The bowels may be kept regular by the administration of aromatic fluidextract of cascara sagrada in 15- to 30-drop doses when necessary, or milk of magnesia in $\frac{1}{2}$- to 2-dram doses as required. If the digestion is poor, an effort should be made to improve it by giving tincture of nux vomica in 1- to 3-drop doses combined with dilute hydrochloric acid, drops 1 to 3, before meals, or by giving quinine sulphate in $\frac{1}{4}$- to $\frac{1}{2}$-grain doses three times a day.

It is also a good plan to give these children occasionally $\frac{1}{2}$ to 1 grain of hydrargyrum cum creta, and thoroughly to cleanse the bowels once or twice a month with a purgative dose of castor oil (1 dram to $\frac{1}{2}$ ounce). If there is a tendency to convulsions, sodium bromide should be given in 2- to 5-grain doses, three times a day.

Treatment of Complications.—Bronchitis and bronchopneumonia are the two most common complications referable to the respiratory tract, and call for the usual treatment; but antirachitic remedies are more effective than any special measures which can be employed. Disturbances of the alimentary tract are by far the most frequent complications, and must be treated in the usual way. In acute attacks with diarrhea, an initial dose of castor oil, 1 to 2 drams, should be given, followed after ten hours by bismuth subnitrate in 10- to 20-grain doses, three or four times daily. The diet should be considerably restricted for several days. But the treatment of the constitutional disease is of the utmost importance, and should always be carried out.

Convulsions form the most common nervous manifestation of rickets. Immediate treatment consists in placing the child in a tub of water at 100° to 106° F. for three or four minutes, after which it should be dried quickly and put into bed. Another effective measure is to wrap the child for five to ten minutes in a blanket wrung out of hot water, and then wrap it up in a hot dry blanket. If these measures do not stop the convulsions, from 5 to 15 grains of potassium bromide with 2 to 5 grains of chloral hydrate may be injected into the rectum. Morphine sulphate, grain $\frac{1}{60}$ to $\frac{1}{30}$, may be given hypodermically in severe cases, or inhalations of chloroform may be tried. An enema of warm salt solution or soapsuds should always be given, and may in itself afford relief.

Laryngismus stridulus, a less frequent complication of rickets, is due to a neurotic spasm of the vocal cords. It should be treated

by the use of a hot pack and an enema, or 1 to 2 drams of castor oil. If there is a tendency to nocturnal attacks, potassium bromide, 5 to 15 grains, and chloral hydrate, 2 to 4 grains, should be given each evening.

In tetany, a rare complication of rickets, there is a painful spasm of the muscles of the hands and feet. It requires but little special treatment, although it is advisable to use a belly band, and to wrap the arms and legs in cotton-wool during an attack. The nervous manifestations of rickets, in common with complications in various parts of the body, call for the recognition of the primary disease and its efficient treatment before a permanent cure can be expected.

Treatment of Deformities.—The prevention of deformities is a very essential part of the management of a case of rickets, and consists in limiting motion during that period of the disease when the bones are soft. The child who shows a tendency to curvature of the spine should be kept in the recumbent posture and not allowed to sit up unsupported.

If kyphosis appears, it should be treated by placing the child on a Bradford frame or a hard bed, and keeping it in the recumbent posture. In severe cases it is advisable to correct the deformity each day by turning the child upon its abdomen, and raising the buttocks while pressure is made upon the spine. Plaster casts should only be used in selected and severe cases, for they interfere with respiration, and increase the tendency to bronchitis and bronchopneumonia. When there is pelvic deformity, the sitting posture must be avoided, especially in girls. Bow-legs and knock-knees can be largely prevented by keeping children off their feet until the bones harden and the rachitic changes have passed away. Creeping also should be prohibited while the bones of the arms are soft. If bowing occurs, properly fitting braces should be worn continuously, and walking discouraged. If the child curls up its legs in bed, external splints should be applied.

Intelligent manipulation of the deformed extremities, if done early, may reduce the curvatures somewhat; but, after the third year, correction of deformities by braces or manipulation is impossible. Massage is useful in strengthening the weakened muscles, and may to a certain extent inhibit the progress of deformity; but it should be performed carefully and judiciously.

Osteotomy for the correction of deformities should be postponed until after the seventh or eighth year, because many rachitic curvatures lessen considerably by this time, and also because there is danger that more or less curvature may follow such an operation if it be done too early.

In very young infants curvatures of the extremities have been absolutely corrected by the use of a plaster-of-Paris cast. The foregoing treatment of deformities from rickets is largely a problem for the orthopedist, and it is always advisable to consult one in order to secure the best results.

CONGENITAL RICKETS.

This form of rickets is seldom encountered in the United States, but a few scattered cases have been reported. It arises during intrauterine life when the fetus is deriving its sustenance through the placental circulation, and is due to malnutrition and chronic disease in the pregnant mother. These infants exhibit at birth the characteristic rachitic changes and deformities, the histological changes in the bony tissue being to a certain extent the same as in older children. The congenital form of rickets is rarely severe, but the rachitic rosary, epiphyseal enlargements, and craniotabes may be apparent in these children at birth.

ADOLESCENT, OR LATE, RICKETS.

This form, also, is rare in America, but not extremely uncommon in Europe. It is said to occur more often in girls than in boys, and may appear at any time between the sixth year and puberty, or even later. In these cases there are both beading of the ribs and epiphyseal enlargement, but the skull is normal, since ossification of the cranial bones is complete before the onset of the disease. Among the deformities caused by late rickets may be mentioned bow-legs, knock-knees, flat-foot, femoral curves, coxa vara, and scoliosis.

ACUTE RICKETS.

Although cases of acute rickets have been described, rachitic changes are essentially chronic, and the existence of an acute form of this disease is extremely doubtful. Most cases of so-called "acute rickets," if investigated thoroughly, will prove to be scurvy or some other disease of similar nature.

CHAPTER XIII.

DISEASES OF THE GASTRO-INTESTINAL TRACT.

In the consideration of the diseases of children, affections of the gastro-intestinal tract are undoubtedly the most important, for they form the basis of many of the illnesses of infancy, and constitute a large proportion of the disorders which occur between infancy and puberty. Several factors may explain the preponderance of gastro-intestinal disturbances over those of other systems of the body which are called upon to functionate after birth.

One of the most significant reasons why the digestive tract so often breaks down under the strain is that, of all the systems of the body, this alone must utilize material foreign to the body. The circulatory system is furnished with a full quota of blood at birth; from birth onward the air utilized by the respiratory system varies only slightly in composition; but even in the breast-fed infant, and vastly more so in the one artificially fed, the digestive system must use material which constantly varies in strength and amount.

Under proper conditions, the digestive apparatus after birth is capable of digesting and assimilating the normal food with reasonable variations for the individual at every period of life; therefore the true explanation of gastro-intestinal affections encountered in infancy and childhood lies, not in the inability of the child to assimilate its food, but in the failure of the parents to appreciate certain peculiarities, both in structure and function, of the digestive system of the infant or child, as compared with the adult, which in most instances leads to overtaxation of the gastro-intestinal tract by the introduction of food unsuitable as to quantity or quality.

DISEASES OF THE LIPS.

HERPES.

Herpes labialis is quite a common affection in children, and receives its synonym "fever blister" because it is frequently associated with a rise in temperature. It is often seen in pneumonia, but rarely occurs in typhoid fever, and the appearance of herpes in a febrile case where typhoid is suspected is of no little significance in discriminating against that disease. The blisters are of little importance except for the fact

that, after the crust forms, healing is sometimes greatly retarded by the habit of picking the sores which children will persist in, unless prevented in some way from reaching them. If this can be done, and they are dusted with either boric acid or zinc oxide, they will disappear in the course of a few days.

PERLECHE.

Perleche is a cracking and ulceration of the mucous membrane of the lips, usually occurring at the angles of the mouth, and affecting the vermilion border of the mucous membrane. It is infectious in nature, but not syphilitic.

Etiology.—Perleche is usually observed in children who are in poor physical condition, and present other signs of malnutrition, such as anemia, chronic nasopharyngitis, adenitis, or scrofula. It is occasionally observed before the second year, but is most common after the period of infancy. The infection is usually conveyed through some tiny fissure in the lip, and manifests itself by swelling and hyperemia, accompanied by smarting and itching which cause the child constantly to lick the corners of the mouth. The tiny fissure which was the port of entry of the infection becomes larger, and other cracks and fissures appear as a result of the constant irritation produced by the tongue. A grayish-white ulcer forms at the angle of the mouth; this is composed of macerated, thickened, and opaque mucous membrane which closely simulates a syphilitic mucous patch. The condition, if not relieved, may persist for several weeks, and in some instances constitutes a grave menace to the health of an already anemic child, for movements of the lips may be so painful as to make the little one refuse its food. The erosions are linear in shape, and involve both corners, where the rhagades may be seen slightly elevated with a reddened base.

Diagnosis.—The diagnosis of perleche cannot be clearly made without considering syphilitic rhagade, eczema of the lips, and herpes labialis. Absence of induration at the base, and of mucous patches inside the mouth, strongly indicates perleche. Eczema of the lips is usually associated with lesions in other parts of the body, and responds much more readily to treatment than perleche. The severity of the infection in perleche, and the pain and smarting present, will generally exclude herpes.

Treatment.—The usual duration of perleche is from two to three weeks, but it should always be treated since it may become chronic if neglected. The best application is silver nitrate, 10 per cent. solution, followed daily by a dusting powder of zinc oxide, or the application of boric acid ointment to the raw surface of the lips. Excellent results are obtained by the use of an ointment of the red oxide of mercury.

Another mode of treatment which has often proven beneficial, although not as quickly effective as those above outlined, is to bathe

the corners of the mouth in a 1 to 2000 bichloride solution, following this immediately and every three hours thereafter by the application of ichthyol, 25 per cent.

DISEASES OF THE TONGUE.

GEOGRAPHICAL TONGUE.

Epithelial desquamation of the dorsum of the tongue is a common affection in children, and is caused by a chronic superficial desquamating glossitis which denudes the tongue of areas of epithelium and gives it a so-called "geographical" aspect, because of the irregular, round, and crescent-shaped patches it presents. These areas are very variable in size, and may coalesce or diminish until they disappear, or suddenly grow again with great rapidity. The denuded patches are red and smooth, the papillæ being absent, the margins are grayish or whitish, but the remainder of the surface of the tongue is normal.

The cause of geographical tongue is unknown. It is observed in rich and poor children alike, and is apparently uninfluenced by hygienic care of the mouth. Once observed in a child it will be found to recur at varying intervals until adolescence, when it tends to disappear. In itself, the condition is of no significance, except that it is often mistaken as a symptom of some other disease. No form of treatment has as yet proven effective, so that spontaneous cure is the ultimate outcome. Most of the cases which have come under my observation occurred in rather weak and debilitated children, but improvement in their general physical health failed to influence the local condition.

GLOSSITIS.

This affection is very rare during childhood, but is occasionally seen as a result of accidental trauma from biting the tongue in a fall, rage, or convulsion. Now and then glossitis is caused by a strong alkali or acid taken by mistake, or by the sting of an insect or a burn. Infection and inflammation of the tongue are always followed by enlargement of the organ, and much pain. If the infection be severe, the tongue may become so large as to protrude from the mouth and also interfere with respiration and deglutition. Recovery usually takes place within a few days; but mechanical obstruction from enlargement of the organ often greatly alarms the parents, and may require urgent measures for relief.

Treatment.—The child's nourishment should be kept up; and, if swallowing is extremely painful, liquids should be introduced by means of a catheter through the nose. When the symptoms are most

acute, ice applied to the under surface of the lower jaw, and kept in the mouth continually, will help to relieve the pain. If the swelling becomes extreme multiple punctures may be made, or the dorsum of the tongue may be scarified on either side of the raphæ.

MICROGLOSSIA.

Microglossia is a very rare condition in which, owing to an arrest in development, the tongue is much smaller than normal. The cause of microglossia is unknown, and since the diminutive tongue causes little or no disturbance in talking or feeding, the condition is of little importance except as a contrast with the opposite condition, macroglossia, which, though rare, occurs with greater frequency.

MACROGLOSSIA.

Enlargement of the tongue may be due to an overgrowth of the lymphogenous structure (lymphangioma or cavernous macroglossia), to an increase in the muscular and connective tissue elements (fibrinous macroglossia), and in some instances to an excess of both lymphogenous and connective tissue elements. Occasionally an abnormally long tongue is rendered very mobile by an unusually lax frenum, which makes it possible for the child to swallow its tongue; several cases of this kind are recorded in literature. Aside from this accident, the tongue may become so large as to fill up the entire mouth, and prevent nursing or the taking of food, or may even protrude from the mouth, which causes it to become bruised and chapped, and the inflammation results in further enlargement.

True macroglossia, which is a congenital condition, should never be confused with the enlarged tongue of the cretin, familiar to all, in whom the tongue is of a deep bluish color, and shows marks of the teeth, is prolapsed, and slightly protrudes from the mouth.

Treatment.—An excessive degree of true hypertrophy of the tongue, resulting in interference with the taking of food, calls for the surgical removal of a wedge-shaped piece of the organ to prevent death from starvation. The cretin's tongue, no matter how large, can always be reduced by the proper administration of thyroid gland, and the tongue with a mild degree of macroglossia is usually accommodated by the natural increase in size of the oral cavity.

DISEASES OF THE MOUTH.

ALVEOLAR ABSCESS.

Many children are allowed to reach the age of eight or ten years before the practice of brushing the teeth is insisted upon, and, as a

result, caries of the teeth is very common during childhood. Caries is followed by infection and inflammation, perhaps by an abscess at the root of the tooth which causes great pain and swelling of the face and jaw. The breath becomes foul. In addition to local signs and symptoms, there are often disturbances of digestion and slight fever from inability to masticate the food properly.

Treatment.—Relief of pain is usually the immediate indication, and this may be met by the application of hot poultices externally over the affected jaw, and by painting the gums hourly with equal parts of tincture of iodin and tincture of opium. If fluctuation can be detected the gums should be incised, as, if allowed to remain unopened, the abscess usually breaks, and the pus is evacuated into the mouth. In some instances these abscesses have been known to rupture into the nose, antrum, or maxillary sinus.

Upon the detection of inflammation at the root, the best treatment by far is immediate extraction of the tooth, which relieves the condition at once. Following the evacuation of the pus, an antiseptic mouth wash should be employed for several days, for which purpose 20 per cent. hydrogen peroxide or 25 per cent. liquor alkalinus antisepticus will prove very efficacious.

ULCER OF THE FRENUM.

Ulceration of the frenum of the tongue is usually observed in weak infants, or as the result of whooping-cough. The ulcer is caused by the contact of the central incisors with the frenum during the act of coughing, this producing a shallow, clean-cut, sharp-edged ulcer on either side of the frenum. It is most frequently seen in children between one and two years of age, and occurs in younger children only after they have cut their central incisors.

Treatment.—The ulcers generally tend to heal spontaneously in the course of a week or ten days; but healing is considerably more rapid if a 2 per cent. solution of silver nitrate is applied daily to the ulcerated areas.

BEDNAR'S APHTHÆ.

Bednar's aphthæ is a symmetrical ulceration which occurs on each side of the palatine ridge over the hamular process of the palate bone, usually in very young infants, and most commonly between the second and third months. It is practically always due to traumatism from too vigorous cleansing of the mouth, which produces abrasions of the mucous membrane. At this particular point these abrasions quickly become ulcers because of the poor local circulation and the tense condition of the mucous membrane. Bednar's aphthæ has been known to follow thrush, and may also be caused by the use of an improperly shaped nipple, but is never due to syphilis.

Symptoms.—The lesions are oval and shallow, usually bean-shaped, and are covered with a grayish necrotic membrane which is very

adherent, and, if removed, reveals a smooth, reddened base. Nursing is so painful that the infant may take the breast for only a few minutes at a time or refuse to nurse at all.

Treatment.—These ulcers should be prevented by cleansing the mouth with the greatest care in order to avoid bruising the mucous membrane, and if a badly shaped artificial nipple is used it should be changed and a proper one substituted. The ulcers should be touched daily with a 10 per cent. silver nitrate solution, and the mouth must be kept clean by following each feeding with a tablespoonful of water. In an ordinary case of Bednar's aphthæ the ulcers usually heal up in the course of a few days.

CATARRHAL STOMATITIS.

Catarrhal stomatitis is an extensive inflammation of the mucous membrane of the mouth, occurring usually during the first two years of life. As a rule, it is due to local irritation from too vigorous cleansing of the mouth, but it is also seen in association with thrush, occasionally during dentition, and in acute contagious diseases.

Symptoms.—The inflammation is at first usually localized in some particular area of the oral mucous membrane, but shows a tendency to spread and involve the whole mouth. The inflamed area is intensely engorged, reddened, and sharply demarcated from the surrounding mucous membrane. As the disease increases in severity the gums become swollen, the tongue is coated, and, although there is an increased secretion of saliva, the mucous membrane of the mouth is hot and dry. Tiny, white, raised dots stud the mouth, tongue, and gums, representing the muciparous follicles, with here and there a patch, grayish-white in color, overlying an affected area of the surface of the mouth. Pain may be so great as to cause the child to refuse its nourishment, and become ill and fretful for a few days with a slight fever; but, as a rule, there are no constitutional disturbances. In some instances the submaxillary glands become enlarged; but the inflammation is rarely severe, and there is never tissue necrosis or ulceration.

Prognosis.—In healthy children this is very favorable, the inflammation disappearing within a few days under proper treatment. Delicate children may become seriously ill from lack of nourishment and gastro-intestinal disturbances, but usually recover in ten days or two weeks.

Treatment.—Cleanliness of the mouth is of more practical value in the treatment of stomatitis than any other procedure. The mouth should be washed out after each feeding with a solution of sodium bicarbonate and sodium borate, using fifteen grains of each to the ounce of water; or, if preferable, a 25 per cent. solution of liquor alkalinus antisepticus may be used, usually with prompt and satisfactory results. If the inflammation tends to persist, the mouth may be swabbed with silver nitrate solution, 0.5 per cent., followed by a

mouth wash of cold water, and a pinch of boric acid or alum dusted on the tongue. If the mucous membranes of the nostrils and conjunctiva are also inflamed, they should be irrigated with the same alkaline washes as are used for the mouth. The child's nutrition must be kept up by full feeding, using gavage if necessary, and a child of one year should always be given an initial purge of two drams of castor oil.

APHTHOUS STOMATITIS (HERPETIC STOMATITIS).

This form of stomatitis is characterized by the formation of small, round, yellowish, superficial ulcerations on the mucous membrane of the lips, cheeks, palate, gums, and tongue, and is always accompanied by a catarrhal stomatitis.

Etiology.—Aphthous stomatitis occurs most frequently during the first three years of life. The actual cause is unknown; a nervous origin has been suggested, but is improbable. Some observers believe it to be an infection derived from the bowels, and caused by toxins generated in contaminated milk; while other authorities regard it as a local infection of the mouth. The theory of the infectious nature of aphthous stomatitis is, to my mind, the most probable. From this stand-point we may regard lack of proper hygiene of the mouth as a strongly predisposing factor, since aphthous stomatitis is most common during that period of infancy when the child crawls over the floor on hands and knees, and puts every object it grasps into the mouth.

The French theory of a relation between aphthous stomatitis and the foot-and-mouth disease of cattle which is transmitted to the child through the medium of cow's milk I consider very obscure, and there is no evidence of the spread of this disease from one child to another. Most cases are seen in connection with some gastro-intestinal disorder, or with one of the acute infections of childhood; but bacteriological examination of the oral secretions and the scrapings from the mouth fail to reveal any organisms other than those found normally in the mouth.

Symptoms.—The initial lesions are small macules, usually found in the anterior part of the mouth, which become vesicles. These vesicles break and leave small ulcers, at first of a grayish-white color, later turning yellow or grayish-yellow in the centre, and having a dark red areola. Ulceration is due to the death of the epithelium, which becomes elevated above the vesicle by an exudation in the mucosa, therefore the ulcers are very superficial. They vary in size from pin head spots to patches as large as a split pea, and, in some instances, are even larger as the result of the coalescence of several smaller ulcers.

Any part of the mouth may be involved, but ulceration is most common on the tongue and inner surface of the cheeks. As a rule not more than a dozen ulcers are visible at one time, although fresh ulcers may appear as others heal. In no instance does the necrosis

of tissue extend further than the mucous membrane, healing always taking place without scar formation.

The gums are swollen, the tongue coated, the mouth is hot and dry, although there is hypersecretion of saliva. Pain is sometimes very severe, which discourages the taking of food. There is usually a somewhat higher fever than in simple acute catarrhal stomatitis, and the child is dull, drowsy, fretful, and restless. The stomach and bowels become affected, there is diarrhea, and sleep is broken by intervals of restlessness; so that, if allowed to persist very long, aphthous stomatitis may precipitate a serious illness. Enlargement of the submaxillary glands is a common finding, but they never suppurate, although there is sometimes pain on moving the jaw.

Prognosis.—The prognosis of aphthous stomatitis is good, and the disease ordinarily runs its course in from one to two weeks, ending in spontaneous recovery. The ulcerations do not heal so quickly in weaklings and marasmic infants, and if vigorous treatment is not instituted, a mixed infection may take place which will render the case much more serious.

Treatment.—The treatment of aphthous stomatitis differs but little from that of the acute catarrhal variety. The ulcerations tend to heal more quickly if touched with a 2 per cent. solution of silver nitrate twice daily, following each application by a mouth wash of cold water. After each feeding the mouth should be washed with a 25 per cent. solution of liquor alkalinus antisepticus, or an alkaline wash containing 15 grains each of bicarbonate of soda and borate of soda to the ounce of water. A 25 per cent. solution of hydrogen peroxide used three times a day as a mouth wash is very effective in clearing away the grayish-white and yellow patches of necrotic epithelium. As in the treatment of catarrhal stomatitis, an initial dose of two drams of castor oil to a child of one year should be given, and the nourishment kept up as fully as possible. Because of the hot and dry condition of the mouth, food will be taken much more readily if chilled before it is given, and the sucking of small pieces of ice affords great relief.

THRUSH—SPRUE.

Thrush is a mycotic infection of the mucous membrane of the mouth, seen most frequently in children of the poorer classes, and is regarded as indicating a poor state of the nutrition and general health rather than the direct result of poor oral hygiene. The fungus, oidium albicans, may be recovered from scrapings of the mouth in every case. It occurs in the yeast form and the mycelium, and under the microscope is revealed as long filaments, which frequently branch and unite to form chains, at each intersection of which will be a rounded cell, or several cells, containing spores. This fungus also propagates by filaments from conidia, and from isolated conidia. In certain respects it resembles the mould fungus and the yeast fungus, but cannot be satisfactorily grouped with either class. As a rule, the

growth is confined solely to the mouth, but may extend to the nose, pharynx, larynx, or even to the stomach, although the gastric mucosa offers a very unfavorable site for thrush, since this fungus thrives best on a dry surface.

Etiology.—Infection with the oïdium albicans may come from a variety of sources, but the most important predisposing factor is a devitalized condition of the infant or child. The fungus, itself, is air borne; it has been found in the vaginal secretion, on the nipples of the breast, and has been isolated from the mouths of normal infants. Since thrush is most common in artificially fed babies, it is probably carried into the mouth in food and on artificial nipples, which have not been properly cleansed and sterilized. Although the devitalized mucous membrane in the mouths of weaklings and marasmic children is most susceptible to thrush infection, yet there must be an abrasion or break in the continuity of the surface of the mouth, such as would be produced by the contact of an improper nipple, before the fungus can find lodgment and grow. The disease is most prevalent in hospitals and foundling asylums, and in many institutions can be held in check only by careful oral hygiene. It is rarely seen before the baby is six months old.

Symptoms.—The first manifestations of thrush are tiny white spots like curds of milk which appear on the tip of the tongue, the mucous membrane of the cheeks, and on the gums. These patches are adherent, grayish-white in color, elevated above the surrounding tissue, and vary in number and size from a comparatively few small patches, in mild cases, to a number of large areas formed by the coalescence of many smaller lesions. There may or may not be any appreciable inflammation of the surrounding mucous membrane; but, as a rule, there is a mild stomatitis, the mouth is dry, and the gums slightly inflamed although there is rarely any pain apparent to interfere with the taking of food.

Gastro-intestinal disturbances are usually present at the height of the disease, and give rise to nausea, vomiting, diarrhea, and slight fever; only, however, in very weak and anemic children do actual lesions of the stomach occur, and these cases usually end fatally. When the growth extends merely to the nasopharynx and esophagus, a more favorable outcome may be expected, although the disease may persist for a greater length of time than when the mouth alone is involved.

Diagnosis.—The diagnosis of thrush is, as a rule, easily made when the characteristic grayish-white patches appear throughout the mouth, accompanied by a very mild stomatitis. If any doubt exists attempts should be made to obtain scrapings from the mouth, and these, when examined under the microscope, reveal the presence of the oidium albicans. On casual inspection one may mistake the small white patches scattered over the mouth for milk curds; but their firm attachment to the mucous membrane shows their true nature, while their elevation above the surrounding surface with an

absence of ulceration serves to exclude the more serious forms of stomatitis.

Prognosis.—Thrush in the normal healthy infant is of little consequence, and usually disappears promptly under appropriate treatment. It is the poorly nourished child, in whom thrush is complicated by severe gastro-intestinal disturbances, or in whom it appears as a complication of an already serious digestive derangement, that suffers greatly, or perhaps dies, from an attack of mycotic stomatitis.

Treatment.—Prophylaxis is of paramount importance. It consists in absolute cleanliness, not only of the infant's mouth, but of everything that comes in contact with the child's mouth or food. Thus, the nurse's hands, the mother's breast, the nursing bottle, and the nipple should be kept as nearly sterile as possible, and the mouth should be cleansed after each feeding by giving the infant a tablespoonful of water.

These precautions should be instituted immediately on the appearance of thrush; in addition, the mouth should be washed out three times daily with a saturated solution of boric acid, using a cotton swab when attempting to remove the various patches, but this must be done very carefully, lest the mucous membrane be abraded. When there are abrasions or superficial ulcerations, a 2 per cent. solution of silver nitrate should be applied daily. If, despite treatment, healing is delayed, it is advisable to discontinue the use of an artificial nipple, and substitute dropper feeding or gavage until there is improvement.

Gastro-intestinal complications should be met by careful regulation of the diet, restriction of the amount of food, and an initial purge of castor oil, two drams to a child of one year. The following prescription is useful, both for its tonic effect and its local beneficial action on the mouth, and may be given with little reservation, regardless of the condition of the stomach:

℞—Tinct. ferri chloridi	. . . .	ʒj
Potassii chloratis	. . . .	gr. xxx
Glycerini	. . . .	ʒiv
Aqua	. . . . q. s.	f ℥iij
Sig.—ʒj in aqua every three hours.		

ULCERATIVE STOMATITIS.

Ulcerative stomatitis is one of the most severe inflammations of the mouth seen in childhood, and occurs chiefly in the children of the poor, neglect being an important factor in its etiology. It does not appear until dentition has been established. The ulcerative process is always first noticed at the line of junction of the gums and teeth, the gums of the lower jaw being most commonly the site of the first ulcers, which rapidly extend along the teeth, and may involve the alveolus.

Etiology.—Ulcerative stomatitis is very rare in private practice. The majority of cases are observed in dispensary patients and inmates

of hospitals and asylums; therefore, lack of proper oral hygiene, together with a poor state of general health, may be considered an essential factor in its production. That improper diet also may be a cause is apparent from the fact that it accompanies scurvy. It may also occur as a sequel of typhoid fever, pneumonia, and the acute contagious diseases of childhood. Formerly, ulcerative stomatitis was not infrequently due to the ingestion of metallic poisons, among which mercury, lead, and phosphorus were the most common; but with the exception of mercurial poisoning, this is now quite rare.

Carious teeth form, perhaps, the most frequent exciting cause; but there must also be a devitalized condition of the gums, due to the poor physical condition of most of these children. Some observers believe this form of stomatitis to be contagious, and claim that it may be transmitted, but this theory is not borne out to any great extent, although epidemics of ulcerative stomatitis have been observed. Bernheim and Pospischill made a bacteriological study of a number of cases, and from all but two of them a fusiform bacillus, resembling the bacillus of diphtheria, and a spirillum were isolated, both being present in each case, one or the other always predominating.

Pathology.—At the onset of this disease the gums, usually about the lower incisors, become swollen and red; as the swelling increases, the teeth may be almost covered by the gums, which become very spongy, are of a dark red color, and bleed when touched. Ulcerations now form at the junction of the gums and teeth, spreading quickly along the whole line of junction, but usually confined to one jaw.

In severe cases the teeth may become exposed and loosened, the lips and cheeks ulcerated; but the process is always limited to the oral cavity, the entire buccal mucous membrane showing an acute catarrhal inflammation. The junction of the gums and teeth is usually represented by a ridge of yellowish necrotic granulations, bathed in a mucopurulent exudation. In some cases the tooth sockets may become involved, and the necrotic process extend to the periosteum of the alveolar process, and even to the jaw-bone.

Symptoms.—Pain is usually so severe when food is taken into the mouth that feeding is quite difficult. The tongue is coated, the breath foul, salivation increased, and neglected cases frequently show an eczema of the lips due to the constant dribbling of blood-streaked saliva mixed with pus. In very young children, there is usually moderate fever, and because of lack of nourishment they become restless, irritable, much weakened, and exhausted. On inspection of the mouth the swollen, inflamed, and bleeding gums are plainly visible; careful investigation may reveal loosened teeth and, perhaps, other ulcerations on the inside of the lips, cheeks, and even upon the palate and tonsils. It is only in most severe cases that the alveolar periosteum and the jaw-bone are found to be necrotic.

Diagnosis.—The diagnosis is, in most instances, readily made from the condition of the gums and the extremely foul breath. The mild-

ness of the constitutional symptoms, in comparison with the severity of the local affection in the mouth, is also an aid in diagnosis.

Bednar's aphthæ may be suggested, but if one recalls the fact that in this rare affection the ulcers are found only on either side of the raphæ over the hamular process of the palate bone, no mistake will be made, since in ulcerative stomatitis the ulcerations may be found anywhere within the mouth. Gangrenous stomatitis may be differentiated from the ulcerative form by the severity of the constitutional symptoms, and the localization of the lesion to one particular area.

Prognosis.—In this disease, as in the other forms of stomatitis, the course and prognosis depend to a great extent upon the vitality of the child. Fairly well-nourished children should show improvement within a week after treatment is instituted; but anemic and marasmic infants may not recover from ulcerative stomatitis for months although, in the majority of cases, the final outcome is favorable.

Treatment.—In the treatment of ulcerative stomatitis, the first consideration should be directed to finding the cause of the attack, and removing it. The mouth should be kept absolutely clean by the use of antiseptic washes and cleansing agents such as a 25 per cent. solution of hydrogen peroxide, or 1 to 5000 solution of potassium permanganate, or a saturated solution of potassium chlorate. The ulcerations should be touched daily with alum, or a 10 per cent. nitrate of silver solution.

Potassium chlorate is also valuable given internally in ulcerative stomatitis, if its administration is properly carried out. My plan has been to give a child of two or three years two grains every two hours the first day, and to reduce the total daily quantity one-half each succeeding day, as improvement is observed. If necrosis of the jaw is suspected, the loosened teeth should be extracted, the jaw-bone carefully examined, and treated surgically if necessary. Because of the poor physical condition of these children, they should be placed in the most healthful environment, with fresh air, sunshine, and nourishing food in abundance. If due to scurvy, orange juice should be given daily; if anemia be marked, full doses of the syrup of ferrous iodide, or an amount of iron equal to this in any other form, will materially promote recovery.

GANGRENOUS STOMATITIS.

Gangrenous stomatitis, cancrum oris, or noma is a rare disease of the mouth which affects children of the poorer classes, and is characterized by the appearance of a small inflammatory spot on the cheek which quickly becomes necrotic, extends with tremendous rapidity, and may end fatally in a few days. This same disease is occasionally observed on the vulva, and more rarely on the anus and prepuce.

17

Etiology.—Gangrenous stomatitis is most frequently seen in institutions, being almost unknown in private practice. It is usually the sequel to a severe illness, perhaps one of the acute infections, particularly measles; a predisposing factor is debility from any cause. Although several organisms have been described in connection with cancrum oris, among them a fusiform bacillus, a spirochete, and the streptococcus, the specificity of any one particular germ has not been satisfactorily established. In many cases catarrhal or ulcerative stomatitis has been the precursor of the disease. It is seen most frequently in that period of childhood between the first and second dentitions, and is as common in boys as in girls.

Symptoms.—Following measles, or any other acute infectious disease, or a long debilitating illness which has been complicated by catarrhal or ulcerative stomatitis, the child with beginning gangrenous stomatitis is at once conspicuous by the foul gangrenous odor

Fig. 28.—Gangrenous stomatitis.

of the breath. On inspection of the mouth, a spot of beginning necrosis will usually be found on the inner side of one cheek, this particular area of mucous membrane being of a darker shade than the surrounding tissue. A bleb forms on the inside, and a corresponding brawny swelling on the outer surface of the cheek. The gangrenous spot increases rapidly in size, and the centre sloughs away, leaving a dark, necrotic, ulcerating surface which may result in perforation of the cheek.

In severe cases, the gums become necrotic, the teeth loosen and fall out, even the jaw-bone becomes necrosed. A fetid discharge covers the affected parts, and emits a foul, penetrating odor which is characteristic of the disease. As a rule pain is very slight, even with perforation, and thrombosis of the vessels at the margin of the ulcer inhibits bleeding.

The constitutional symptoms vary somewhat, but become severe;

fever, as a rule, is moderate; the child is dull, apathetic, extremely depressed or prostrated, and may become delirious because of the severe toxemia, further evidence of which is the extremely feeble action of the heart. The lymph nodes of the face and neck show general enlargement, diarrhea is always present, and the disease usually comes to a fatal termination by septic pneumonia.

Diagnosis.—The diagnosis of gangrenous stomatitis is easily made when the disease is well established; but, at the onset, the initial lesion is extremely difficult to differentiate from simple ulcer of the mouth. Anthrax may be excluded by the history of the case and by bacteriological examination of scrapings of the mouth.

Prognosis.—The prognosis of cancrum oris is unfavorable; few children survive; therefore only slight hope of recovery can be held out to the parents.

Treatment.—Prophylaxis is the most important element in any treatment which can be said to be effective. The mouth should always receive the utmost attention during the course of any debilitating disease. It should be cleansed very gently, for too vigorous cleansing which results in abrasions of the mucous membrane is more harmful than none at all. If ulceration appears, the mouth should be carefully inspected daily so that, at the first indication of approaching gangrene, the necrotic tissue may be widely excised. If necrosis be already advanced and extensive, the case is desperate; but an attempt may be made to cauterize the edge of the slough, going well into the living tissue at all points. For this purpose the actual cautery must be used, caustics and other cauterizing agents being of no value whatsoever.

Attempts have been made to stop the ravages of this disease by means of the x-ray, incandescent lamps, and injections of either the perchloride of mercury, carbolic acid, or tincture of iodin into the tissues at points in advance of the approaching necrosis; but radical surgical procedures, such as wide excision of the gangrenous tissue, multiple tooth extraction, and curettage of the jaw-bone have been more successful, when the condition of the patient warranted them. The mouth should be cleansed frequently with a 25 per cent. solution of liquor alkalinus antisepticus, and the ulcerated surface protected by a dressing.

Although sometimes very difficult to prevent it, the child's strength, if possible, should not be allowed to fail. It should be given the most nourishing liquid food in small quantities every two hours, and be kept in the sunshine and fresh air as long as the slightest hope of recovery is entertained. Stimulation is necessary in every case, and 20 or 30 drops of brandy may be given a child of two years, every two hours; if borne by the stomach, a half teaspoonful of the mixture of iron, quinine, and strychnine should also be given three times a day. In recent years, antistreptococcic and antidiphtheritic serum have been used to combat cancrum oris, but with little appreciable result.

PYORRHEA ALVEOLARIS.

Pyorrhea alveolaris is a subacute or chronic inflammation of the pericemental membrane, and may appear at any time after the eruption of the teeth. The exciting cause is always of bacterial nature but the primary underlying factor of pyorrhea in childhood is gastro-intestinal toxemia. Bacterial invasion is favored by irritation produced by the tartar deposited on the teeth and gums, and may also be brought on by trauma to the gums or by chemical and mechanical irritation.

Symptoms.—The onset of pyorrhea alveolaris is, as a rule, sudden, and pain is usually the first symptom. The gums become swollen, are painful on pressure, and the teeth are loosened by recession of the gum margin. On inspection of the mouth a purulent discharge may be observed to exude from the gum margin, and this accounts, in a measure, for the foul breath and coated tongue which these children present. In most instances this pus is swallowed, and by its presence either gives rise to fermentation of the stomach or to direct absorption of the toxins into the child's system. The effect of pyorrhea locally is to favor to a great degree the entrance and growth of many pathogenic organisms, particularly the bacilli of influenza and diphtheria.

Treatment.—The child affected with pyorrhea alveolaris should be sent to see a dentist in order that the teeth may be thoroughly cleansed and deposits of tartar removed, after which an antiseptic mouth wash composed of equal parts of hydrogen peroxide and extract of witch hazel should be used at least every two hours the first day or so, and less frequently thereafter. The diet should not be reduced in amount, but the food must be so soft that mastication is unnecessary, lest the gums become traumatized by the act of chewing.

When pyorrhea does not respond to the foregoing treatment, a culture should be obtained from the discharge, and an autogenous vaccine be made, the dosage of which would depend largely upon the organism grown and the effect of the treatment outlined upon the disease. Recent observations of the beneficial effect, in most cases, of the hypodermic administration of emetin hydrochloride, seem to recommend a trial of this therapeutic measure.

UVULITIS.

Acute inflammation of the uvula is rare during childhood, and practically unknown in infants. Children with congenital elongation of the uvula are especially predisposed to attacks of uvulitis; and, since each attack still further elongates the organ, recurrent uvulitis is quite common.

Etiology.—Uvulitis not infrequently occurs in association with gastro-intestinal disturbance and rachitis, and in rare instances may be produced by the ingestion of hot liquids, strong acids, or alkalies. The

most common cause of uvulitis is the extension of an inflammatory process in the pharynx or tonsils.

Symptoms.—The most distinctive symptom of uvulitis is a persistent unproductive cough, due to an attempt to relieve the irritation produced by the pressure of the uvula on the base of the tongue, fauces, or upper part of the larynx. There is also a constant desire to swallow, even though swallowing be painful, for the swollen uvula feels like a foreign body in the pharynx, and in severe cases it may attain such a size that the oropharyngeal opening is occluded, thus causing dyspnea and attacks of suffocation. When the swelling of the uvula reaches these proportions, it interferes with the taking of food, and the child may become weak and exhausted from lack of nourishment. Other constitutional symptoms are rare.

Diagnosis.—The diagnosis of uvulitis is very simple, and readily made by inspection of the mouth, which reveals the swollen, boggy, edematous uvula, very much altered in shape, and so enlarged as to be in contact with the base of the tongue and the fauces.

Treatment.—At the onset of inflammation, uvulitis may sometimes be aborted by swabbing the uvula every hour with a 1 to 20,000 solution of adrenalin chloride. After the uvula has become edematous, adrenalin has little permanent effect; but the swelling may be greatly relieved by the application of a 2 per cent. solution of tannic acid three times daily. A gargle of Dobell's solution should be used every three hours; if the child be too young to gargle, this solution may be sprayed upon the throat. Great relief will be afforded by allowing the child to suck small pieces of ice, and by cold applications to the neck.

In mild cases of uvulitis the foregoing measures are usually effectual; but when swelling of the organ becomes so great as to produce dyspnea, more radical treatment is often necessary. Multiple puncture of the uvula with a short bistoury or double-cutting aspirating needle is the quickest and most satisfactory method of relieving the tension, and is not attended by any danger. If the inflammation shows a tendency to chronicity, the tip of the uvula should be excised.

DISEASES OF THE ESOPHAGUS.

ESOPHAGITIS.

Acute inflammation of the esophagus is much more rare in children than in adults. Occasionally it is produced by the extension of inflammatory processes in the mouth or pharynx; but usually it is due either to the ingestion of strong acids or alkalies, or to the impaction of a foreign body which the child has attempted to swallow.

Symptoms.—The symptoms of acute esophagitis are largely dependent upon the cause. If there be but slight injury to the mucous mem-

brane, there is merely a little pain on swallowing and slight elevation
of temperature for a few days, but when strong acids or corrosives
have been swallowed the symptoms are most severe, and death may
ensue in a few hours.   The child is usually prostrated.   It vomits
shreds of bloody mucus.   Thirst is extreme.   There is severe burning
pain under the sternum, and every attempt to swallow causes such
suffering that the taking of liquids is exceedingly difficult.   If the
patient survives this period of acute symptoms, there still remains
the danger of edema of the glottis which sometimes comes on during
the succeeding two days.   Not until a year has elapsed may we feel
sure that no ill effects will follow; for stricture of the esophagus may
appear at any time from two weeks to several months after the
accident.

Treatment.—The mild form of esophagitis heals spontaneously
within a few days, hence treatment is unnecessary except the restric-
tion of the diet to bland liquids at a moderate temperature.   Cases
due to poison should receive antidotes immediately, and an effort
be made to empty the stomach.   Stimulation is often necessary, and
the strength may be supported by hypodermic injections of morphine
sulphate, gr. $\frac{1}{20}$, and atropine sulphate, gr. $\frac{1}{150}$ (for a child of five
years), also by the rectal injection of brandy, ℥ij, in an ounce of black
coffee.   This may be repeated every three hours until all danger from
collapse is passed, and then the child should be closely watched for
signs of edema of the glottis.   Nourishment should be kept up by
means of nutrient enemata, and codeine sulphate, gr. $\frac{1}{10}$, may be
given for the relief of pain.   If stricture occurs as a sequel, surgical
intervention is necessary.

RETROESOPHAGEAL ABSCESS.

Retroesophageal abscess is a very rare affection, caused either by
the breaking down of the retroesophageal lymph nodes or by the
extension of a suppurative process due to Pott's disease.   It is most
frequently seen in association with tuberculosis, but may follow
measles, scarlet fever, or diphtheria.

Symptoms.—Among these are an irritating spasmodic cough and a
decided change in the voice.   The breathing is stertorous.   Dyspnea
occurs spasmodically, and is most marked on inspiration.   The neck
is swollen externally, and all the cervical glands are greatly enlarged.
The temperature often runs up to 102.5° F. or above, and the pulse
and respiration are accelerated.

Prognosis.—In retroesophageal abscess the prognosis is unfavorable.
Most cases die from pressure on the pneumogastric nerve or rupture
of the abscess into adjacent structures.   An instance is reported of
recovery after rupture of such an abscess into the esophagus.

Treatment.—The treatment is surgical; the abscess should be opened
and drained.   If the child is tuberculous, it should be removed from
any crowded surroundings and sent to the seashore.   The diet should

PLATE II
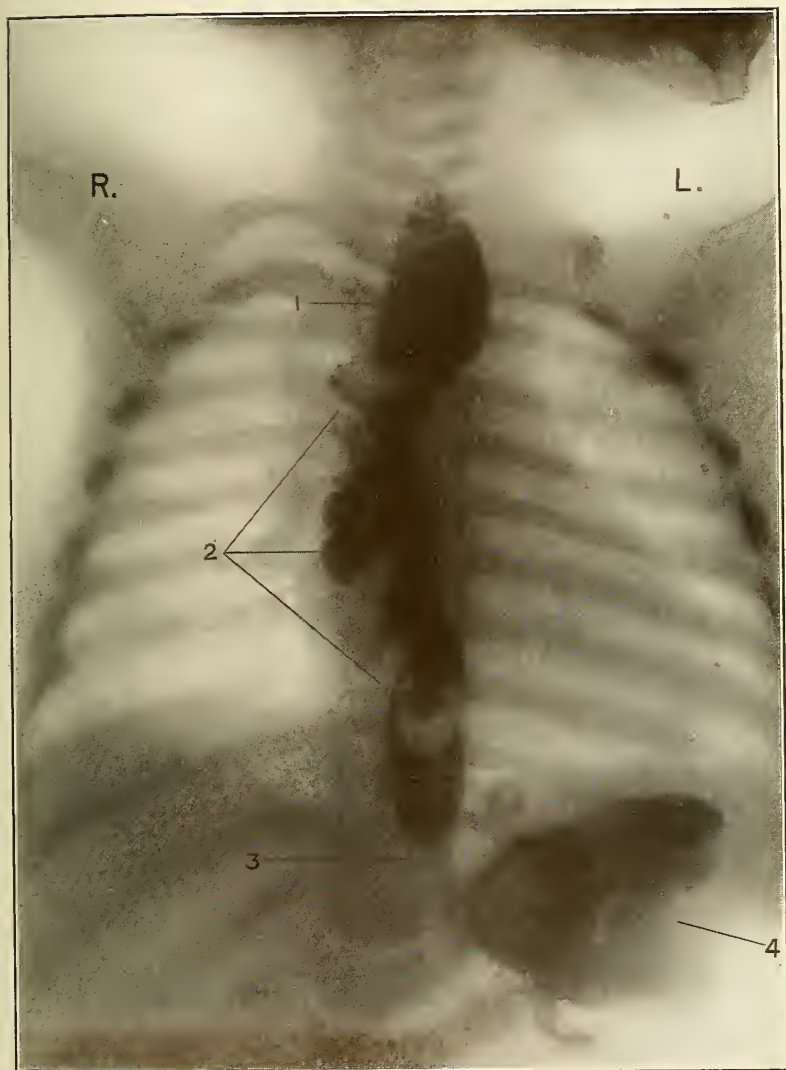
Stricture of the Esophagus in a Child Aged Two and a Half
Years, due to Swallowing Lye.

R., right side; L., left side. 1, dilated portion of esophagus above
constricted portion; 2, marked irregularity in lumen of esophagus;
3, narrowed portion of lumen; 4, the stomach.

be so adjusted as to consist of most nourishing food, and a tonic, such as cod-liver oil or the syrup of the iodide of iron, may be given if the stomach is not upset by this medicine.

DISEASES OF THE STOMACH AND INTESTINES.

VOMITING.

Vomiting, although merely a symptom, occurs so frequently in infancy and childhood, and arises from such a variety of causes, that special discussion of this subject is warranted.

In Early Infancy.—The newborn infant may vomit immediately after each nursing, even though perfectly healthy. This is usually caused by the infant swallowing the breast milk too quickly or by overfeeding, and is a conservative measure on the part of the stomach to prevent overtaxation of the digestive organs. Various reasons have been adduced to explain this symptom, and it is probable that other factors may be responsible for vomiting in the breast-fed infant, since regulation of the quantity and the time of feeding does not always correct the condition.

The stomach of an infant, lying as it does in an almost upright position so that it forms a nearly continuous line with the esophagus, is easily emptied by slight pressure such as might be created by the movements of the diaphragm during respiration. There is neither nausea nor epigastric pain in this form of vomiting; the milk usually returns only slightly changed or curdled; and, although the condition may persist, no appreciable loss of weight results. It is always well to investigate the feeding of an infant in whom this form of vomiting is observed; but if correction and regulation of the nursing do not relieve it, no further treatment can be instituted, and the condition may with safety be allowed to continue, as it will cease spontaneously in due time.

Symptomatic Vomiting.—Vomiting in later infancy and in childhood is always significant of some disorder, and should never be regarded or treated as an independent affection. In extremely few cases it may be said to have become a habit, the attack being apparently a voluntary act on the part of the child during a crying spell or fit of anger. By far the most common cause of vomiting is indigestion, either chronic or acute, and when due to this cause it is accompanied by pain in the stomach as well as nausea. The vomited material indicates clearly the source of irritation, for it is composed of undigested sour-smelling food or curds of milk. This form of vomiting should receive careful attention and treatment from its very beginning, since it is indicative of digestive disturbances which, if allowed to continue, may become chronic.

In young children any cough, if at all severe, will produce vomiting, and in pertussis, especially, the child vomits at each paroxysm. Reflex vomiting is also provoked by irritation of the pharynx from an elongated nipple, by the habit of hand sucking, or by eye-strain, earache, dentition, or intestinal worms. The projectile vomiting of meningitis is reflex in character, and although it commonly appears at the onset of the illness it rarely recurs. The same form of vomiting is observed in cases of brain tumor and in cerebellar disease, but without other signs of digestive disturbance.

Aside from indigestion, perhaps the most common cause of vomiting is intestinal derangement, which need not necessarily be of inflammatory nature, since it often results from the absorption of toxins in the upper and lower bowel. Vomiting is often the first symptom of intestinal obstruction, whether from volvulus, intussusception, or fecal impaction, and persists until the obstruction is relieved, becoming stercoraceous in the end. In appendicitis also it is a prominent symptom at the onset, but does not continue throughout the attack unless peritonitis sets in, and in peritonitis it invariably indicates extreme irritability of the visceral peritoneum. Obstruction higher up in the alimentary canal, and commonly at the pylorus or esophagus, is also accompanied by vomiting which, however, is more of the nature of regurgitation. In pyloric stenosis particularly, there is constant regurgitation of small quantities of food with intermittent periods in which the amount of vomitus is far in excess of the quantity of food taken at the preceding nursing, since it represents the residue of several previous feedings which have been retained by the greatly dilated stomach.

Some children, and many infants, vomit whenever there is an elevation of the body temperature of 2° or 3°, regardless of the cause of the pyrexia, and, in most of the acute infections, vomiting is one of the premonitory symptoms. There undoubtedly occurs a neurotic form of vomiting which is induced by excitement, fright, fear, or fatigue, especially in children with a tainted nervous heredity. There are other, and trivial, causes of vomiting which have not been discussed; but, before attributing vomiting to some slight incident or occurrence, the patient should be thoroughly examined for signs of one of the grave diseases which may possibly be present.

Cyclic Vomiting.—Cyclic vomiting is comparatively rare and is peculiar to children. As the term implies, there are periodical attacks of vomiting which occur at intervals of weeks or months, without other signs of gastric disturbance. This condition is of such importance that it is described as a distinct disease, although it has not been proven to be a primary affection of the stomach. During the attacks the stomach is extremely intolerant to food of any kind or even water, and prostration is sudden and extreme.

Etiology.—The direct cause of cyclic vomiting is unknown, but from observation of a number of cases it is evident that a variety of factors are operative in its production. Feeding seems to be of minor

importance, since the affection is observed in children whose diet is ideal; hence it may be assumed in the majority of cases that the impulse arises from a source outside the stomach, in further support of which assumption is the apparent absence of gastric derangement in the intervals between the attacks. A great many children who suffer from cyclic vomiting are of a neurotic disposition, and since it practically occurs only during childhood, when the nervous system is as yet unstable, it is quite possible that a nervous element is active in its causation.

The fact that in a number of instances acetone and diacetic acid have been demonstrated in the urine of these patients, and that the breath has had a pear-like odor, has firmly supported the view that cyclic vomiting is a result of acidosis. This theory is further borne out by the effects of alkaline treatment. It is quite probable that in cyclic vomiting we have the symptom-complex of gastro-intestinal lithemia due to an increased acidity of the body fluids as a result of disturbed metabolism, and the importance of gastro-intestinal auto-intoxication as a causative factor cannot be overlooked, since in many of these children there is a history of constipation for long periods before each attack.

Symptoms.—An attack of cyclic vomiting usually lasts from one to three or four days, during which time there are intervals of from three to six hours in which vomiting ceases, only to be repeated, until the attack is over. The vomitus, at first, is composed of undigested food, although little or no other evidence of indigestion is present; later the vomited material contains mucus which may be tinged with blood or bile. The child becomes extremely thirsty, but ejects every drop of water swallowed; consequently, after a few attempts, drinking is abandoned and the little one simply lies quietly on its back. There is occasional complaint of pain in the stomach, both before and during an attack, and constipation is usually present. The temperature, as a rule, is normal or subnormal. The pulse at first is slightly accelerated and strong, but becomes weak when exhaustion sets in.

After two or three days the attack may suddenly cease or, in some instances, the vomiting gradually decrease in severity and frequency until it stops altogether, when the child quickly regains strength, and may be perfectly well for weeks or months until another attack supervenes. As a rule, the attacks cease entirely at puberty, although rare cases have been reported in which cyclic vomiting continued to recur until adult life.

Diagnosis.—Unless one can obtain a history of preceding periodical attacks, the diagnosis of cyclic vomiting is difficult, and should only be made after careful exclusion of the more common causes of vomiting, and in the absence of any signs or symptoms of gastro-intestinal derangement. Among the many causes of vomiting which must be excluded are tuberculous meningitis, volvulus, intussusception, and appendicitis. Absence of fever is significant as eliminating inflam-

matory lesions of the gastro-intestinal tract, and a rapid pulse and good physical condition of the child usually exclude tuberculous meningitis.

Prognosis.—The prognosis of cyclic vomiting is, on the whole, favorable, since the majority of children survive the most severe attacks, and quickly regain their normal health and vigor when the vomiting ceases.

Treatment.—When an attack of cyclic vomiting comes on, the child should be put to bed, and for twenty-four hours nothing should be given it by mouth but a few sips of water. It is a good plan to give an enema as soon as an approaching attack is suspected, and the injection may be repeated once or twice daily until vomiting ceases. After twenty-four hours the very lightest nourishment, preferably albumin water or weak broth, may be allowed in small quantities, and, if this is not retained, nutrient enemata may be resorted to. If fluids do not excite vomiting, soft food may be given the child on the following day, and full diet gradually resumed.

For the control of vomiting, morphin and codein are the most efficient drugs, but the dosage should be carefully graduated according to the age, because of the idiosyncrasy of children to opiates. In view of the fact that acidosis is frequently responsible for cyclic vomiting, an alkali, such as sodium bicarbonate, which may be given in 10- to 20-grain doses every hour, will often bring about excellent results. Older children may be given larger doses; and, if the infant or child does not retain this drug when given by mouth, twice the dose should be administered by rectum. In the course of a day the infant should receive 1 dram, and the child 2 drams, if the drug is to have any appreciable effect.

The diet of these children should be very much restricted in its carbohydrate content because of the acidosis and evident relation between cyclic vomiting and inefficient carbohydrate metabolism. Salt water bathing and massage are valuable adjuncts to the treatment. In addition to the specific measures outlined above, these children often improve if given the advantage of hygienic surroundings. As a prophylactic measure against future attacks, the bowels should be so regulated as to make sure of a daily movement.

GASTRALGIA.

Gastralgia is neuralgic pain in the abdomen, usually located in or about the stomach, and due to irritation of the sensory gastric nerves. It is quite common in infancy. Little or nothing is definitely known as to its etiology, since it occurs regardless of whether the stomach is empty or full; therefore it is apparently reflex, and due to external, constitutional, or visceral disturbances.

Symptoms.—The attacks usually come on suddenly, and may last for a few minutes or several hours, during which time the patient may be prostrated. Vomiting is rare. The epigastrium shows no

sign of tenderness upon palpation, and recovery is prompt when the pain ceases.

Diagnosis.—The diagnosis of gastralgia should always be held in reserve until every other possible cause of abdominal pain is excluded. This involves careful and thorough history taking, as well as physical examination, especially in very young children who are unable to locate the pain.

Treatment.—The child should be put to bed, and a hot application, such as a mustard plaster, hot water bag, or a turpentine stupe, may be placed over the epigastrium. Internally, hot water with 5 to 10 drops of spirits of chloroform, or 10 to 30 drops of brandy or gin, or a teaspoonful of peppermint water, will often give relief. If these measures fail and the condition grows worse, the child should be closely watched for signs or symptoms of more serious nature.

Children who suffer from repeated attacks of gastralgia need a carefully regulated diet, and their bowels should be moved at least once daily. The administration of Fowler's solution, 1 drop doses three times a day, to a child of five years, and smaller doses to younger children, gradually increased to the point of tolerance, is of great value in the prevention of subsequent attacks, if kept up throughout the interim. Tincture of nux vomica, in 2 drop doses, may also be given, either alone or in combination with arsenic. These children should spend most of their time in the open air, but overexercise must be guarded against, as fatigue is very harmful.

INDIGESTION.

Fat Indigestion.—Indigestion during infancy, both in the nursling and in the bottle-fed baby, is quite often to be attributed to an excess of fat; and since in infancy the digestion of fat is accomplished for the most part in the small intestines, the symptoms of fat indigestion are chiefly referable to the intestines, and are to be discerned in the stools. Vomiting is a common symptom in these cases. In the infant's stomach a fat-splitting ferment is present.

Etiology.—Indigestion caused by an excess of fat in the ingested milk is far less common in the breast-fed infant than in the bottle-fed baby; but the nursling may also suffer from fat indigestion because of an idiosyncrasy to fats. This, however, is quite rare.

In another case the mother, believing that her milk is poor, may give her child a teaspoonful or two of cream with each nursing under the erroneous impression that the more fat the baby takes the fatter it will grow. As a result, the child ingests an amount of fat far in excess of its actual need or digestive capability. In artificially fed infants fat indigestion is quite common because of the prevailing tendency among physicians, when making up formulas, to prescribe an excessive percentage of fat rather than one too low.

Symptoms.—The breast-fed infant rarely suffers much from an excess of fat, even though the mother's milk be too rich; for, if exces-

sively fat, regurgitation follows each feeding, and the child usually soon becomes able to digest a slightly higher percentage than normal. In addition to regurgitation the baby loses its appetite, this also being a conservative measure on the part of Nature to limit the quantity of fats ingested. The bowels become loose, the stools contain fat curds and fat-free globules, and there is usually much flatulence and colic.

Continued disturbance in the digestion of fats results, first, in a failure to gain weight, and, subsequently, in loss of weight; but, as a rule, in the nursling fat indigestion is not attended by any serious impairment of health. The bottle-fed baby, however, usually fares worse. The symptoms of fat indigestion already enumerated become exaggerated, and, in addition, high fever generally accompanies the acute digestive disturbance. The stools may be either very loose, green in color, and composed of curds and mucus; or very watery and acid in reaction; or dry and well formed, and of a whitish or grayish color—the "soap stool," formed by a combination of fat and an alkaline salt. In some instances the loss of alkaline salts in the stools may be so great as to cause a relative acidosis with the characteristic symptoms of acid intoxication, such as increase in the respiratory rate, stupor, or extreme restlessness.

If acute fat indigestion is not relieved within a short time it soon becomes chronic, and, not only is the digestive power of the infant impaired, but other disturbances of metabolism arise and result in rachitis, infantile atrophy, or "marasmus." Loss of weight is progressive and continuous, and even though the child be given excellent care recovery is at best a slow and tedious process.

Treatment.—When indigestion is due to an excess of fat in the mother's milk, the amount taken at each feeding should be reduced by shortening the nursing period. We can also lower the fat content of the breast milk by giving the baby a small quantity of water immediately before it nurses. If the infant is losing weight because of insufficient food, the interval between feedings may be shortened so that the child will get the same amount of nourishment at each feeding as it previously received, but be fed more often in the twenty-four hours and, consequently, receive a larger amount of food.

The percentage of fat in the mother's milk may also be to some extent decreased by reducing her diet, especially with regard to protein, and by increasing the amount of exercise she takes. Since the milk obtained when the breast is almost empty is richest in fat, it is advisable to let the infant nurse only half the contents of each breast rather than to take the full feeding from one breast. If these measures fail to adapt the breast milk to the infant's digestive powers, it is sometimes necessary to procure a wet nurse.

In these cases where the mother has been in the habit of giving the child a teaspoonful or so of cream in addition to the breast milk, it is a simple matter to relieve fat indigestion; for on discontinuing the practice the condition soon passes away. Intolerance to fat is

fortunately very rare; but, when encountered, the sole resource is skimmed milk, and it is only with greatest difficulty that the caloric requirements of the infant can be supplied.

When fat indigestion arises in artificially fed infants, a fat-free diet should be instituted, and, for two or three days at least, no fats whatever be allowed. After this time a small amount may be added to each feeding, and the percentage of fat gradually increased from time to time, according to the degree of tolerance established.

An indication as to the proportion of fat being digested is readily furnished by observation of the stools, which should be carefully examined whenever the amount of fat is to be increased. Any evidence of excessive fat ingestion calls for immediate reduction in the percentage of fat, as intolerance is very quickly precipitated; and it is much safer to increase the amount of protein and carbohydrates if the formula be too weak, although it is difficult to furnish in this way an equal number of calories without setting up digestive disturbance. When there is an idiosyncrasy to the fat of cow's milk, a wet-nurse should be secured, since, as a general rule, in these cases human milk is well borne, whereas it is impossible to change the character of the fat in cow's milk or to modify it in any way.

In the summer months many cases of fat indigestion may be prevented by reducing the percentage of fat in the feeding mixtures, as even healthy infants show a greater intolerance to fat at this season of the year. In the treatment of fat indigestion it is usually wise to give an initial purge, castor oil in full dosage being most effective. If the case be very severe, the stomach should be given a rest by withholding all food for from twelve to twenty-four hours after purgation.

Carbohydrate Indigestion.—Sugar indigestion in the breast-fed infant is quite rare, but is readily induced in artificially fed babies, either because they have ingested an excessive amount of carbohydrates or have taken an unsuitable form of starch or sugar.

Etiology.—The amount of sugar in human milk seldom varies more than 1 or 2 per cent. As a rule, this excess is readily digested by the healthy infant. Only in exceptional cases do digestive disturbances arise, and they are usually very mild, being marked by vomiting, diarrhea, eructations of gas, and more or less colic. The stools are thin, green in color, acid in reaction, and they frequently irritate and excoriate the buttocks. When carbohydrates are given in addition to the breast milk for their laxative effect, or mixed feeding is resorted to, and an excess thus received, the symptoms are more severe. In artificially fed babies sugar indigestion can most frequently be attributed to the substitution of cane sugar for lactose or to the too liberal use of sugar of milk. In rare instances there is a marked intolerance to milk sugar in even the smallest quantities, and a more suitable carbohydrate must be substituted; but, as a rule, sugar of milk is very well borne by artificially fed infants if the feeding mixture does not contain more than 6 or 7 per cent.

Symptoms.—Two forms of carbohydrate indigestion are met with—the acute and the chronic—and the symptoms vary according to the particular form of starch or sugar which causes the disturbance. In all cases of this nature diarrhea is the most prominent symptom, the stools being very loose, watery, and at times frothy. They are grass-green in color, acid in reaction, and frequently contain mucus. Colic is severe owing to excessive fermentation and flatulence, the latter being demonstrated by frequent eructations of gas which usually afford relief. The buttocks are irritated and excoriated by the highly acid stools, while the excessive ingestion of sugar causes an eczematous condition of the face, and of the scalp as well. The vomiting in sugar indigestion is not severe, and the vomitus has no special characteristics, although it is usually highly acid in reaction.

In acute cases there is often a sharp rise of temperature which is of short duration, and loss of weight may be quite rapid. Associated with this high fever and rapid loss in weight, there may be decided toxemia with dulness or even decided stupor. In chronic cases, however, there is no fever and very little, if any, wasting, for the assimilation of large quantities of sugar causes an increase in body weight. Carbohydrate intoxication may occur in acute and severe cases, and is then usually attended by marked disturbance of the nervous system and prostration.

Cane sugar when given in excess produces the same symptoms as sugar of milk, but is less irritating to the intestinal mucosa. Starch ingested in excess usually causes chronic indigestion, which results in disturbances of nutrition rather than digestion. Pure maltose is never used in infant feeding; but preparations of dextrin-maltose are sometimes employed, and if given too freely produce much the same symptoms as those caused by too much milk sugar except that there is more fermentation, and colic and flatulence are more distressing. The stools, too, are dissimilar, being usually of a dark brown color. As a rule infants suffering from carbohydrate indigestion do not apparently lose in weight and may even seem to gain; but they are pale and anemic, and if closely scrutinized their muscle tissue is found to be loose and flabby so that, in reality, they are in poor physical condition and are less able than normal children to combat a severe illness, or to endure the prolonged strain often associated with an intercurrent infection.

Prognosis.—In acute forms of carbohydrate indigestion the infant may be quite ill, but the prognosis is somewhat more favorable when the disturbance is due to dextrin-maltose or starchy preparations than when caused by an excess of some other form of carbohydrate. Chronic cases usually recover, but improvement is apt to be slow, and not a few of these infants succumb to some acute intercurrent infection.

Treatment.—An excess of lactose in the mother's milk is usually ascribed to a too generous diet—too rich, not only in carbohydrates, but in other food elements as well. Therefore, when sugar indigestion

appears in a suckling, the diet of the mother should be cut down, and other steps taken to render her milk less rich. When sugar of milk is the causative factor in artificially fed infants, the amount ingested should be restricted as far as possible, and the percentage of fat also decreased. In these cases fats act as an irritant to the intestinal mucosa, and consequently are not well borne in full quantities. If possible, milk sugar should be absolutely withheld for a few days, as lactic acid fermentation in the intestine will persist if even a small quantity is given.

Since mother's milk contains 7 per cent. of lactose, and cow's milk about 4 per cent., the elimination of sugar of milk from a mixture without a dangerous reduction in its caloric value is not an easy task. Protein is well borne, however, and these infants show a tolerance to small quantities of starch, therefore by giving a mixture of skimmed milk with a cereal diluent, the caloric needs of the infant are fairly well supplied, and at the same time but a low percentage of fats and carbohydrates is being ingested. After a few days it is usually possible to add dextrin-maltose to the mixture, and then gradually to return to milk sugar. Cream whey mixtures are contraindicated because of the relatively high percentage of milk sugar in whey, but Eiweissmilch or, as it is often called, albumin milk, is usually well borne.

When an excess of dextrin-maltose has set up indigestion, the treatment is practically the same as in acute carbohydrate indigestion from an excess of lactose; but, after dextrin-maltose has been omitted from the feedings for a few days, sugar of milk should be substituted. Indigestion due to an excess of starch is usually chronic, and the persistent gastro-intestinal derangement so weakens the digestive powers of the infant that, in addition to withdrawing starch from the food, fats and protein also must be considerably reduced, or, better still, if possible, mother's milk be given instead of a feeding mixture. If a wet-nurse cannot be procured, a cream whey mixture, or a rather weak formula containing not more than 2 per cent. of fat, 4 per cent. of sugar, and 1 per cent. of protein should be given, and these proportions gradually increased. At the onset it is always well to give the gastro-intestinal tract a thorough cleansing by a full dose of castor oil, and in severe cases the stomach should be given complete rest by withholding everything but water for twelve to twenty-four hours following the purge.

Protein Indigestion.—Food injuries in the breast-fed infant are more often due to an excess of protein in the mother's milk than to an excess of either fat or sugar. The artificially fed baby suffers from protein indigestion only when cow's milk forms a part of the feeding mixture; for even though it is able to digest a higher percentage of protein than is found in mother's milk, the protein in cow's milk contains so much casein that an infant fed on a cow's milk mixture can easily receive an excessive amount of casein, and indigestion be the result.

Etiology.—A high percentage of protein in mother's milk is usually the result of an excessive protein diet on her part, and either an excess or a lack of exercise. The milk of neurotic mothers and those who have been subjected to nervous shock, such as grief, worry, or fright, is apt to contain a higher percentage of protein than normal, and during the first ten days of lactation, before the equilibrium of milk secretion is definitely established, the milk contains more protein than it does later. The quantity of protein in human milk may be as high as 4 per cent.; but the excess is rarely more than 2 per cent. above normal. In artificially fed infants protein indigestion is usually induced by an excess of casein, and is but rarely caused by a high percentage of whey. Exceptional cases have been recorded of infants who have shown evidence of anaphylaxis to the protein of cow's milk, and were unable to digest the smallest quantities, owing to sensitization of the system from absorption of the protein of cow's milk during the first few days after birth, when the intestines were in an abnormal condition.

Symptoms.—Habitual colic is one of the most characteristic symptoms of excessive protein ingestion during early infancy, whether in the breast-fed or bottle-fed infant. In the nursling the other symptoms of protein indigestion are slight. Vomiting is rare, but there is usually considerable flatulence. The stools are loose, watery, and of a brown color; they contain mucus, and quite frequently fat curds due to an accompanying inability to digest fats. The temperature is not elevated except in extremely acute cases, when a sharp rise may occur. Breast-fed infants, as a rule, show no marked disturbance of nutrition as a result of protein excess, and lose very little weight. In infants who are being fed an excessive amount of whey protein digestive disorders can usually be attributed to an excess of sugar and salts in the whey, since whey itself rarely gives rise to indigestion.

Casein, on the other hand, owing to the large, hard curds it forms, may derange the digestion. The symptoms produced by an excess of casein may be quite severe. Vomiting is not unusual, the vomitus containing large curds which are hard and tough in contrast to the soft curds found in fat indigestion. These curds may also be seen in the stools as white, gray, or green particles, frequently covered with a coating of mucus when passed. As a rule, diarrhea accompanies this form of the affection, but constipation is not uncommon, and in some instances the stools are normal in every respect except for the presence of curds. Colic and flatulence are usually severe, and evacuations of the bowels may be accompanied by much pain. Ordinarily there is little or no elevation of temperature.

Prognosis.—The prognosis in protein indigestion is much more favorable than when the disturbance of digestion is due to an excess of fat or carbohydrates. Breast-fed infants, as a rule, rapidly recover when the excess of protein is removed from the mother's milk; and, since it is a simple matter to reduce the percentage of protein in

feeding mixtures, artificially fed babies rarely suffer any serious or lasting ill effects.

Treatment.—When the breast milk is found to contain an excess of protein, the cause of this excess should be ascertained and counteracted as quickly as possible. If the diet be too rich it should be cut down; if the mother is taking too much exercise she must stop short of fatigue in the future; and if her life has been too sedentary she must be induced to take a moderate amount of exercise. Grief, worry, anxiety, and other nervous states should always be guarded against during lactation. In the bottle-fed baby protein indigestion can usually be relieved by reducing the percentage of casein in the feeding mixture, and substituting whey protein in order to satisfy the protein needs of the infant.

The formation of large casein curds may also be prevented by peptonizing the milk or by the addition of alkalies, such as lime water, or solution of sodium bicarbonate, or cereal diluents, such as barleywater or rice-water. Sodium citrate added to milk also prevents the precipitation of casein curds, but has the disadvantage of being constipating. When protein indigestion causes watery brown stools, it is advisable to cut out the protein for a day or two, substituting carbohydrates in its place, and then restore the protein to the mixture. In addition to dietetic regulation an initial purge of castor oil should be given to sweep all of the large casein curds out of the gastro-intestinal tract.

Feces.—The first stools after birth are of a dark and almost black color, and they remain so until the infant takes milk from the breast, after which they become yellow, are of a pasty consistency, and acid in reaction. If the infant is fed by the bottle the stools are lighter in color and larger in bulk than those of the breast-fed infant, but, as a rule, are fewer in number, the normal nursling having two to five stools a day. Curds found in the infant stool may be composed either of fat or protein (casein), the former being soft, and soluble in ether, whereas casein curds are hard, tough, usually larger than fat curds, and insoluble in ether. Green stools often cause the mother great concern; but, if the bowel discharges are otherwise normal, this change is of no significance, being due to the conversion of bilirubin into biliverdin. White and gray stools indicate the presence of fats in the form of soap and the absence of bile. Black stools are usually caused by the ingestion of drugs, such as iron, bismuth subnitrate or charcoal; but, in rare instances, the presence of blood from the intestinal tract gives the discharges a black, tarry color. If bright scarlet blood appears in the stool, its origin is an anal fissure or rectal polyp.

Pus in the discharges indicates some severe inflammation of the intestines; and, while a small quantity of mucus may be found in the normal stool with the aid of a microscope, a considerable amount is abnormal and signifies intestinal inflammation. There is only a faint odor to the stools of breast-fed infants, while in those of the bottle-fed baby it may be quite pronounced, and varies according

18

to the diet. The stools in fat indigestion are loose and contain fat curds and fat in the form of soap. If the infant receives too little of fats the stools become dry and hard. Casein curds are characteristic of protein indigestion, and the stools are usually increased in number, alkaline in reaction, with a decidedly fecal odor. Thin, watery, highly acid stools, which irritate the buttocks and genitalia, are characteristic of sugar indigestion. At the present time the bacteriological examination of infants' stools is of little clinical value, owing to our limited knowledge of the relation of bacteria to digestive disturbance and inflammation of the intestines.

Acute Gastric Indigestion.—Acute gastric indigestion is one of the most common affections of infancy and childhood.

Etiology.—Improper feeding, whether irregularity of the feeding periods or an excessive amount of food, is most frequently the cause of gastric indigestion in infancy; but a change of diet, if too radical, will often bring on a severe attack of indigestion, while occasionally a diet, which up to a certain period has been perfectly suitable, will overtax the stomach because an existing illness has lowered the power of digestion.

Artificially fed infants furnish the majority of cases, for rarely is there change enough in breast milk, from nervous excitement or any other cause in the mother, to make it indigestible; but a protracted spell of hot weather, or the substitution of a prepared food or cow's milk for one nursing period, will often produce a severe attack of gastric indigestion in an otherwise healthy breast-fed infant. In artificially fed babies the attack is brought on by either the poor quality of the food given or the strength of the preparation. Older children suffer from acute gastric indigestion because of indulgence in pastry, candies, unripe fruits, and other indigestible articles, and by eating continually between meals or too hurriedly at table. The disease is most prevalent during the summer months, and frequently occurs during the period of dentition.

Symptoms.—The principal symptoms of acute gastric indigestion are pain referred to the hypochondrium, vomiting, nausea, headache, and fever. Preceding these acute symptoms there is usually a period of malaise during which the child is tired, peevish, and shows no desire to play. Vomiting is accompanied by much retching. The vomitus is sour, and composed of undigested food. The stomach is generally somewhat distended. In severe cases the child may be prostrated. The temperature may rise to 104° F. or above. Convulsions sometimes occur. The pulse becomes weak and rapid. There may be an occasional chill. The breath is always foul, the tongue thickly coated. An attack such as this is usually preceded by constipation, but may be followed by diarrhea. For a few days afterward the stomach is very sensitive, and unless extreme care is exercised nausea and vomiting may again occur.

Diagnosis.—The diagnosis of acute gastric indigestion is usually a simple matter because of its frequency in childhood; but one should

always remember that the gastric disturbance may be merely a symptom of one of the acute infectious diseases.

Prognosis.—Recovery, as a rule, promptly follows the removal of the cause, and rarely does a case terminate fatally if properly treated at the onset. Occasionally a convulsion will occur which, in a very weak infant or child, may prove fatal.

Treatment.—In every case the stomach should be emptied as quickly as possible with the aid of a stomach-tube or by the administration of an emetic. Stomach washing is accomplished in infants by passing a small catheter (No. 16 American) into the baby's stomach while the child is held in an upright position, after which it may be laid on the nurse's lap on its side. A glass connecting rod with rubber tubing and a funnel at the distal end is attached to the catheter, and warm water or salt solution poured into the funnel, and subsequently siphoned off. This is done repeatedly until the water returned from the stomach is perfectly clear. In older children it is so difficult to pass a stomach-tube that this procedure is not often attempted; but they should be compelled to drink glass after glass of warm water and to take one or two drams of the syrup of ipecac.

Once the stomach is emptied, nothing should be taken by mouth for some hours (twelve to twenty-four) with the exception of a little water. Calomel is, perhaps, the best drug we can use, and this may be given in grain doses, corresponding to the age of the child up to five years. When nausea disappears albumen-water, barley-water, or whey may be given cautiously in small quantities for twenty-four hours, after which the breast-fed infant may be put to the breast for a nursing period of not more than five minutes every three hours for the following day or two, gradually increasing the length of this period until the normal amount of food is being taken.

The somewhat older infant may be given weak broths on the second day, and gruels or light semisolids following this, according to the improvement noted. Constipation is usually relieved by the calomel given; but, should it persist, a tablespoonful of milk of magnesia, or a half-glass of magnesium citrate, may be given a child of three years, or a soapsuds enema or glycerin suppository may be resorted to.

ACUTE GASTRITIS.

Acute gastritis is an acute inflammatory condition of the stomach which rarely occurs primarily, but is seen frequently during childhood accompanying that common disease, gastro-enteritis. The line of demarcation between severe acute gastric indigestion and acute gastritis is very obscure; but in acute gastritis we assume that there are lesions of the stomach, however slight, and we also recognize five types of this affection; *i. e.*, catarrhal, membranous, toxic, ulcerative, and suppurative. All but the catarrhal form, however, are so rare that they need only to be mentioned as definite clinical entities.

Etiology.—The most frequent causes of acute gastritis are improper food, too frequent or too hasty eating, or any of the other factors active in the causation of acute gastric or gastro-intestinal indigestion. It is often observed during the course of the acute infectious diseases, and occurs secondarily in many inflammatory states of the intestinal tract. The most severe form of acute gastritis is that which results from the ingestion of strong acids or alkalies.

Pathology.—In catarrhal gastritis the gross changes are not marked, but the gastric mucosa in certain areas is reddened and swollen, while the stomach is either contracted or dilated, and contains undigested food and great quantities of mucus which may be blood-stained from slight hemorrhages. Microscopically, patches of mucosa are seen to be infiltrated with round cells, and there are numerous erosions of the epithelium with minute extravasations of blood scattered over the mucosa.

The gastritis produced by corrosives or acids is characterized by large ulcerated areas denuded entirely of mucous membrane, though but rarely does the ulcer penetrate the stomach wall. In membranous gastritis a false membrane forms on the lining of the stomach, the cause of which is either the diphtheria or pseudodiphtheria bacillus. Ulcerative gastritis is merely an inflammation of the gastric mucosa, characterized by the formation of numerous small erosions or ulcerations. If pus be present in the stomach wall the condition is known as suppurative gastritis, but this is very rare.

Symptoms.—At the onset of the disease the symptoms of acute catarrhal gastritis differ from those of acute gastric indigestion only in the degree of severity. Vomiting is decidedly worse, and may persist for several days or even a week. The vomitus is at first composed of undigested food, but later consists of mucus tinged with bile or even blood. There is a sudden rise of temperature to 104° or 105° F., which is significant of rapid absorption of toxins, while pain may be so severe that, in addition to rigidity of the abdominal muscles, the muscles of the legs are contracted and fixed, drawing the thighs up toward the abdomen. The belly is visibly distended, and so tender that the child protests by screaming if any attempt be made to palpate it. The pulse is accelerated, and ranges between 140 and 160, the respiratory rate also being increased. Thirst is extreme and hard to relieve; the mouth is dry; the tongue heavily coated; the breath has a fetid odor.

In severe cases convulsions may occur at the onset of the attack; but, if vomiting continues, exhaustion sets in and the child may become prostrated. Constipation usually precedes the attack, but many times there is diarrhea, numerous foul-smelling, semiformed stools being passed daily. The urine throughout an attack of acute gastritis is scanty, high-colored, acid, and concentrated owing to the loss of body fluids through other channels.

Suppurative gastritis is characterized by the same symptoms, although it is usually of longer duration.

Membranous gastritis can be diagnosed only after death, since the symptoms are not characteristic, and the affection is such a rarity as to afford no opportunity for its study.

The symptoms of ulcerative gastritis are much the same as in the acute catarrhal form, but the former is marked by more hemorrhages from the mucous membrane of the stomach.

Acute gastritis not infrequently extends into the duodenum, which is indicated by the appearance of jaundice, especially in older children.

Diagnosis.—Acute gastritis is only to be differentiated from acute gastric indigestion by the severity and persistence of the symptoms. Close observation for several days is also necessary before one can definitely say that the gastric disturbance is not merely the beginning of one of the acute infections. Meningitis is often closely simulated by acute gastritis; but in meningitis the pulse rate is slow, while in acute gastritis it is accelerated, and this differential point, together with the local signs and symptoms in acute gastritis, should make the correct diagnosis easy. Pneumonia is sometimes suggested; but careful examination of the chest will fail to elicit any physical sign of this disease; while if typhoid fever be suspected abdominal examination will reveal neither the enlarged spleen and liver nor the typical rose spots, and a Widal test will verify these negative findings. A valuable aid in the diagnosis of acute gastritis is an accurately taken history in which particular attention is directed to the nature of the child's diet.

Course and Prognosis.—The usual duration of an attack of acute catarrhal gastritis is from several days to a week, after which time the infant or child who has previously enjoyed good health will have an uneventful recovery. On the contrary, in weak, marasmic infants and poorly nourished, anemic children, acute gastritis sometimes proves quickly fatal or, more often, shows a tendency to become chronic, and the patient gradually so loses weight and strength that it easily succumbs to some slight infection.

Treatment.—This differs but little from the treatment of acute gastric indigestion. The stomach should be emptied immediately, and as long as vomiting persists the best way to accomplish this is either to wash the stomach out in the way described under the treatment of acute gastric indigestion, or, if this is impossible, as is usually the case in older children, emesis should be secured by administering 1 or 2 drams of syrup of ipecac and then compelling the child to drink glass after glass of warm salt solution.

Purgation should be produced if possible, and 1 grain of calomel given to infants in $\frac{1}{10}$-grain tablets every half-hour, or 2 grains of calomel given in $\frac{1}{4}$-grain doses to older children, if followed by a purgative dose of magnesium sulphate, is an excellent therapeutic aid in treatment. Nothing but this medicine should be put in the stomach for from twenty-four to forty-eight hours. If thirst is extreme small bits of ice or sips of cold water may be given the child.

The symptoms of acute gastritis rarely require special attention;

but if fever runs high the child may be sponged with tepid water, and if convulsions appear it should be put in a warm bath. Children and infants in poor physical condition occasionally require stimulation by means of the hypodermic administration of camphorated oil, 1 drop for each year of the child's age. Aromatic spirits of ammonia, 10 to 40 drops, diluted with water, if retained when given by the mouth, is often followed by marked stimulation. If oral administration of drugs be impossible, an ounce of black coffee may be given by rectum. If vomiting persists, cerium oxalate in $\frac{1}{2}$-grain doses, combined with bismuth subnitrate, gr. x, should be administered every three hours, this dosage being suitable for a child of three years. The stomach should be washed out two or three times daily.

After the active symptoms have subsided and nausea has ceased, feeding may be very gradually resumed. For the first day or so after a period of starvation, the breast-fed infant may be allowed a nursing period of at first three minutes, and later five minutes, every four hours, each nursing to be preceded by the administration of two or three ounces of sweetened water to dilute the milk. If improvement continues, and the baby seems unsatisfied, the nursing periods may be gradually lengthened until the child gets the normal quantity of breast milk at each feeding.

Artificially fed infants should be deprived of milk for even a longer period—barley-water, albumen-water, rice-water, or whey taking the place of the usual formula for several days, or until the stomach can again retain food, when a little milk may be added to these preparations, increasing the amount at each feeding until the full quota is being taken. If, while milk is being given, any symptoms of gastritis appear, the milk should be immediately stopped. Lime-water is an excellent diluent for milk in such cases, and if milk or cane sugar is not borne well by the stomach, saccharin or glycerin may be added to the formula to make it palatable.

Older children should be kept on gruels and broths for the first day or so after it is deemed advisable to begin feeding, and if the stomach continues to improve, junket, jellies, stewed fruits, and other semi-solid foods should be given, gradually increasing the strength of the nourishment, until the child is again on full diet. Tincture of nux vomica is sometimes a great aid to digestion in these children, and may be given in three minim doses three times a day combined, for a child of five years, with dilute hydrochloric acid in 5-minim doses.

In the treatment of corrosive gastritis, much the same procedure is carried out, except that the stomach-tube should never be passed because of the danger of perforation. An antidote should be given immediately, and emesis must be secured by forcing the patient to drink excessive quantities of warm water until vomiting ensues, and the water returns clear from the stomach. Morphine is required for the relief of pain, and when nourishment is craved nothing should be given by mouth but oils and milk. Stimulants such as are prescribed

under the treatment of acute catarrhal gastritis are even more necessary, but in corrosive gastritis are often useless, since, if much of the poison has been swallowed; no treatment available will prevent a fatal termination.

GASTRODUODENITIS.

Gastroduodenitis occurs commonly as a result of the extension of acute catarrhal inflammation of the stomach into the duodenum, causing jaundice by involvement of the bile ducts. It is usually most alarming to the parents, but is not regarded as a serious complication of gastritis.

Symptoms.—The symptoms of acute catarrhal gastritis vary little with involvement of the duodenum. Jaundice is the most prominent feature. The stools are clay-colored, the bowels constipated. The urine is stained with bile. On examination of the abdomen, the liver is usually found to be enlarged.

Treatment.—The diet should be restricted to liquids. The infant may be given whey, albumen-water, or barley-water instead of milk, while the older child should take only broths and diluted milk until improvement is noted. Cholagogues are indicated in gastroduodenitis, and there are several which may be used with equally good results. The phosphate of soda may be given daily in from 10- to 30-grain doses to either infant or child, or podophyllum may be administered twice daily in from $\frac{1}{10}$- to $\frac{1}{4}$-grain doses, according to the age of the patient. Under careful treatment the jaundice should disappear in the course of three to ten days.

ACUTE GASTRO-ENTERITIS—SUMMER DIARRHEA—SUMMER COMPLAINT.

Acute gastro-enteritis is the most common form of infectious diarrhea met with in children. It may be caused by either the dysentery bacillus of Shiga and Flexner, the streptococcus, the colon bacillus, the Bacillus pyocyaneus, or the gas bacillus. The disease differs from ordinary diarrhea with fermentation in that the bacteria are found in the walls of the intestines where they may produce lesions, while in fermentative indigestion bacterial activity is confined to the intestinal contents.

Etiology.—Acute gastro-enteritis has been aptly termed "summer complaint" because of its prevalence in hot weather, a fact explained by the lowered vitality of infants in hot weather, and by the readiness with which milk and other foods become contaminated and spoil at this season.

Digestion also is inhibited to such a degree in hot weather that a feeding mixture, which might be perfectly suited to an infant's digestive powers in cooler weather, is too strong for summer feeding, and must be reduced lest it cause gastro-enteritis.

It can be readily understood that if correct feeding for winter,

when kept up until hot weather, may cause summer diarrhea, injudicious feeding, whether the fault be overfeeding, irregular hours, or carelessness in mixing the food, is also an important factor in its causation.

Bottle-fed babies are the chief sufferers, but the disease is not extremely rare in summer in nursing infants who are kept too much at the breast. This is usually because the mother allows the infant to nurse whenever it is fretful, under the impression that it is hungry, when in reality it is thirsty and requires, not breast milk, but water.

Mother's milk is practically sterile, but the artificially fed infant must take a milk which is often contaminated and, even though sterilized, may contain toxins generated by bacteria or introduced into the milk through poisonous weeds the cow has eaten.

In institutions epidemics occur even among breast-fed infants, so that the possibility of direct contagion must be recognized, although it is probably a potent factor only in the production of summer diarrhea when children are closely segregated, being transmitted by means of soiled diapers, wash rags, towels, etc. When, however, all the children in a ward contract the disease simultaneously, the cause is most likely to be found in some unfit article of food.

The children of the poorer classes are especially prone to the disease, its frequency and severity diminishing with improvement in hygiene and environment. Overcrowding and poor food account in large measure for the number of cases in tenement houses and congested districts. Age is also a predisposing factor, since the majority of cases are seen in children less than two years old, probably, and chiefly, because of injudicious feeding at this time of life.

Pathology.—In many instances there is nothing characteristic in the appearance of the stomach and intestines, and, even when present, the lesions are varied and fail to correspond exactly with the nature and severity of the symptoms. As a rule, the duodenum and jejunum show no pathological lesions, but in the colon and the distal end of the ileum there may be evidences of catarrhal inflammation throughout the mucosa, with here and there a congested area, superficial erosion, or ulceration. There is an excessive outpouring of mucus which bathes the mucous membrane, while the solitary follicles and Peyer's patches are hyperplastic, or in some instances ulcerated.

As a rule, the lesions are most marked in the large intestine, and almost invariably there is hyperplasia of the mesenteric lymph nodes. The stomach and intestines are dilated, distended with gas, and contain undigested food and mucus.

Microscopic examination of the mucous membrane shows that the epithelium is degenerating and desquamating, and bacteria may be detected under the epithelial layers. A pseudomembrane sometimes forms and covers a considerable area. The changes in the viscera comprise cloudy swelling of the renal tubular epithelium, fatty degeneration of the hepatic cells, and degenerative changes in the cells of the nerve centres.

Symptoms.—In mild cases of summer diarrhea the child has moderate fever, passes three or four loose curdy greenish-yellow stools a day, and may vomit several times daily after feeding. As a rule, it is restless and irritable, and may occasionally cry out with colicky pain. There is no desire for food, yet the child shows no prostration, and at no time seems seriously ill.

This type of the disease, however, is not so grave as the usual acute form, which presents a far different picture. The onset is quite sudden, and is marked by persistent vomiting and frequent bowel movements which at first consist of fecal matter, but subsequently contain mucus and blood, and little else. The stomach may or may not reject food; but the appetite is so impaired that there is rapid emaciation. There may be severe pain, caused by the distention of the abdomen and the gas and tenesmus which accompany each bowel movement. The temperature range is from 103° to 105° F. at the onset of the attack, but may drop to 100° or 101° F. after several days of diarrhea.

The infant is prostrated, restless, gets but little sleep, and takes practically no nourishment. Moreover, as a result of the constant straining at stool, it may suffer from prolapse of the rectum. The buttocks become excoriated, the thighs may be eczematous because of the irritation of the frequent bowel movements, which may aggregate from fifteen to twenty-five a day.

The heart may also show signs of weakening and the pulse become feeble; but unless the infant is in poor physical condition at the onset because of malnutrition, rachitis, pertussis, or other constitutional devitalizing disease, death does not ensue except in the very severe or neglected cases. If the child is going to recover, a gradual amelioration in the severity of the symptoms is noticed, the stools decrease from twenty to ten daily, and contain less mucus and more fecal matter. Vomiting, if persistent during the attack, gradually diminishes in frequency and severity, and the stomach may retain a little light nourishment. In fatal cases the diarrhea continues; the stools contain nothing but mucus and blood; the high temperature and extreme prostration indicate severe toxemia; and death follows from convulsions and coma or exhaustion.

In some cases neither death nor recovery ensues immediately, but the acute gastro-enteritis passes into a subacute stage, with cessation of vomiting, moderately copious diarrhea, mucus stools, and steady loss of weight. There may or may not be colic.

The infant is usually very restless or apathetic. The abdomen is sunken or, in rare cases, distended; the spleen and liver are enlarged; the heart sounds are weak; albuminuria is usually present, and is due in most instances to cloudy swelling of the tubular epithelium since acute nephritis is rare. The blood, as a rule, shows a leukocytosis of 15,000 to 20,000, with a decided increase in the polynuclear leukocytes; in severe cases these figures may be much higher unless the system is too weak to react, and then leukopenia is present.

Holt describes an acute intestinal intoxication in which there is no diarrhea but, on the contrary, the child is constipated. To quote from his article:

"These cases are puzzling and frequently most serious, but fortunately they are not of common occurrence. I have, however, seen several striking examples with very high temperature, grave nervous symptoms, and sometimes marked abdominal distention, in which it seemed almost impossible to move the bowels by drugs. Castor oil, calomel, and salines have in some cases been tried in succession in four or five times the ordinary doses without the slightest effect, even when supplemented by frequent intestinal irrigation. It has sometimes been nearly two days before free movements were finally produced. These are often exceedingly foul. It is somewhat difficult to explain such cases. There seems to exist for the time almost complete intestinal paralysis. The toxic materials are locked up in the small intestine, for the colon is frequently quite empty."

Diagnosis.—The diagnosis of acute gastro-enteritis is not difficult, especially in those cases which occur during the summer months; but the differentiation between this disease and acute intestinal indigestion, or ileocolitis, can be made only after several days' study of a case, since at the onset the three diseases present like symptoms. The severity of the symptoms in acute infectious diarrhea is, perhaps, the most significant differentiating point between that disease and acute indigestion.

On the other hand, if the symptoms become aggravated and there is pain with persistent high temperature after several days of diarrhea, ileocolitis is suggested rather than summer complaint. Since the exanthemata are frequently ushered in by premonitory diarrhea and vomiting, one should always reserve the diagnosis of infectious diarrhea until several days have elapsed without the appearance of a rash. A careful examination of the chest should be made for physical signs of pneumonia, and of the abdomen for rose spots and enlargement of the spleen, since in both typhoid and pneumonia diarrhea may be the predominant initial symptom.

In severe cases of infectious diarrhea there may be distinct signs of meningeal irritation from toxemia; consequently, in meningitis with diarrhea the diagnosis of the meningeal condition should be made only tentatively until the diarrhea has subsided.

Course and Prognosis.—An infant previously healthy, attacked by a mild summer diarrhea, usually puts forth its powers of resistance, and recovery follows in the course of a week or so; but acute gastro-enteritis yields a high mortality rate among the weak, anemic, poorly-nourished children of the slums. In severe cases the outcome depends to a large extent upon the physical condition of the child; but the presence or absence of complications, such as nephritis, broncho-pneumonia, or ulceration of the intestines, also affects the chances of recovery. In a favorable case the active symptoms should subside within a week, but diarrhea may persist for a month. The prospect

of recovery is also greatly promoted by instituting proper treatment immediately, and removing the child from its crowded, unhygienic surroundings to the seashore or country. In the fatal cases death usually supervenes between the seventh and fourteenth days. Many of the chronic cases die.

Treatment.—Prophylaxis has done far more to reduce the mortality in infants during the summer than we may ever hope to accomplish by the most skilful treatment; therefore a thorough understanding of preventive measures against summer diarrhea is of more vital importance than the actual management of a case.

The breast-fed infant should, if possible, never be weaned during the summer months. On the contrary, the mother should continue to nurse her baby, and with strict regularity as to intervals. Particular attention should be paid to this phase of nursing in the summer, for, owing to the hot weather, the infant is constantly fretting from thirst, but is too often given the breast instead because the mother believes it to be hungry. In this way the amount of food is increased during the hot weather instead of diminished, as it should be, and gastro-intestinal disturbances result. Water that has been boiled should be given freely, and if this be done the number of feedings may be decreased during the summer months.

The breasts should be kept perfectly clean, and the nipples cleansed before and after each feeding with boric acid solution. The baby's mouth may be kept clean by giving after each nursing a spoonful of boiled water.

Fissured nipples and caked breasts are a potential source of infection, and warrant the removal of the infant from the breast until the condition is relieved. These few precautions, together with the hygienic measures outlined below, will serve to protect the breast-fed infant from summer diarrhea.

Far greater care must be exercised to insure the safety of the bottle-fed infant during the hot summer months. In the first place, the purity of the milk should be guaranteed, and, if possible, but a few hours should elapse between the milking period and the time the milk is used. Regardless of its purity, if the farm is located at a considerable distance from the consumer, the milk should be either pasteurized or sterilized at home before using it for the infant, and, as many babies are taken to the mountains, country, or seashore in summer, it is often necessary to do this with the milk.

Cleanliness of the nursing bottle is also absolutely essential. It should be washed thoroughly with a bottle brush after each using. The nipples must be boiled each day, and kept in a saturated solution of boric acid until needed for use. It is well to decrease the quantity of each feeding by about one-half in real hot weather, and the strength of the formula may also be reduced to considerably below the standard for a normal infant. As a general rule, the less frequent the feedings the better the infant's digestion, and in the summer, particularly, three- or four-hour intervals between feedings are sometimes advisable.

The child's clothing and body should also be kept absolutely clean, and a bath given once or twice daily. It should be kept out of doors in a cool place all day long, but direct exposure to the sun is very dangerous, and must be avoided. At night it should sleep in a cool, well-ventilated room, and be well protected against drafts. Whenever possible young children and infants should be sent away to the seashore or mountains for the summer months, preferably to coast resorts because of their equable climate.

The stools of these infants should be carefully disinfected with 5 per cent. carbolic acid solution before being disposed of, and the diapers thoroughly washed and disinfected before using them the second time. Better still is a cheap napkin that can be destroyed after use. Cleanliness on the part of the mother or nurse is essential to prevent contamination of the food by her hands, since reinfection from this source may happen.

Every case of diarrhea or digestive disturbance occurring in babies during the summer must be considered serious, and prompt measures taken for its cure. Feeding must be stopped at once, and a purgative, preferably castor oil, should be administered. Milk, especially, should be withheld for several days, even from the breast-fed infant, and for the first twenty-four to forty-eight hours nothing but boiled water given. This can be taken as frequently as desired. If improvement is noted on the second day, and the infant seems hungry, weak barley-water or albumen-water may be given, one or two ounces at a time every three hours for a day or so, after which breast feeding may be resumed. The baby should nurse for only three to five minutes at first, and there should be an interval of three or four hours between the feedings. If improvement continues, the length of the nursing period may be gradually increased until the child nurses full time. If, on the other hand, the symptoms grow worse when the infant is put to the breast, nursing should again be stopped for several days, when another attempt at breast feeding may be made.

The artificially fed infant may be deprived of food for an even longer time than the nursling, and if there are no visible signs of improvement, all nourishment, especially milk, may be withheld for several days or even a week, and nothing but albumen-water or weak barley-water permitted. Water may always be given freely, but barley-water and egg albumen should be allowed only when the stomach becomes retentive, and then in one-half the usual amount for a child of a given age.

The strength of the food must be increased cautiously, and it is well to keep the child on weak broths, beef juice, rice-water, barley-water, or predigested preparations for a week or more before attempting to give milk. Eiweiss milk is very well borne in some cases; skimmed milk, well diluted, may be tried at first; but no attempt should be made to give milk until the stools show their normal characteristics.

A good way in which to resume milk feeding is to add a teaspoonful of milk to each feeding; then, if no untoward symptoms develop

gradually to increase the amount of milk until a formula may be given. The strength of this formula, however, should be much below that given to the same child when well, and only one-half the usual quantity should be allowed at a feeding. If a whey-cream mixture is well borne, this may be given with barley-water or rice-water and sugar of milk. It is generally advisable to peptonize the milk in order to insure its assimilation; for malnutrition is usually extreme, and the prognosis frequently depends upon the infant's ability to utilize its nourishment.

Recent investigations have shown that if the infectious organism is the colon bacillus, the Bacillus dysenteriæ, or the streptococcus, a carbohydrate diet will aid in effecting a cure, while a protein, fat-free food is more beneficial when the diarrhea is caused by the gas bacillus and its allied organisms. Further laboratory investigations are necessary before these finer points in dietetic management can be applied. In particularly stubborn cases where attempts at feeding are ineffectual, dextrinized cereals and preparations, such as Keller's malt soup, offer nourishment in the form easily digested and assimilated. Better still, an effort should be made to secure a wet-nurse, since breast milk is the most easily digested and highly nutritious food the infant can take.

In no case should the strength or quantity of the food be increased rapidly, owing to the danger of relapse. Reinfection can only be avoided by careful after-treatment and strict regulation of the diet. In feeding children during the summer it is a good rule never to give them as much as they desire, and never to encourage them to eat when they display no appetite.

Hygienic Management.—Next in importance to the dietetic treatment of acute gastro-enteritis is its hygienic management. The child should be kept in the open air, if possible, or in a cool room which is sunny and well ventilated. The clothing should be very scanty, and in extremely hot weather a diaper is all that is necessary. A bath should be given at least once or, better, twice a day. The buttocks and thighs should either be anointed with sterile vaselin or powdered with boric acid to prevent chafing and eczema from the irritation of the frequent stools.

These hygienic measures, while essential and sometimes effectual in ameliorating summer diarrhea, should be carried out at home only when the child cannot be sent away; for immediate removal of the city child to seashore or mountains offers a far better chance for improvement than any hygienic measures employed at home. The nurse or mother should be instructed to observe the strictest cleanliness with regard to the child and its food and clothing in order that, once cured of the disease, reinfection does not occur.

Symptomatic and Medicinal Treatment.—Of the acute symptoms vomiting and diarrhea often call for immediate relief. If the attack be especially severe, stimulation may be necessary. As a rule, vomiting is not exhausting, and ceases if food is withheld; but, if it per-

sists, the stomach should be washed out with warm saline solution. It is scarcely ever necessary to repeat this procedure, since one washing not only checks vomiting but cuts short the attack. If lavage is of no benefit emetics, such as warm mustard water in large quantities, or syrup of ipecac in $\frac{1}{2}$- to 1-dram doses, should be administered to evacuate the stomach.

Since diarrhea, like vomiting, is a conservative process at the onset of acute gastro-enteritis, increased evacuation of the bowels should be promoted by the administration of a purgative to clean them out. Castor oil is probably the best drug for this purpose; but calomel or a saline may be amply effectual. The proper dose of castor oil would be 2 drams for an infant of six months, 4 drams for a one-year-old child, and an additional dram for each year of age above this. One grain of calomel is sufficient for the infant of one or two years, and may be given in $\frac{1}{10}$ of a grain dose every hour. Magnesium citrate should be administered in dram doses diluted with water, while the sulphate of magnesium is retained best by the stomach if the usual dose of 1 dram is given, well diluted, and in small portions at frequent intervals.

In addition to free catharsis, colonic irrigations are of great benefit in cleaning out the lower bowel, as well as in stimulating peristalsis and supplying fluid for absorption by the gut. They should be given daily, or even twice a day, at the onset of the attack, using a pint of normal salt solution for very young infants and a quart each time for older children. If tenesmus is severe, starch enemata may be used temporarily in place of the saline solution, and cocaine suppositories each containing $\frac{1}{4}$ to $\frac{1}{2}$ grain may be inserted after the irrigation. Care should be taken that the temperature of the solution when used is at least one degree above that of the body; for, especially in weak infants, and if the fluid is cold, irrigation may be followed by marked depression and even collapse. When the stomach does not retain food, nutrient enemata may be given, and salt solution administered by slow proctoclysis.

Despite the colonic irrigations it may become evident during the course of an attack that toxins are again being absorbed from the intestinal tract; and when this happens free purgation is again indicated. If stimulation is necessary, atropine sulphate and strychnine sulphate may be given in $\frac{1}{100}$ to $\frac{1}{200}$ of a grain dose hypodermically, or 1 to 5 minims of camphorated oil may be administered in the same manner. Alcohol in the form of brandy or whisky is also valuable if given in 10- to 30-drop doses every two hours when collapse seems imminent, but should not be used routinely in every case. Hypodermoclysis has been employed with success in cases of threatened collapse; but, unless great care is taken to prevent infection by sterilizing the solution and apparatus used, an abscess may form at the site of injection and prove a serious complication.

Hot baths are very stimulating, but the water must be hot enough to bring about a reaction yet, at the same time, not burn the patient.

The child should not be allowed to remain in the water more than three to five minutes, and should be carefully protected from draughts after the bath.

The use of drugs in summer diarrhea for other purposes than purgation and stimulation does not appreciably affect the course or duration of the disease; but the administration of such intestinal antiseptics as bismuth, salol, and resorcin is always advisable since they, at least, inhibit bacterial growth. To be effectual subnitrate of bismuth must be given in 5- to 10-grain doses every two hours to a child from one to two years of age. Salol and sodium salicylate are less well borne by the stomach, but may be given in 1-grain doses every two hours to infants of one year and over. Resorcin may cause vomiting; but if small doses of $\frac{1}{2}$ to 1 grain are administered every two or three hours to an infant one year old, the stomach may be able to retain it. Sulphur given in the same manner is equally effective and less liable to provoke vomiting.

Because of its constipating effect, opium should never be used until the intestinal tract has been thoroughly purged, unless pain is so severe that collapse is threatened. Later in the course of summer diarrhea, when it becomes advisable to check the number of bowel movements, paregoric may be given once, twice, or oftener daily in 5- to 10-drop doses to the child of one to two years; or Dover's powder, $\frac{1}{5}$ of a grain, in repeated doses every three hours until relieved. Morphine should be resorted to only when agonizing pain requires instant relief, and in these cases $\frac{1}{40}$ to $\frac{1}{30}$ of a grain of the sulphate may be administered hypodermically.

ACUTE ENTEROCOLITIS.

Acute enterocolitis is an inflammation of the small and large intestines caused by the same factors which give rise to summer diarrhea, but differing from that disease pathologically in being sometimes accompanied by quite distinct ulcerations in the bowels. This affection is also characterized by the passage of blood-streaked mucus stools with violent tenesmus, so that it may be mistaken for true dysentery. As a matter of fact, severe cases of acute enterocolitis cannot be differentiated from dysentery; but, as a rule, the symptoms are much milder.

Etiology.—Acute enterocolitis is a disease of infancy, but few cases being seen in later childhood. It is most common among children of the poorer classes, hence improper food, unhygienic surroundings, lack of fresh air, uncleanliness, and segregation are evidently among the chief predisposing factors. The majority of cases occur during the summer months, and in children whose physical resistance has been impaired by an attack of acute gastro-enteritis or some other gastro-intestinal disturbance. Syphilis, tuberculosis, rachitis, and other chronic systemic conditions predispose a child to enterocolitis; in many instances we find a history of recent recovery from broncho-

pneumonia or some acute contagious disease. Numerous investigators have found the colon bacillus, the streptococcus, and the bacillus of Shiga in large numbers in the stools of these cases; therefore we are justified in believing that acute enterocolitis is infectious in origin.

Pathology.—Two distinct types of acute enterocolitis are demonstrated by postmortem findings. In the catarrhal form the lesions are mild, and resemble those found in the intestines in cases of acute gastro-enteritis. The mucous membrane of the lower end of the ileum and the colon is swollen and hyperemic, while hemorrhagic areas may be scattered throughout. The lymphoid structures are swollen and elevated, and there is apt to be marked congestion of Peyer's patches. The gut has a rough feeling due to epithelial desquamation, and in the pseudomembranous form of enterocolitis a false membrane covers the mucosa.

The ulcerative form is characterized by the formation of large and small ulcers, particularly in the large bowel. This usually occurs in children who are debilitated by preceding illness. Large ulcers are, as a rule, quite superficial; but the smaller ones are deep and may extend down to the muscular coat of the intestine. They are generally the result of involvement of the lymphoid follicles with subsequent ulceration and excavation. The most common lesions found associated at autopsy are those of bronchopneumonia.

Symptoms.—The onset of acute enterocolitis is usually very sudden, the symptoms, for the most part, being referable to the intestines. The bowels are very loose, and as many as ten to fifteen greenish watery stools may be passed daily. Later the discharges are composed of mucus and blood, each movement being attended by great pain and tenesmus. Vomiting occurs only in severe cases, and in these prostration may come on quickly. The temperature is elevated, ranging from 103° to 105° F., the pulse is correspondingly rapid, and the respiratory rate accelerated. In mild cases the temperature drops to normal in a few days, vomiting ceases, the diarrhea subsides, and recovery takes place within ten days to two weeks from the onset. In severe cases, however, the inflammation may become chronic; or, if the child's resistance is very low, bronchopneumonia may set in as a dangerous, or even fatal complication.

Diagnosis.—The diagnosis of acute enterocolitis is obvious from the frequent and painful bowel movements, and the presence of mucus and blood therein. Dysentery may be closely simulated, but in the ordinary case of acute enterocolitis is ruled out by the comparatively mild symptoms. Intussusception may be thought of in some cases; but abdominal palpation and rectal examination reveal no abdominal tumor, and the fever in acute enterocolitis is too high to be accounted for by intussusception.

Prognosis.—Acute enterocolitis occurring in an infant previously healthy usually ends in recovery; but when it is secondary to a severe or wasting disease, or complicated by bronchopneumonia, the outlook is grave.

Treatment.—All feeding should be stopped immediately, and a purgative dose of castor oil given. If pain is severe it may be necessary to administer $\frac{1}{32}$ to $\frac{1}{20}$ of a grain of codeine sulphate, or 1 to 3 drops of tincture of opium, every two or three hours. After twenty-four to forty-eight hours of starvation, an ounce or two of barley-water, rice-water, albumen-water, or weak broths should be given every two hours. Milk should be omitted from the diet for at least a week. After the bowels have been thoroughly evacuated, bismuth in full doses of 10 to 20 grains, according to age, should be administered frequently (every two or three hours) in order to control the diarrhea. When the number of stools has decreased considerably and other symptoms have subsided, boiled milk, at first in small quantities, may be added to the feeding mixture, and this amount cautiously increased as improvement is noted, until the child is again taking its full quota of milk.

If the constitutional symptoms are severe and collapse threatens, stimulation may be required; and in these grave cases it is well to support the child by 10- to 30-drop doses of brandy every two hours, together with atropine sulphate, $\frac{1}{800}$ to $\frac{1}{400}$ of a grain, or strychnine sulphate, $\frac{1}{600}$ to $\frac{1}{300}$ of a grain, according to age. These children should be sent to the seashore or mountains as soon as convalescence is established, for, midst healthful surroundings, recovery is more rapid and relapses are much less apt to occur.

DYSENTERY.

Dysentery is an acute diarrheal affection of infancy and childhood, in most instances caused by bacterial invasion of the intestines, and occasionally by the Ameba coli. In the United States amebic dysentery is rare, but sporadic cases occur here and there throughout the country. It is highly probable that there are more cases of amebic dysentery among children in the southern states than is ordinarily presumed.

Etiology.—In most cases of dysentery the essential etiological factor is invasion of the gastro-intestinal tract by bacteria, of which the bacillus of Shiga and the organism isolated by Flexner are the most important. In this country the Shiga-Kruse bacillus is but rarely found in dysenteric stools, most cases being due to the Flexner bacillus, which is acid-forming, and does not ferment in milk or sugar media. Numerous organisms allied to the Flexner bacillus have been isolated and, no doubt, give rise to many of the milder attacks of diarrhea so prevalent in summer. It is probable that the colon bacillus and the streptococcus, when found in large numbers in the stools of children with dysentery, constitute a mixed infection. In temperate climates bacillary dysentery occurs most frequently during hot weather either in sporadic cases or in epidemics which affect children who are on a mixed diet. The amebic form is usually contracted by the ingestion of raw fruits and vegetables or is conveyed by contaminated water.

19

Pathology.—The intestinal lesions are confined to the lower portion of the ileum and the colon, and in every case the large bowel is most markedly affected. The changes produced vary greatly in character according to the virulence of the organism, the resistance of the patient, and the duration of the attack. In mild cases there is merely catarrhal inflammation with congestion of the mucosa, swelling of Peyer's patches and, occasionally, hemorrhages scattered throughout the mucous membrane. More rarely, similar changes may be observed in the stomach and, exceptionally, the small intestine bears the brunt of the disease, the villi being so swollen and edematous that they project above the surface of the mucosa. The entire mucous membrane is covered by a thick tenacious layer of mucus, sometimes resembling a pseudomembrane. In mild cases microscopic examination reveals epithelial desquamation. In more severe types of catarrhal inflammation, superficial ulceration occurs. These ulcers are almost invariably found in the colon alone, and while they may cover considerable areas they rarely burrow below the mucosa.

The follicular and ulcerative type of dysentery is marked by the formation of small, punched-out ulcers at the site of the lymph nodules and solitary follicles. They are caused by necrosis of the nodule and evacuation of the softened and disintegrated tissue into the intestine. In some cases larger ulcers are found, formed by the coalescence of several small ones. Follicular ulceration frequently extends to the muscular coat, but perforation is a rarity; as a rule, the wall of the intestine becomes greatly thickened, and presents a worm-eaten appearance because of the ulcerated areas. In the more severe cases the mesenteric glands are congested and swollen, but there is no necrosis. In some instances the spleen is enlarged, while in the amebic form abscess of the liver is occasionally observed. Bronchopneumonia is a frequent complication, and urinary examination may reveal a mild nephritis or, even more probable, acute degeneration of the renal epithelium.

Symptoms.—An attack of dysentery usually comes on suddenly with diarrhea as the most prominent symptom. This may be preceded by a feeling of malaise, with headache and a rise in temperature. Most cases present the picture of acute indigestion for the first day or two, but vomiting is usually neither severe nor persistent. The stools are at first composed of fecal matter; but, after several such have been evacuated, they become watery and consist for the most part of mucus and blood. There may be as many as twenty or thirty movements daily, all accompanied by such pain and straining that prolapse of the rectum is not uncommon. Relaxation of the sphincter may also take place, and result in the almost continuous passage of small amounts of mucus and blood in which may be found shreds of epithelium resembling particles of washed raw meat. The abdomen is usually distended and tender, and abdominal pain intense. Loss of fluid from the tissues causes great thirst, a dry mouth, and highly concentrated urine. In the most severe forms prostration from the

overwhelming toxemia is alarming, and the child may die in collapse on the second or third day. There is always more or less nervous depression and prostration, the degree depending upon the absorption of toxins. The pulse is rapid and feeble, the feet and hands are cold, and convulsions may occur. Emaciation is rapid and out of all proportion to the duration of the attack. As a result of the poor state of the nutrition bedsores frequently develop in protracted cases.

Amebic dysentery is characterized by the same symptoms as the bacillary form, but the attacks are milder and the disease tends to run a subacute course marked by periods of quiescence and exacerbation.

Diagnosis.—Dysentery must sometimes be differentiated from typhoid fever and intussusception, and in cases where nervous symptoms predominate it may simulate meningitis. Typhoid fever may be ruled out by a Widal test and by examination of the abdomen for rose spots and an enlarged spleen. The absence of abdominal tumor on palpation and rectal examination and the high fever we find in dysentery, serve to exclude intussusception. The diagnosis of dysentery is based upon the number of stools, tenesmus and straining at stool, and the passage of blood and mucus. Microscopical examination will reveal the infecting bacteria or the ameba coli.

Course and Duration.—The usual duration of an attack of dysentery is from two to three weeks, but recovery has been known to occur within ten days. In fatal cases, as a rule, death supervenes in the third week, although very severe ones may succumb in a few days. When recovery is about to take place, the diarrhea lessens at the end of the first week, the stools become less watery, and no longer contain blood. Amebic colitis is characterized by its chronicity, and may persist for a year or longer.

Complications.—Perforation of the intestines and peritonitis are extremely rare. In the amebic type of dysentery abscess of the liver and spleen may occur, but is most infrequent. There is usually evidence of acute degenerative processes in the heart, liver, and spleen. Bronchopneumonia, while a common occurrence in dysentery, should be regarded as an associated condition due to extreme inanition rather than as a complication.

Prognosis.—The prognosis should always be guarded until after the first week or so of the attack. Healthy children, previously well nourished, usually recover, but the outlook is serious if there has been an antecedent gastro-enteritis.

Treatment.—Prophylaxis is an important phase in the consideration of dysentery. It consists chiefly in the prevention of contamination of food and drink. During the prevalence of epidemics care should be exercised to prevent the infection of healthy children by attendants, and the patient's hands must be kept clean lest he reinfect himself.

At the onset of the disease a full purgative dose of castor oil should be administered, and this supplemented by colonic irrigations of normal salt solution at a temperature of 100° F., given twice a day.

If vomiting is severe the stomach must be washed out, and all food withheld for from twenty-four to forty-eight hours. Milk is not well borne in these cases, and for at least a week only barley-water, rice-water, albumen-water, or weak broths should be allowed. After the first few days one saline irrigation a day is sufficient, and even this should be discontinued as soon as possible because of its irritating effect upon the rectum. If there be ulceration of the bowel with much bleeding a mild astringent solution, such as tannic acid, 1 dram to a pint of water, or extract of hamamelis, a half-dram to a pint of water, should be used instead of the normal saline solution.

Bismuth subnitrate is excellent to check the diarrhea after castor oil has had its effect, but must be given in 10- to 20-grain doses every two or three hours. Opium is indicated if abdominal pain and tenesmus are severe, and may be administered in the form of Dover's powder, $\frac{1}{8}$ to $\frac{1}{2}$ of a grain at a dose, or 1 to 5 drops of the deodorized tincture of opium may be injected into the rectum in a solution of starch. In giving opium care should be taken not to lock up the bowels or allow the child to become stuporous; and before repeating a dose the effect of that given previously should be noted. Hot applications, such as very mild mustard plasters or hot-water bags, placed over the abdomen, serve to relieve pain, and if collapse is threatened the child should be surrounded by hot-water bags and be kept well covered. Stimulation, when required, may be furnished by hypodermic injections of atropine sulphate, $\frac{1}{800}$ to $\frac{1}{400}$ of a grain, strychnine sulphate in $\frac{1}{400}$- to $\frac{1}{200}$-grain doses, and 1 or 2 grains of camphor in oily solution.

The hygienic care of infants and children suffering from dysentery is exceedingly important, and whenever possible a change of climate should be insisted upon, for the reason that under proper environment recovery takes place more quickly and relapses are not as liable to occur. Syrup of the iodide of iron is a valuable adjunct in the treatment of the secondary anemia which follows, and quite large doses (15 to 30 drops) may be given three times a day. In treating amebic dysentery, a fluid diet should be maintained for a longer period than is necessary when treating older children with the bacillary form of the disease, and a 1 to 1000 or a 1 to 500 solution of quinine should be used for irrigations instead of salt solution.

CHOLERA INFANTUM.

Cholera infantum is the gravest form of summer diarrhea; but, fortunately, is more rarely met with than acute gastro-enteritis. It is characterized by sudden onset, incessant vomiting, diarrhea and prostration. In a considerable proportion of cases it terminates fatally since its course is with difficulty influenced by treatment.

Etiology.—Although German investigators have proven that faulty assimilation of fat and sugar will produce cholera infantum, other observers have recently demonstrated that large numbers of the

bacillus of dysentery (Flexner), the colon bacillus, the streptococcus, the Bacillus pyocyaneus, or the Bacillus acidophilus, or several of these organisms in combination, are present in every case; hence, for the present, this malady must be considered infectious in nature. Cholera infantum is so often associated with the use of impure milk that we are forced to the conclusion that it is due to toxins generated by bacteria ingested with the milk, and that these are either liberated in the milk before it is taken into the system or formed by bacterial growth in the milk after it has reached the stomach or intestines.

In further support of the theory that impure milk is the cause of the disease is the fact that it appears only in the summer, also its frequency in bottle-fed infants and its rarity in the breast-fed. Cholera infantum occurs almost exclusively in children under three years of age, and rarely attacks previously healthy babies, so that the majority of cases are seen in the marasmic, anemic weaklings of the slums whose nutrition is reduced and health impaired by overcrowding, unhygienic surroundings, lack of fresh air, and unsuitable food.

Pathology.—The postmortem findings are few and insignificant in comparison with the symptoms. At death emaciation is extreme; the abdomen is retracted, the eyes are sunken, the skin lies in folds; yet the gastro-intestinal tract may show evidence of merely a catarrhal inflammation, with here and there minute hemorrhages and superficial erosions of the epithelium. The stomach and intestinal mucous membranes may either be hyperemic or have a washed-out appearance. Even upon microscopic examination they show nothing more than intense inflammation with epithelial desquamation. The liver cells reveal fatty degeneration, while the renal epithelium of the convoluted tubules is in a state of cloudy swelling.

Degenerative changes are also detected in the heart muscle, while the lungs show areas of consolidation from collapse, also hypostatic pneumonia at their bases. Because of the great loss of body fluid which reduces the amount of serum, the blood is thick, concentrated, and dark red in color.

Symptoms.—The onset of cholera infantum is very sudden. The disease is usually ushered in by a rise of temperature and prostration, quickly followed by violent and persistent vomiting and diarrhea. It generally appears in an infant who has been ill with a mild, subacute, or chronic digestive disturbance of such trivial nature as scarcely to cause the mother to seek medical advice, but cases have been reported in babies previously healthy.

Vomiting usually precedes the diarrhea. At first the vomitus consists of undigested food and sour-smelling curds; later, bile and mucus are expelled. After this the vomitus is composed of a thin serous fluid containing flakes of mucus. Diarrhea is profuse at the onset, and after the intestinal contents are evacuated the stools become extremely watery. From twenty to thirty movements a day are not unusual; but when the tissues of the body are drained of fluid

the stools become smaller. They have a peculiar, musty odor, and may be either yellow, greenish, or gray in color.

The temperature is usually high at first, ranging from 103° to 106° F., but may drop to normal or subnormal from the loss of body heat. In some cases it rises to 107° or 108° F. at death. Thirst is intense, but appetite is completely lost, and food is refused. The pulse become rapid and feeble; the respirations are shallow. Prostration comes on quickly; the skin becomes cold and clammy; the eyes are sunken; the abdomen is retracted; the anus is often so relaxed that the passage of intestinal contents is continuous.

Delirium now sets in, and the infant either dies in convulsions or passes into stupor, finally into coma, and death supervenes. Loss of weight is so rapid and extensive that it may amount to 25 per cent. of the entire body substance in forty-eight hours. In a few cases the prostration is not so extreme, and the symptoms' tend to subside after a day or so, but recovery is, unfortunately, not the rule.

Diagnosis.—In cholera infantum the diagnosis is largely determined by the severity of the symptoms, since in some respects it may be closely simulated by acute gastro-enteritis; but the symptoms of the latter are milder. Asiatic cholera is the only disease which presents symptoms identical with those of cholera infantum; but, fortunately, in this country it is rarely necessary to suspect the comma bacillus to be a causative factor. Typhoid fever and appendicitis may give rise to symptoms simulating cholera infantum, but marked and localized tenderness and rigidity over the appendix in the one case, and concomitant signs of typhoid fever in the other, will exclude cholera infantum.

Prognosis.—The prognosis is invariably unfavorable, and little hope of recovery can be entertained, even though the case be seen at the very onset; for a favorable termination seems to depend as much upon the previous state of health of the infant as upon promptitude or skill in treatment.

Treatment.—The prophylaxis of summer diarrhea is carried out by means of the same precautions as are recommended to prevent acute gastro-enteritis. It consists in the proper care of milk during hot weather, hygienic living conditions in summer, and the prompt and efficient treatment of every case of gastro-intestinal derangement, however mild.

When cholera infantum is suspected the first indication is to empty the stomach and bowels by means of gastric lavage and colonic irrigation, using saline solution at a temperature of 90° to 100° F. This may be repeated every four to six hours unless collapse is threatened. If prostration is extreme, stimulants should be given in the form of hypodermic injections of atropine sulphate, $\frac{1}{800}$ to $\frac{1}{400}$ of a grain, with digitalin, $\frac{1}{200}$ to $\frac{1}{100}$ of a grain, or camphorated oil, 3 to 5 minims. Morphine sulphate is very efficacious in controlling diarrhea and counteracting the effects of the toxins on the nervous system, but should be used cautiously in $\frac{1}{80}$- to $\frac{1}{40}$-grain doses hypodermically, and should never be given if the infant is stuporous or comatose.

Rectal or oral administration of stimulants is impossible; but excellent results follow the supplying of normal salt solution to the tissues by hypodermoclysis. From 200 to 400 c.c. of warm normal salt solution may be injected into the loose tissues of the abdomen or back several times a day. If the temperature is subnormal, a hot mustard bath should be given, and the infant be placed in bed and surrounded by hot water bags.

If the temperature is dangerously high, the baby should be put in a bath with the water at 100° F., and this gradually reduced to 85° F. An ice-bag should be kept on the head, and rectal injections of cold water given. If the stomach is at all retentive, 5 to 10 drops of champagne or brandy may be administered every hour or so, and cracked ice put in the mouth to allay the intense thirst.

When improvement sets in, an attempt should be made to resume feeding, but nothing should be given by mouth until the symptoms have well abated, and the first food must be predigested or peptonized. If this is retained, very weak broths or whey mixtures may be cautiously given; but milk should be withheld for at least a week.

As soon as possible the child should be taken to the seashore, where the fresh sea air and change of surroundings will hasten convalescence.

CHRONIC GASTRITIS.

Chronic gastritis is of common occurrence in infancy and childhood. It is usually associated with chronic gastro-enteritis, and is secondary to the intestinal disturbance. Occasionally the intestinal symptoms are so mild that the affection may be regarded as an uncomplicated chronic inflammation of the stomach; but such cases are rare, and in the majority of them the intestines sooner or later become involved.

Etiology.—The chief cause of chronic gastritis is prolonged and improper feeding, which results in successive attacks of acute gastritis, and eventuates in chronic inflammation of the gastric mucosa. It is rare in breast-fed infants, and among its predisposing factors are congenital syphilis, tuberculosis, and organic disease of the heart and lungs; therefore if the feeding be not at fault one must suspect some constitutional dyscrasia.

Artificially fed babies, whose digestive systems have been severely overtaxed and impaired by improper feeding in early infancy, furnish us with the majority of the cases of gastritis which are chronic in nature. Chronic gastric indigestion usually precedes this condition and is often due to the high percentage of fats contained in the food which these babies are made to take.

Pathology.—The lesions observed in the stomach of such infants at postmortem simply denote a more advanced stage of inflammation than is seen in acute gastritis. The stomach is usually dilated, and inflammatory changes are diffused throughout its mucosa; in a certain number of cases in which the organic damage is confined to the pylorus, the stomach may be of natural size. The mucosa is roughened;

it varies in color from a dark dusky red to faded gray; it is covered with a layer of thick tenacious mucus. The submucosa is usually thickened, while the muscular coat of the stomach is atrophied and weakened.

Atrophy of the mucosa is sometimes observed in very early infancy as the result of chronic interstitial changes produced by long-continued irritation from fermenting residue continually present in the stomach. When sectioned and observed under the microscope, the mucosa shows a degeneration of the epithelium of the tubules, which are either enlarged or obliterated, also enlargement of the glandular structures as a result of chronic adenitis.

Symptoms.—The symptoms of chronic gastritis are both local and constitutional. Vomiting is often persistent, and regularly follows each feeding period. The vomitus is composed of partly digested food and foul-smelling curds which may be bile-stained; in some instances an acid mucus is vomited by these children in the morning. The tongue is coated, the breath foul. In addition to vomiting there are eructations of gas, and frequently the passage of considerable flatus.

In infants diarrhea is usually present, but older children with chronic gastritis are, as a rule, constipated. The stomach is dilated and tympanitic on percussion; in protracted cases, gastroptosis may be so pronounced that the lower border of the stomach extends far below the umbilicus. The abdomen is tender on pressure. There may be severe pain after eating, and colic of moderate degree is not uncommon in infants, although older children with chronic gastritis rarely complain of pain.

The appetite is either very poor or capricious, and the child looks anemic and flabby, is always listless and fatigued, and very apt to be fretful and peevish during the day and restless at night. There is progressive loss of weight; and, because of lack of nutrition and the impoverished condition of the blood and tissues, eczematous lesions of the skin appear. These children also frequently develop a persistent pharyngeal cough which, if severe, leads to the diagnosis of tuberculosis.

Infants with chronic gastritis quickly become emaciated. As the disease progresses all the symptoms are aggravated. There is constant diarrhea; vomiting persists. The appetite is ravenous but, owing to the failure of the digestive system to assimilate food, loss in weight continues. The temperature is frequently subnormal; the extremities are often cold and blue because of the poor circulation. Parasitic stomatitis or "thrush" is a usual accompaniment at this stage of the disease, and the infant sinks rapidly from exhaustion and malnutrition, being wasted to a mere skeleton.

In older children chronic gastritis, while not quickly responsive to treatment, does not present the serious aspect observed during infancy. After infancy it is of less frequent occurrence, and the symptoms differ slightly. Vomiting after meals is characteristic of chronic gastritis in the child, but constipation is more apt to be present than

diarrhea. The appetite is perverted, and in many instances the articles of food craved are most harmful. The abdomen is distended because of gaseous accumulations in the stomach and bowels. In some instances there may be fever at night.

Nearly all these children show a chronic inflammation of the mucous membrane of the nose or throat, although the intestines frequently escape. The older child does not present the pitiful spectacle that the infant with chronic gastritis furnishes, but is pale, anemic, fails to gain in weight, and easily falls a prey to acute infections.

Diagnosis.—In these cases the cough, if persistent, and the emaciation often suggest pulmonary tuberculosis; but a careful chest examination, a microscopic study of the sputum for the tubercle bacillus, and a von Pirquet test will be of valuable service in differentiating the two conditions.

Chronic gastritis must also be differentiated from tuberculosis of the peritoneum, which it resembles because of the persistent diarrhea and distended abdomen. The absence of fluid in the peritoneal cavity, which can be demonstrated by physical examination, and a skin test for tuberculosis in addition, will usually exclude the tuberculous condition, since this differentiation is not at all difficult.

The history of the case is of extreme importance in establishing the diagnosis of chronic gastritis; while a test meal will often aid us in demonstrating the absence of those digestive changes which are produced by an insufficiency of hydrochloric acid and pepsin, together with an excessive formation of lactic and butyric acid, and a continuous outpouring of mucus.

Occasionally a case of chronic gastritis may suggest, in the earlier stages, typhoid fever, but there is really little similarity, and a Widal test will usually rule out this infection. If congenital syphilis is thought to be the primary cause, a therapeutic test should be made, since it rarely does harm and is often followed by improvement.

Prognosis.—In chronic gastritis the prognosis depends largely upon the age and the physical condition of the patient when treatment is begun. Young infants, in whom atrophic changes in the gastric mucosa have already taken place, have but slight chance for a favorable outcome. In children one or two years old, the chances for recovery are somewhat more favorable than in early infancy, as the debility of the infant renders it extremely susceptible to secondary infection. In older children there is practically no actual mortality from chronic gastritis; but it is most difficult to treat, and runs a very protracted course.

Treatment.—The treatment of chronic gastritis depends for its results upon the fidelity with which the dietary and hygienic instructions and regulations are followed by those in charge of the child. The diet should receive the utmost consideration. It is advisable at first to restrict the strength and amount of food to the minimum, and gradually to increase it as improvement is noted.

When treatment is first instituted milk should be withheld from

the infant for a short time, and barley water, albumin water, or weak broths substituted. If breast-fed, the infant may be allowed to resume nursing in forty-eight hours; but full quantities should not be permitted until there is marked improvement in the stomach symptoms. Artificially fed babies should be deprived of milk for an even longer time than the breast-fed; and, when milk is once more allowed, it should be given in small quantities at first, and gradually increased as digestion improves.

In some cases digestion is so very poor that it may be necessary to give predigested foods, or to add a peptonizing powder to the milk at each feeding, and if nothing given by mouth is satisfactorily digested, rectal feeding must be resorted to in order to keep up the child's nutrition, and the stomach may be given a complete rest. Older children rarely require so great restriction of diet, but should not be allowed the articles of food they prefer if they are at all indigestible.

In the treatment of gastritis a liquid diet should be maintained until the stomach shows marked improvement. Broths and clear soups with dry toast should first be given; and, if this food is well borne, scraped beef or tender chops may be permitted, adding a little to the daily menu until the full amount of nourishment is taken, and thus improve the poor physical condition of the patient. The period between nursings should be lengthened an hour in infants, and older children forbidden to eat between meals. If vomiting continues after restriction of the diet, the stomach should be washed out daily before the midday meal to bring away the coating of mucus which collects on the gastric mucosa, and seriously inhibits digestion. In these cases warm sterile water or salt solution may be used for washing the stomach; but a solution of sodium bicarbonate, ℈j to the pint of water, is preferable if fermentation is present.

Drugs alone will have no appreciable effect upon chronic gastritis; but because of the frequent deficiency of hydrochloric acid in the gastric secretion of infants this acid is often given in 1- or 2-drop doses, and may to advantage be combined with tincture of nux vomica in 1-drop doses. Older children are given correspondingly larger doses of these two drugs. In addition a daily dose before breakfast of sodium phosphate is necessary to regulate the bowels. Cerium oxalate, 2 grains, and menthol, 1 grain, given every three hours, are often valuable in controlling persistent vomiting.

After the diet, the most important considerations in the treatment of a child with chronic gastritis are the environment and living conditions, and proper hygienic surroundings will aid materially in hastening the recovery of these little ones. An infant should be kept out of doors all day on a bed taken to the roof, or it may be put on the porch in its coach. Older children should be encouraged to play out of doors, but not to the point of fatigue. If possible, they should be sent to the country or seashore after steady improvement has set in, since relapses are quite common, and can be prevented only by ideal management of the case and hygienic environment.

DILATATION OF THE STOMACH.

Chronic dilatation of the stomach is quite common in infancy and childhood, and is most frequently the result of chronic gastric indigestion or chronic gastro-enteritis. Acute dilatation of the stomach, while less common than the chronic form, is observed oftener in children and infants than in adults.

Etiology.—In infants and young children the stomach becomes dilated from quite trivial causes, but the condition is often merely temporary. Only when there are continuous or oft-repeated signs of gastric derangement does permanent dilatation take place. Children with a constitutional dyscrasia, such as tuberculosis, syphilis, rachitis, or marasmus, frequently suffer from dilatation of the stomach due to the atonic condition of the stomach wall, which impairs gastric motility, and results in the retention of a residue and consequent fermentation.

A less common cause of gastric dilatation is an obstruction within the gastro-intestinal tract, such as pyloric stenosis or partial obstruction of the bowel. In a great many cases gastric dilatation is the result of recurring or continuous distention of the stomach due to fermentative gastric indigestion. A severe and acute form of dilatation of the stomach sometimes occurs in scarlet fever, in typhoid fever, in pneumonia, and in chloroform poisoning.

Pathology.—At postmortem the stomach is generally found to be much larger than has been suspected from clinical evidence during life. Cases have been reported in which the gastric capacity was three times that of a normal infant. Chronic catarrhal gastritis is usually present, and the thinness of the stomach wall is evidence of considerable atrophy of its muscular coat.

Symptoms.—The symptoms presented by a child with chronic dilatation of the stomach are principally those of the associated chronic gastritis. Vomiting is persistent, and may occur periodically at intervals of twelve to twenty-four hours. Mucus may be vomited in the morning when the stomach is empty; but, as a rule, vomiting follows the nursing or feeding period, and the vomitus contains undigested food or milk curds from several preceding feedings, showing that although food is retained in the stomach much longer than normally, digestion is so faulty that the stomach contents are practically unaltered by the action of the gastric juice.

The tongue is coated, the breath is fetid. The child either has chronic diarrhea or is markedly constipated. Eructations of gas occur at frequent intervals, and there is usually considerable flatus. The stomach is ballooned, and tympantic on percussion; the lower border may extend well below the umbilicus. The abdomen is tender, especially around the epigastrium. There may be considerable pain after eating.

As a rule the appetite is poor, and the child quickly loses weight, becomes anemic and flabby looking, and is exhausted from lack of

nourishment. It is apt to be peevish during the day and restless at night. Occasionally convulsions are noted. Extreme thirst is one of the few characteristic symptoms of chronic dilatation of the stomach, and is very difficult to assuage. Chronic gastric dilatation gives rise to indefinite and vague symptoms; acute dilatation of the stomach may manifest itself suddenly, and its symptoms are of greater severity. Dyspnea may appear from pressure of the distended stomach upon the heart.

The infant or child soon loses color, and appears to be very ill. Upon percussion the epigastrium is found to be tympanitic from the ensiform cartilage down to well below the umbilicus; but it is difficult to differentiate the gastric tympany from that caused by colonic distention. Acute dilatation of the stomach to a moderate degree often occurs during infancy; but is relieved by the eructation of gas, and does no harm unless repeated too frequently.

Diagnosis.—When there is a history of long-standing chronic gastritis, and a large area of tympany is found over the epigastrium, the diagnosis of chronic dilatation of the stomach is not difficult; but, unless some practical method of gauging the stomach contents is used, there will be extremely wide variations between the clinical estimation of the degree of dilatation and the postmortem findings.

In the case of an infant or young child it is sometimes possible to calculate the capacity of the stomach by filling it with water, and noting the amount required. Transillumination may be a valuable aid in outlining the boundaries of the organ, and in differentiating gastric dilatation from colonic distention, which is the only condition that can possibly be confounded with dilatation of the stomach.

Prognosis.—Chronic dilatation of the stomach does not necessarily endanger the life of the child, but is often the cause of a prolonged chronic gastritis inasmuch as, when present as a complication of a chronic stomach affection, it renders treatment very difficult.

This superadded condition may be the indirect cause of death during infancy by reducing the digestive power of the stomach to a dangerous degree. When acute dilatation of the stomach exists alone, recovery is much more rapid; but in those cases where the dilatation is due to organic lesions, such as stenosis of the pylorus, or congenital stenosis of the duodenum and ileum, the outlook is very unfavorable.

Treatment.—The restriction and regulation of the diet is the most important measure for the relief of chronic dilatation of the stomach. The breast-fed infant should be deprived of milk for a day or so, and barley-water, albumen-water, and weak broths be substituted. At the end of this period the child may again be put to the breast, but only for a few minutes at each nursing period.

At first it is well to increase the length of time between the feedings; but when improvement sets in the nursing periods may be gradually increased and the intervals between them shortened, until the child is again taking its full quota of nourishment daily. Artificially fed infants may be taken off milk for a week or ten days if necessary;

if the digestion be very poor, the food may be either predigested or given by rectum.

In older children chronic dilatation of the stomach is never as serious as in infancy, and frequently careful regulation of the diet and abstention from pastry, candies, fried and other indigestible foods, are sufficient to bring about amelioration. The ingestion of large quantities of liquids should be avoided. The stomach should be washed out daily as long as vomiting continues, using warm saline solution or an aqueous solution of bicarbonate of soda, ℥j to a pint of water.

The tincture of nux vomica is often of great value in the treatment of chronic dilatation of the stomach because of the atonic condition of the stomach wall. It may be given in 1-drop doses, three times a day, to a child of one year, or in 5-drop doses, three times a day, to a five-year-old child, and is best administered at meal times in combination with an organic preparation of iron.

Children with chronic dilatation of the stomach often show marked improvement if sent to the seashore or country; but when this is impossible much can be accomplished at home by insisting on outdoor life, and fresh air and sunshine.

Acute dilatation of the stomach is often relieved spontaneously by the eructation of gas, which can sometimes be made more effectual by the administration of a few drops of spirits of chloroform, Hoffman's anodyne, or peppermint-water. If stimulation is required, camphorated oil in drop doses may be given to an infant hypodermically, or 1 dram of brandy and a half-ounce of black coffee by rectal injection. If the diet is regulated after the first attack of acute dilatation of the stomach, it will go far toward preventing further trouble.

PYLOROSPASM.

Spasm of the pylorus, while by no means a common affection in infancy, occurs with much greater frequency than does hypertrophic pyloric stenosis, although few cases are recognized. In the majority of instances it is thought to be hypertrophy of the pylorus until the results of operation or the effects of medical treatment exclude the possibility of its being an organic lesion.

Etiology.—Pylorospasm is usually seen in bottle-fed babies, rarely in the breast-fed. It is often brought on by a sudden change from mother's to cow's milk, and is usually accompanied by either hypersecretion of gastric juice or hyperacidity. The frequency of gastric disturbances in infancy and the rarity of pylorospasm make it evident that other contributing factors must be active in causing this condition; but the exact nature of these influences is as yet most obscure.

It has been noted that the majority of infants with pylorospasm are nervous and excitable, manifesting a hereditary neurotic tendency which results in hyperirritability of the pyloric sphincter in common with all other muscles of the body. The normal extreme irritability

of muscle tissue in early infancy probably explains to a certain degree the occurrence of the disease at this period.

Pathology.—On postmortem examination the stomach and esophagus are usually greatly dilated, the mucous membrane is congested and formed into folds much like those observed in hypertrophic stenosis of the pylorus; but in uncomplicated cases of spasm there is neither tumor formation nor increase of tissue at the pyloric end of the stomach, and the degree of stenosis is not so extreme.

Symptoms.—Vomiting is the chief, and sometimes the only marked symptom of pylorospasm; especially is this true in mild cases where the infant is apparently healthy in every other respect. Weeks and even months may elapse after birth before the infant begins to vomit; but from the time vomiting sets in it is persistent.

The baby usually vomits soon after food reaches the stomach; either the stomach contents are all ejected at once or small quantities are brought up at frequent intervals. Rarely does the amount of vomitus exceed the quantity of food taken at the last feeding. On examination of the vomited material there is no evidence of fermentation or other gastric disturbance. The stools may be nearly normal in size, notwithstanding the constipation, and the child may be fairly well nourished because of the amount of food retained by the stomach and passed into the intestine.

In serious cases of pylorospasm the symptoms closely simulate hypertrophic pyloric stenosis. Vomiting is severe, and may be explosive in character. Practically everything eaten is rejected by the stomach, so that the infant rapidly loses weight. Obstinate constipation with very small stools is caused by the deficiency of digestive residue in the intestines.

Antiperistaltic waves can be observed on inspection of the abdomen immediately after food is taken; but these disappear after the stomach contents are ejected or passed into the duodenum. Occasionally a tumor is palpable at the pylorus, but it is much smaller than the mass felt in cases of hypertrophic pyloric stenosis, and if it is palpated carefully a change in size will be noted corresponding to the contraction and relaxation of the pyloric sphincter (Fig. 29).

In mild cases no peristaltic wave may be visible and no tumor felt, but if x-rays are taken after a bismuth meal, marked interference with the passage of the stomach contents into the duodenum will always be observed (Plate III).

Diagnosis.—As a rule the diagnosis of obstruction at the pyloric end of the stomach is not difficult, but a severe case of pylorospasm may so simulate a mild case of hypertrophic pyloric stenosis, or a mild case of hypertrophic pyloric stenosis may so much resemble a severe case of pylorospasm, that the differentiation is sometimes impossible.

The chief symptoms which favor pylorospasm, however, are the slight impairment of nutrition, the moderate constipation, the fairly normal gastric digestion, and the absence of a palpable tumor of fixed size at the pylorus. Although the symptoms of pylorospasm

PLATE III

Boy, Aged Four and One-half Years; Weight, Thirty-three and One-half Pounds, Frequent Attacks of Vomiting. Palpable Tumor at Pylorus.

There is constant irregularity in the outline of the pylorus, and upon palpation under fluoroscopic observation there is partial fixation as well as palpable thickening. At six hours there is almost complete retention of the barium meal in the stomach. In the erect posture the stomach does not empty. When the patient lies on the right side, the barium meal leaves the stomach in a very small stream. Stomach dilated.

are much milder than those of hypertrophic pyloric stenosis, there is a marked contrast between vomiting due to spasm of the pylorus and habitual vomiting. In the latter condition the child does not suffer from loss of nourishment, the stools are normal in size and number, and the vomiting, never projectile, can be attributed to a certain extent to conditions outside the stomach.

Prognosis.—In the majority of cases the prognosis is good if treatment is carried out faithfully, and the amount of intestinal residue, as shown by the size of the stools, is a fair indication of the progress of the disease. The course of a case of pylorospasm is usually protracted, but recovery under medical treatment should be anticipated except in the gravest cases where surgical intervention is sometimes necessary to save life.

Fig. 29.—Pylorospasm in an infant two months old, showing peristaltic waves.

Treatment.—Dietetic regulation is the most important phase in the treatment of pylorospasm, and it is a question whether appreciable results can be obtained from any other therapeutic measures. The breast-fed infant should be allowed to nurse for only a few minutes at a time, and the intervals between nursing should be an hour longer than is normally required for the stomach to empty itself. If the mother's milk contains a high percentage of fat, the quantity allowed at each feeding should be reduced still further, and this deficiency in the amount of the feeding made up by giving the infant from a dram to a half-ounce of lime-water after each nursing.

Since, however, it is most frequently the artificially fed infant who suffers from pylorospasm, proper modification of cow's milk in these

cases is, perhaps, the most important consideration in the regulation of the diet. The strength of the formula given will depend, of course, upon the age and weight of the infant with pylorospasm, just as in any other feeding case; but, bearing in mind the fact that it is most desirable to have as much of the stomach contents pass the pylorus as possible, an attempt should be made to give a mixture which will pass readily into the duodenum, because of the relatively rapid digestion of its constituents and the lack of curd formation.

From this point of view it will be readily seen that the carbohydrate content may be unchanged, inasmuch as lactose leaves the stomach early, and does not give rise to curd formation; that protein is better given in the form of whey to prevent so far as possible the formation of large casein curds; and that the fat content of such a formula should be greatly reduced because of the length of time required for the stomach to empty itself after a feeding rich in fats. Olive oil, five drops three times a day, is often retained and well digested, and frequently the dose may gradually be increased to 10 or 15 drops three times a day. I have often seen fat in this form retained and well digested when cream, even in the smallest amounts, could not be tolerated.

The addition of lime-water to such a formula should be much in excess of that used for the ordinary feeding case, on account of the hyperacidity of the gastric juice in these cases. It is the safest plan, however, to allow the degree of alkalinity of the mixture to depend upon the amount of hyperacidity as determined by gastric analysis, since hyperalkalinity might prove to be as active an exciting factor in provoking pylorospasm as hyperacidity. Besides, in addition to the beneficial effects of neutralizing the hyperacidity of the gastric juice, the addition of an alkali to the formula delays the coagulation of casein by rennin, and thus helps to eliminate the protein curds in the gastric contents, and favors their easy passage into the duodenum.

It is, perhaps, the best plan to reduce considerably the amount given at each feeding. To a certain extent this quantity will depend upon the size and age of the child, but the severity of the symptoms should also serve as a guide. In aggravated cases it may be advisable to give but a dram or two of the mixture at each feeding, increasing this amount gradually as improvement is noted.

If too little food is retained to nourish the infant or sustain life, a No. 15 catheter may be passed through the pylorus, and food be thus introduced into the duodenum. The stomach should be washed out daily with a 5 per cent. solution of sodium bicarbonate. Rectal injection of normal saline or Ringer's solution is also advocated because of the theory that instillation of salt solution in the rectum diminishes the secretion of the gastric juice.

In these cases there are no drugs which have any appreciable effect upon the frequency or severity of the vomiting; but paregoric is sometimes given in 5- to 10-drop doses, and cocaine also by mouth in weak solution for its anesthetizing effect upon the mucous mem-

brane of the stomach. Bromide of soda in 2-grain doses, three times a day, tends in some cases to lessen the tendency to spasm at the pylorus. In addition, warm spice poultices are sometimes applied to the epigastrium immediately before and after feeding.

The majority of cases of pylorospasm will show slow but progressive improvement under medical treatment, and eventually recover; but in a few instances, despite careful regulation of the diet and the best attention, an infant will fail to improve, and steadily grow worse, and then surgical intervention is warranted. If the physical condition be good, posterior gastro-enterostomy is not attended by high mortality, and it offers the only possible chance for life.

HYPERTROPHIC PYLORIC STENOSIS.

Hypertrophic pyloric stenosis is a condition which usually occurs in early infancy, and is the result of congenital hypertrophy of the sphincter muscle at the pyloric end of the stomach. The excess of muscular tissue at the pylorus forms a tumor there which so narrows the inner diameter of the pyloric orifice that, instead of measuring 3 to $3\frac{1}{2}$ mm., as in the normal infant, it will barely admit a small probe.

Etiology.—Hypertrophic pyloric stenosis is essentially a disease of infancy, and in more than 50 per cent. of the cases the symptoms appear before the second week. After the eighth week the disease is comparatively rare. However, a number of such cases have been reported in older children.

I have recently seen, in consultation with Dr. Betts, a boy, four years of age, with a palpable tumor at the pylorus. This boy was nursed until he was a year old, and developed normally. From the age of one to two years he gained slowly in weight. Since he was two and a half years old, he has had, every four to six weeks, attacks of severe vomiting with a slight fever. These attacks last ordinarily for forty-eight hours. During the past year and a half he has not seemed as well or as strong as he ought to be, and he is always pale and constipated. On December 2, 1915, in one of these brief illnesses, he vomited some spinach that he had eaten fourteen hours previously. The x-ray plates show that at six hours there was almost complete retention of the barium meal in the stomach. In the erect posture the stomach did not empty at all; but when the patient lay on the right side we got a fairly good view of the barium meal leaving the stomach in a very small stream. At twenty-four hours all the barium meal was found in the colon, the stomach being entirely empty of it.

Many of the cases occur in the first-born child, and boys are more subject to the disease than girls. Observers are not united in the opinion that the hypertrophy of the sphincter muscle is congenital; some insist that it is the result of too frequent muscular spasms during the first few days of life, caused by gastric or duodenal irritation. But while it is true that hyperacidity is usually associated with pyloric stenosis, no conclusive evidence has been brought to light

20

that would lead us to believe that the condition can be attributed to improper feeding or hyperacidity. The actual cause of this disease is therefore still a mooted question.

Pathology.—On examination of the stomach the pylorus is readily detected as a hard tumor mass the lumen of which is so small that a thin probe can be passed through only with difficulty. The stomach is usually dilated, the muscular coat hypertrophied throughout. The mucous membrane lining the stomach is thickened, roughened, covered with mucus, and also shows signs of gastritis. In some cases the longitudinal folds of mucous membrane extend into the pylorus, and completely occlude that orifice. The intestines are contracted, atrophic, and, for the most part, empty. When examined under the microscope, a longitudinal section of the pylorus reveals intensely hypertrophied muscle fibers, and also hypertrophy of the mucous membrane in this region; but the longitudinal muscle fibers are but little, if at all, affected.

Symptoms.—At birth the infant with hypertrophic pyloric stenosis is apparently normal, and usually remains in good health for several days or a week; but, sooner or later, the chief symptom of this disease appears, and the child vomits persistently after each feeding without regard to what is fed. Sometimes two or three feedings are retained, and then expelled simultaneously. As the disease progresses the vomiting becomes forcible and projectile in character; it tends to persist, and grows worse despite regulation of the diet and all other measures instituted to control it. When several nursings are retained before being vomited the stomach may become greatly dilated, and gastric peristalsis is visible. A palpable tumor can usually be detected in the pyloric region, and easily identified as the hypertrophied pylorus. Although the epigastrium may be quite prominent, the lower abdomen is concave and sunken owing to the fact that the intestines contain very little fecal matter or food residue, since it is impossible for much food to pass into the duodenum. The infant is constipated because of the lack of intestinal contents, and in those cases where hypertrophy of the pylorus is accompanied by pylorospasm, very little fecal matter is passed from the time the disease becomes manifest until death. The stools sometimes resemble meconium, and contain bile and a small amount of food residue. The loss in weight is rapid and excessive, and unless relieved the child eventually starves to death. In a few weeks the infant is reduced to a mere skeleton with sunken eyes, flaccid abdomen, and dry skin and lips. The large dilated stomach makes the epigastrium the most prominent part of the body, and as the infant lies on its back in a state of exhaustion peristaltic waves, perhaps several at a time, become visible and can be seen to run slowly across the upper abdomen from left to right. The temperature is normal or subnormal. The appetite is usually ravenous, but there is no other symptom of gastric disturbance.

Diagnosis.—The diagnosis of a typical case of pyloric stenosis is not difficult, and can sometimes be made with reasonable certainty

from a history of the vomiting. If an otherwise healthy breast-fed infant vomits persistently, having begun to do so a few days after birth, and if the vomiting has become more forcible and projectile in character, and continues in spite of all regulation of diet and other measures to stop it, and if no other symptoms of gastric derangement are present, we should certainly be inclined to consider the case pyloric stenosis. A careful physical examination of the abdomen should be made shortly after feeding, and, after the epigastrium has been closely scrutinized for the peristaltic wave, an endeavor made to palpate the tumor at the pylorus. If the peristaltic wave be seen and the tumor found, the diagnosis is amply confirmed.

Indigestion accompanied by vomiting is easily excluded if other symptoms of indigestion are absent and by the facts that the infant is being breast fed and there is no cause for any derangement of digestion. Emaciation is much more rapid in hypertrophic pyloric stenosis, and there is neither foul breath, coated tongue, anorexia, nor diarrhea such as is found in gastric indigestion. Simple pylorospasm presents most of the symptoms of hypertrophic pyloric stenosis, and in some instances it is impossible to differentiate these two conditions positively.

In a typical case of hypertrophic stenosis, however, a differentiation can be made with reasonable certainty. Constipation is much more marked in stenosis than in spasm; the baby is usually breast fed in a case of stenosis and artificially fed in pylorospasm; moreover, if the tumor on palpation does not vary in size and shape, the evidence is almost conclusively in favor of hypertrophic stenosis. Dilatation of the stomach is strongly suggestive of hypertrophic stenosis, and failure to improve under proper treatment is reasonable evidence of an organic lesion. Habitual vomiting of infancy may be mistaken for hypertrophic pyloric stenosis; but in habitual vomiting of infancy there is no constipation or loss of weight, and the baby continues to thrive, whereas in hypertrophic pyloric stenosis it fails rapidly.

Prognosis.—The prognosis in hypertrophic pyloric stenosis depends upon the extent of the hypertrophy and the physical condition of the child when brought under observation. If there is a considerable degree of hypertrophy, medical treatment affords no hope of recovery; and if it seems likely that the child will survive the shock of an operation pyloroplasty or posterior gastro-enterostomy should be performed, and will be followed by recovery in a large majority of cases, if done by a skilful surgeon. If but a slight degree of hypertrophy is present, there may be great improvement after proper medical treatment.

Treatment.—From many points of view, surgical treatment of hypertrophic pyloric stenosis is far preferable to medical attention. Extensive hypertrophy at the pylorus demands immediate operation as the only chance for recovery. With a moderate degree, the patient improves but little under medical treatment, and it is doubtful whether even a very slight hypertrophy of the pylorus is benefited at all.

Unless the obstruction, however insignificant, be removed, there

remains a potential factor for benign obstruction of the pylorus in later years. In some cases the pylorus may be dilated by means of forceps introduced into the stomach; but the best results follow pyloroplasty and posterior gastro-enterostomy. Before operation the stomach should be washed out and salt solution given by rectum for absorption. Enteroclysis may be continued after operation, or the saline may be given subcutaneously in an emergency.

As soon as postanesthetic nausea has subsided the child may be put to the breast; but only a dram or two of milk should be allowed at a time, and this should be followed by a half ounce of water after each nursing. The breast is allowed every hour, however, until the amount at each feeding can be increased, after which normal feeding is gradually resumed.

If operation is contra-indicated, the feedings are reduced and gradually again increased, just as in the postoperative cases, and the stomach is washed out daily. If spasm of the pylorus is complicating a mild degree of hypertrophy, it is sometimes possible to feed these infants by catheter. Bicarbonate of soda in 2- to 5-grain doses, three times a day, acts very well. If a sedative is required, 5 drops of paregoric may be given. When all means of introducing food into the stomach fail, an effort must be made to keep up the nutrition of the infant by means of nutrient enemata.

PYLORIC STENOSIS IN OLDER CHILDREN.

It is not my intention to study here the condition spoken of as congenital pyloric stenosis in infants, but to consider stenosis of the pylorus in children who have passed the period of infancy. The study of the condition in older children must, however, embrace those patients in whom the condition has persisted from infancy into later childhood, as well as those cases in which infancy has been free from all symptoms of pyloric stenosis, but in which the symptoms appeared months or years later.

At the outset of this study one is immediately impressed by the fact that while medical literature is crowded with articles on congenital hypertrophic pyloric stenosis and pyloric spasm in infants, there has been, as far as I am aware, little written exclusively on the condition in older children.

The discussion in all of these papers deals with the problem as to whether the condition is congenital or not; as to the cause of the hypertrophy of the circular muscular fibers at the pylorus; as to whether in a given case the condition is one of pyloric spasm simply, or hypertrophic stenosis simply, or both spasm and hypertrophy, and as to the necessity for an operation.

The etiology of these congenital or early cases of pyloric stenosis has been considered as due, perhaps, to gastric hyperacidity, or at least to some disturbance of gastric secretion and consequent gastric indigestion. Other causes are hyperplasia at the pylorus, edema of

the mucous membrane of the pylorus and pyloric spasm. The causes of spasm are not clear. It may be due to a toxemia, to gastric dilatation, to a change in the gastric secretion, or it may be nervous in origin. The classical symptoms are vomiting, constipation, progressive loss in weight, scanty urine, visible peristaltic waves, and possibly the finding of a tumor at the pylorus.

The treatment ordinarily advised is feeding, stomach-washing, opium, poultices to the epigastrium, saline enemata for the purpose of absorption of liquid, nutrient enemata, operation.

A study of the subject of organic pyloric stenosis as found in American, English, German and French literature has impressed forcibly on me the belief that a careful clinical examination of children and young adults will result in bringing to light a fair proportion of cases of organic stenosis during this period of life. The condition was long overlooked in infants; and while not flattering to clinicians, it is, I believe, nevertheless true that the condition is now being overlooked in the older child and young adult.

Children and young adults[1] who present gastric symptoms that continue for months or years, and especially if vomiting and epigastric pain are present, may be suffering from organic pyloric stenosis, and if such patients have been treated for a long time medicinally without benefit, the probability of there being organic pyloric stenosis increases. Many such cases have finally come to operation, and a pyloric stenosis found by the surgeon at operation.

The reason why medical treatment failed in these patients is then clear. Permanent changes have taken place in the gastric secretory apparatus as the result of the long continuance of the hypertrophic stenosis, and a cure by medical treatment is impossible. An early recognition of the pyloric obstruction should in a fair, or probably in a large, proportion of these cases lead to their cure by medical rather than surgical treatment. This seems at least probable, if one is to reason by the results obtained in pyloric stenosis in infants and older children. A study of the cases in infants and older children treated medically shows conclusively that the majority of those infants who suffer from a mild degree of hypertrophic pyloric stenosis are cured by the physician and do not come to operation. Heubner[2] reports 19 cured in 21 infants; Bendix,[3] 30 in 32 cases; Hutchinson,[4] 13 in 14 cases. Stark,[5] 11 out of 12 cases; Bloch,[6] 6 patients, all recovered.

I do not wish to be understood as implying that there are not patients who require operation. I believe that the position taken on the question of operation by Robert Hutchinson,[7] "that operation is never in any circumstances justified in these cases," is an extreme one, but that most of the cases of slight and moderate pyloric stenosis can be cured by medical treatment is my firm belief.

[1] Maylard, British Med. Jour., July 11, 1908, p. 71.
[2] Therap. d. Gegenw., 1906, vol. viii.   [3] Med. Klin., 1909, vi, 1813.
[4] Clin. Jour., September 9, 1908.   [5] Zentralbl. f. Kinderh., 1909, xiv, 5.
[6] Jahrb. f. Kinderh., 1907, lxv, 317.   [7] Loc. cit.

It is an accepted fact that many patients live into advanced adult life who at autopsy show congenital pyloric stenosis. Rudolph Mayer[1] reports 31 such cases. Cautley and Dent[2] report 3 cases of pyloric stenosis at the age of six, eleven and twenty-two years, respectively, and believe they were probably congenital. Mayo Robson reports 1 case, Lauderer[3] 10 and William Russell[4] 3 cases.

There is probably no doubt that congenital pyloric stenosis of a mild grade may remain latent for months and probably for years, and cases of organic pyloric stenosis have been observed during the second and third years of life, where a distinct insufficiency of motility, together with hyperacidity existed. There is probably hardly any year between early infancy and well-advanced old age that does not show cases of pyloric stenosis in which the patients have come to operation. Some of these patients have had gastric symptoms from childhood; in others the symptoms vary from a few months to a number of years. Rosenheim[5] reports a case in a child who had always enjoyed good health until an attack of measles at the age of five years, from which he made a good recovery. Three months later the child began to vomit and continued to vomit almost every day. When six and a half years of age he was operated on and found to be suffering from hypertrophic pyloric stenosis. The case ended in recovery. Osler reports a case of hypertrophic pyloric stenosis in a child that lived until the third year.

There is much room for thought in the study of the cases in which the hypertrophic stenosis is not sufficient in amount to produce dangerous symptoms in infancy. The hypertrophy gradually increases in degree, the stenosis becomes more marked, and in later years the child presents the symptoms directly and indirectly produced by the stenosed pylorus. Beardsley reports a case at the age of four years, Sonnenberg one at five years, and Hansy one at eleven years (Cautley[6]).

Then there are cases that present exactly the opposite picture. The symptoms of hypertrophic pyloric stenosis are very severe in infancy; the babies are so ill that it seems as if they must surely die. They recover, however, and at the age of three or four years are apparently perfectly well. Robert Hutchinson has reported such cases.

The question naturally arises, what has occurred in these cases? Has the congenital hypertrophy of the pylorus largely or partly disappeared? Has the compensatory hypertrophy of the stomach muscles been sufficient to overcome the original condition, and has no gastric dilatation occurred, or has it been a combination of both conditions? Cases are reported in which apparently no gastric dilatation has occurred, and the evidence in such cases points strongly to absorption of the hypertrophy.

[1] Virchow's Arch. f. Path. Anat., 1885, cii, 413.
[2] Tr. Roy. Med. and Chir. Soc., 1902, lxxxvi.
[3] Tübingen, 1879.   [4] British Med. Jour., July 11, 1908.
[5] Berl. klin. Wchnschr., 1899, xxxii, 703.   [6] Diseases of Children, 1910, p. 264.

What is the explanation of the very large percentage of recoveries reported by certain observers—Heubner, Bendix, Hutchinson, Stark, and Bloch? I believe it is as follows: Many of these cases are instances of pyloric spasm; others are cases of slight hypertrophic pyloric stenosis; in still others, both spasm and a mild degree of hypertrophy exist, and if one is ready to admit that a slight amount of hypertrophic pyloric stenosis may be largely or in part absorbed, the explanation is quite satisfactory. Stiles, who has operated on many of these patients, claims that true congenital hypertrophic stenosis of the pylorus is rare, and, when it does occur, should be treated surgically. Cautley is of about the same opinion, but believes that very mild cases of this disease may recover under medical treatment. Cases of this character may, and probably do, in some instances, show gastric symptoms and evidence of pyloric stenosis in later life, since persistent stenosis in the older child is represented by, first, a period of compensatory stomachic muscular hypertrophy; this is followed by a period which may exist from the inception of the stenosis which represents stagnation and the slow emptying of the stomachic contents; and lastly, retention, or the failure of the stomach ever to completely empty itself. Is it not possible to recognize these cases clinically? Should they come to operation undiagnosed?

The following conclusions in regard to hypertrophic pyloric stenosis in older children and young adults seem to me to be justifiable:

1. Pyloric stenosis is present in children and young adults more commonly than is supposed.

2. The age at which it manifests itself depends on the degree of stenosis present.

3. Pyloric stenosis may be latent for years.

4. It is found by the surgeon during childhood and young adult life, and its early recognition by the physician is important from the standpoint of early medical or, if necessary, surgical treatment.

5. The entire disappearance of all the classical symptoms of congenital hypertrophic stenosis, and the apparent health of the infant during its subsequent early childhood, suggest the probability of an absorption of the hypertrophy, especially as no gastric dilatation may develop later in life.

The stomach through the sympathetic and cerebrospinal nerves is connected with practically all organs and tissues in the human body, and it has been demonstrated that pyloric spasm can be produced artificially by stimulation of the vagus. The contraction of involuntary muscular tissue, the source of the contraction being outside the stomach, is at least probable.

Is pyloric spasm unassociated with pain and with persistent vomiting, a common condition, or even an occasional condition in older children? It certainly clinically does not resemble the cases described as associated with gastric ulcer,[1] and gall-stone colic in which the

---

[1] Fairchilds, Iowa Med. Jour., Des Moines, 1910–11, xvii, 212.

pain is excessive and vomiting not a marked symptom. Periodic pyloric spasm may occur at the menstrual period,[1] unassociated with any disease of the stomach, or with spasm in any other portion of the body, and under such conditions is probably a primary spasm of the pylorus, a motor neurosis. Lauder Brunton's observations tend to show that pyloric spasm may accompany migraine, and be a manifestation of a neurosis.

A number of cases of infantile pyloric spasm have been followed up to the age of five years and older, and found to be in first-class health, and in quite a considerable number of such cases there is no neurotic family history. The clinician should not lose interest in these babies after they pass beyond the period of infancy, and an effort should be made to follow their subsequent histories, especially with reference to any gastric symptoms.

Cases of infantile pyloric spasm have been kept under observation for years.[2] All the symptoms may persist until the third year, and the child may remain nervous and anemic for years afterward. In other cases, solid food can not be given until the child is three, or even five years old. In still other cases of spasm, vomiting may persist until the patient is four years old or older, and the peristaltic waves may continue until the child is over four years of age.

Cases diagnosed as cyclic vomiting have come to autopsy[3] and the lumen of the pylorus has been found to be very small, with a distinct hypertrophy. In these cases the children may be four or five years of age, the symptoms of epigastric pain and vomiting having been present since birth, returning at irregular intervals of months, and the children being apparently perfectly well between the attacks.

I do not mean to give the impression that cyclic vomiting and pyloric spasm are not two entirely different conditions, but to suggest the advisability of considering the possibility of pyloric spasm producing symptoms that resemble closely those of cyclic vomiting.

What are the causes of the pylorospasm in infancy that disappears in later childhood? Half of these children are breast-fed, and it seems to me unnatural to claim, as does von Starck, that the unaccustomed presence of food in the stomach produces an irritation of the gastric mucosa and so causes the spasm.

A clearer understanding of the etiology in infants may help us in the recognition and study of the condition in older children.

ENTERALGIA OR COLIC.

Colic is paroxysmal pain caused by a spasm of the intestinal muscles; and, although merely a symptom of gastro-intestinal disorder, it is so common in infancy and early childhood that it merits special consideration. Artificially fed babies suffer much more from colic than

---

[1] Hemmeter, Diseases of the Stomach, Ed. 3, p. 744.
[2] Heubner, Berlin, Therap. d. Gegenw., 1906, viii, 433.
[3] Proc. Roy Soc. Med., London, 1909–10, iii, Sect. Dis. Child., 78.

do the breast-fed, but it is by no means infrequent when the feeding is apparently ideal.

The most common cause of colic is gas in the stomach and intestines, which accumulates rapidly as a result of fermentation due to indigestion and improper feeding, especially when there is intolerance to sugar, or when the carbohydrate or protein content of the food is excessive. In many instances, air swallowed while nursing from the breast or bottle is also responsible for intestinal pain after feeding.

Colic may occasionally be a symptom of far more serious import than in the cases mentioned, since it is present in appendicitis, obstruction of the bowel, peritonitis, and intussusception. It may also be caused by intestinal parasites, by chilling of the abdomen, and by drinking ice-water.

Symptoms.—The symptoms of colic need but little description. The child cries out in pain; its face is distorted, its legs are drawn up; the knees and elbows are bent, and the hands clinched. The abdomen is hard and tense, but there is no tenderness, and the pressure of the warm hand in palpating brings relief. There is no fever; but the child may break into cold sweat during a paroxysm and turn pale, its extremities grow cold, and it may go into collapse.

These symptoms usually appear shortly after the child is fed, causing it to moan and cry until there is an eructation of gas from the stomach and discharge of flatus from the bowel, after which the pain disappears and relief is evident. The pain may sometimes be mitigated by giving the infant some food as soon as it begins, but usually it returns in a short time. In carbohydrate indigestion colic does not set in until several hours after the first or second feeding.

Diagnosis.—Simple colic will need to be differentiated from appendicitis, otitis media, and intussusception. The localized tenderness and rising fever which accompany appendicitis usually reveal the source of pain in that disease. In intussusception, there is early tumor formation, and the bowels are constipated, the discharges containing blood and mucus. If acute otitis media be present, and the child be watched closely, it will usually indicate in some way that the supposed colicky pain in the abdomen is really in the ear. Peritonitis may also be excluded by the severity of the vomiting and the short duration of the pain.

Treatment.—The most effectual treatment of colic is to relieve the distention of the intestines by promoting the expulsion of the gas, and to prevent further fermentation and gas formation by careful regulation of the diet. The child or infant should lie on its belly and hot-water bags or hot poultices may be applied. An enema of warm water almost always brings relief, and warm peppermint-water should be administered by mouth.

If the peppermint does not relieve the pain, we should not resort to alcohol or opiates, but the infant may be given a drop or two of Hoffman's anodyne, 1 or 2 grains of bicarbonate of soda, 15 drops of

elixir of anise, or 5- to 10-drop doses of Wyeth's elixir of catnip and fennel. It is sometimes well to give a teaspoonful of castor oil or a tablespoonful of milk of magnesia to children who habitually suffer from colic; and if, during the nursing period, they are taken from the breast and held upright, at the same time being patted on the back, the gas will escape and the tendency to attacks of colic be greatly lessened. If the intervals between feedings are lengthened, and the amount of food is decreased for a short time, these attacks usually cease. The strength and quantity of the food may then be gradually increased until the child is taking ample nourishment. Strict attention should be paid to the clothing these children wear, in order to guard against any chilling of the abdomen, and to make sure that there are no constricting bands around the waist.

HEMATEMESIS.

Hematemesis is not an uncommon occurrence in childhood, though infrequent during infancy. True hematemesis may be due to the hemorrhagic diseases which affect the newborn, to gastric ulcer, also to scurvy, purpura, hemophilia, vicarious menstruation, acute leukemia, and splenomegaly, as well as to accidental causes. In some cases the blood is swallowed and then ejected from the stomach, thus simulating the hematemesis seen in breast-fed infants when the blood from cracked and fissured nipples is swallowed with the milk and then vomited.

It also occurs in children who swallow blood from the nose, gums, or throat. Blood which is immediately ejected as soon as it enters the stomach is apt to be of a bright scarlet color; but it is dark and coffee-colored if it has remained there for any length of time. When passed by the bowel, blood always makes the stools black and tarry.

Treatment.—If the blood actually comes from the stomach the case is one of true hematemesis, and the child should be put to bed and deprived of food and liquids until the bleeding stops. Calcium lactate may be given in 5-grain doses, three times a day, or adrenaline chloride in 1 to 1000 solution in 5-drop doses, three times a day.

An injection of fresh human blood serum is by far the most effectual measure to stop the bleeding, and as much as an ounce may be given to an infant, three times a day, with no danger of untoward effects. Smaller amounts often fail to control the hemorrhage; therefore, if it be obtainable, the maximum dose should be given. Horse serum, diphtheria antitoxin or coagulose may be used instead of fresh human blood serum. Coagulose is supplied in 15 c.c. glass bulbs which contain 0.65 gram of desiccated powder, equivalent to 10 c.c. of human blood serum. Coagulose will keep for a long time without losing any of its properties.

GASTRIC ULCER.

Ulceration of the stomach is very rare before adult life, but during childhood is more frequently observed in infants than in older children. There may be but one small perforating ulcer or many areas of shallow ulceration which is hardly more than erosion. Follicular ulcers caused by acute gastritis are more common in infancy and childhood than in adult life. Tuberculous ulcers are exceedingly rare, yet they occur either as one large ulcerated area or several smaller ones. Acute ulceration of the stomach shows a distinct tendency to perforation, and usually appears at the cardia, while the chronic gastric ulcer which results in scar formation and cicatrical contraction is generally located near the pylorus.

Etiology.—When a newborn infant suddenly develops a gastric ulcer, many good reasons incline us to believe it either of embolic origin or due to some other circulatory change, such as venous stagnation or ecchymosis. In older children the same etiological factors are active which are potent in adult life. In some cases chronic ulceration of the stomach and gastric erosions are thought to be secondary to catarrhal gastritis, while gastric ulcer is observed in association with the hemorrhagic diseases of the newborn, with scorbutus, and with septicemia, and occasionally follows one of the acute infectious diseases. Congenital ulceration of the stomach has been reported.

Symptoms.—During infancy and childhood vomiting of blood and the passage of bloody stools are the only characteristic symptoms of gastric ulcer, and in the absence of these a diagnosis is impossible, inasmuch as the additional signs and symptoms are simply those of gastritis. The pain may be mistaken for colic since, as a rule, it immediately follows the ingestion of food and causes vomiting; but if the amount of blood in the vomitus be small, as is frequently the case in follicular ulceration, it escapes notice and the true condition is not suspected. Blood vomited soon after a hemorrhage is scarlet-red, and practically unchanged; if it has been retained for any length of time in the stomach it is coffee colored, and when passed in the stools very dark and tarry. When a gastric ulcer is present, hemorrhage is often induced by active exercise or a heavy meal, and if the amount of blood lost is small, frequent hemorrhages, while unnoticed, may cause secondary anemia which develops rapidly and may be one of the first marked symptoms. The epigastrium is usually tender on palpation, although the pain may be referred to the back. Frequently there is an associated catarrhal gastritis. The bowels may be either loose or constipated.

Diagnosis.—In children the diagnosis of gastric ulcer can frequently be made if there is vomiting of blood and melena, but when there is little or no hemorrhage the disease is recognizable only at postmortem.

Prognosis.—The prognosis is often unfavorable, since death usually follows hemorrhage or perforation. In the absence of these complications, and when the ulcer is situated at the cardia or the pylorus,

cicatrization may ensue and cause stenosis and dilatation of the stomach.

Treatment.—If gastric ulcer be suspected and the child is vomiting blood, it should be put to bed and kept there until all symptoms have subsided. Food and drink by mouth should be withheld, and nourishment given by rectum in the form of nutrient enemata. When thirst is extreme pieces of ice may be given the child to suck, and small sips of water allowed. As improvement sets in, weak broths should be added to the diet, and the quantity and strength of the food gradually increased until the normal diet is established. Bleeding may be so severe that the child will sink into collapse and die before any measures to control it can be taken. If stimulation is required, camphorated oil, administered in one to five doses hypodermically, or black coffee, 2 to 6 drams, with 20 to 80 drops of brandy, injected into the rectum, will frequently be of service. Gelatin in dram doses, given by mouth every hour, has controlled bleeding in not a few cases.

Although drugs are usually ineffectual, adrenalin chloride solution, 1 to 1000, in 1- to 3-drop doses every hour, or bismuth subnitrate, in 10-grain doses every three hours, may be tried. The injection of human serum, one ounce of which may be used subcutaneously in an infant, will often be followed by cessation of bleeding. Excepting surgery this is, perhaps, the most reliable method of checking internal hemorrhage. Injections of one to three ounces of human blood, the blood being withdrawn from the donor by means of a sterile syringe and immediately injected into the patient, have a decided influence in controlling hemorrhage. If human serum or whole blood cannot be quickly obtained, horse serum, or diphtheria antitoxin which contains horse serum, can usually soon be secured and injected. The results from these injections are often most satisfactory.

Pain is sometimes so severe as to require the hypodermic administration of morphine, or the rectal injection of chloral, bromides, or paregoric, which should be given in double the dosage prescribed by mouth. Hot fomentations are also of service in relieving epigastric pain; but if there is hemorrhage, cold applications are indicated. Perforation of the stomach demands immediate surgical intervention; but the mortality rate from such operations is very high, even though a competent surgeon operate immediately on a case in which perforation has been diagnosed.

CHRONIC INTESTINAL INDIGESTION.

Chronic intestinal indigestion is usually associated with chronic gastric indigestion, and occurs, as a rule, in children who have had repeated acute attacks of indigestion.

Etiology.—It is most common during infancy, and may be either congenital or acquired. The congenital form is due to an inherent weakness of the intestinal digestive power, while the acquired type is usually brought on by a prolonged period of injudicious feeding. Chil-

dren whose health is undermined by syphilis, tuberculosis, rachitis, or other chronic disease, are especially liable to it, and it may follow a severe attack of scarlet or typhoid fever, diphtheria, or other acute infection. In the majority of cases, chronic intestinal indigestion is observed in the children of the poor who are segregated amid unhygienic surroundings, and who do not receive proper care or food. The breast-fed baby rarely suffers unless the mother is extremely careless and the infant is nursed without regard to regularity, but the condition is very common in bottle-fed babies, and constitutes one of the most difficult affections which the practitioner is called upon to treat in infancy and childhood.

Perhaps the most common source of trouble in artificial feeding is the faulty composition of the feeding mixture which results in either fat, carbohydrate, or protein indigestion. Overfeeding is another frequent cause of trouble, due to the mother's habit of giving the baby the bottle whenever it cries to quiet it. Whether the food be too rich in one or all constituents, or too great a quantity be given, the result is an overtaxing of the intestinal digestion which, if greatly prolonged, weakens the digestive function and produces a state of chronic indigestion.

While chronic digestive disturbances are especially common during the first year of infancy, intestinal indigestion is not uncommon in older children. Here the usual cause is an excess of carbohydrates from overindulgence in cakes, candies, pastries, and other rich and indigestible foods. In some cases the condition arises because the child is fed for a year or two after weaning on too large a proportion of cereals, bread, and potatoes. Condensed milk and patent foods are often the cause of infantile indigestion, for they are rarely prepared properly, and, although the child's stomach usually tolerates them for a short time, their continued use sets up digestive disturbances which are hard to correct. Regularity in feeding is just as important with older children as with infants, and the habit of eating between meals, which is so common during childhood, often gives rise to chronic intestinal indigestion, because of the continuous strain upon the digestion.

Pathology.—As a rule there are no clearly defined pathological lesions of the intestines, since this condition is usually a functional disturbance, therefore is not accompanied by organic changes. In protracted cases, evidences of a low-grade catarrhal inflammation of the intestinal mucosa may be found, and, in addition, the lymphoid tissue of the intestines and mesenteric glands may be enlarged and swollen.

Symptoms.—There is usually a history of injudicious feeding or other dietetic error, as a result of which the infant has repeatedly had acute attacks of gastro-enteritis. Partial recovery has followed, and then the series of acute attacks has yielded to a state of chronic and persistent indigestion. The weight at first remains stationary, then begins to fall, and other signs of impaired nutrition, such as anemia, subnormal temperature, failing circulation, irritability, restlessness and disturbed sleep, develop. The bowels are usually loose, diarrhea alternating

with occasional and irregular periods of constipation. The stools are usually a greenish color, and composed of undigested food, as is shown by the presence of fat and protein curds.

In some cases they are extremely watery, highly acid, and frothy, and are passed with much flatus, which indicates a disturbance of carbohydrate digestion. There is generally a moderate amount of mucus in the stool in the form of shreds which may surround the fecal masses. The stools average five or six daily, but during acute exacerbations the infant may have from twelve to twenty a day. Although constipation usually alternates with diarrhea in intestinal indigestion, yet now and then the constipation stubbornly persists, and in these cases the stools are hard, dry, covered with mucus, and may be passed with extreme difficulty.

Intestinal colic and flatulence are much more distressing in the cases that are constipated than in those with diarrhea, and fever is more likely to be present. Vomiting occurs only occasionally, and is never a constant feature of the disease. The appetite may be good or impaired, but is usually capricious; the tongue is always coated; the breath has a fetid odor. In both infants and children the abdomen is distended, protuberant, and tympanitic; but, as a rule, there is little or no tenderness. The skin is usually dry, the extremities are cold, the temperature is subnormal except during acute exacerbations, and when the child is constipated. An infant may remain in this condition almost indefinitely, but its development is retarded, both mentally and physically, so that it may be two or three years of age before it can walk or talk.

In the older child the symptoms of chronic intestinal indigestion are somewhat different from those in infants. Constipation is present instead of diarrhea; but in exceptional cases the bowels may be loose. The stools are hard, either gray or white and of foul odor. Colic is sometimes severe, and there is usually much flatulence. The appetite may be good or poor, but is usually either impaired or so capricious as to fail to provide sufficient nourishment. In consequence these children become thin, pale, and anemic, with dry skin, sunken eyes, and cold extremities. They are usually listless and apathetic, show no desire to play, and are easily fatigued. The nervous system is sometimes markedly affected, causing the child to be extremely irritable and cross. In some cases attacks of syncope and dizziness are quite common. Convulsions are rare; but acute attacks of vomiting with severe headache are not infrequent.

The absorption of toxins from the intestine produces a mild but continuous fever with occasional sharp rises in temperature when the toxemia becomes more profound. The urine is high colored, concentrated, and, as the result of intestinal stupor, contains an excess of indican. In older children the skin frequently shows evidence of urticaria or other lesion due to the gastro-intestinal disturbance while in infancy eczema and intertrigo on the thighs and buttocks are a common result of the irritating action of the stools.

Diagnosis.—In the majority of cases the diagnosis of chronic intestinal indigestion is easily made if the symptoms and history of continued digestive derangement are taken into consideration; but in order to treat the condition successfully its exact nature must be ascertained. The character of the stools will be a clue to the form of indigestion present, and a microscopic examination of the feces will determine whether there is intolerance to fat, proteins, or carbohydrates. In fat indigestion the bowels are loose, semisolid, of a yellowish-green color, and contain fat curds. Carbohydrate indigestion is indicated by very loose or watery and highly acid movements, which irritate the buttocks and are passed with much flatus. If the bowels are constipated, the infant is usually suffering from protein indigestion, and large casein curds are found in the stools. Colic is apt to be more severe in this form of indigestion, and there is more or less vomiting. In some cases tuberculosis may be suspected, but examination of the lungs is negative, there is no cough, and the temperature range is unlike that of tubercular infection.

Prognosis.—The prognosis depends upon the vitality of the child, the duration of the disease, and, to a certain extent, upon the mode of treatment and the thoroughness with which it is carried out. As a rule, the outlook is more favorable in breast-fed infants and in older children; but, at the best, improvement is very slow and recovery gradual.

Treatment.—The most important phase of treatment is the regulation of the diet. When the infant is breast-fed, if the stools show an excess of fat, which indicates that the milk is too rich, the time the baby is allowed to nurse should be shortened, and an ounce or two of water given the child in addition to each nursing to dilute the mother's milk. The diet of the mother may also be restricted. The baby should be nursed at regular intervals of not less than three hours. In exceptional cases it may be necessary to pump the milk from the mother's breasts and skim it before giving it to the child; but under no circumstances should any other food be substituted for the milk of the mother unless a suitable wet-nurse be found. If the child is bottle-fed, and a wet-nurse can be procured, the giving of human milk forms an easy solution of the dietetic problem.

In every case it is advisable at the beginning of treatment to withhold all milk for a day or two, and then, if it be necessary to give a feeding mixture, the formula should contain a very low percentage of fats and proteins. The amount of fats, carbohydrates, or proteins to be given an infant should always depend upon the character of the stools; and if intolerance to any one of these constituents is evident the amount of that one ingredient to be put into the feeding mixture should be decreased until improvement in digestion is noted.

As a rule, it is well to give a fat-free mixture at the onset of treatment to babies who are exclusively bottle-fed, and to stop all starchy food in children two or three years old who are on a mixed diet. Bottle-fed babies should be given dextrinized gruels and cereals, fat-free milk that has been peptonized, fat-free whey, and albumen-water in small

quantities at frequent intervals as a substitute for the milk mixture. As the child improves fat may be very slowly added, $\frac{1}{4}$ to $\frac{1}{2}$ per cent. at a time, and the percentage of sugar also increased as the digestive power of the patient improves.

A strong tendency prevails to substitute patent infant foods when milk is not borne well; but in these cases they are apt to do harm and, as a rule, it is wiser to resort to Eiweiss milk, broths, buttermilk, or malt soup formulas. In older infants broths, beef juice, scraped meat, fruit juices, and a little dry bread, or a few crackers may be given; but all starchy foods should be much restricted for at least a year. Children who have been allowed to eat between meals and indulge in sweets and pastries should be kept upon a very plain but nutritious diet for a month or two, and allowed to eat only at meal time. The child of two years may be fed five meals daily, the child of three or four should eat four times a day, and at five years three meals are sufficient. As a rule, cakes, candies, pastries, potatoes, hot bread, fried food, and raw fruits should be prohibited in the dietary of children with weak digestion; but beef, lamb, chicken, fish, eggs, and rice are usually well digested.

The medicinal treatment of chronic intestinal indigestion should consist of an initial purgative dose of castor oil, repeated at intervals during the course of the disease, and the bowels kept open by the use of a mild laxative to insure at least one passage a day. In the cases with diarrhea it is well to administer bismuth subnitrate in 5- to 10-grain doses, also tincture of nux vomica in one- to three-drop doses for its tonic effect on the system. The syrup of the iodide of iron is also of service during convalescence, given in full tonic doses of 10 to 30 drops, according to the age of the child. Intestinal irrigations are to be resorted to only during acute exacerbations; for, if too long continued, they are liable to irritate the colon and do more harm than good.

The general treatment of these children should be carefully carried out. The clothing must be warm enough to protect the child from changes in temperature. An abdominal binder gives support to the distended abdomen, and should be worn continuously. The importance of the daily bath should be emphasized, and a cold sponge each morning is most beneficial unless the child is too weak for it, as shown by coldness or blueness of the extremities after the brisk rubbing which should always follow a cold sponge.

If sent to the mountains or seashore, children improve rapidly; but when such change of environment is impossible, abundance of fresh air should be given the child by keeping it out of doors during pleasant weather and having the sleeping room well ventilated at night.

CHRONIC ILEOCOLITIS.

Chronic ileocolitis is induced either by severe attacks of acute ileocolitis or by chronic intestinal indigestion; but the most severe cases are those which follow acute ileocolitis.

Etiology.—An acute attack of ileocolitis usually becomes chronic because of poor management of the diet and lack of hygienic care. In a few instances it may follow typhoid fever, pneumonia, and other acute infectious diseases, especially scarlet fever and measles. After severe and protracted cases of chronic intestinal indigestion, we find at postmortem chronic inflammation of the ileum and colon, which is the result of constant irritation of the intestinal mucosa by bacterial toxins, fermentation, and the products of decomposition.

Pathology.—The most common pathological lesions found in the intestines are a change in color to dull gray; slight pigmentation; swelling of the lymphoid tissue; and a thickening of the mucosa of the ileum and colon. In addition to these evidences of chronic catarrhal inflammation of the mucosa, there are hemorrhagic and ecchymotic areas scattered throughout the walls of the bowels, while an excessive secretion of mucus covers the mucosa. In severe and protracted cases, a section of the intestinal wall studied under the microscope will show the tubular glands of the mucosa to be atrophied, and the villi and follicles undergoing degenerative changes. The mesenteric glands are swollen and enlarged, and occasionally show caseation in the centre. Ulceration is rare in chronic ileocolitis; but at postmortem follicular ulcers are occasionally found, and less frequently large superficial ulcers.

Bronchopneumonia and hypostatic congestion of the lungs are the most frequent accompanying diseases. In severe cases there is cloudy swelling of the heart muscle, also fatty degeneration of the liver cells. Nephritis is rare; but in most cases the renal epithelium is found in a state of cloudy swelling.

Symptoms.—In the majority of cases the most important symptoms of chronic ileocolitis are constant diarrhea and progressive emaciation. The stools are usually yellowish green or brown in color, and number three to six a day. They consist of undigested food and mucus, but there is rarely any blood except in ulcerative cases. The abdomen is generally distended and tympanitic; and there may or may not be a great amount of colic and flatulence. Tenesmus is not as severe, nor prolapse of the rectum as common, as in acute ileocolitis.

In many cases the appetite is unaffected and the child rarely vomits; but emaciation is continuous and progressive, and the face acquires a wizened and pinched expression. The skin is dry and coarse, and may be eczematous about the anus and genitalia. The constitutional symptoms become aggravated as the disease progresses, and in advanced cases the eyes are sunken, the fontanelle is depressed, the temperature subnormal, the heart beat weak and rapid. The child becomes so exhausted that it rarely moves, and the cry is merely a whine. The feet and hands are cold because of the poor circulation, and the extremities may become edematous. At the onset of the disease the child is fretful, cross, and irritable, perhaps crying continually, and throughout the attack it is nervous and peevish. The nervous symptoms are varied, delirium and stupor being common, but con-

21

vulsions are rare. Death usually supervenes as the result of some complication or intercurrent disease, such as bronchopneumonia, or one of the exanthemata.

Diagnosis.—Chronic ileocolitis may simulate general tuberculosis, and the child should be thoroughly examined with the view of excluding this disease as an underlying factor. Absence of fever and of demonstrable foci in the lungs and a negative von Pirquet reaction will usually preclude the possibility of tubercular infection. Syphilis should be excluded by careful inquiry into the history of the mother and by blood examination if necessary. Rachitis presents such characteristic physical signs that its presence is perfectly obvious. It is practically impossible to form any idea of the extent of the ulceration, but the catarrhal form of chronic ileocolitis may be diagnosed by the absence of blood in the stools and the comparatively mild character of the disease.

Prognosis.—The prognosis in the severe cases is, as a rule, unfavorable, and is rendered still more so by existing systemic diseases or by the poor environment so often observed in this disease. The outcome also depends upon the vitality of the child when brought under observation, its age, and the severity of the attack. Infants may die, but the chances of recovery are much more favorable in older children.

Treatment.—Whenever possible these children should be removed from their unhygienic surroundings and sent to the country or seashore; or, at least, should be given the advantage of plenty of fresh air, for improvement is much more rapid when the child lives under sanitary conditions. Much depends upon the thoroughness with which treatment is instituted; hence it is important that the mother of the child carry out orders implicitly or that she secure a reliable trained nurse.

An initial purgative dose of castor oil should be given, and thereafter the bowels should be moved every day; milk of magnesia may be given to infants, and cascara sagrada to older children. Colonic irrigations should only be used during the acute exacerbations; for, if employed continuously, they may irritate the rectum and colon and cause an increased secretion of mucus by aggravating the inflamed mucosa. When by absorption an accumulation of fecal matter gives rise to autointoxication, the colon should be irrigated with normal saline solution, after which several ounces of a 2 per cent. tannic acid solution should be injected and allowed to remain for fifteen to twenty minutes.

Regulation of the diet is perhaps the most important part of treatment. The feeding should be carefully supervised throughout the attack and for months after recovery has taken place, because indiscretions in diet cause relapses, one of which may prove fatal. Mother's milk should be given whenever possible. If the baby is bottle-fed, milk should be withheld for several days, and albumen-water, dextrinized cereals, or weak broths substituted. After improvement sets in peptonized milk, Eiweiss milk, malted soups, or buttermilk may be tried in lieu of ordinary milk.

When it seems wise to give milk, a weak formula containing 2 per cent. of fats, 4 per cent. of sugar, and 1 per cent. of protein should be used for the infant of six months, and weaker formulas for younger babies. These mixtures may gradually be made stronger as the digestive power of the infant increases. An arrest of the progressive loss of weight is a favorable indication, and a steady gain in weight may be regarded as a sign of ultimate recovery, provided there are no relapses.

INTUSSUSCEPTION.

Intussusception is the slipping of one portion of the intestine into another, and is one of the causes of acute obstruction of the bowels. The most common site for this affection is the ileocecal region, although it may occur in any part of the intestinal tract. There are three anatomical forms: the enteric, in which one portion of the small intestine is invaginated into another section of the small bowel; the colic, in which the large bowel is invaginated within the large bowel; and the ileocecal, caused by the invagination of the ileum and cecum into the colon.

Etiology.—Intussusception is a rare disease, but occurs much more frequently in childhood and infancy than in adult life. The majority of cases are seen in children less than a year old, and males babies are affected more frequently than females. Its exact cause is unknown. Several theories have been advanced, the most probable of which are based upon the irregular muscular action (spasmodic in character) of the intestinal wall and paresis of the muscular coats of the bowel. In a certain number of cases Meckel's diverticulum and other abnormalities have been found, and exceptionally there is a history of antecedent digestive disturbance and inflammation of the bowels. Typhoid fever may also be followed by intussusception; here the weakened condition of the intestines is probably a factor in its causation. In most cases, however, the patient is a healthy and well-nourished infant who has never been ill; therefore the disease is caused more frequently by overactive and spasmodic intestinal movements than by lack of muscular tone. A history of abdominal injury is occasionally elicited; but the relation between trauma and intussusception has not been established.

Pathology.—Invagination takes place from above downward, hence the distal portion of the affected intestine is found drawn over the proximal end. At the site of the lesion we find three layers of bowel: the outer, or intussuscipiens, which receives the invaginated portion; the internal, or entering layer; and the returning layer. These last two layers are invaginated and enclosed by the first, and are known as the intussusceptum. The intussusceptum is thickened and congested, and in the course of a few days adhesions form between this and the enclosing layer of intestine. Obstruction of the bowel is the result of congestion and edema, and the blood supply is cut off by traction on the mesentery which, if unrelieved, causes gangrene and sloughing. At

postmortem there is frequently found a condition known as agonal intussusception, in which multiple intussusceptions have taken place throughout the intestinal canal.

Symptoms.—In the usual form of the disease the symptoms develop suddenly, and are so severe that they may cause shock and collapse. Vomiting is persistent and violent, and occurs earliest in the attack when the lesion is high up in the small bowel. Pain is severe and comes in paroxysms, during which the child lies on its back with the thighs drawn up on the abdomen. There are no prodromes; therefore healthy children are sometimes attacked while nursing or asleep, and may be quickly prostrated or go into collapse. The bowel discharges are loose and evacuated with much tenesmus. After the fecal matter below the site of the obstruction has been discharged, the stools are composed of blood and mucus.

The temperature becomes subnormal; the face is pale; the pulse is rapid and feeble. Pain subsides when gangrene sets in, but vomiting continues, although it is not as a rule stercoraceous. The bowels usually become absolutely constipated about the third or fourth day.

The abdomen, at first, is neither tender nor distended, but so relaxed that abdominal palpation is easily accomplished. During the first day or two the presence of a tumor is readily ascertained but, later on, the abdomen becomes distended and tympanitic, and, if peritonitis develop, is tender and rigid throughout.

In a considerable proportion of the cases a tumor may be detected by digital examination through the rectum, and it should always be sought for, since it is a valuable aid to diagnosis. The mass is sausage-shaped, from four to six inches long, and of putty-like consistency. It is rendered more prominent by abdominal examination or by an attack of severe pain. It may be located in any part of the intestinal tract, and is sometimes found projecting from the anus.

Intussusception generally runs an acute course. If the obstruction is not relieved prostration becomes extreme, the temperature ascends because of the toxemia and peritonitis, the child's face assumes an anxious expression, and it finally dies in collapse.

Diagnosis.—The diagnosis of intussusception can be made with certainty in any case where there is a history of sudden severe pain, vomiting, and bloody stools, and when a tumor can be felt along the intestinal tract. The sudden onset, the violence of the symptoms, the absence of fever, the shock, sharply differentiate this disease from ileo-colitis, gastro-enteritis, or other intestinal inflammation.

Prognosis.—During infancy an attack of intussusception runs a rapid course; and, in the majority of cases, unless relieved spontaneously or by operation, will terminate in death within five days. The earlier the operation and the older the child the better the outcome. Sub-acute and chronic cases offer a more favorable outlook than do those of the acute type, in which the chances of recovery are reduced considerably by each succeeding day of the disease.
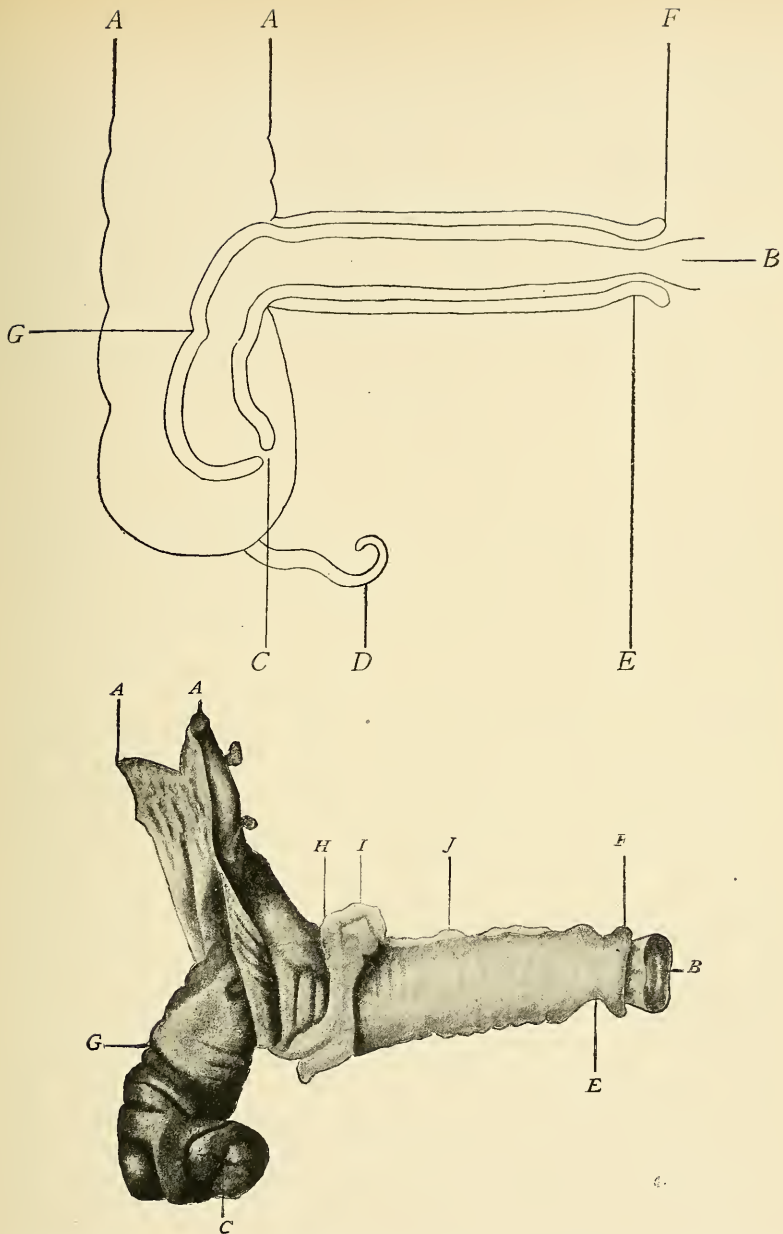
FIG. 30.—Drawing of specimen from a case of intussusception in an infant. In the diagram the letters have the same signification. *A A*, colon; *B*, point where the ileum enters the intussusception. The letter *B* points directly within the intestinal lumen. A probe passes in at *B* and comes out at *C*. The diagram illustrates the course which it must pursue. *D* is the appendix; *E*, point of constriction of the ileum; *F*, point of slight eversion where the serous coat turns to pass within the bowel; *G* shows the line of constriction in the ileum caused by the ileocecal valve. The specimen has been pulled through the valve to show this line of constriction and the gangrenous mass of intestine beyond extending from *G* to *C*. This point of constriction in the diagram is indicated by a slight depression at *G*. *H*, *I* and *J* show the line of attachment of the mesentery. Between *H* and *I*, and extending slightly beyond the line *I*, is a fold produced by pulling the intestine through the ileocecal valve sufficiently far to show the gangrenous process.

Treatment.—Surgical intervention should immediately follow the making of a diagnosis; for, if the gut is not gangrenous, the condition may easily be corrected by a laparotomy. Attempts at reduction by other measures are justifiable, but should not delay operation. Warm water may be injected into the colon, and, if no force is used, will do no harm. Inflation of the intestines by air under pressure is a dangerous procedure, and, if employed, the air should be introduced very slowly. The child should be inverted when these methods of reduction are being tried. If, perchance, reduction is accomplished, no food should be allowed for from twelve to twenty-four hours, the bowels should be kept quiet by suitable doses of paregoric, and no cathartics or purgatives given for at least a week.

CHRONIC CONSTIPATION.

Chronic constipation, which is quite common in infancy and childhood, is characterized by a failure of the bowels to move regularly, and by stools which are smaller than normal, very dry, and passed with much difficulty. An infant whose bowels are regular should have two or three movements daily, and the older child at least one stool a day.

Etiology.—Constipation in the breast-fed infant is usually due either to a deficiency in the quantity of food or a deficiency of fat in the breast milk. These two factors must also be considered in the constipation of artificially fed babies; in addition there may be in the milk formula an excess of protein which, in itself, will result in hard, dry stools. Infants are all predisposed to constipation because of the position of the intestines during infancy; for situated, as they are, mainly in the pelvis, the great length of the large intestine in proportion to the size of the child's pelvis results in multiple flexures which retard to a great extent the movement of the fecal contents, and favor the absorption of the liquid portion of the stool, making the mass hard and dry.

Atony of the intestines is a frequent cause of constipation in children who suffer from rachitis, malnutrition, anemia, tuberculosis, or any other chronic disease. In the acute infections, owing to the reduction in body fluid caused by fever, the stools are often hard and dry. Occasionally a steady milk diet will constipate, especially if no water is given, and in older children a diet lacking in residue, as would be the case if vegetables and fruits were restricted or omitted, will tend to produce constipation. In rare cases constipation will prove to be due to an organic cause, such as stricture of the anus, imperforate anus, anal fissure, proctitis, or hemorrhoids. In some families there undoubtedly exists a hereditary predisposition to defective musculature of the bowels, which manifests itself early in life by stubborn chronic constipation in the child.

Symptoms.—The most common indication of constipation is a diminution in the normal number of bowel movements; but in some cases, while the number of stools is normal, they are very hard, dry, and

smaller than they should be, indicating that the waste products of the intestines are not all being excreted. In constipation, evacuation of the bowels is usually attended by much pain and straining which may lead to inflammation of the rectum and the passage of blood and mucus in the stools. If, for an unusual length of time, the bowels are insufficiently evacuated, constitutional symptoms appear and the child becomes restless, feverish, nervous, and irritable, and may complain of persistent abdominal pain. The tongue is heavily coated, the breath foul, and all desire for food is lost.

Diagnosis.—Constipation in itself is but a symptom the underlying cause of which should be sought, and the diagnosis of this primary condition is sometimes extremely difficult.

Prognisis.—The prognosis of chronic constipation depends largely upon the cause; as a rule, it is very resistant to treatment and difficult to correct.

Treatment.—The most important point in the treatment of chronic constipation is to ascertain the cause and remove it. If there is muscular atony of the intestines, due to constitutional dyscrasia, an effort should be made to build up the child's general health by careful regulation of the diet and the administration of such tonics as cod-liver oil, or drop doses of the tincture of nux vomica; in addition, special attention should be directed to the treatment of the underlying disease, whether tuberculosis, syphilis, or rachitis. Inasmuch as faulty diet alone is responsible for many cases of constipation, the quantity, strength, and quality of the food should always be carefully investigated and adjusted, although the results obtained are not always as encouraging as might be expected, probably because of secondary atony due to prolonged distention of the bowels.

In breast-fed infants constipation is usually due to insufficient food or to a deficiency of fat in the mother's milk; therefore the nursings should be supplemented by artificial food in carefully prepared formulas or by additional breast milk from a wet-nurse. If the amount of food is insufficient and the milk is also poor in regard to its fat content, an effort should be made to improve the quality by increasing the amount of fat in the mother's diet. This, however, often fails to raise the fat content, so that it may be necessary to give a dram or two of cream before each nursing. In artificially fed babies who suffer from constipation the formulas and mixtures should be analyzed to ascertain if the fat content is not too low, and to reduce the percentage of protein if in excess. A steady milk diet in later infancy, supplemented at times by the addition of oat meal gruel, orange or prune juice, or an occasional baked apple, will often correct or prevent constipation.

With older children if the food does not leave enough residue to force the bowels to move regularly, the amount of fruit and vegetables should be increased. In regulating the diet of constipated infants and children so as to secure the laxative action of certain articles of food, the mistake is too often made of overdoing the matter; hence

the artificially fed infant, in particular, is sometimes given as high as 6 per cent. of fat mixtures in order to facilitate bowel evacuations, notwithstanding the fact that to raise the fat content above 4 per cent. will tend to constipate. The older child, too, is stuffed with fruits and cereals which overtax the stomach and intestines, not only producing obstinate constipation but chronic gastro-enteritis as well. Too often flatulent dyspepsia is begun in infancy in consequence of the frequent administration of cane sugar or oatmeal for the relief of constipation when the judicious use of specially prepared carbohydrates, such as Mellin's food or Horlick's malted milk given once or twice a day, is all that the infant requires to insure regularity of the bowels.

In some children constipation is due largely to a lack of proper training with regard to the bowels. As soon as it can sit up, the infant should be placed upon the chamber each morning after its first nursing, and if this practice is continued throughout infancy and early childhood a habit is formed which will persist through life. The school child should not be allowed to depart from the house in the morning until an effort has been made to evacuate the bowels, and schools should be so conducted that children have an opportunity of using the toilet whenever necessary, instead of being compelled to suppress this desire as is often the case. Water may always be given freely to the constipated child. In the treatment of chronic constipation, particularly, it is very beneficial to take a glassful before breakfast and at night before retiring.

The use of enemata and suppositories is so liable to be abused that we frequently find in them a cause of, rather than a remedy for chronic constipation, since by habitually resorting to them the bowel becomes so accustomed to the powerful local stimulus produced that there is little or no response to natural stimulation alone. If only occasionally employed, however, they are of unquestionable value in securing evacuation of the lower bowel when the purgative action of drugs is not desirable. In infants, especially, the simple expedient of inserting a cone of Castile soap into the rectum will cause the bowels to move, but the mother should not make a practice of this, as it may give rise to severe irritation of the rectum and anus. Glycerin suppositories are far more irritating than cones of soap, hence should only be used occasionally, and suppositories containing drugs are rarely of any value in infancy and childhood.

Furthermore, enemata, while they are always effectual in giving temporary relief, should never be used frequently or indiscriminately, since they have a tendency to balloon the rectum, also to favor the accumulation of feces, and may cause atony of the bowel, thus greatly aggravating the constipation. If, however, the amount of fluid injected is but small, dilatation of the rectum is not likely to happen. For this reason it is advisable to inject an ounce of sweet oil or olive oil, or twice that amount of warm soapy water, rather than to wash out the rectum and colon with a half-pint or more of plain water or saline solution. Greater activity on the part of the infant or child should be encouraged

as a valuable adjunct to treatment, and massage of the abdomen, especially along the course of the colon, is often beneficial.

Constipation which is due to a local lesion of the anus or rectum depends for its relief upon the cure of that condition. If there be spasmodic contraction of the anus with no apparent organic lesion, such as fissure in ano, etc., sodium bromide in 2-grain doses or tincture of belladonna in drop doses may be given the infant, and warm applications made locally. Stricture of the anus requires dilatation either by the finger or by more forcible divulsion, and fissure in ano is best treated by keeping the bowels loose, and anointing the lower part of the rectum and anus with unguenti zinci oxidi, unguenti hydrargyri, or a simple boric acid ointment.

Electricity is the least valuable of all therapeutic measures in the treatment of constipation, and therefore is practically to be regarded as a last resort, and of service only in those cases where constipation is due solely to atony of the intestinal musculature. The galvanic current is more effectual than the faradic; when employed, one electrode should be placed in the rectum and the other passed over the colon. If it is desirable to use the faradic current, one electrode may be placed over the spine and the other passed with pressure over the abdomen along the course of the colon. While neither electricity, massage, nor hydrotherapy in the form of the cold morning bath, if used alone, is effectual in the treatment of chronic constipation, yet the judicious combination of these measures with an occasional enema, persevered in faithfully for several weeks, will in many cases yield most gratifying results.

Medicinal treatment of constipation is a rather unsatisfactory and objectionable mode of correcting it, since drugs are of merely temporary service at the most, and if their use is prolonged the bowels become tolerant to all but massive doses, and it becomes impossible to secure a movement by any of the other methods suggested. If used at all, drugs should be given only occasionally during the course of more effectual methods of treating the condition, and the physician should aim gradually to lengthen the interval between doses, as well as to reduce the amount of the drug given. Now and then a course of calomel, 1 grain in divided doses, may be given the child of two or three years whose stools are grayish white. If there is atony of the muscular coat of the bowels, tincture of belladonna or tincture of nux vomica should be administered in drop doses for its stimulating effect. Cascara sagrada is, theoretically, the best medicinal agent if drug treatment is to be continued for any length of time since, in addition to its purgative action, cascara has a tonic effect on the bowels. The dose of the fluidextract is from two to ten drops according to the age of the child, and the elixir may be given in one-half to one teaspoonful doses. Maltine and cascara is very widely and wisely used. Aside from being an agreeable preparation, this is a valuable adjunct to treatment if administered properly, since it tones up the whole system in addition to its beneficial action on the bowels. Rhubarb, either in the form of

the aromatic syrup or the mixture of rhubarb and soda, is frequently given to children, but should be used cautiously since its continuous administration may aggravate the constipation. The same advice applies to the prescribing of senna, either in the form of the infusion, of which a half-ounce three times a day is the proper dose for a child of three years, or as the syrup, which is administered in 10- to 20-drop doses.

If colic and flatulence are present salines are indicated, and 10 to 30 grains of sodium phosphate should be given daily in cold water before the morning meal. This, however, should only be continued until the child's bowels move every day after breakfast, and then the dose should be gradually reduced until the drug can be withheld altogether. Olive oil in dram doses is frequently sufficient to cause an evacuation of the bowels in the young infant, and does not gripe as does castor oil. Liquid petrolatum, liquid albolene, and Russian mineral oil, if pure, are also preferable to less inert agents, since their action is for the most part mechanical. The dosage of any of the mineral oils is from 15 to 100 drops. If given in milk their presence is unsuspected. For bottle-fed babies, milk of magnesia is quite a popular laxative, a teaspoonful being added to the feeding mixture. In most cases it acts very well; but in common with all other drugs used for chronic constipation it should be given only temporarily, and should be discontinued as soon as possible.

INCONTINENCE OF FECES.

The age at which a child exercises control over the act of defecation depends a great deal upon the training it receives. If no effort is made to form a habit, voluntary control of the bowels will in most cases be spontaneously established at about two years of age; but if, at the third or fourth month, the infant is placed upon the chamber each morning after feeding, the habit of having a stool at that time every day will have become fixed by the sixth month. Incontinence of feces, therefore, should not be considered abnormal before the second year; but after this it is a pathological condition usually the result of an organic lesion elsewhere in the body, but occasionally functional or due to local irritation.

Etiology.—Fecal incontinence is seen most frequently as the result of injury to the spinal cord, transverse myelitis, paraplegia, and comatose conditions; also in cases of severe asthenia with marked adynamic nervous state, such as occurs in typhoid fever, pneumonia, or cholera infantum, and in grave attacks of scarlet fever, diphtheria, or other acute infections. Except when found in association with the foregoing conditions it is exceedingly rare, and may not be observed in the otherwise apparently healthy child, as is urinary incontinence. Sometimes incontinence of feces is associated with enuresis in extremely nervous children who show other signs of nervous instability, such as night terrors, stuttering, and habit spasms; occasionally there is fecal incon-

tinence without enuresis. The stools are abnormal, but not neces-
sarily diarrheal in character, since the bowels may be moved but once
daily, yet the movements have an offensive odor, are slimy, and some-
times show fermentation. This condition is regarded as a nervous
affection of the intestines, probably in some way related to the mucous
colitis of adult life.

Prognosis.—The prognosis is always good unless there is associated
organic brain or cord lesion or mental deficiency. As a rule, the habit
may be stopped within a week. When observed during the course
of a severe acute illness it is merely a transient sign of asthenia, and
disappears promptly with recovery from the primary disease. As in
enuresis, relapses are common, and should be guarded against rigidly.

Treatment.—The child with incontinence of feces should, first of
all, be restricted as to diet, and all foods which have a laxative action
on the bowels, such as fresh fruits and green vegetables, should be
prohibited. In most cases a nerve tonic is needed, and excellent results
may be obtained from the administration of one to three drops of tinc-
ture of nux vomica or Fowler's solution, to which a nerve sedative,
such as Dover's powder, 1 to 3 grains, or a combination of tincture
of belladonna and sodium bromide should be added to relieve the
excessive nervous irritability. Bismuth subnitrate in from 2- to 10-
grain doses, according to the age of the child, should be given every
three hours for one or two days if the bowels are loose.

DIARRHEA.

Diarrhea, one of the most common affections of infancy and child-
hood, is said to exist when the stools become loose and watery, and are
much increased in number. It is always a sign of some intestinal dis-
order, since it is caused by increased peristalsis and increased intes-
tinal secretion. Moreover, since diarrhea results in marked disturb-
ance of digestion and nutrition, the diseases of infancy in which it is
the most prominent symptom have by far the highest mortality.

Etiology.—Diarrhea is, as a rule, observed in children from six
months to two years of age, a fact largely accounted for by the liquid
diet which they receive at this period of life. It does not occur in the
breast-fed infant unless there is some disturbance of lactation, hence
its comparative infrequency before the sixth month. But the fact that
90 per cent. of cases of diarrhea are seen in bottle-fed infants seems
almost proof positive that in improper feeding lies the chief cause.
The error in diet may be either overfeeding, faulty composition of the
formula, or contamination of the milk; here, again, the great prevalence
of diarrhea in the summer time would seem to indicate that unsuitable
or, more likely, spoilt milk, plays a large part in the diarrheas of infancy.

In large cities the number of cases of diarrhea and the mortality vary
each summer in direct proportion to the height of the temperature
and duration of the hot spells, which shows also the effect of excessive
heat in the production of this disease. Milk may also be unfit for use

because of contamination by filth from the stable, street, or store; hence the poorer classes furnish most of the cases of diarrhea which are due, in an indirect way, to unhygienic surroundings, poorly kept food, also to lack of fresh air, cleanliness, and sunshine. Fatalities from infantile diarrhea are far more numerous where there is a constitutional dyscrasia, such as tuberculosis, syphilis, rachitis, or malformation, from any cause whatsoever.

Classification.—There are two main groups of diarrheas: the simple, or non-infectious, and the infectious. Simple diarrhea may be classified as follows:

Mechanical.—That produced by indigestible articles of food which act as foreign bodies in the intestinal tract. In these cases the diarrhea is an indication of Nature's desire to rid the intestines of the irritating material, hence the best treatment is an initial purge to sweep out the offending substance, after which recovery is usually prompt.

Medicinal.—In some infants the laxative effect of an ordinary cathartic will persist indefinitely. The milk of the mother, too, has a cathartic effect after she has taken certain purgatives.

Eliminative.—Here diarrhea may be a demonstration of Nature's effort to rid the system of toxins by way of the bowel; for instance, eliminative diarrhea is seen in uremia, and occasionally in acute infectious diseases.

Reflex.—Reflex diarrhea due to nervous shock, such as great fright, or excitement, is rather rare in children, but does occur, and is characterized by an excess of mucus in the stools. Occasionally diarrhea is brought on by chilling the surface of the body.

Treatment of Diarrheas.—The first step in the treatment of a simple diarrhea is to ascertain the cause and remove it. In the majority of instances it is good therapy to give a full dose of castor oil, after which bismuth may be administered in fairly large doses, 5 to 10 grains, every two or three hours for a day or two. In the severe diarrhea of infancy it is well to stop all milk, and give nothing but water for 24 to 48 hours. In less severe cases barley-water, rice-water, egg albumen, or very weak broth may be substituted for the milk in order that the child should not suffer from utter lack of nourishment, and as soon as improvement sets in milk feeding should be resumed, beginning with very small quantities.

INTESTINAL BACTERIA.

At birth the intestines contain no bacteria, but shortly after nursing is begun the B. lactis aerogenes, B. coli communis, and putrefactive organisms may be found in the intestinal contents. The stool of a normal infant may reveal just as many organisms as that of the child who is ill; but, for the most part, they are non-pathogenic, and practically include all the bacteria found in the mouth. It is quite generally believed that the presence of certain bacteria in the intestinal tract is essential to good health, since if they are continually destroyed in the milk, the infant is liable to develop scurvy. The Bacillus acidophilus

is invariably to be found in the mouth and intestines, and the B. bifidus communis is often detected in the stools of breast-fed infants. Lactic acid is formed in the intestines by the action of the Bacillus lactis aërogenes, which depends for its growth upon milk sugar, and therefore invades the upper intestine, also by the action of the Bacillus coli, which lives upon intestinal secretions. In addition to these organisms, saprophytes, non-pathogenic cocci, the B. subtilis and, occasionally, the bacillus of Shiga, are found in the stools of normal infants, although the Shiga bacillus is the organism responsible for acute gastro-enteric infection in infancy.

MUCOUS COLITIS.

Mucous colitis, or mucous disease, is a chronic inflammation of the intestines characterized by intestinal indigestion and an excessive production of mucus, with the formation on the mucosa of a pseudo-membrane which is passed at intervals in the form of casts of the intestine and in ropy masses. The disease is essentially the same as that which occurs in the adult and, as a rule, the signs and symptoms are similar.

Etiology.—This disease is most common in delicate, neurotic children who are of neuropathic ancestry and precocious. It is very rare during infancy, usually occurring between the ages of five and twelve years. There may be a history of antecedent catarrhal colitis or of frequent attacks of intestinal indigestion.

Symptoms.—Mucous colitis is apt to appear in recurrent attacks during which the child is acutely ill, complains of severe abdominal pain, and passes casts of the intestine and shreds of mucus in the stools. As a rule, there is constipation, with hard dry movements which are covered with mucus. Occasionally a stool is normal except that it contains large quantitites of mucus. The appetite is capricious, the tongue coated, the skin pale and anemic-looking. The child is irritable and peevish during the day and very restless at night; often it is undernourished and loses weight. The attacks are brought on by indiscretions in diet or by nervous excitement such as that which fright and competitive games produce.

Diagnosis.—The diagnosis is, as a rule, readily made upon finding mucous casts of the intestines and an excessive number of mucous shreds in the stools. The character of the stools will enable us to differentiate attacks of mucous colitis from the abdominal pain due to appendicitis; while a careful study of the signs and symptoms and consideration of the history will differentiate it from tuberculous peritonitis, which it occasionally resembles.

Prognosis.—The outlook is favorable for final recovery; but the disease runs a protracted course with frequent exacerbations and relapses.

Treatment.—If necessary the bowels should be regulated by an evening dose of fluidextract of cascara sagrada, 20 to 30 drops. Enemata and colonic irrigations are contraindicated because of their tendency

still further to irritate the colon, but a glycerin suppository may be used occasionally.

Dietetic treatment is of the utmost importance, the progress of the case depending upon the careful and continued regulation of the diet, since each indiscretion is followed by a relapse. Carbohydrates are especially harmful in these cases, and all sugars and starches should be limited. Pastries, cakes, candies, potatoes, and bread must be prohibited or greatly restricted, and broths, stewed fruits, cereals, and meats substituted. Toast may be given in place of fresh bread. As a rule, milk is harmful.

These children are anemic and below par, and they require tonic doses of tincture of nux vomica, one to three drops, or Fowler's solution, one to three drops, after meals. Improvement is much more rapid if a change of climate can be secured by sending the child to the sea-shore or mountains.

AMYLOID DISEASE OF THE INTESTINES.

Amyloid disease of the intestines is rare during infancy, but may be seen occasionally in association with amyloid disease of the liver, spleen, or kidney. The exact metabolic changes which cause the deposition of lardaceous material is not understood, but the disease attacks children who are afflicted with syphilis, tuberculosis, or some chronic suppurative process.

Pathology.—At the onset of the disease, a lardaceous substance is deposited in the walls of the small vessels of the intestinal villi, and is later found also in the mucosa and submucosa.

Symptoms.—This disease gives rise to no definite or characteristic symptoms, and the diagnosis is made only at autopsy.

Treatment.—There is no specific treatment for amyloid disease; but, in cases where it is suspected, the underlying cause should be ascertained, and removed if possible.

INTESTINAL OBSTRUCTION.

Obstruction of the intestines may be either congenital or acquired and, while the congenital form is the one usually met with during childhood, the acquired type is by no means uncommon. The affection when acquired is the result of intussusception, strangulated hernia, volvulus, impaction of a foreign body in the intestine, peritonitis, constricting bands, or the pressure of intra-abdominal tumors. The congenital form is due to malformation of the intestinal tract, hernia of the umbilical cord, imperforate anus or rectum, or other anatomical anomaly.

Symptoms.—The symptoms of complete obstruction of the bowels are vomiting and the passage of a small amount of feces mixed with blood and mucus, after which there is absolutely no movement of the bowels. Vomiting persists, and becomes stercoraceous if the

obstruction is not relieved, while pain is severe and paroxysmal. The pulse soon becomes weak, exhaustion and prostration ensue, and sepsis quickly supervenes from the absorption of toxins. The temperature is high; the abdomen is distended and tympanitic. If the obstruction is not relieved the gut now becomes gangrenous, pain ceases, sepsis is profound, and the child dies in collapse within twenty-four hours from the time gangrene sets in.

Diagnosis.—The diagnosis is based upon the history of absolute constipation, persistent vomiting which becomes stercoraceous, and symptoms of collapse with severe abdominal pain, together with other symptoms which vary according to the nature of the obstruction.

Prognosis.—The prognosis of intestinal obstruction is at the best unfavorable. The chances of recovery are greatest in those cases in which the diagnosis is made early. After gangrene has set in death is almost a certainty.

Treatment.—The treatment is wholly surgical, and consists in abdominal section and removal of the obstruction. If a portion of the gut is gangrenous, resection is also necessary.

VOLVULUS.

Volvulus is a twisting of the bowel upon itself in such a way that its lumen is occluded and obstruction is the result. The onset is sudden, and is marked by severe pain followed by absolute stoppage of the bowels. The abdomen is distended and extremely tender, but there is rarely stercoraceous vomiting. It is impossible to find a tumor either by abdominal palpation or rectal examination. The symptoms rapidly become aggravated, but collapse does not appear as quickly as in other forms of bowel obstruction. This condition is very rare in children; when it occurs the sigmoid is most frequently the portion of bowel involved, and the treatment is surgical.

APPENDICITIS.

This affection is of much less frequent occurrence in children than in adults, and is extremely rare during infancy. The majority of cases are observed in children between five and fifteen years of age. The acute form is the more common and, as a rule, an attack of appendicitis runs a much more rapid course in children than in adults. Chronic appendicitis is very infrequent during childhood. In many respects the disease presents quite a different aspect in the child, the symptoms being more obscure, and the appendix lying farther above the pelvic brim than in the adult, which makes the seat of tenderness and pain above McBurney's point. The treatment also differs to a certain extent from that in the adult.

Etiology.—Appendicitis is more common in boys than in girls, and becomes more frequent as the child advances from infancy. Its rarity in children before the second year is due to the infant's liquid diet

and the relatively wider lumen of the appendix at this age. Heredity may be considered a predisposing cause when a tendency to constipation is inherited or when certain anomalies of the appendix are observed in successive generations of a family. Fecal concretions which block the lumen of the appendix are the cause of most of the acute attacks, but obstruction of the appendix is rarely due to a foreign body.

In many instances there is a history of digestive disturbance and constipation; but these conditions may be regarded merely as predisposing factors, since bacteria are the exciting cause in every case. An extension of intestinal inflammation into the appendix may produce appendicitis; but this is infrequent, and only in exceptional cases do we elicit a history of preceding injury to the abdomen or of intestinal parasites. The affection is occasionally associated with acute infectious diseases, such as scarlet fever, follicular tonsillitis, and typhoid fever.

Pathology.—The same forms of acute appendicitis are observed in children as in the adult—the catarrhal, suppurative, perforative, and gangrenous. Ulceration or perforation is quite common, and increases the gravity of appendicitis in children. Catarrhal appendicitis is characterized by swelling and rigidity of the appendix, hyperemia of the mucous membrane, and infiltration of the walls with round cells. The lumen of the appendix is almost obliterated, and usually contains fecal concretions and mucus or mucopus.

In inflammation of the appendix in children there is a marked tendency to pus formation and, as a consequence, the catarrhal type is usually followed by suppuration. In a few instances recovery ensues without pus formation, while more rarely chronic catarrhal inflammation may follow. In suppurative appendicitis the inflammation of the appendix is more severe owing to the deficient blood supply and more virulent infection; all the coats of the organ are involved, and its lumen is filled with pus. In acute appendicitis in children the tendency is toward abscess formation; but, although perforation is not infrequent, peritonitis is rare, for the abscess is usually walled off by adherent omentum.

Gangrenous appendicitis is the result of an obstruction to the circulation in the appendix which causes the tip to become necrotic. As in the perforating form, the contents of the appendix are discharged through the opening thus made, and this sets up a localized or general peritonitis according to whether or not there are inflammatory adhesions to limit the spread of infection.

Localized gangrene of the appendiceal wall from pressure of a large fecal concretion is the usual cause of perforation of the appendix; but in these cases peritonitis does not assume the grave septic aspect commonly observed in gangrenous appendicitis. Intestinal obstruction following acute appendicitis, and due to strangulation of the gut and inflammatory adhesions about the appendix, while comparatively rare, occurs with greater frequency in children than in adults. Chronic

appendicitis, although seldom seen in children, may be the sequel to several acute catarrhal inflammations. At autopsy the appendix is found to be thickened, tortuous, and bound down by adhesions to the adjacent tissues.

Bacteriology.—While inflammation of the appendix can be attributed to no special organism, yet in the majority of cases in which bacteriological studies have been made the colon bacillus has been found in large numbers. Among the other organisms isolated are typhoid and tubercle bacilli, and the streptococcus and staphylococcus.

Associated Lesions.—Appendicitis, especially the acute suppurative form with abscess formation, is occasionally the cause of suppuration in other organs. This tendency to secondary abscess is more marked in children than in adults. Not infrequently an abscess may be discovered in the liver, brain, parotid gland, lung, or in tissues adjacent to the appendix.

Symptoms.—Mild catarrhal appendicitis often escapes recognition in children, owing to the fact that the symptoms are not unlike those of acute gastric or intestinal indigestion. There are pain and tenderness in the abdomen, but these are rarely referable to the right iliac fossa; and, unless appendicitis be suspected and an attempt made to elicit tenderness over the appendiceal region, no local symptoms may be apparent.

The onset of such an attack is sudden; it may or may not be preceded by gastro-intestinal disturbance as manifested by anorexia, general malaise, and either constipation or diarrhea. Vomiting is rather a constant feature, often persisting until the attack subsides. There is usually a hypersensitive state of the bladder with frequent and, sometimes, painful micturition, and by rectal examination of the cecal region we can often detect inflammation of these parts.

Rigidity of the abdomen is not so valuable a diagnostic sign in children as in adults, since, even though there be neither pain nor tenderness, the child may resist all efforts to palpate the abdomen. If the inflammation is but slight, the temperature does not ascend above 101° or 102° F., and in the course of three or four days subsides. There is, however, greater elevation of temperature in children than in adults, the height always depending upon the severity of the attack except when gangrene or perforation has caused the temperature to fall.

In a severe attack of *acute catarrhal appendicitis* the symptoms are much more pronounced and the disease more clearly defined. The fever may rise above 105° F.; the pulse is rapid; vomiting and pain are severe. There is distinct tenderness over the appendiceal region, which in children is a little above McBurney's point, and the right rectus muscle is rigid. If the abdomen be examined before rigidity appears, it may be possible to palpate the firm, swollen appendix by making pressure on the back opposite to McBurney's point with the left hand while palpating deeply in the region of the appendix and down into the pelvis with the fingers of the right hand.

22

In *ulcerative* or *perforative appendicitis* the symptoms at the onset are those of a severe form of acute catarrhal inflammation. The attack comes on suddenly with vomiting, constipation, fever of 102° to 105° F., and severe abdominal pain. At first the increase in the pulse rate corresponds with the elevation of temperature; but after perforation the temperature usually falls, while the pulse becomes more rapid and thready.

If the appendix is well walled off by the omentum and inflammatory adhesions, perforation causes merely a localized peritonitis which is indicated by an area of induration about McBurney's point. When suppuration takes place an abscess forms, and the patient suffers from chills and sweats. At this stage the child usually assumes a characteristic posture; it lies on its back with the knees drawn up, and the abdomen held quite rigid. The pulse is rapid, the tongue coated; vomiting continues; the face wears an anxious, pinched expression.

If an abscess forms, a tumor-like mass may be felt in the right iliac fossa and the whole abdomen be distended; but fluctuation is difficult to make out because of the depth of the suppurative process. Its location depends, however, upon the situation of the appendix. If the organ lies in the pelvis, the abscess is often readily perceptible upon rectal examination.

Few cases are allowed to proceed beyond this stage without operation; but until the abscess is opened it continues to increase in size, pain and tenderness persist, the child stoops or limps if placed upon its feet, and the temperature remains elevated. As a rule, fluctuation is easily detected. The abscess either ruptures externally through the skin or empties its contents into the general peritoneal cavity, and thus gives rise to peritonitis. In rare instances the pus is evacuated into the rectum, bladder, or vagina, and peritonitis does not occur.

Gangrenous appendicitis, while most severe, is not accompanied by any characteristic symptoms, and the course of an attack is so deceptive that the mortality is very high. This is because the child's exact condition is not recognized, and the decrease in pain and tenderness, and decline of the fever which accompanies gangrene of the appendix, are mistaken for signs of recovery, whereupon operation is postponed, and septic peritonitis develops a day or two later.

The shock from rupture of a gangrenous appendix is profound, and is usually attended by vomiting, acute pain, and collapse, while ordinary perforation or ulceration of the appendix causes sharp pain, a fall in temperature, and accelerated pulse rate.

General peritonitis—which follows perforation or gangrene of the appendix when the appendiceal region is not walled off—is marked by a rise of temperature, weak, thready pulse, rapid and shallow respirations, cold and clammy skin, and often persistent hiccoughs. The face wears an anxious expression; nausea and vomiting continue. The child is constipated. The abdomen is distended and tympanitic, and there is a board-like rigidity of all the muscles of the abdominal

wall. General peritonitis is most common in gangrenous appendicitis, the disease being so acute that there is usually insufficient time for the walling off of the appendix; consequently, when it ruptures the contents escape into the peritoneal cavity. Recovery from general peritonitis is rare, especially from the form which follows gangrene of the appendix, this being usually of extremely septic type.

Chronic Appendicitis.—The symptoms of chronic appendicitis are much milder than those of the acute form. There is a history of one or more acute attacks, followed by more or less complete recovery; but the child constantly complains of pain and discomfort in the appendiceal region. There may be slight fever at intervals, and occasionally an attack of appendiceal colic with vomiting and constipation. These children, while, perhaps, not as healthy as the normal child, do not show the nervous effects of chronic appendicitis which are so frequently observed in adults who suffer from this disease; and, as a rule, there is little emaciation or debility.

Diagnosis.—The all-important aid to the diagnosis of appendicitis in children is rigidity of the abdominal wall, especially on the right side, together with accompanying localized symptoms, such as pain and tenderness in the right iliac fossa. In the absence of these indications of appendiceal inflammation the diagnosis is very difficult, and may be impossible, since all of the additional signs and symptoms are those which may also be found in other gastro-intestinal affections. If the appendix lies in an anomalous position, appendicitis is rarely thought of, and may altogether escape recognition unless an exploratory operation be performed.

It is highly important that appendicitis be recognized early; at the same time, because of its similarity to many other diseases of the gastro-intestinal tract, careful differentiation is often necessary. Intestinal colic may be ruled out by the absence of fever and localized tenderness, also by the tendency of the symptoms to subside within a short time. In bowel obstruction there is neither tenderness nor pain in the right iliac fossa. In intussusception the tumor can usually be palpated in the centre of the abdomen or on the left side, and may often be detected by rectal examination. The stools contain blood and mucus; there is no fever at the onset; constipation becomes absolute. Intussusception is also rare after infancy, while volvulus is so extremely rare in children that it needs no consideration.

For a day or so it may be impossible to exclude acute indigestion, enterocolitis, and colic with fever; but, after the lapse of twenty-four hours, colic and acute indigestion usually show marked improvement with cessation of the pain or localized tenderness in the right iliac fossa, while in enterocolitis the constant diarrhea with mucous stools discloses the nature of the affection.

Right-sided pneumonia, especially when accompanied by diaphragmatic pleurisy, may produce signs closely simulating acute appendicitis. The right rectus muscle may be rigid, and, until physical signs appear in the lungs, a diagnosis is frequently impossible. Rapid

respiratory rate, limited motion of the right chest, and negative rectal examination of the cecal region may all favor pneumonia. The leukocyte count, while not of great service in differentiating appendicitis from pneumonia, is a valuable adjunct in the exclusion of gastrointestinal disturbances, such as acute indigestion, colic, ileocolitis, and intussusception.

To a certain extent one can also differentiate acute catarrhal inflammation from suppurative appendicitis; but it is by no means always possible. As a rule, a count of 12,000 or less usually signifies catarrhal inflammation, while a leukocytosis of 20,000 or over indicates suppuration. It must also be borne in mind that in severe acute cases there may be no leukocytosis. In order to obtain the most accurate data from a blood examination in appendicitis, a series of counts should be made, since a rising leukocytosis is far more significant than a single high count.

Prognosis.—When an early diagnosis is made, and the case is operated upon immediately, the prognosis is favorable. In mild cases of acute catarrhal appendicitis the outlook is, as a rule, also favorable; but suppuration and abscess formation are more common than in adults. Cases of appendicitis which are operated upon late, and those occurring in children under five years of age, are alike more unfavorable as to outcome.

Treatment.—Rest in bed is imperative. During the early stages of inflammation, an ice-bag should be kept over the appendiceal region. Nothing should be given by mouth but water or albumen-water. If pain be severe, and an operation is to be performed, opiates may be given in small doses—$\frac{1}{8}$ to $\frac{1}{2}$ grain Dover's powder. But in those cases which the physician must treat expectantly, either because the diagnosis is not clearly established or operation is refused, opiates are contra-indicated, since they mask important symptoms.

An enema may be given to open the bowels; in some cases it is well to place the child in a sitting posture with the knees flexed, as suggested by Fowler, and give salt solution by slow proctoclysis. When the diagnosis is positive, surgical interference should be urged, and no further attempt need be made to treat the case medicinally. If operation is refused, the child should stay in bed on a liquid diet, and the bowels be kept moving daily by the use of enemata.

These cases must be closely watched. If the fever continues and no improvement is noted, operation is demanded. Any sign of perforation or gangrene, as shown by a drop in the temperature with accelerated pulse rate and evidences of shock, calls for immediate operation. If, on the other hand, there is improvement under medicinal treatment, the case may be allowed to proceed to recovery. An interval operation should, however, be advised, since one attack of appendicitis is usually succeeded by others until the appendix is removed.

The treatment of chronic appendicitis is surgical. An appendectomy should be performed in the interim between acute exacerbations. In

treating appendicitis in children the medical man should always secure the assistance of a surgeon. Owing to the tendency to abscess formation, operation is the wisest procedure in every case.

INTESTINAL WORMS.

Children are nowadays much less frequently infested with worms than they were a generation or so ago, and it is today the consensus of opinion that the injurious effects of intestinal parasites have been greatly overestimated. The same varieties are found in children as in adults; but in the majority of children the parasites present are the threadworm and the roundworm.

Etiology.—Children of the poorer classes are the usual hosts of these parasites, the disease being seen less commonly in private practice. Infants seldom harbor worms, for the reason that parasites gain entrance to the body through the ova which are swallowed with food and water, or are carried to the mouth by means of the fingers, or are on articles picked up off the floor or street by young children.

Symptoms.—The general symptoms produced by intestinal worms are indefinite and vague. Anemia, loss of weight, diminished appetite, and peevishness may be the only signs that the child is ailing; but in severe cases we find vomiting, colic, restlessness and moaning at night, a constant desire to pick at the nose, gritting of the teeth, and more or less nervousness, sometimes even convulsions.

When the body is infested by intestinal parasites there is always secondary anemia; but, in addition, there are other and more characteristic changes. Eosinophilia usually accompanies all forms of intestinal infestation, but is most marked when trichinæ are present. In trichiniasis also there is usually leukocytosis. Severe secondary anemia as a result of intestinal parasites may exhibit a blood picture not unlike that of pernicious anemia. Aside from these general constitutional symptoms caused by animal parasites, there are other signs and symptoms which vary more or less according to the particular type of worm present.

Cestodes.—The Tænia mediocanellata, or beef tapeworm, and the Tænia solium, or pork tapeworm, are the most common varieties. The Bothriocephalus latus, or fish tapeworm, and the Tænia cucumerina, the Tænia nana, and the Tænia echinococcus are quite rare. Among the nematodes, the Oxyuris vermicularis, or threadworm, the whipworm, and the Ascaris lumbricoides, or roundworm, are the most common forms found in children. The hookworm, or Ankylostoma duodenale, or Uncinaria duodenalis, while quite common in the South, is rarely seen in the northern part of the United States. Trichinæ seldom infest children, since the ova are ingested by eating uncooked pork.

Tæniæ, or Tapeworms.—Tapeworms are matured or fully developed larvæ from the muscles and solid viscera of animals. The ova are taken into the bodies of various animals, and carried to the

solid tissues, especially to the muscles, where they lodge, and in two or three months they produce cysts the size of a pea within which there forms a tænia head or scolex. If the flesh containing these cysts is eaten uncooked, a tapeworm will develop from the scolex, and the segments of the tapeworm will mature several months after the fixation of the scolex in the intestine. When mature, the segments develop male and female generative organs.

Tænia Solium, or Pork Tapeworm.—The pork tapeworm is less common than the Tænia mediocanellata, or beef tapeworm. It develops in the small intestine after the ingestion of raw or underdone measly pork. Usually there is but one worm in the intestine, but there may be more than one. The Tænia solium ranges from six to thirteen feet in length. Its head is rounded, of pin-head size, and is succeeded by a thread-like neck, then by segments which gradually become shorter and broader. There are four suckers and a projecting circle of twenty-six long and short hooklets about the head of the tænia.

The segments, when mature, become detached and are passed out of the intestine with the feces. They are a little less than a half-inch in length and one-quarter of an inch in breadth, but vary so much in shape that at about three feet from the head they are quadrilateral. The female matrix occupies the middle of each segment, and is provided with eight to fourteen tree-like branches on each side. The male generative organs are small vesicles in the anterior portion of the segment. The sexual opening is on one side near the middle. The ovarian or uterine apparatus of a mature segment contains many thick-shelled eggs, each of which holds within it an embryo with six hooklets.

Tænia Mediocanellata, or Beef Tapeworm.—The beef tapeworm is the most common variety met with in the human being. It varies from 12 to 30 feet in length. In comparison with the Tænia solium, or pork tapeworm, its segments are thicker and broader, being approximately $\frac{2}{3}$ of an inch long and half as broad. The head of the worm is larger and thicker, and contains sucking disks, but no hooklets. The ova are larger, and the ovarian branches of the female matrix are more numerous.

Bothriocephalus Latus, or Fish Tapeworm.—This variety of tapeworm is found most commonly in foreign countries. It is longer than any other intestinal parasite, measuring 20 to 30 feet. The head is unarmed and club-shaped; it has two longitudinal suckers. The segments are broader than those of the other varieties of tapeworm, and the ovarian apparatus is rosette-shaped and situated in the centre. The ova are larger than those of the beef or pork tapeworm, and have a lid at one end. They develop only in fresh water, and here they form an embryo which is eaten by fish, These embryos form cysts in the viscera and muscles of the fish, and if the fish are eaten raw or only partially cooked scolices develop in the intestine.

Dwarf Tapeworm—Tænia Nana.—This tapeworm is the smallest variety found in man, measuring from $\frac{1}{3}$ to 1 inch in length, and $\frac{1}{50}$

of an inch in width. The head has four suckers, a rostellum, and hooklets. The segments are yellowish, short, and broad. Tænia nana is more common in children than is generally supposed.

Symptoms.—The subjective symptoms of tapeworm are vague and indefinite, and are directly due to the irritation produced by the worm within the intestine. There is always a certain amount of gastro-intestinal disturbance, which is often very slight, but in some cases may cause diarrhea, colicky pains in the abdomen, and either a capricious appetite or anorexia. The breath is foul; at intervals there may be nausea and vomiting. None of these symptoms, however, is at all characteristic of intestinal worms any more than it is pathognomonic of other derangements of digestion.

These children are usually irritable and peevish during the day and extremely restless at night from nervous excitation; but the symptoms referable to the nervous system are not as severe as those caused by the roundworm, and convulsions are rarely seen. In many cases the child may be apparently in good health; but there is usually a gradual loss in weight, accompanied by anemia which may reach an extreme degree, but is usually slight and of secondary nature. Eosinophilia is also a common finding on blood examination.

Diagnosis.—The diagnosis of tapeworm can be made only when the segments appear in the stools, and when their presence in the intestine is suspected a purge should be given to bring about their expulsion. Since the treatment for the different varieties of tapeworm differs slightly, the segments and the head should be examined microscopically in order to identify the particular type of worm present. If no segments are passed, the feces should be examined microscopically for the ova, which are quite numerous and easily found if a tapeworm inhabits the intestinal tract.

Prognosis.—It is seldom a difficult matter to expel a tapeworm, and since its presence causes no severe symptoms the outlook is in most cases very good. The only possible danger to life arises when the eggs of the pork tapeworm find their way into the stomach. When this occurs the embryos are formed in the intestinal canal and may pass through the intestinal wall, and become lodged in the muscles, the viscera, or even in the brain.

Treatment.—Prophylaxis is important, and consists for the most part in prohibiting for the use of the child any beef or pork which has not been inspected. This is especially imperative in those cases where rare beef or beef juice is being given to children, and here the mother should personally inspect the meat for cysticerci before she prepares it for the child. These cysticerci will look like small cysts the size of a pea in the beef. When practicable, the safest and surest precaution against tapeworm is thoroughly to cook all meat and fish, as this absolutely destroys the cysticerci.

Fecal discharges from children who harbor tapeworms should be carefully disinfected with 5 per cent. carbolic acid solution before being disposed of, to prevent infection from being transmitted to other

children or to animals by the ova which they contain. Care is also necessary to keep the child from reinfecting itself by its soiled hands or by the receptacle into which the stools are passed.

The most widely used drug for the expulsion of the tapeworm is aspidium, or male fern (filix mas), which should be given in doses of 5 to 30 drops, according to the age of the child. Both this and other drugs used for the expulsion of the tapeworm cause more or less irritation of the gastro-intestinal tract; therefore, it is advisable to use as little as possible, and in order to facilitate the action of the vermifuge the following plan of treatment is usually resorted to:

The night before the vermifuge is to be taken, the child is given a very light supper, and then a full dose of castor oil, $\frac{1}{2}$ to 1 ounce. Another dose of castor oil is administered before a very light breakfast the next morning. Then the aspidium is given, preferably in divided doses three hours apart until the proper amount has been taken, when another dose of castor oil is administered.

The stools are carefully examined for the head and segments of the worm. If the segments continue to come away or the symptoms persist, it may be necessary to repeat this treatment over and over until the head is found in the stool; for, unless it is passed, the worm will grow again. One or, at the most, two treatments will usually bring away the head.

While the child is being treated for tapeworm special attention should be paid to its diet, which should be as light as possible to augment the action of the vermifuge, and also to lessen the danger of gastro-intestinal disturbances from its irritating effect.

Other drugs used to expel tapeworms are pelletierin, dose 1 to 2 grains, infusion of pepo or pumpkin seeds, dose 1 dram, and an emulsion of turpentine containing 10 drops of turpentine to the dram. The latter is given in teaspoonful doses every three hours for a day or two, being preceded and followed by castor oil, or the turpentine may be given in 1-dram doses combined with an ounce of castor oil. An infusion of kooso or brayera may be made by adding a half-ounce of powdered leaves to a pint of water and of acacia in equal parts. From 2 to 8 ounces of this may be given, according to the age of the child, and should be followed by castor oil. Thymol in 5- to 30-grain doses has also been found efficacious.

Nematodes.

This variety of worms is much more common in children than the cestodes. More than one worm is usually present at a time, but they do not attain the size of the cestodes. Among the most common nematodes found in children are the round worm, threadworm, whipworm, trichina, and hookworm.

Oxyuris Vermicularis—Threadworm.—This is a very small worm, also called the pin- or seatworm, which inhabits the colon, and especially the rectum. It forms in such great numbers that large masses

composed of the worms may be passed by the bowel. They are quite commonly found in children, but may also infest adults. The female worm is white in color, and about half an inch long, while the male is but $\frac{1}{6}$ of an inch in length. They develop from ova in about two weeks after the ingestion of the eggs, which are of an irregular ovoid shape, and $\frac{1}{500}$ of an inch in length.

By the time the embryos reach the cecum they are sexually mature, and when the female arrives in the rectum she deposits there immense numbers of eggs that mature into myriads of worms which are discharged with the feces. Infection with the ova may take place by means of water or food, and also through the uncleanliness of persons infested. Scratching about the anus results in contamination of the hands which soil anything they touch, and thus spread the infection.

Symptoms.—Itching and irritation about the anus and genitalia are the most characteristic symptoms of threadworms. The irritation of the colon may also cause a constant discharge of mucus from the rectum. The child suffers most from the itching in the evening, for at this time the worms migrate and get into the folds of the rectum. During the day the child is very restless, and constantly scratches the buttocks.

Incontinence of urine is occasionally due to threadworms, and frequent micturition is a common symptom. As with roundworms, picking at the nose and grinding the teeth are rather characteristic, and indicate disturbance of the nervous system; while anemia, loss of appetite, and loss of weight show the effects of threadworms upon the general health of the child.

Sometimes there is an accompanying catarrhal colitis, and vulvovaginitis or balanitis may also be attributed to threadworms because the itching produced so easily leads to the habit of masturbation. Severe consequences from threadworms are rare; but appendicitis has been traced to an accumulation of these parasites in the intestine, and convulsions occasionally supervene.

Diagnosis.—Obstinate pruritus in children should make the physician suspect the presence of threadworms as a causative factor, and the stools should be examined for ova. A positive diagnosis is, however, impossible until either the worms or their ova are found in the feces.

Prognosis.—In the majority of cases, all of the worms may be swept out of the intestine in a short time by proper treatment; but, occasionally, it is difficult to rid the child of the parasites, either because they are in the cecum where injections do not reach them or because the child is allowed to become contaminated and continuously reinfected *via* the mouth.

Treatment.—When the worms are all in the lower part of the colon, enemata will often suffice for their removal. The lower bowel should first be irrigated with warm salt solution to wash out all of the feces and mucus, and then an infusion of quassia or garlic, or a 1 to 10,000 solution of bichloride of mercury, should be injected, 6 or 8 ounces being allowed to remain within the bowel for an hour. In order to

clean out the colon effectually a long rectal tube should be used for the injection.

But the threadworms are often lodged so far up in the bowel that this treatment is unsuccessful, therefore santonin and calomel, of each $\frac{1}{4}$ to $\frac{1}{2}$ a grain, or the fluid extract of quassia, 10 to 20 drops, should be given prior to the irrigation, and in addition a purge of castor oil, 2 drams to an ounce, or magnesium sulphate, 1 dram to half an ounce, to drive the parasites into the lower bowel. It is advisable to repeat the saline irrigation and the injections every evening for a week, by which time most children will have been cured.

In many instances examination of the stools will fail to reveal either ova or worms after the second or third injection; but when they have lodged in the cecum and also inhabit the small intestine, a much longer course of treatment is frequently necessary.

Treatment, while beneficial, may not wholly rid the child of threadworms because of the ease with which reinfection takes place. This is usually due to the transmission of the ova from the contaminated clothing and buttocks to the mouth by means of the hands, and especially the finger nails, beneath which the ova lodge when the child scratches. For this reason the buttocks should be carefully cleansed and bathed with a 1 to 10,000 bichloride solution after each bowel movement, and the itching should be controlled by anointing the anus with a dilute mercurial ointment at night and after each stool. All underclothing, diapers, nightgowns, and bed linen should be thoroughly boiled as well as cleansed when soiled.

The stools should be treated with 5 per cent. carbolic acid solution before being disposed of, and the receptacle they are passed in disinfected. The child, if old enough, must be taught to keep the hands clean by washing them whenever they have come in contact with the buttocks, and the finger nails should be scrubbed with soap and water to dislodge the ova. At night scratching may be prevented by sewing up the sleeves of the sleeping garment.

If the general health of these children has been affected, they should be put upon a nourishing but light diet, encouraged to play out of doors, and given a tonic containing tincture of gentian or quassia which will serve the double purpose of building up the system and, to a certain extent, acting as a vermifuge.

Ascaris Lumbricoides—Roundworm.—The roundworm occurs more frequently in children than any other intestinal parasite, particularly in children between the ages of three and ten years. It inhabits the upper portion of the small intestine. In appearance it is very much like the common earthworm, its body being round, fusiform, and of a reddish-brown color. The female is 7 to 14 inches long, while the male measures from 4 to 8 inches. It is about as thick as a goose quill, and the head is furnished with three oval papillæ which have fine teeth.

The ova are elliptical in shape, of a dark reddish color, and about 0.05 mm. long. They are, in all likelihood, ingested with water and

food. At times these worms migrate from the small intestine, having been found even in the pharynx, mouth, nares, larynx, and trachea, and have caused asphyxia, pulmonary gangrene, and hepatic abscess.

Symptoms.—A child may harbor a number of roundworms in the intestine without showing any sign whatsoever of their presence. Intestinal irritation may give rise to digestive disturbances with colic, tympanites, constipation or diarrhea, and loss of appetite, or even nausea or vomiting. These children are usually very restless at night and peevish during the day, with a tendency to pick at the nose continually and to grind the teeth.

Evidence of rectal irritation may also be noticed, but is not characteristic, while headache is not uncommon. Nervous symptoms are usually mild; but occasionally round worms may cause dizziness, hysteria, syncope, or convulsions. The harmful effects of roundworms on the nervous system are supposed to be due to the absorption of poisons which they excrete into the intestinal canal. Eosinophilia is a common finding.

Diagnosis.—It is scarcely possible to make a diagnosis of roundworm infestation upon the symptoms enumerated above; therefore, unless their presence is suspected and the stools are examined for ova, the diagnosis is not made until a worm is passed in the stool, and discovered by the child or its parent. The stools of children who harbor roundworms always contain a multitude of ova which are easily detected by microscopic examination. As a rule, several worms are present in the intestinal tract at one time; hence the persistence of the symptoms after the passage of one or more worms indicates that there are still others in the bowel.

Treatment.—The best drug for expelling roundworms is santonin. The child should be given a light supper and a half-ounce to one ounce of castor oil before going to bed. The following morning santonin should be given, combined preferably with calomel. A child one year old should take a half-grain three times during the day with the same quantity of calomel. This dose may be doubled for children over three years old. The last dose of santonin is given in the evening, and the next morning from 1 to 3 drams of magnesium sulphate should be administered before breakfast.

If more worms are passed after this treatment it must be repeated until they are no longer seen in the stools and microscopic examination of the feces shows the absence of ova. Other vermifuges which may be used are fluidextract of spigelia, dose $\frac{1}{2}$ to 1 dram, and senna. To prevent the child from reinfecting itself the hands and anus must be kept perfectly clean and as free from ova as possible. The expulsion of all the worms is usually followed by a cessation of the symptoms, after which no further treatment is necessary unless the child has become anemic, when an iron tonic should be prescribed.

Trichocephalus Dispar.—The Trichocephalus dispar, or whipworm, frequently accompanies threadworms when they infest the colon, but its presence is rarely discovered. This parasite closely resembles

the threadworm. The male measures 3 to 4 cm. in length, while the female is somewhat longer and sometimes attains a length of 5 cm., or 2 inches. The tail end of the female is conical and pointed, while that of the male is blunt and coiled up like a spring. The rest of the worm up to the head is slender and hair-like. The head is very small, but has the power of attaching itself most firmly to the intestinal wall.

The ova are but 0.05 mm. (0.0012 of an inch) long, and have a button-like projection at one end. In most instances this parasite lodges in the cecum, although it may also inhabit the small intestine and the appendix. It is very rarely found in infants; but of all the parasites that infest man the trichocephalus is said to be more common during childhood and adult life than any other. It is supposed to gain entrance to the intestine by the ingestion of the ova in drinking water.

Symptoms.—As a rule, only a few worms are in the intestine at one time, therefore the symptoms are few and very slight; but when a large number of these parasites collect in the cecum the consequences may be serious. In such cases anemia is quite marked, and even brain symptoms have been attributed to the whipworm. On account of the usual association of the threadworm with the whipworm, the threadworm being present in far greater numbers, it seems reasonable to suppose that these symptoms are only in part due to the Tricho-cephalus dispar.

Diagnosis.—The presence of this parasite is rarely diagnosed during life, although the ova may be found in the stools and identified under the microscope.

Treatment.—In order to prevent the whipworm from effecting entrance to the intestine, precautions should be taken to ascertain the purity of all drinking water. Salt solution and infusion of garlic or quassia, or a solution of 1 to 10,000 bichloride of mercury, should be used as injections in the manner described in the treatment of threadworms; but it is usually necessary to supplement these measures by the oral administration of drugs because of the habitat of the whipworm, which is too high in the colon to be effectively reached by rectal irrigation alone. Calomel, 1 to 3 grains, should be given, followed by a full dose of castor oil.

After the bowels have been moved a vermifuge, such as santonin, $\frac{1}{4}$ to $\frac{1}{2}$ of a grain, repeated in six hours, fluidextract of quassia, 10 to 20 drops, given every six hours for three doses, or tincture of podophyllin, 1 to 2 drops every six hours for three doses, may be administered.

Ankylostomum Duodenale—Hookworm.—The hookworm is a nematode which inhabits the duodenum and jejunum. The female is about a half inch, and the male one-third of an inch in length. The body is thread-like. The head is conical; the mouth is bell-shaped, is surrounded by a horny capsule, and contains four hook-like teeth. These are ventrally situated, and on the dorsal side are two smaller vertical teeth by which the worm fastens itself to the mucous membrane.

The eggs are deposited in muddy water or in warm, moist earth, and there the embryos are liberated. These develop into larvæ which soon pass into the dormant state and remain quiescent for an indefinite period until they are taken into the stomach by means of drinking water, food, or dirt—more commonly dirt that has collected on the hands, and particularly under the finger nails.

Direct infection through the skin is now thought to be the usual mode of transmission, the parasite entering the body through the skin of the feet and legs by contact with contaminated soil. It then is carried by the blood stream to the lungs, passes into the air spaces, bronchi, trachea, esophagus, or stomach, and finally reaches the small intestine. Here sexual characteristics develop in the parasites, reproduction ensues, and the ova are deposited in the bowel, but they do not multiply within the intestine. Males are affected with hookworm to the same extent as females, and the parasite is most prevalent in children between the ages of six and sixteen.

Symptoms.—The symptoms of hookworm disease, or uncinariasis, are to be attributed chiefly to severe toxemia and anemia. There is usually abdominal discomfort or pain, progressive emaciation, and high-grade anemia resembling the pernicious form. Children who are infested have an apathetic, languid expression, and look much older than they really are. They are stunted mentally and physically, and become shiftless, untruthful, dishonest, disobedient, and closely resemble cretins. The face is puffy, the skin muddy and sallow, the abdomen distended, and the extremities are either very thin or swollen and edematous.

These children have either a poor or ravenous appetite which is usually capricious, and they not infrequently eat earth and dirt, hence their name, "dirt-eaters." The subjective symptoms include headache, dizziness, palpitation of the heart, dyspnea, nausea, and spells of vomiting. The bowels are usually constipated, and the stools contain microscopic and occult blood. The changes in the viscera revealed at postmortem are cerebral anemia with effusion into the ventricles of the brain, flabbiness of the heart muscle, fatty degeneration of the liver, softening of the spleen, and the lesions typical of nephritis.

Diagnosis.—A definite diagnosis can be made only by finding the ova and the hookworms in the stools, although the symptoms in most cases of uncinariasis are very characteristic.

Prognosis.—The prognosis is good if the case is diagnosed early and is properly treated; but, occasionally, the disease runs a rapid course and the patient succumbs from exhaustion within a few weeks.

Treatment.—Thymol is universally recognized as a specific for hookworm disease. The maximum amount which should be given is $7\frac{1}{2}$ grains to children up to the age of five years, 15 grains to children from five to ten years, and 30 grains to those between ten and fifteen years. Two days before the thymol is to be given the diet should be restricted to liquids, and a dose of magnesium sulphate adminis-

tered, this being repeated on the following day. On the next day the thymol is given, one-half the dose at 6 A.M., and the remaining half at 8 A.M., followed two hours later by another dose of magnesium sulphate. The child should then be made to lie on the right side for two hours.

This treatment should be repeated at the end of a week, and ten days later the stools should be examined for ova or parasites. If none is found, no more thymol need be given; but the child should have plenty of fresh air, good nourishing food, and should take a tonic such as Fowler's solution, 1 to 3 drops three times a day, or the syrup of the iodide of iron, 10 to 30 drops three times a day, for the anemia.

Trichina.—The Trichina spiralis is a vivaporous worm occasionally found in children. The male, when mature, measures $\frac{1}{20}$ of an inch, and the female $\frac{1}{12}$ to $\frac{1}{6}$ of an inch in length. The embryo is about $\frac{1}{25}$ of an inch long, and lies coiled up in a spiral within an ovoid capsule in the sarcolemma sheath of the muscle fiber. The mature worm has a pointed, unarmed head. The neck is long and more slender than the body, which has a round, blunt end.

The life history of the trichina begins when the larvæ become encysted in the muscles. When this flesh is eaten by another animal or by man, the larvæ are liberated by the digestive processes and pass into the intestines. In from two to four days they become sexually mature, and five to seven days thereafter they produce hundreds of living embryos. The intestinal trichinæ reach their full growth and then die in from four to five weeks.

During her life period in the intestine the female trichina may bring forth several broods of embryos. The living embryos migrate from the intestine at once, and invade the muscles through various channels, principally along the connective-tissue routes, so that the symptoms of muscular irritation develop in from seven to ten days after eating trichinous meat.

The embryos attain maturity about two weeks after they invade the muscle tissue, where their presence sets up an irritation which in from four to six weeks causes the formation of a fibrous capsule about the trichina. Usually but one trichina is found within a capsule, and this encapsulated trichina may live for years in the muscle, the capsule finally becoming calcified so that it may be easily discerned by the naked eye.

Symptoms.—After meat infested with trichinæ has been eaten there is usually a period of gastro-intestinal disturbance, followed by the symptoms of trichiniasis, which are not unlike those of influenza, rheumatic fever, malaria, or typhoid fever. There are severe pain and soreness in the muscles, and edema of the face and eyelids which suggests nephritis. These symptoms usually persist for from ten days to two weeks, during which time the disease is rarely recognized.

Diagnosis.—A positive diagnosis can be made only by removing a section of muscle tissue, and identifying the trichinæ within it.

Treatment.—As soon as the diagnosis is made, thorough purging with calomel, 1 to 3 grains, should be instituted, and six hours later $\frac{1}{2}$ to 2 drams of magnesium sulphate should be administered. Further treatment is merely for the relief of symptoms, since it is doubtful whether anything can be done to arrest the progress of the disease after the muscles have once been invaded.

Morphin sulphate, $\frac{1}{50}$ to $\frac{1}{10}$ of a grain, may be given hypodermically for the relief of pain if simpler measures, such as warm baths and hot applications to the muscles, fail to give relief. Since death sometimes occurs from exhaustion or the severe irritation, the child's strength should be kept up, and stimulation resorted to whenever necessary.

Among the drugs said to be effective in destroying the trichinæ are glycerin, given in dram doses hourly, benzine in 5- to 20-grain doses, and picric acid in 1- to 2-grain doses.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Acute peritonitis is extremely rare during childhood, and non-tuberculous chronic peritonitis is practically never encountered. Acute inflammation of the peritoneum is always a secondary process, although in some cases the primary factor may be very obscure. It may be circumscribed or diffuse, and is also classified as serous or suppurative, according to the nature of the exudate.

Etiology.—Peritonitis may occur in the fetus from syphilis or septic infection of the mother; but it is seen more frequently in the newborn than at any other period of childhood, and is the result of infection of the umbilicus. Appendicitis is the most common cause of acute peritonitis in older children, but it is also occasionally seen in combination with inflammation of the abdominal viscera, lungs, pleura, or pericardium. Direct violence to the abdomen is responsible for a few cases; in other instances it may be traced to an attack of typhoid fever, pneumonia, one of the acute infectious diseases, or to gonorrheal vulvovaginitis.

Perforation of an abdominal viscus, such as a rupture of the gall-bladder, abscess of the liver, ulcer of the duodenum or stomach, or a typhoid ulcer, always produces peritonitis. Sometimes infection may be transmitted from an empyema. Intussusception and strangulated hernia, if not reduced, are soon followed by peritonitis, and in rare cases the peritoneum has become infected through a ruptured cyst of the liver, spleen, or kidney. Occasionally the infection is carried through the blood stream, and in this manner peritonitis may complicate suppurative otitis media or an attack of meningitis. Infection by way of the lymph channels has also been proven.

Bacteriological studies have shown that the colon bacillus, pneumococcus, Staphylococcus aureus, Streptococcus pyocyaneus, and Streptococcus pyogenes are the organisms most frequently present. Gonococci may occasionally be found when peritonitis complicates gonorrheal vulvovaginitis; but this is rare. In appendicitis accompanied by peritonitis, the colon bacillus has been isolated in the great majority of cases. Pneumococcal peritonitis, which has a special predilection for children, may complicate pneumonia or pneumococcemia; and peritonitis in which the staphylococcus or streptococcus predominates is usually due to an acute infectious disease or ruptured abdominal viscus.

Pathology.—The postmortem findings depend altogether upon the character of the inflammatory process. In every case the visceral and parietal peritoneum is found to be intensely congested and hyperemic. If there be but little serous exudate, and the intestines are covered by an exudate of lymph, or are bound together by a fibrous exudate, fibrinous peritonitis is recognized. In serous peritonitis there is but little fibrin in the exudate, but a large amount of serum is found in the peritoneal cavity.

Suppurative peritonitis is characterized by the formation of pus within the peritoneum. This is usually confined to that portion of the peritoneal cavity surrounding the site of infection, where an abscess forms; but in severe cases it may be present throughout the whole abdominal cavity. When a collection of pus is found walled off within the peritoneal cavity by the formation of inflammatory adhesions, the condition is known as circumscribed peritonitis, in contradistinction to the diffuse form in which the pus is free.

Symptoms.—In acute peritonitis the symptoms are usually very frank and acute; but occasionally they are masked by the primary and associated condition. This causes great difficulty in diagnosis, especially in infants during the first few days of the disease when the condition is not suspected. The prominent symptoms are pain, tympanites, absolute constipation, persistent vomiting, and high fever. Pain appears first at the point of infection, *e. g.*, the right iliac fossa or umbilicus—but soon becomes general and extends over the whole abdomen.

Vomiting is persistent, occurring usually at intervals, and increases in severity as the disease progresses. The vomitus is composed for the most part of a greenish, watery liquid which contains mucus. Hiccough is often distressing, and may be regarded as an unfavorable sign. In cases where there has been an antecedent diarrhea, this condition may persist; and, although constipation may afterward take its place as a result of paralysis of the intestinal muscles which inhibits peristalsis, diarrhea is more common in the peritonitis of childhood than in adults.

There is usually an initial chill, followed by a sharp rise in temperature to 103° F. or even higher. The pulse is weak and rapid; the respirations are short and quick; and, while the body may be

hot and dry, the extremities are cold. The tongue is dry and brown; the eyes are sunken; the child lies upon its back with its knees drawn up to relieve the tension upon the abdominal muscles. The urine is high-colored, concentrated, and scanty, or it may even be suppressed; upon examination it is found to contain an excessive amount of indican. There is always leukocytosis in peritonitis; but unless a blood count has been made before the disease is suspected this is of no diagnostic value, since it may be due to the primary condition—appendicitis.

Physical examination in peritonitis reveals a distended, tender, tympanitic abdomen with marked rigidity of all the abdominal muscles. In circumscribed peritonitis the pain and tenderness are confined to the site of the abscess, and the symptoms are not as severe as in the diffuse form. Pneumococcal peritonitis, which is to a certain extent localized, is characterized by an encapsulated collection of pus behind the anterior abdominal wall just below the umbilicus, which forms a tumor at this point.

In some cases of this variety of peritonitis, the skin about the navel becomes perforated and the abscess drains externally, whereupon recovery ensues. As a rule, the symptoms of peritonitis due to gonorrheal vulvovaginitis are very mild, and the infection is confined to the pelvic peritoneum; but this form of inflammation of the peritoneum is not to be regarded too lightly, since the infection may spread throughout the peritoneal cavity and rapidly prove fatal.

Diagnosis.—In children the diagnosis of peritonitis is difficult, and in infants the disease is often unrecognized. The history of an associated affection which might cause peritonitis and the physical signs are the most reliable points upon which to base the diagnosis. Distention of the abdomen and extreme rigidity of the abdominal muscles, with persistent vomiting and severe constitutional disturbances, make up a group of signs and symptoms significant of acute peritonitis; but appendicitis should always be ruled out, if possible, by rectal examination. If this can not be done, and no other cause for the symptoms can be found, exploratory laparotomy is justifiable.

Acute peritonitis may be differentiated from typhoid fever by its shorter and more acute course, by the relative severity of the symptoms, and by the Widal reaction. Intussusception may resemble peritonitis, but it can be differentiated by the absence of fever and the bloody, mucous stools which result from invagination of the gut.

Prognosis.—Acute peritonitis is a fatal disease during infancy, and death usually occurs within the first few days. Pneumococcal peritonitis and diffuse peritonitis caused by the rupture of an abdominal viscus also present an unfavorable outlook. Gonorrheal peritonitis, however, ends in recovery in a great many cases. In circumscribed and traumatic peritonitis recovery depends upon the severity of the infection and the time when operation is performed.

Treatment.—Surgical interference offers a better chance for recovery in most cases of acute peritonitis than does any other remedial measure.

23

Medical treatment is warranted only in those cases where peritonitis is due to mild trauma or an acute infection, or when it is associated with nephritis or gonorrheal vulvovaginitis; even in these cases, if the disease fails to respond to medication within two days, laparotomy is justifiable. Operation is always indicated when there has been a rupture of an abdominal viscus, and should be performed immediately; for in diffuse peritonitis surgery avails but little if the condition has persisted for several days. Medical treatment of peritonitis consists, for the most part, in the relief of pain and in keeping the intestines at rest. The child should be kept in Fowler's position and cold applied to the abdomen for the first few days while the acute inflammatory symptoms persist.

If one feels sure that there is no danger of perforation of the intestines, a thorough purge of $\frac{1}{2}$ to 2 drams of magnesium sulphate should be given. Nothing should be taken by mouth until vomiting ceases; but the child may be allowed to suck cracked ice if the mouth is parched. After the first three or four days warm applications, turpentine stupes, and poultices may be put on the abdomen, and the child be given whiskey or brandy in 5- to 20-drop doses at frequent intervals during the day. So long as the stomach rejects food, nourishment may be furnished by means of nutrient enemata; but, as the patient's condition improves, the breast-fed infant may resume its nursing and the bottle-fed baby be given small quantities of a weak milk mixture, gruel, or broth.

When the temperature runs very high the child may be sponged with tepid water or rubbed with alcohol. If tympanites becomes severe, a long rectal tube may be inserted into the bowel and allowed to remain there for several hours. If stimulation is indicated, champagne or whiskey in 5- to 20-drop doses should be given at frequent intervals, and strychnine sulphate, grain $\frac{1}{500}$ to grain $\frac{1}{250}$, or camphorated oil, 1 to 5 drops, may be administered hypodermically. The intestines may be kept immobile by hypodermic injections of morphine sulphate, grain $\frac{1}{60}$ to $\frac{1}{20}$, combined with atropine sulphate, grain $\frac{1}{1000}$ to $\frac{1}{500}$; the morphine thus given will also alleviate the pain.

In addition to these immediate measures, much can be done to improve the general condition of the child by giving it good hygienic care, plenty of fresh air, and by sending it away to the seashore or country during convalescence.

The treatment of circumscribed peritonitis is essentially surgical, and consists of incision and drainage, followed by the tonic measures outlined above.

CHRONIC PERITONITIS.

Chronic peritonitis which is not caused by the tubercle bacillus is of such extreme rarity during childhood that its occurrence is doubted by various competent authorities. This, however, applies only to diffuse inflammation of the peritoneum, for localized chronic peritonitis may accompany disease of any of the abdominal viscera.

Etiology.—The exact cause of this form of chronic peritonitis is never clear, but it may follow measles, rheumatism, exposure, or injury. The visceral layer of peritoneum becomes inflamed, while the organ which it covers becomes diseased, and circumscribed peritonitis of chronic type may follow appendicitis. Chronic non-tuberculous peritonitis usually occurs after the fifth year.

Pathology.—Chronic peritonitis may be either localized or diffuse, serous or fibrinous. The serous form, or peritonitis with ascites, is characterized by the accumulation of a large quantity of straw-colored fluid within the peritoneal cavity. There is only a relatively small amount of fibrinous exudate, and few adhesions are found. This picture is far different from that of fibrinous, dry, or proliferative chronic peritonitis in which the abdominal cavity contains little fluid, but the intestines are covered with fibrin, and bound down and matted together by fibrous adhesions. In chronic localized peritonitis there is an increase in fibrous tissue over the serous covering of an abdominal viscus or about an area of preceding inflammation within the peritoneal cavity—for example, appendicitis or salpingitis.

Symptoms.—The symptoms of chronic peritonitis come on most insidiously. Enlargement of the abdomen is usually the first perceptible sign of disease within the abdominal cavity, although there may have been gradual loss of weight and strength, and indefinite and vague symptoms, such as slight pain and tenderness of the abdomen. Dyspepsia usually precedes the collection of ascitic fluid. The appetite is generally poor; the bowels are alternately constipated and loose. There is usually slight fever, highest in the evening, and the child has secondary anemia from malassimilation and indigestion. In some cases there is marked disturbance of the general nervous system, but, as a rule, convulsions do not occur.

On inspection, in ascitic cases, the abdomen usually appears to be enlarged and distended, and a fluctuation wave can be detected. The area of flatness on percussion will be found to change with change in posture, and its exact extent varies according to the amount of fluid present.

In dry, or plastic, peritonitis, occurring in thin or emaciated children, the greatly thickened and rolled-up omentum may sometimes be palpated. Chronic non-tubercular peritonitis runs a very irregular course with alternating periods of relapse and improvement; but in many cases the fluid is slowly absorbed, and permanent recovery usually follows.

Diagnosis.—The most important point in the diagnosis of chronic non-tubercular peritonitis in a child is the presence of ascitic fluid in the abdomen with absence of any disease of the heart, liver, or kidneys, and no demonstrable tuberculous lesion. Tuberculosis must always be excluded in these cases, although this exclusion is invariably an extremely difficult task.

Failure to find a tuberculous lesion in other parts of the body is not conclusive evidence against tuberculosis, neither is a negative von Pirquet reaction. Moreover, examination of the ascitic fluid for the

tubercle bacillus is of little value in determining its presence, since it is often impossible to find it in known tubercular peritonitis. But when, in a given case, all of these determining factors are negative and when guinea-pig inoculation with the ascitic fluid also fails to give a tuberculous reaction, one is justified in pronouncing the case non-tubercular.

Prognosis.—The prognosis of chronic non-tubercular peritonitis is more favorable in the child than in the adult, and a number of cases end in complete recovery. There is always, however, the danger of death from an intercurrent infection or from extreme debility and exhaustion. The prognosis should be guarded in every case, because of the difficulty of definitely excluding tuberculosis.

Treatment.—Rest in bed and a carefully regulated and nourishing diet should be insisted upon. The child should also be given tonics, such as the syrup of the iodide of iron, 10 to 30 drops, tincture of nux vomica, 2 to 5 drops, or cod-liver oil in teaspoonful doses after meals; but the physician should watch to see that this medication does not impair the appetite. The bowels must be regulated by the administration of fluidextract of cascara sagrada (aromatic), 15 to 40 drops, or the compound syrup of rhubarb, $\frac{1}{2}$ to 1 dram. If the absorption of the ascitic fluid is very slow, saline cathartics, such as either magnesium sulphate or Rochelle salts, $\frac{1}{2}$ to 1 dram, should be employed routinely.

When these measures fail, and the accumulation of fluid becomes so great as to cause discomfort, the abdomen should be tapped with a trocar and the fluid slowly withdrawn. If it rapidly reaccumulates after each tapping, great improvement, and sometimes recovery, can be brought about merely by opening the abdomen and washing out the peritoneal cavity with warm normal saline solution.

ASCITES.

Ascites is an accumulation of serum in the peritoneal cavity. It is usually due either to obstruction of the portal circulation or inflammation of the peritoneum, although a dropsical condition of the peritoneum with no apparent cause also occurs, and is more common in children than in adults.

Ascitic fluid is usually clear, straw-colored, alkaline in reaction, with a specific gravity of 1010 to 1015; under the microscope it is found to contain leukocytes, red corpuscles, endothelium, fat cells, and cholesterin crystals. When ascites is due to malignancy or to tuberculous peritonitis the serum may be blood-stained, and it is occasionally also bile-stained. In chylous ascites the accumulated fluid has a milky appearance, and contains fat droplets, sugar, and a few lymphocytes.

Etiology.—Ascites is frequently associated with general edema in cases of nephritis, cardiac disease, chronic pleurisy, pernicious anemia, and leukemia. It is also a prominent symptom in most cases of peri-

tonitis in children, especially those due to tuberculous infection. A common cause is portal obstruction, which may be the result either of diseases of the liver, such as cirrhosis, syphilis, lardaceous disease, or hepatic tumors, or of interstitial pneumonia, thrombosis of the portal vein, pressure of abdominal tumors, or obstruction of the vena cava by enlarged retroperitoneal lymph nodes.

Simple dropsy, or that form without apparent cause, is in all probability in most instances due to subacute peritonitis. Chylous ascites is due to obstruction of, or injury or ulceration along the thoracic duct; it is sometimes observed in association with tuberculosis of the mesenteric glands.

Symptoms.—The symptoms of ascites are very mild and usually imperceptible until the accumulation of fluid is large enough to cause a sensation of weight and pressure in the lower abdomen. As the amount of fluid increases, the symptoms due to pressure become more decided, and constipation, dragging pains in the loins, dyspnea, and frequent micturition result.

At this stage the presence of fluid in the abdomen is quite obvious, and in some cases the abdomen becomes enormously large, the enlargement being symmetrical. The superficial veins in the skin about the umbilicus are distended and prominent, and a fluctuation wave may easily be detected. On percussion there is flatness in the flanks, also tympany over the centre of the abdomen while the child lies on its back, with alteration in the areas of flatness and tympany on change of posture. The liver, spleen, and even the heart are pressed upon by the accumulated fluid, and finally displaced.

Diagnosis.—The diagnosis of ascites should be based upon a history of one of the causative factors that have been mentioned, together with the physical signs and symptoms of an unsacculated accumulation of fluid within the peritoneal cavity. On seeking in a given case to find the cause of ascites, examination of the heart, blood, and urine will quickly show whether cardiac disease, anemia, or nephritis is the underlying cause, while removal of the ascitic fluid followed by deep abdominal palpation will often reveal the presence of a tumor in those cases due to portal obstruction by a neoplasm.

Abdominal cysts may grow so large that they closely simulate ascites, hence such conditions as hypernephroma, ovarian cysts, and hydatid disease of the liver must always be excluded. The protuberant abdomen seen so often in children with chronic intestinal indigestion, rachitis, or marasmus, sometimes suggests ascites; but careful physical examination will reveal no shifting flatness or tympany on change of posture, no fluctuation wave, and the apparent distention of the abdomen will disappear when the child lies down. Chylous ascites can be diagnosed only when some of the ascitic fluid has been withdrawn from the abdominal cavity and examined.

Prognosis.—Ascites is but a symptom, not a disease; therefore the prognosis depends upon the underlying factor. Except in idiopathic or simple dropsy, the outlook is generally unfavorable.

Treatment.—Obviously the most essential point in curing ascites is to remove the cause, all other measures being designed merely to alleviate the symptoms caused by the presence of the fluid within the abdominal cavity. The child should be placed on a light nutritious diet containing a high nitrogen content, and the liquid intake restricted as much as possible.

Hydragogue cathartics, such as magnesium sulphate, dose 20 to 60 grains, or powdered jalap, dose 1 to 3 grains, and diuretics such as potassium bitartrate, 5 to 20 grains, or digitalis leaves powdered, $\frac{1}{10}$ to 1 grain, or powdered squills, $\frac{1}{20}$ to $\frac{1}{2}$ of a grain, should be given in order to drain the tissues of fluid and promote the absorption of the ascitic serum in the peritoneum. Tonics, such as the syrup of the iodide of iron, dose 10 to 30 minims, or the saccharated carbonate of iron in $\frac{1}{5}$- to 1-grain doses, are speciaelly beneficial in ascites due to anemia, or in so-called simple dropsy.

If, in spite of the treatment above outlined, absorption of the fluid does not take place and the pressure symptoms become worse, the abdomen should be tapped and the serum withdrawn slowly through a trocar. Several hours should be consumed in completely draining off the fluid, and the abdomen should be tightly bandaged during and after its removal in order to prevent such a sudden and great fall in intra-abdominal pressure as would produce harmful consequences.

INGUINAL HERNIA.

Inguinal hernia is not an uncommon affection in childhood, occurring most frequently in little boys. Three forms are recognizable; the congenital, funicular, and infantile. The congenital type is that in which a loop of intestine forces its way through the still open funicular process, and thus effects entrance to the scrotum, where it usually surrounds the testicle.

In the funicular type the tunica vaginalis is shut off from the funicular process above the testicle, so that the hernia occupies the funicular canal, but does not envelop the testicle. The infantile, or encysted, form is very rare, and can be diagnosed only at operation. The intestine, encased in a pouch of peritoneum, forces its way into the funicular process and descends, although this canal is closed above and open below.

Etiology.—Hernia may exist at birth or it may develop as the child grows older and begins to lead an active life; but in children it is always regarded as a congenital condition. It usually occurs on the right side, but both sides may be involved, and thus give rise to a double inguinal hernia. Boys are more frequently affected with hernia than girls because of the presence in boys of the inguinal canal, which is a weak spot made by the testicle in its descent to the scrotum.

The infant is predisposed to hernia because of the relatively short and direct course of the inguinal canal, which allows easy passage of the gut through the internal ring if this inner opening is not entirely

closed, or if the peritoneum at this point is weak or lax. Femoral hernia, on the other hand, is very rare in children because of the proximity of the pubic spine, the anterior spine of the ilium, and Poupart's ligament, which in the child lie so close together that there is really insufficient space for a hernia to form. It is more common in girls than in boys.

Pathology.—The hernial sac usually contains loops of the small intestine, and only occasionally is the omentum found within it.

Symptoms.—In most cases the only symptom is the presence of a tumor in the inguinoscrotal region. This tumor becomes smaller when the child lies down, and again enlarges when the upright position is assumed, or whenever intra-abdominal pressure is increased by crying or coughing. Upon examination the external ring is found to be enlarged, an impulse is transmitted to the tumor during coughing, and the mass on being manipulated and pushed back into the abdomen makes a gurgling sound. The entire scrotum may be filled by the tumor, so that it is difficult to palpate the testicles, but they can usually be found above and behind the coils of intestine. It is important that both testicles be located in order to eliminate the possibility of an undescended testicle. When an inguinal hernia occurs in girls, the tumor occupies one of the labia majora.

Diagnosis.—The diagnosis of inguinal hernia is more perplexing in children than in adults because of the possibility of mistaking it for other conditions which may produce a tumor in this location during childhood, also because of various affections which may coexist with hernia, such as a cyst of the spermatic cord, a hydrocele, or an undescended testicle. Congenital hydrocele, which is comparatively common in children, presents the following differentiating features:

HERNIA.	HYDROCELE.
Reducible, accompanied by gurgling on reduction.	Irreducible, or slowly so, with no gurgling sounds.
Dulness or tympany on percussion.	Flat on percussion.
Increase in size on crying and coughing.	No change in size.
Impulse on coughing.	No impulse.
Opaque.	Translucent.

Encysted hydrocele of the cord presents the same differentiating features as congenital hydrocele; but, owing to its movability, it may be pushed into the internal ring and an apparent reduction thus be accomplished, which is very confusing. The scrotum should always be carefully examined in order to determine the position of the testicles; for if they can be located in this manner undescended testicle can be ruled out. If the testicle is not found on that side of the scrotum in which there is a tumor, the hernia is probably complicated by an undescended testicle, and on examination of the mass the testicle will be felt as a firm hard tumor which is tender and painful on pressure. There is no pain or tenderness associated with uncomplicated hernia, and the tumor is elastic and soft.

Femoral hernia can be easily distinguished from the inguinal variety

by the location of the tumor just beneath the saphenous opening, with its origin from the outer side of the pubic spine; while the origin of inguinal hernia is from the inner side of the spine of the pubic bone. Enlarged inguinal glands, while sometimes giving rise to a tumor resembling that of hernia, are easily differentiated, for on palpation they are found to be hard, firm, and non-reducible; they transmit no impulse; and are accompanied by enlargement of the whole chain of glands in this region.

Complications.—In addition to other congenital conditions, such as undescended testicle and hydrocele, hernia may be complicated by obstruction of the bowels caused by strangulation of the loop of intestine caught within the hernial sac. This occurrence, however, is quite rare in children because of the elasticity of the tissues of the inguinal canal; but, strange as it may seem, it happens more frequently in infants than in older children. In the child the symptoms of strangulation are very acute; there is sudden pain, with vomiting and absolute constipation after the fecal contents of the bowel below the obstruction have been passed. The hernia is irreducible and very painful; the child lies with the leg on the affected side drawn up to the abdomen.

Prognosis.—The prognosis in uncomplicated hernia in the child is very favorable, and in the majority of cases recovery ensues if properly fitting appliances are obtained and worn to keep the hernia from coming down into the inguinal canal.

Treatment.—Operation for the cure of hernia is necessary only in older children; for, during the first few years of life, the application of a truss, which must be worn continuously day and night, will generally effect a cure. If the wearing of a truss is successful it will bring about obliteration of the hernial sac, and close the ring within a year, so that, at the end of this period, the pressure on the ring can be removed and the tumor will not appear. But the truss should always be worn for at least six months after this time in order to prevent recurrence.

When a child is wearing a truss, the physician in charge of the case should instruct those in attendance to see that the pressure upon the hernial opening is never relaxed; for if the hernia should reappear during bathing, and this should happen frequently, it will indefinitely postpone cure. Care should also be taken to see that the skin beneath the truss pad does not become irritated or excoriated, and to this end special effort must be made to keep the skin in this region dry. As the child grows, the truss should frequently be made larger to accommodate the increasing size of the child; but it should not be worn indefinitely.

After the second year the prospect of cure by non-operative means diminishes greatly, so that operation is advisable if the condition persists without improvement. Owing to the difficulty of properly fitting a truss for femoral hernia, operation is advisable in children as soon as the patient's general condition will allow it. If inguinal

hernia becomes incarcerated or strangulated, operation is imperative, and should be performed immediately.

The results of operation in uncomplicated herniæ are so favorable that it is unquestionably the surest and safest mode of cure, provided the child is old enough and strong enough to survive the operation. In children suffering from hernia the bowels should be kept regular before and after operation, and after a cure has been effected, either by operation or non-operative measures, active exercise should be prohibited for months, in order to prevent a possible recurrence.

Strangulated or incarcerated hernia calls for immediate reduction by taxis and manipulation, or by operation. In attempting to reduce a strangulated hernia, which is an easier procedure in the child than in later life, the child should first be placed in a warm bath to relax the muscles, and then a few drops of ether be administered. If the contents of the hernial sac can not be pushed back, immediate operation is demanded, the nature and extent of which will depend upon the condition of the strangulated intestine. If gangrene is present, resection is advisable, and in older children the shock of resection is frequently borne quite well, although it is usually fatal in infants.

DISEASES OF THE RECTUM AND ANUS.

PROCTITIS.

Proctitis, or inflammation of the rectum, is not uncommon during childhood, and may be either primary or secondary. Of the simple varieties the catarrhal is the most common, and the membranous and ulcerative types are the most infrequent. The specific form is rare; it is spread from the genitalia in cases of gonorrheal vaginitis and diphtheritic vulvovaginitis.

Etiology.—Among the primary causes of proctitis must be included trauma (although this is rare), threadworms, the use of drastic purgatives, and rough manipulation of thermometers and syringe nozzles when inserted within the rectum. The prolonged use of soap or glycerin suppositories may also produce irritation and inflammation of the rectum. Proctitis occurs secondarily in cases of gastro-enteritis, dysentery, prolapse of the rectum, and rectal polypi.

Except when it accompanies general diphtheritic infection, the membranous form is more often due to the streptococcus than to the Klebs-Loeffler bacillus; while gonorrheal proctitis is, in the majority of cases, caused by a spreading of the profuse discharge from the vagina backward into the anus, but may also be produced by careless handling of thermometers and syringe nozzles.

Pathology.—In catarrhal proctitis the mucous membrane is swollen and hyperemic, and there is an exudation of blood-stained mucus. The ulcerative form is but a more advanced stage of the catarrhal

type, and is marked by the formation of large superficial ulcers scattered throughout the rectum. Follicular ulceration of the rectum is also occasionally seen in association with follicular ulcerative colitis; in this type the ulcerations, while small, are quite deep, extending far down to the muscular coat of the bowel.

In membranous proctitis many superficial ulcerations form; they are covered with a grayish-white membrane which is very tenacious and leaves a raw, bleeding surface when removed. Gonorrheal infection of the rectum gives rise to a severe catarrhal inflammation, distinguished by an excessive purulent secretion.

Symptoms.—Among these, rectal tenesmus is the most prominent, and is extremely severe during defecation, the pain lasting a considerable time after each bowel movement. The child is usually constipated, but there are frequent discharges from the rectum of bloody mucus which may or may not be mixed with feces. The stools contain shreds of mucus and, in the diphtheritic type, sometimes casts of the rectum. In ulcerative proctitis much blood is usually passed by the bowel. In gonorrheal proctitis the discharge from the rectum is distinctly purulent and, if examined microscopically, may be found to contain the gonococcus. Prolapse of the rectum quite often accompanies the condition as a result of the straining and tenesmus at stool.

Treatment.—The child suffering with proctitis should be put to bed, and kept upon a nourishing liquid diet which will not cause constipation. If necessary, the stools can be kept loose by the administration of mild laxatives, such as the syrup of rhubarb, in 20- to 60-drop doses, or milk of magnesia, 1 to 2 teaspoonfuls at a dose. Pain may be relieved by the use of suppositories of $\frac{1}{20}$ to $\frac{1}{4}$ grain of opium or $\frac{1}{20}$ to $\frac{1}{8}$ grain of cocaine, or by painting the mucous membrane of the rectum with cocaine solution.

Sweet oil, olive oil, saline solutions, lime-water, boric acid, and potassium permanganate may all be used for injection in the catarrhal form of the disease; but care should be taken not to further irritate the already inflamed rectum.

In the ulcerative form a weak astringent, such as $\frac{1}{2}$ of 1 per cent. of silver nitrate solution, is very effectual, while in gonorrheal proctitis injections of antiseptic solutions, such as tincture of iodine, a half-dram to a pint of water, or 1 to 2000 silver nitrate solution, or a 1 per cent. solution of protargol or witchhazel, are all indicated. In diphtheritic proctitis the child should be given large doses of antitoxin in addition to the local treatment.

PROLAPSE OF THE RECTUM (PROCIDENTIA RECTI).

Prolapse of the rectum may be either partial or complete, according to whether the mucous membrane alone or the entire rectal wall protrudes from the anus. Partial prolapse, or prolapse of the anus, as it is sometimes called, is due to relaxation of the mucous membrane

which, to a certain extent, becomes detached from the muscular coat of the rectum underlying it, and is everted by the straining at stool. It may, perhaps, return to its place after each movement of the bowels, and the affection usually shows a tendency to spontaneous cure.

A predisposition to total prolapse may be accounted for by the almost vertical position of the rectum during infancy and early childhood, and by its weak attachments which fail to hold it in place. Constipation and diarrhea, as well as dysentery which causes severe tenesmus and straining, are the most frequent causes of rectal prolapse, owing to the increased abdominal pressure they produce.

Other conditions, such as fecal concretions, rectal polypi, vesical calculi, and irritation of the rectum, bladder, or genitalia from other causes, may be contributory factors. Pertussis may produce it because of the increased abdominal pressure during paroxysms of

Fig. 31.—Procidentia recti; cretin eight years old.

coughing. Rectal prolapse occurs most commonly in children under three years of age, especially if they are undernourished, weak, or anemic, and is not infrequent in rachitic children (Fig. 31).

Symptoms.—In cases of prolapse of the anus inspection reveals an everted mass of mucous membrane which may be covered with mucus and blood, and surrounds the anal orifice. It has a central aperture, and is easily reducible. Prolapse of the rectum forms a much longer and thicker mass, with an orifice in the centre of the distal end. The mucous membrane is at first bluish-red in color, and shows marked congestion.

Later, if the prolapse is not reduced, ulceration may develop, pain become severe, and the tumor swells and bleeds constantly. Reduction is now difficult; but if the case is seen just after prolapse has occurred it can usually be corrected merely by manipulation.

Diagnosis.—Actual prolapse of the rectum is easily diagnosed by inspection; but sometimes rectal polypi and intussusception may simulate a prolapse, in which case the examining finger can be inserted between the tumor and the rectal wall, which obviously would be impossible in rectal prolapse.

Treatment.—In mild cases, the tumor can usually be replaced by simple manipulation, and this should be done after every bowel movement which brings on a prolapse. A suppository of tannic acid, 2 grains, or some other astringent, should be inserted within the rectum as soon as the prolapse has been reduced; a cold-water douche after each bowel evacuation will strengthen the muscular tone of the rectum.

Severe cases of prolapse require the application of cold to the swollen and inflamed tumor, and in some instances it may be found necessary to administer a few whiffs of chloroform to relax the contracted muscles before the rectum can be pushed back into position. The child should be laid upon the lap, face down, with the legs higher than the buttocks, while reduction is being accomplished, and after the rectum has been replaced the buttocks should be held together by strips of adhesive plaster in order to prevent another prolapse. In severe cases rest in bed is advisable, and should be insisted upon until marked improvement takes place. Even in mild cases the child should always lie down before his bowels move.

In addition to these immediate measures, much can be done to benefit these children by ascertaining and removing the cause of the prolapse, whether it be constipation, diarrhea, intestinal parasites, rectal polypi, or any other source of irritation which increases intra-abdominal pressure or straining at stool. The bowels should be regulated and kept slightly loose. An effort should also be made to build up the child's general health by a nourishing diet, improved surroundings, and other hygienic measures.

Under this treatment the ordinary case of prolapsed rectum will recover unless the sphincter ani has altogether lost its tonicity, when operation will be necessary. But surgical procedures which, in the adult, are attended by excellent results may be much too severe for the child; therefore, as a general rule, we should choose the simplest operation which will give results. Thus, fixation of the rectum at a higher level is too formidable, hence is contraindicated, and scarification of the mucous membrane with the actual cautery or amputation of the prolapsed portion of rectum should be chosen instead.

RECTAL POLYPI.

These growths in the rectum are rarely found in infants, but the condition is not uncommon during later childhood. They are usually pedunculated, but may have a sessile base, and consist merely of hypertrophied mucous membrane. As a rule, there is but one polypus present, and that is commonly situated on the posterior wall of the

rectum just above the internal sphincter. These tumors may be either adenomata, myxomata, or fibromata. They are hard and firm, and are composed of villi and rugæ of the mucous membrane. In exceptional cases the rectum may be nearly filled with small tumors.

Symptoms.—The most common symptoms of rectal polypi are tenesmus, prolapse, and hemorrhage from the bowel. Pain is very severe whenever the bowels move, and the stool is covered with bright red blood.

Diagnosis.—The diagnosis should be made only after digital examination and exploration of the rectum with a speculum and reflected light.

Treatment.—When the growth is pedunculated it may be easily ligated at the base and snipped off. Sessile tumors are quite difficult to remove, and a rectal speculum should be used in order to see that the ligature embraces all of the stump. After removal of polypi, an opium suppository or iodoform ointment should be inserted in the rectum to relieve the pain. It is dangerous to allow these growths to remain in the rectum, since they are apt to become malignant.

HEMORRHOIDS.

Children rarely suffer from hemorrhoids, but either the external or internal variety may occasionally be observed. They are practically never seen in infancy, but are less rare as the child advances beyond the third year.

Etiology.—The most common cause of these growths is chronic constipation.

Symptoms.—The internal form of hemorrhoids is the only one likely to be met with in children, so that usually no tumor is found on inspection of the anus. The only important symptoms are pain and the passage of stools containing bright red, unchanged blood.

Treatment.—The bowels should be kept loose by the administration of cascara sagrada, 10 to 30 drops, each evening, or milk of magnesia, 1 to 3 drams. The anus should be kept clean, and the lower rectum flushed with salt solution twice a day, after which an astringent ointment containing 2 per cent. of tannic acid may be applied, or the following solution may be injected into the rectum each morning:

> ℞—Acidi tannici . . . . gr. ij
> Ichthyoli . . . . ℥ss
> Alcoholis . . . . ℥j
> Aqua . . . . q. s. ad. fl ℥iss
> Sig.—Use as injection each morning.

FISSURE IN ANO.

Fissure of the anus is usually the result of severe constipation, and is caused by the stretching of the anal mucosa in passing large hard fecal masses. In exceptional cases it is produced by rough manipula-

tion of the nozzle of a syringe or rectal tube, and occasionally it is associated with eczema of the anus and the presence of intestinal parasites.

Symptoms.—Pain is so extremely severe and agonizing when the bowels are moved that the child voluntarily retains the feces within the bowel as long as possible, thus increasing the constipation and aggravating the condition. Bright red blood may appear upon the diaper or in the stool.

Diagnosis.—The diagnosis is made by physical examination, which reveals in acute cases a small narrow break at the mucocutaneous junction, and an ulcerated area if the fissure be chronic.

Treatment.—The bowels should be kept moving freely by the use of laxatives to prevent the formation of hard masses of feces. The mucous membrane should be thoroughly cleansed every day with a mild antiseptic solution, such as a saturated solution of boric acid, and the fissure then touched with a 10 per cent. solution of nitrate of silver. Relief is often afforded by keeping the anus well greased with vaseline or oxide of zinc ointment containing 1 per cent. of phenol. If there is any tendency to chronicity the anus should be forcibly dilated with the fingers.

SPASM OF THE ANUS.

Spasm of the anus is most commonly associated with fissure in ano, but may happen also in neurotic children when no organic lesion exists.

Symptoms.—Tenesmus and pain during defecation are the principal symptoms. Constipation is caused by the natural aversion of the patient to evacuate the bowels because of the pain which the act produces. Examination of the anus in these cases will reveal a markedly contracted sphincter ani.

Treatment.—The bowels should be regulated by the administration of mild laxatives, such as aromatic syrup of rhubarb, $\frac{1}{2}$ to 1 fluidram; cascara sagrada, 1 to 2 grains; or milk of magnesia, 1 to 2 drams. Injections of olive or sweet oil also render the lower end of the rectum less sensitive; but care should be taken not to cause irritation.

In some cases it may be found necessary forcibly to dilate the sphincter. The following prescription will be found valuable in inhibiting rectal spasm, and is especially useful if there is a slight abrasion of the mucous membrane or a fissure in the anus:

```
℞—Unguenti belladonnæ,
      Unguenti hyoscyami  .  .  .  .  .  .  .  .  .  . āā   ℨij
      Petrolati .  .  .  .  .  .  .  .  .  .  .  . q. s. ad.   ℥j
   Sig.—Apply freely.
```

ANAL FISTULÆ.

Anal fistulæ are rare during childhood, but may occasionally be the sequelæ of proctitis and hemorrhoids. In many cases the infection is tuberculous. The course of the disease is usually chronic.

Symptoms.—The affection first manifests itself as a small abscess in the cellular tissue surrounding the anus. Instead of forcing its way to the surface and rupturing externally, this abscess burrows into the surrounding tissues. The openings of the fistula are usually quite small, even though the fistulous tract is long and tortuous. When one opening is in the rectal mucous membrane and the other on the skin surface the fistula is called a complete one.

A complete internal fistula is one which has both openings in the mucous membrane, and a complete external fistula is one with both of its openings on the skin surface. Blind fistulæ have but one opening, and may be either internal or external, according to whether that opening is on the skin or in the rectum. External fistulæ manifest themselves by the formation of this opening on the skin which does not heal, and continually discharges pus. If the fistula is internal, pruritus often accompanies it, and is thought to be due to irritation of the anus by the pus which exudes from the orifice of the fistula within the rectum.

Diagnosis.—The subjective symptoms are very mild, and until pus is detected oozing from one of the orifices the existence of the affection may not even be suspected. If, however, such an opening be found, the diagnosis is easily confirmed by inserting a probe and following its course. In this manner, also, the nature of the fistula can be determined; or, better still, the course and ending of a fistulous tract can be ascertained by injecting hydrogen peroxide into one orifice, and noting its appearance at the other.

Treatment.—The treatment of fistula in ano is surgical. If the fistula is complete, the entire length of the tract should be incised and laid open; if incomplete, it should be converted into a complete one by artificially continuing the tract until it has an internal opening into the rectum, and then be incised. After incision the pyogenic membrane lining the fistulous tract should be curetted away, and the cavity packed with iodoform gauze. By these means a cure is usually brought about, but relapses are common.

ISCHIORECTAL ABSCESS.

An ischiorectal abscess is one which forms in the ischiorectal fossa midway between the anus and the tuberosity of the ischium, and is due to infection from an abrasion, fissure, or ulceration of the rectum or anus. The lymph nodes about the rectum first become involved, and thence the invading organisms effect entrance into the fossa. Occasionally the abscess is tuberculous as a result of tuberculosis of the bony pelvis.

Symptoms.—Tenderness, induration, swelling, and redness are found on the affected side; but no fluctuation can be detected until late in the course of the disease, owing to the depth of the abscess and the density of the fascia. Defecation is painful. The child cannot bear its weight on the affected buttock while sitting down. The

temperature usually runs high, and other constitutional symptoms of pus formation appear.

Treatment.—The abscess should be freely incised and the cavity washed out with hydrogen peroxide, after which free drainage should be kept up until the discharge of pus ceases. If the case is one of tubercular infection, the abscess should be opened and the affected bone curetted. After the cavity is perfectly clean it should be closed without drainage.

If pus formation again becomes evident, the same treatment should be repeated, and several incisions of the abscess are sometimes required. It is well to give these children a purgative dose of castor oil. If tuberculosis be evident they should be kept out of doors, put on a nourishing diet, and sent away to the country, mountains, or seashore if practicable.

DISEASES OF THE LIVER.

Organic diseases of the liver are extremely rare during childhood; on the other hand, this organ frequently becomes affected during the course of the acute infectious diseases so common among children.

SIZE AND LOCATION OF THE LIVER.

The liver is relatively much larger in the child than in the adult, its weight being from $\frac{1}{30}$ to $\frac{1}{20}$ of the entire weight of the body. At birth it weighs about four ounces (128 Gm.), which is approximately 4 per cent. of the body weight; but its subsequent growth is not in proportion to that of the other tissues of the body, therefore in the adult its weight represents only $\frac{1}{40}$ of that of the entire body. When the organ is outlined upon the body surface, liver dulness is found to extend from the fifth interspace in the mammary line to about an inch below the border of the ribs; in the axillary line it reaches the seventh intercostal space; and posteriorly it extends to the ninth intercostal space.

In order to examine the liver to the best advantage, the child should lie upon its back with the knees flexed to relax the abdominal muscles. The lower border may be outlined by percussion, and by light palpation from below upward using only the finger tips. The upper border is easily outlined by percussion, since there is a marked contrast between liver dulness and pulmonary resonance.

In rare instances the liver occupies an abnormal position. It may be found on the left side of the body in cases of transposition of the viscera; it may be forced downward by right-sided pleural effusion, or by contraction of the chest wall from rickets; or its weight may stretch and elongate the ligaments which hold it in place, and make it sink to a lower level in the abdomen than is normal. This accident occurs most commonly in infants and young children who are ill-nourished and anemic.

BILE.

The composition of bile, which is the product of the secretory activity of the liver cells, differs slightly in the child from that in the adult. It contains a larger quantity of mucin and less of acids. The relatively small proportion of acids is largely responsible for the difficulty with which an infant digests fats, and accounts in some degree for the ease with which fermentation is set up in the intestinal canal. The other constituents of bile are fat, organic salts, lecithin, cholesterin, and about 97 per cent. of water.

JAUNDICE, OR ICTERUS.

Icterus neonatorum, which is usually a physiological phenomenon, is the most common form of jaundice in infancy. After the third or fourth year acute catarrhal jaundice, which is quite rare during infancy, is as common in children as in adults; but other forms of jaundice are seldom observed.

Obstructive Jaundice.—**Etiology.**—Jaundice is merely a symptom of disease. It is a staining of the skin and secretions with pigment derived either from the bile or from the blood. In the latter case the jaundice is called hematogenous, because of its origin; and when the pigment is derived from the bile as a result of obstruction to the flow of bile through the bile ducts it is called hepatogenous. Obstructive jaundice is another term for the hepatogenous variety, while the hematogenous is sometimes called unobstructive jaundice.

Obstructive jaundice is by far the more common, and may be due either to congenital obliteration or stenosis of the bile ducts, to impaction of a gall-stone in the common duct, to pressure upon the lumen of the bile ducts by enlarged glands or tumors in adjacent organs and tissues, or to fecal accumulations within the intestines. Very rarely the bile ducts may be obstructed by an impacted round-worm or by hydatid or echinococcic cysts.

The most common form of obstructive jaundice is the acute catarrhal type which arises when the mucous membrane of the bile ducts becomes swollen, and obliterates the lumen of the bile passages. This inflammation of the bile passages is, as a rule, the result of extension of inflammation from the small intestine; but, although it is usually associated with gastro-intestinal disturbance, it is now generally believed to be of infectious origin, a belief which is supported by the occurrence of several epidemics of acute catarrhal jaundice. It is sometimes a complication in the acute infections, especially scarlet fever and measles. So far bacteriological investigations have failed to demonstrate the presence of any specific organism in acute catarrhal jaundice, although Jaeger isolated a bacillus of the proteus group (proteus fluorescens) in the urine of these patients.

Symptoms.—The most characteristic sign of jaundice of the obstructive type is the yellowish discoloration of the skin caused by the

24

deposition of bile pigments. The skin is also irritated by these pigments; it itches intensely, and may even present lesions of urticaria lichen, or furunculosis. In addition to the discoloration of the skin, the sclera has a yellowish tinge, and the urine and perspiration may also be stained. The stools, on the other hand, are clay-colored, and absolutely devoid of biliary coloring matter, while the tears, saliva, and mucus are also free from discoloration.

In order to test the urine for the presence of bile, put a few drops of urine and half as many drops of nitric acid on a porcelain plate, and allow them gradually to approach each other and fuse. If bile pigment be present a play of colors appears in which red, violet, green, and yellow predominate.

A slow pulse is very characteristic of jaundice in the adult, but is, as a rule, not so invariable in children; it is due to the sedative action of the bile salts upon the heart mechanism. The principal subjective symptoms of the ordinary case of jaundice are vertigo, headache, nervous irritability, and depression of spirits which in the child is apt to manifest itself as stupidity.

In acute catarrhal jaundice the foregoing symptoms are usually preceded by more or less gastro-intestinal disturbance with nausea, vomiting, anorexia, diarrhea or constipation, and pain and tenderness in the epigastrium. The liver is generally somewhat enlarged, and may be quite tender. There is a rise in temperature to 101° or 102° F.; but, aside from a mild feeling of malaise, the child does not especially complain, and many of these cases are treated in the out-patient dispensaries.

Severe obstructive jaundice is rare, but may occasionally be met with. The involvement of the nervous system is pronounced, and is marked by delirium, convulsions, and unconsciousness. There is also high fever, and rapid, irregular pulse and respirations. In these cases death usually supervenes quickly from exhaustion. Important features in jaundice are the tendency to hemorrhage and the lengthened coagulation time of the blood, which contraindicate any but the most urgent operations on these children.

Diagnosis.—The diagnosis of jaundice is easy; but the real purpose in diagnosis is to discover the cause and the seat of the obstruction. The acute catarrhal type may be diagnosed by its mild symptoms together with a history of associated gastro-intestinal derangement and by excluding, as far as possible, other causes of obstruction of the bile ducts.

Prognosis.—In acute catarrhal jaundice, unless associated with an acute infectious disease, the prognosis is favorable. In jaundice due to other causes, the prognosis depends absolutely upon the gravity of the causative factor. The course and duration of an attack of jaundice also depend largely upon the cause. Ordinary simple catarrhal jaundice runs a course of two to six weeks' duration, but in exceptional cases may last much longer.

Treatment.—Unless there is elevation of temperature, the child need not remain in bed but should be kept quiet. An initial course of calomel should be given, 1 grain in doses of $\frac{1}{10}$ of a grain every hour to a child under five years of age, and 2 grains in doses of $\frac{1}{4}$ of a grain to the child above five.

The diet must be carefully regulated, and starches, sugars, and fatty foods restricted. Bland and easily digestible articles of food, such as broth, skimmed milk, albumen-water, and toast are permissible at the onset and, as improvement is noted, lean meat, fish, chicken, and vegetables may be allowed.

The bowels must be kept regular, and for this purpose sodium phosphate, 10 to 20 grains, calomel, $\frac{1}{4}$ to $\frac{1}{2}$ of a grain, or saline mineral waters, such as Carlsbad or Vichy, may be given daily.

Alkaline baths are beneficial, and may easily be prepared simply by adding 2 ounces each of sodium bicarbonate and sodium chloride to a tub of water. When the itching is distressing, a wash containing 1 drop of phenol to the ounce may be applied to the skin, and will usually relieve it. Fresh air is particularly needful in these cases, and the child who is able to play about should be kept out of doors as much as possible.

In severe attacks of jaundice the patient should be put to bed and a hot-water bag or mustard plaster applied to the epigastrium to relieve the pain. If this does not bring relief paregoric may be administered in 5- to 10-drop doses every three hours. To control the vomiting, it may occasionally be necessary to give cocaine, $\frac{1}{40}$ to $\frac{1}{20}$ of a grain, every three hours until the vomiting ceases. In the grave types of jaundice the child's strength must be kept up by stimulation, and brandy, 10 to 20 drops, and aromatic spirits of ammonia, 5 to 10 drops, may be given every two or three hours by mouth, as well as camphorated oil, 1 to 3 drops, and strychnine sulphate, $\frac{1}{400}$ to $\frac{1}{200}$ of a grain, hypodermically. If there are severe hemorrhages, salt solution by slow proctoclysis, and lead acetate, $\frac{1}{10}$ to $\frac{1}{4}$ of a grain, or ergot, 1 to 2 grains, should also be given every three hours.

Hematogenic Jaundice.—Hematogenic, or non-obstructive, jaundice, which is due to alteration in the state of the blood, may be observed during the course of a great number of diseases among which are the following: sepsis, typhoid fever, malaria, congestion of the liver from cardiac or pulmonary disease, syphilis, tuberculosis, specific fevers, acute yellow atrophy of the liver, Winckel's disease, Weil's disease, also after severe hemorrhage, in phosphorus and phenol poisoning, and in cyclic vomiting.

Symptoms.—The symptoms of hematogenous jaundice are chiefly those of the underlying causative factor. The jaundice is less intense than in the obstructive type, and the urine is less bile-stained, although the amount of true urinary pigments, especially urobilin, may be very much increased. In this type of jaundice the stools are not clay-colored.

Course and Prognosis.—The duration of hematogenous jaundice is usually brief, since many of the conditions which cause it are fatal.

Treatment.—The treatment of unobstructive jaundice is essentially that of the underlying condition.

CONGESTION OF THE LIVER.

There are two forms of congestion of the liver—active and passive. Active congestion, which is much less grave than the passive type, occurs physiologically after each meal, but may be much aggravated by overfeeding. This form of congestion may also be associated with acute gastro-intestinal disturbances and infectious diseases. Rarely are any symptoms referable to the liver except an occasional dull ache, or a feeling of fulness in the hepatic region.

Chronic, or passive, congestion of the liver is much more common during childhood than the acute form, and is always due to some obstruction to the flow of blood toward or through the heart. Valvular heart disease is the most common cause of passive congestion; but it may also be due to pulmonary disease, such as fibroid pneumonia, chronic tuberculosis, or fibrosis and adhesions of the pleura.

Pathology.—If chronic congestion of the liver persists for a long period, organic changes take place. The liver becomes reduced in size, and on section it has a nutmeg appearance, due to an alternation of dark and light tints produced by the unequal distribution of the blood within the liver. Its external surface is smooth.

Symptoms.—In chronic congestion the symptoms are, as a rule, mild. There may be slight tenderness in the hepatic region with, in some cases, gastro-intestinal disturbance, such as nausea, anorexia, and constipation. The liver is at first enlarged and tender; at times it may be possible to detect pulsation of the organ. Jaundice is rare; ascites occurs late in the course of the disease; the urine is scanty, and of high specific gravity. In most cases the spleen also is enlarged.

Prognosis.—In passive congestion of the liver the prognosis depends entirely upon the causative factor and is, as a rule, less favorable than when the congestion is active.

Treatment.—The most important point in the treatment of passive congestion of the liver is the relief of the primary disease. Purgation and depletion are beneficial in both the active and passive forms, and may be effected by the administration of 1 to 3 grains of calomel, followed by 1 dram of sodium phosphate, or 2 to 4 drams of magnesium sulphate. The diet should be greatly restricted, and until improvement takes place only liquids allowed.

The following prescription for a child of five years has been found valuable in the after-treatment of these cases:

R—Tinct. nucis vomici . . . . f ʒj
　　Acidi nitrici diluti . . . . f ʒj
　　Aqua . . . . q. s. ad. f ʒiij
Sig.—Teaspoonful in water three times a day.

ENLARGEMENT OF THE LIVER.

An enlarged liver is quite a common finding in children, and is met with much more frequently than in adult life. Simple enlargement is observed in congestion due to pulmonary or cardiac disease, and occasionally accompanies the acute infections. The most common forms of enlargement are those due to syphilis and rachitis. There is also a certain degree of enlargement of the liver in all diseases of the blood, especially in the pseudoleukemia of von Jaksch; but in these cases enlargement of the spleen is more marked.

Other maladies which cause increase in the size of the liver are hypertrophic cirrhosis, fatty infiltration, hepatic and subphrenic abscess, amyloid and hydatid disease, and Still's disease. The association of enlarged spleen with enlargement of the liver, and *vice versa*, is quite common in children, and in the majority of the forms of enlarged liver mentioned above the spleen also is increased in size.

There are a number of intra-abdominal and thoracic conditions which at first may lead the physician to think the liver enlarged, but these can be differentiated from hepatic enlargement by careful examination. The liver may be displaced downward by a right-sided empyema, pleural effusion, or a circumscribed peritoneal effusion between the liver and diaphragm; but in these conditions there is flatness over and above the upper part of the normal area of liver dulness. Tumors of the right kidney or other organs adjacent to the liver may by their proximity either displace the liver or cause an apparent increase in liver dulness; but upon careful examination a line of demarcation between the tumor and the liver can usually be made out.

CONGENITAL ACHOLURIC JAUNDICE.

This disease, which is also known as familial jaundice and congenital family jaundice, is hereditary, occurring usually in several members of a family, and is due to an abnormal state of the blood which is believed to be inherited.

Two forms of familial jaundice are recognized: one type in which the jaundice appears at birth or shortly afterward, and another in which the affection does not manifest itself until late childhood or early adult life. A peculiar feature of the malady is the absence of bile in the urine, hence its name acholuric jaundice. Enlargement of the spleen and anemia are also characteristic features of familial jaundice.

Etiology.—Heredity is the most important etiological factor, and one can usually obtain a history of the disease having been transmitted through successive generations of the family. Several members of a family may suffer from it at the same time, males and females being affected equally. The primary cause is obscure, but it is supposed to be a congenital defect in the hemopoietic system which produces an abnormal state of the blood.

Pathology.—Familial jaundice is hematogenic in origin, and is believed to be due to a congenital abnormality of the red corpuscles which renders them more fragile than they normally are. This leads to an excessive degree of hemolysis, and consequent enlargement of the spleen owing to increased functional activity, also to the production of an excessive amount of bile pigment by the liver.

Symptoms.—In the majority of cases the jaundice is present at birth, but the discoloration of the skin is usually slight, and the other symptoms are mild. In some cases the sclera alone shows pigmentation, but the degree of discoloration may vary greatly from time to time in the same patient. In mild cases the jaundice tends to fade and disappears within a few weeks.

As a rule, there are no marked symptoms of illness, although the child may occasionally have a slight fever with malaise and so-called biliousness. The liver may be slightly increased in size, and the spleen is always decidedly enlarged. Although the urine in these cases shows no bile pigment, yet the stools are bile-stained, and upon examination of the urine an excess of urobilin may be found.

Changes in the blood vary from anemia so severe that it causes death to merely a slight alteration in the blood picture. As a rule, the blood changes are most marked in infancy, and when the blood is microscopically examined a great reduction in the number of red cells is noted; many of them are smaller than normal, and usually a few nucleated red cells are present. There is a great reduction in hemoglobin and a low color index; but the leukocytes are practically unchanged.

These children may continue to have such attacks all through life, but they are less severe after they are grown up, and, as a rule, do not affect the general health.

Diagnosis.—In familial jaundice the diagnosis can be made, as a rule, on the history of the disease appearing in more than one member of a family, and in successive generations. Absence of bile in the urine, and alteration of the blood picture, with very mild subjective symptoms, are also characteristic findings which confirm the diagnosis.

Prognosis.—This is least favorable in very young infants in whom the disease is associated with anemia. Older children and adults suffer but little; and, although the affection is liable to persist throughout life, the attacks occur after longer intervals with increasing age.

Treatment.—There is no known cure for this disease, but prophylaxis may accomplish much in preventing attacks of jaundice in the children who have inherited it. The diet should be carefully regulated so as to prevent gastro-intestinal disturbance, and the surface of the body should be kept warm and never allowed to become chilled. Fowler's solution in 1- to 3-drop doses, or tincture of ferric chloride, 2 to 10 drops, should be administered for the anemic condition of the blood. Any measures which help to build up the general health, such as moderate outdoor exercise, ample rest, and change of surroundings, are beneficial in these cases.

CONGENITAL OBLITERATION OF THE BILE DUCTS.

This affection is quite rare, but a number of cases are recorded in medical literature. In some instances it has been observed in more than one member of a family.

Etiology.—The disease occurs more frequently in male than in female children, and is to a certain extent hereditary. The exact cause is unknown; but it is thought to be due to maldevelopment, since it is occasionally accompanied by other congenital anomalies. It may also be the result of an idiopathic inflammation of the bile ducts in early life, or possibly may be due to congenital syphilis.

Pathology.—In these cases the liver shows marked cirrhotic change, being hard, and olive green in color from staining by the obstructed bile. The gall-bladder may be completely obliterated, or represented merely by a small fibrous sac with scarcely any lumen. In some cases it contains a little clear mucus; exceptionally, when the common duct alone is obstructed, it may be distended. The common bile duct and the hepatic ducts are often entirely obliterated, nothing remaining but fibrous cords which when opened may or may not disclose a narrow lumen. The bile capillaries are dilated and distended with bile, which is usually thick and inspissated.

Microscopic examination of a section of liver tissue shows but little change in the liver substance and practically no degeneration of the liver cells; but the bile capillaries are irregularly distended and choked up with biliary secretion. The spleen is also enlarged, and together with the pancreas may show extensive fibrosis.

Symptoms.—Infants suffering from this condition may appear normal at birth, but if jaundice is not present then it usually develops in the course of two or three days, and rapidly becomes intense. In exceptional cases jaundice may not appear for two or three weeks, even though the bile ducts were obliterated at birth. The intensity of the jaundice varies; but the pigmentation never leaves the skin, and in addition there may be subcutaneous hemorrhages and extravasations of blood into the mucous membranes. The stools are white because of the absence of bile, and may be quite dry and hard. The urine is bile-colored, and stains the napkin, but the amount of biliary coloring matter varies considerably.

On examination, the abdomen is often found to be distended, and may contain an abnormal amount of peritoneal fluid, although the distention is partly due to enlargement of the liver and spleen, which may be quite marked. In a number of cases the general health is seemingly unaffected; but in the course of a few weeks or months the body nutrition begins to fail, exhaustion and emaciation are progressively worse, and death usually occurs within a year. The majority, however, succumb in much less than a year, and death may ensue shortly after birth from hemorrhages about the navel, under the skin, or from the stomach or intestines.

Diagnosis.—To the general practitioner the diagnosis is difficult; and, unless something strongly suggests the condition it may not be recognized. When the disease is suspected the following symptoms will point to the diagnosis: intense jaundice of obstructive type, coming on within a few days after birth, and associated with hemorrhages into the skin and from the mucous membranes.

Prognosis.—The outlook is most unfavorable, since the disease cannot possibly be influenced by medicine or surgery except in syphilitic cases, and here slight improvement may follow antisyphilitic treatment.

Treatment.—It is perfectly obvious that no treatment will avail when the bile ducts are obliterated, and even surgical intervention can be of use only in those cases where there is merely obstruction to the flow of bile. Since a few of these cases appear to be of syphilitic origin, mercury may be administered orally in the form of calomel, $\frac{1}{8}$ to $\frac{1}{4}$ of a grain, or in gray powder, 1 to 3 grains daily; or it may be given by inunction, $\frac{1}{2}$ to 1 dram of mercurial ointment being rubbed into the skin every day. Potassium iodide in 1- to 5-grain doses three times daily may also be given for its antisyphilitic properties, and because it is theoretically supposed to dissolve fibrous tissue, and thus widen the lumen of the stenosed ducts.

Aside from this mode of treatment, nothing can be done but to keep up the general health of the child by judicious feeding and other hygienic measures.

STENOSIS OF THE BILE DUCTS.

Stenosis of the biliary ducts may also occur in children from inflammatory changes within the lumen of the ducts, such as might be caused by the passage of a large gall-stone. Pressure from without may also occlude the biliary tracts. Occasionally stenosis is produced by neoplasms of adjacent tissues, by perihepatitis, and by syphilis. Most of the symptoms are due to the underlying cause of the stenosis, and an accurate diagnosis of the affection is rarely made.

Treatment.—The treatment depends entirely upon the cause, and consists in the removal of the stenosis and the establishment of free drainage of the bile.

ACUTE YELLOW ATROPHY OF THE LIVER.

This very rare disease is, fortunately, especially uncommon in children. It is characterized by fatty degeneration and atrophy of the liver accompanied by toxic symptoms, and is almost invariably fatal.

Etiology.—Acute yellow atrophy sometimes accompanies the acute infections, notably diphtheria, erysipelas, and typhoid fever; occasionally it follows chloroform poisoning. It has also been seen in association with syphilis, but its true cause is as yet undiscovered.

Boys are more liable to contract the disease than girls; but in adult life more women suffer from it than men, owing to the predisposing influence of pregnancy.

Pathology.—Postmortem examination reveals a liver about one-half the normal size, reddish in color, its surface presenting a mottled appearance, with areas of red, green, gray, and yellow. In consequence of the great destruction of liver cells the organ rapidly dwindles in size, sometimes even in four or five days. The capsule is wrinkled and loose, the tissue is soft and flabby, the lobular markings may be wholly obliterated. On section the surface is either of a uniform yellow color or presents alternate areas of red and yellow. The yellow areas represent an earlier stage of the disease; they contain degenerated liver cells within which are fat droplets of all sizes. The red areas are composed of cellular débris and connective tissue in the meshes of which may be found fat drops and biliary coloring matter.

Pseudo-bile ducts, or canaliculi, and a certain degree of cell infiltration of the interstitial tissue may be observed under the microscope, and indicate an attempt at regeneration of the liver cells and beginning fibrosis. The bile ducts are in a state of catarrhal inflammation. Leucin and tyrosin may be found in the interior of the hepatic and portal veins. The skin and other viscera are usually deeply bile-stained. There may be small hemorrhages in various parts of the body. The spleen is enlarged; there are fatty changes in the heart muscle and renal epithelium, and an abnormal amount of serum within the serous cavities.

Symptoms.—This disease begins insidiously, and for several days there may be merely slight evidences of gastro-intestinal derangement, such as anorexia, nausea, vomiting, jaundice, malaise, headache, and abdominal distress. The liver is at this period believed to be enlarged; but the symptoms are so mild that the cases rarely come under observation at this early stage. Following these symptoms, the disease suddenly assumes a grave aspect; the jaundice deepens, vomiting becomes severe, and there is an increase in abdominal pain.

Blood may be found in the vomitus or passed in the stool. Subcutaneous hemorrhages throughout the body may follow slight injuries, and there may be bleeding from the gums. The blood picture usually shows a moderate leukocytosis (15,000 to 20,000). Cerebral symptoms, such as delirium, convulsions, and drowsiness or coma, are not uncommon. The urine decreases in quantity, is deeply bile-stained, of high specific gravity, and contains leucin and tyrosin. The temperature is generally a little above normal, but may be subnormal, although there is always a sharp rise before death. The decrease in the size of the liver is remarkable; in some cases, a few days after the onset of the disease, it can no longer be outlined either by percussion or palpation.

Diagnosis.—At the onset of the disease an accurate diagnosis is impossible, since there are no characteristic symptoms; but, when fully developed, a case of acute yellow atrophy of the liver should

not be difficult to recognize except because of its rarity. The cardinal features upon which the diagnosis should be based are the intense jaundice with ecchymoses, slight fever, persistent vomiting, and symptoms of cerebral irritation. The diagnosis may sometimes be confirmed by physical examination, which reveals a great reduction in the size of the liver and enlargement of the spleen.

Prognosis.—The disease usually ends fatally within a short time, and the mortality is so great that reported recoveries may probably be attributed to error in diagnosis. The usual duration of the disease is from two to three weeks.

Treatment.—The treatment of acute yellow atrophy of the liver is almost purely symptomatic, for little can be done to arrest the progress of the disease. At the onset a course of 1 or 2 grains of calomel in divided doses should be given, followed by ½ to 2 drams of magnesium sulphate. Rest in bed is, of course, imperative. The diet, while light, should contain the maximum of nourishment in order to support the child's vitality. In the cases with cerebral symptoms an ice-bag may be applied to the head, and headache relieved by the administration of phenacetin or antipyrin, 2 to 3 grains. Saline solution, given by slow proctoclysis, may possibly diminish toxemia and keep up the child's strength.

CHOLELITHIASIS.

Gall-stones are extremely rare in children, but may occur at any age from infancy to puberty. In the newborn they are usually fatal, and even in older infants cause death within a few weeks. Jaundice is intense; in infancy it may be the only perceptible symptom; but in older children gall-stones give rise to the same symptoms as are observed in the adult.

Diagnosis.—In older children the diagnosis can usually be made from the symptoms; but in infants few cases are recognized.

Treatment.—The treatment is almost wholly surgical, and consists in the operative removal of the stones, which are generally found in the common duct.

ABSCESS OF THE LIVER.

This affection is extremely rare during childhood. As in the adult, abscess of the liver may be either single or multiple, but is always of microbic origin.

Etiology.—Most abscesses in this organ arise from infection in the portal area or from suppurative processes elsewhere in the abdomen, such as appendicitis or suppuration of the mesenteric glands. Abscess formation in the liver may also be due to sepsis, pyemia, peritonitis, typhoid fever, traumatism, phlebitis, or tuberculosis, and has been known to follow the migration of round worms into the biliary passages. Amebic abscess of the liver is rarely seen in children, even when amebic dysentery is prevalent.

Pathology.—The right lobe of the liver is the most common seat of an abscess, although in some cases the whole lobe may form an abscess cavity. The liver is usually enlarged; but there may be no visible change in its contour if the abscess be deep-seated. The content of the abscess is usually pus which may rupture into the right pleural cavity, the peritoneum, or the pericardium, and cause death; in exceptional cases it may be discharged through the abdominal wall by means of a fistulous opening.

Symptoms.—In abscess of the liver these are usually severe, but in rare cases they may be latent. As a rule, there is pain in the hepatic region, and the liver is tender. Chills and sweats usually accompany the fever which is the most constant feature of the disease, and ranges from 103° to 105° F. Jaundice is not invariably present, but occurs in about 50 per cent. of the cases, although it is never intense. Vomiting and diarrhea are not uncommon. In severe cases prostration comes on rapidly.

On physical examination the liver is usually, but not always, found to be enlarged. When there is enlargement, it spreads upward in the midaxillary and mammary lines, and is due to the pus present and to hyperemia and swelling of the hepatic cells. Fluctuation is a late symptom, but can generally be demonstrated, and a characteristic edematous condition of the skin and abdominal wall over the hepatic region is observed. Pronounced nervous symptoms and nephritis are not uncommon in the latter stages of this disease.

Diagnosis.—Hepatic abscess is extremely difficult to diagnose, especially in the early stages. Pain in the hepatic region, and referred to the right shoulder, tenderness over the liver, and an intermittent fever which is not malarial, are significant diagnostic points; but it may be impossible to state positively that there is an abscess unless pus is withdrawn by means of an aspirating needle.

Prognosis.—The prognosis is usually unfavorable, even when a case is treated early. Death is due to toxemia and exhaustion. The mortality in children averages about 75 to 85 per cent.

Treatment.—The treatment of hepatic abscess is essentially surgical, and unless the child is too weak for operation incision and drainage should be advised. Operative interference is useless when there are multiple suppurating foci within the liver.

The child's vitality and strength should be supported by a nourishing diet and the administration of tonics, such as tincture of ferric chloride, 2 to 5 drops, or quinine sulphate $\frac{1}{2}$ to 1 grain, three times a day. Morphine may be necessary for the relief of pain, and can be given hypodermically in $\frac{1}{40}$ to $\frac{1}{20}$ of a grain dose, according to the age of the child.

SUBPHRENIC ABSCESS.

Subphrenic abscesses are rare in children, but are occasionally found in association with suppurative conditions in the region of the liver. The most common site of such an abscess is the space behind the right

coronary ligament and extending around its right border to the sub-hepatic space. Sometimes the pus accumulates either to the right or left of the falciform ligament, or in the lesser peritoneal cavity.

Etiology.—In most cases subphrenic abscess is secondary to appendicitis; but it may follow the perforation of a gastric or duodenal ulcer, traumatism, cholelithiasis, Pott's disease, perinephric abscess, empyema, pneumonia, abscess of the liver, spleen, or pancreas, or diffuse peritonitis.

Symptoms.—The symptoms are pain in the right lower chest and about the diaphragm and liver, alternating chills, fever, and sweats, dyspnea, and cough. Examination of the blood in these cases reveals a leukocytosis of 15,000 to 25,000. The abscess cavity may contain pus and, in some instances, gas which causes either tympany or flatness on percussion over this area.

Diagnosis.—In subphrenic abscess the diagnosis is based upon the history of some disorder which might possibly cause an abscess to form in the subphrenic region; upon the presence of dulness, pain, tenderness, a tumor, and rigidity of the overlying muscles; upon thoracic signs and symptoms, *e. g.*, pleural friction or effusion, upward displacement of the right lung, and increased dulness over the liver; and upon additional general signs and symptoms of suppuration, such as fever, chills, sweats, leukocytosis, and progressive wasting.

Prognosis.—In children the prognosis is very unfavorable, and without operation practically all of these patients die. Operation is attended with such great shock that the mortality-rate where there is surgical intervention is over 50 per cent.

Treatment.—The treatment is essentially surgical, and consists in securing free drainage. The incision is usually made through the lower chest wall—very rarely through the abdomen. Should this mode of treatment be successful, perfect recovery may be expedited by administering tonics, such as syrup of the iodide of iron, 10 to 20 drops, or the elixir of iron, quinine, and strychnine phosphates, 5 to 10 drops, three times daily; by putting the child on a full nourishing diet; and, if living in the city, by sending it away to the country or seashore for pure air and change of scene.

FUNCTIONAL DISORDERS OF THE LIVER.

Functional disorders of the liver are quite common in children, and are usually referred to as "bilious attacks." The exact nature of such derangements is unknown; but they are usually accompanied by intestinal indigestion, and seem to depend upon a lack or an impoverished quality of the biliary secretion, for the stools are grayish-white or clay-colored, and very hard and dry. In some instances a predisposition to these attacks seems to be inherited, the father or mother also being affected in the same manner.

Symptoms.—During such a bilious spell symptoms indicative of intestinal indigestion appear, such as coated tongue, offensive breath,

anorexia, nausea, flatulence, and constipation or diarrhea with foul-smelling stools. For a day or two preceding the attack the child is fretful and peevish, and if old enough complains of not feeling well. The characteristic appearance of the stools is caused by incomplete absorption in consequence of the lack of bile elements in the intestinal tract. The temperature is usually elevated to 100° or 101° F., but rarely reaches 103° F. The urine is highly colored and concentrated.

These children are usually pale and sallow, and during an attack may become very weak; but, as a rule, the symptoms subside in a day or two, and recovery rapidly ensues.

Treatment.—Prophylaxis is important. Children who are subject to bilious attacks should have their diet carefully regulated and be prevented from overeating. Actual treatment consists in stopping all food for twelve to twenty-four hours, and giving water freely. Cholagogues are very useful, either calomel, podophyllum, or dilute nitric acid, the calomel preferably administered in $\frac{1}{10}$ to $\frac{1}{5}$ of a grain doses every hour until 1 or 2 grains have been taken, followed in six to eight hours by magnesium sulphate, $\frac{1}{2}$ to 1 dram; or, sodium phosphate in 10- to 20-grain doses may be given each morning in hot water. The dose of podophyllum is $\frac{1}{4}$ to $\frac{1}{2}$ grain, and of dilute nitric acid 1 to 2 drops, three times daily.

When the symptoms have disappeared, the diet may be increased, and cereals, broths, junket, and custards allowed until the full diet is gradually resumed. The bowels should be regulated by an occasional course of calomel followed by a saline laxative. Abdominal massage, moderate exercise, and an outdoor life are especially beneficial in these cases.

CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is uncommon during childhood, and most rare in infancy, the cases increasing in frequency as adult life is approached. It is more common in boys than in girls. It is caused by an overgrowth of connective tissue at the expense of the functionating cellular structure of the organ. As a rule, the liver becomes much firmer and smaller than normal; but it may be considerably enlarged, and in some cases there is no apparent alteration in size. Two forms are recognizable in children—the atrophic in which the liver dwindles in size, and hypertrophic cirrhosis which results in enlargement of the organ.

Etiology.—Syphilis is one of the chief causes of this affection, and it is in association with congenital lues that most infantile cases of cirrhosis occur. Alcohol, the most common cause of cirrhosis in later life, plays but an insignificant role during childhood.

A cardiotubercular type of cirrhosis has been described in association with polyserositis, but is comparatively unimportant. The eruptive fevers may also be mentioned as having in some way a possible influence in the production of cirrhosis. In many cases the cause of the disease is obscure.

Pathology.—In atrophic cirrhosis the liver is small, hard, and lobulated, and has an uneven surface. There is an excess of connective tissue in the organ, this being found around the liver lobules, about the bile ducts, and dipping down from the capsule into the liver substance, causing the capsule to become adherent. In hypertrophic cirrhosis the liver is usually enlarged, the fibrous tissue not showing the tendency to contract which it does in the atrophic form. There is practically no compression of the portal vein; but the biliary channels are obstructed, the flow of bile impeded, and this produces jaundice. In the majority of cases of cirrhosis of the liver the spleen is enlarged and usually shows a certain degree of fibrosis.

Symptoms.—This disease may remain latent for a considerable time, but symptoms of secondary conditions due to cirrhosis usually appear quite early. Among these are gastro-intestinal disturbances due to chronic congestion of the gastric mucous membrane, such as nausea, anorexia, irregularity of the bowels, and abdominal discomfort. Epistaxis, vomiting of blood, and melena also appear as the result of congestion of the mucous membranes, but by relieving the engorgement are rather beneficial than harmful.

These children are pale and sallow; the veins of the upper abdomen and lower thorax are distended and quite prominent; their flesh is flabby; there is a steady loss in weight. There may be slight fever in the early stages of atrophic cirrhosis, but jaundice appears less frequently than would be expected. In many cases abdominal dropsy is the first symptom which calls attention to the cirrhotic liver, the accumulation of fluid being enormous, and causing marked distention of the abdomen, and often distress from mechanical interference with the diaphragm.

Edema of the legs is not so common, but may occur in consequence of obstruction from pressure of the ascitic fluid on the veins which return the blood from the lower extremities. Physical examination reveals a diminished area of liver dulness, and, as a rule, enlargement of the spleen. In these cases death is due to exhaustion, and may be preceded by drowsiness, delirium, and coma.

In hypertrophic cirrhosis, jaundice is present at the onset, grows deeper as the disease progresses, and is one of the diagnostic features. There is no ascites. Both liver and spleen are enlarged. The course of the hypertrophic is more rapid than that of the atrophic form of cirrhosis, and the children affected usually die within a year or eighteen months.

Diagnosis.—The cardinal features in the diagnosis of atrophic cirrhosis are the decrease in the size of the liver and the ascites. In hypertrophic cirrhosis the enlargement of the liver and spleen and deepening jaundice are significant, but the diagnosis is often most difficult and is frequently made only at postmortem.

Prognosis.—In most cases the prognosis is unfavorable, and there is certainly no possibility of curing the disease and restoring the liver to normal; but in some instances we may remove the cause of cirrhosis,

and thus arrest the disease before the liver is too badly damaged to functionate properly. In syphilitic cirrhosis the outlook is thus made favorable if antisyphilitic treatment be instituted early.

Treatment.—In all but syphilitic cases the treatment is largely symptomatic, since little can be done to restore the organ to its normal state. When syphilis is suspected, mercurial inunctions should be given, using $\frac{1}{2}$ to 1 dram of mercurial ointment daily, and rubbing it into a different part of the body each day.

Mercury should also be administered internally in the form of gray powder, dose 1 to 3 grains three times a day; or calomel may be given, $\frac{1}{10}$ to $\frac{1}{6}$ of a grain three times a day. It is advisable to continue the mercurial treatment for at least a year, and potassium iodide should be given in addition in 1- to 5-grain doses after meals, gradually increasing the dosage to the point of tolerance.

In cirrhosis from other causes the underlying factor should be removed, whenever possible, in order to arrest the progress of the disease.

These children are, as a rule, poorly nourished and in bad health, consequently an attempt must be made to build up their general constitution by adequate rest, moderate exercise, plenty of fresh air, and frequent bathing. Particular attention should be paid to the diet, which must be nourishing but light on account of the congestion of the gastric and intestinal mucosa. The body must be kept warm, and its surface never allowed to become chilled.

If relief of the gastro-intestinal symptoms is to be expected, the gastric congestion must be removed, and this is best accomplished by giving the child alkaline mineral waters before meals. Carlsbad and Vichy are the best, and act most effectually when taken hot; in fact, hot water is of so much value when taken by the tumblerful before meals that, in cases where mineral waters cannot be procured on account of their cost, plain hot water should be taken instead. Saline purgatives are also of service in relieving the congested stomach, and sodium phosphate, 10 to 20 grains, or magnesium sulphate, $\frac{1}{2}$ to 2 drams, may be taken each morning in hot water.

Besides relieving the symptoms due to congestion, these saline depletents also carry off ascitic fluid; but it may be necessary to give diuretics as well, or even to tap the abdomen if the accumulation of fluid becomes so great as to cause mechanical interference with the heart action or the diaphragm. The following prescription for a diuretic has proven of service:

R—Potassii bitartratis . . . . ℥iiss
   Syrupi limonis,
   Aqua . . . . āā f℥j
Sig.—℥j, every three hours in water.

If the heart becomes weak and stimulation is needed, strychnine sulphate, $\frac{1}{400}$ to $\frac{1}{200}$, and atropine sulphate, $\frac{1}{800}$ to $\frac{1}{400}$ of a grain, may be given by hypodermic; but if the cardiac weakness is due to over-

action caused by the pressure of the ascitic fluid tapping should be resorted to.

To tap the abdomen of a child, the little patient should be held in a sitting posture until the trocar has penetrated the abdominal wall. The fluid should be drained off very slowly, a little being allowed to remain. Before plunging in the needle, care should be taken to see that the bladder is empty, since this organ has not infrequently been punctured while tapping the abdomen.

In many cases the fluid returns rapidly and tapping is again necessary. Laparotomy has recently been advocated as a possible means of preventing this recurrence of ascitic fluid.

AMYLOID LIVER.

Amyloid degeneration, or lardaceous disease, of the liver, although quite rare during childhood, sometimes occurs in association with amyloid disease of other organs, and is characterized by an infiltration of all the tissues of the liver with amyloid material. The infiltration begins in the walls of the smaller arteries, then invades the cells and capillaries and, in extreme cases, permeates even the connective tissue.

Etiology.—Tuberculosis, syphilis, and rachitis are the diseases most often accompanied by lardaceous infiltration. The usual cause of amyloid liver, however, is continued suppuration, especially that of chronic bone disease, and it is occasionally observed in association with tuberculosis of the hip and Pott's disease. Pulmonary tuberculosis does not, as a rule, cause amyloid disease; but other chronic suppurative processes in the lung, such as empyema and bronchiectasis, are recognized factors in the formation of lardaceous substance. Syphilis of the bone, pernicious anemia, leukemia, and the infectious diseases are also predisposing causes.

Pathology.—The amyloid liver is enlarged, hard, firm, and heavier than the normal organ. The surface has a waxy appearance, and on section the liver tissue resembles the fat of bacon. When examined under the microscope, degeneration of the liver cells is apparent, which has been caused by deposits of an amyloid substance resembling starch granules; occasionally there is also fatty infiltration.

If a section of the affected liver tissue is painted with a weak solution of iodine, it turns to a mahogany-red color, and on adding sulphuric acid the color again changes to violet or blue. When the liver is thus affected, deposits of amyloid substance may also be found in the spleen and kidneys.

Symptoms.—In these cases there are no typical symptoms referable to the liver aside from the marked enlargement, which can be demonstrated by physical examination of the child. An increasing pallor of the skin, with edema and puffiness about the eyes, is a constant feature, but there is no jaundice. Occasionally gastro-intestinal disturbances appear, with nausea, vomiting, and diarrhea. The kidneys

are usually involved to such an extent that the urine is increased in amount and contains albumin and hyalin casts.

In the terminal stages of the disease, ascites and edema may be present, and the child become extremely emaciated from progressive wasting. On physical examination both the liver and spleen are found to be enlarged. The enlargement of the liver is uniform, the edges are rounded, the surface of the organ is smooth. As a rule, there is no tenderness.

Diagnosis.—This is readily made when we find in a child enlargement of the liver with a chronic suppurating process elsewhere in the body, and when there is accompanying enlargement of the spleen and albuminuria, but no jaundice.

Prognosis.—In children the prognosis of lardaceous disease of the liver is, as a rule, unfavorable because of the gravity of the primary disease. When it is possible to remove its cause, improvement in so far as this is indicated by a cessation of symptoms may occur, but it is questionable whether the liver tissue ever returns to normal.

Treatment.—Aside from the removal of the cause, the treatment of lardaceous disease is largely symptomatic. If syphilis is the underlying factor this disease should be treated. In those cases associated with chronic suppuration, the suppurative process must be healed, if possible. Since the general health of these children is usually poor, they should be put upon a light nourishing diet, take moderate exercise, and have adequate rest and plenty of fresh air. Tonics containing iron, such as the syrup of ferrous iodide, dose 5 to 20 drops three times a day, may be given; and, if the circulation be poor, also 1 to 3 drops of digitalis after meals.

FATTY LIVER.

This disease is common during childhood, and is the cause of many cases of enlargement of the liver. It may be due either to fatty infiltration or fatty degeneration of the liver cells, which are more or less completely converted into fat. When fatty infiltration takes place, fat droplets are found within the liver cells which become larger, push aside the protoplasm, and cause it to disappear by interfering with its nutrition. In fatty degeneration the protoplasm of the liver cell disintegrates, the nucleus loses its staining power, and fat droplets representing the principal part of the residue fill the cell. Of these two conditions fatty infiltration is by far the more common during childhood.

Etiology.—Fatty infiltration takes place in children who are overfed, and who indulge in an excessive amount of sweets and pastry; to a certain extent, too, it is present in every case of adiposity. Children in cachectic states in which oxidation of fat is interfered with have a fatty liver, and this condition is also associated with gastro-intestinal indigestion, tuberculosis, rachitis, and wasting diseases. Fatty degeneration of the liver occurs as a result of the action of various toxins upon the liver cells, and is seen in phosphorus and chloroform poisoning; in acute infections, such as diphtheria, measles, scarlet fever,

25

smallpox, typhoid fever, miliary tuberculosis, acute leukemia, and bronchopneumonia; also in acute yellow atrophy of the liver, congenital syphilis, and chronic tuberculosis.

Pathology.—It has been estimated that more than one-half of all the children who come to autopsy have an abnormal deposit of fat in the liver. In fatty infiltration the liver is greatly enlarged in all its dimensions, is soft, and has a smooth surface. It is lighter in color than the normal organ, owing to the presence of fat, and also to anemia of the liver. On cross-section fat droplets may be expressed from the cut surfaces with ease, and on microscopic examination the fat content of the liver cell is readily detected.

Symptoms.—There are no subjective symptoms of fatty liver, and unless revealed by palpation and percussion the condition may not be suspected. The area of liver dulness will be found to be increased, but not so greatly as in amyloid disease. The edge of the liver can usually be palpated, and is smooth and rounded. In the latter stages of fatty degeneration the liver becomes much smaller than usual, owing to the rapid destruction of liver tissue.

Prognosis.—In fatty infiltration the prognosis depends on the underlying factor, and if the cause can be removed the liver returns to normal. Fatty degeneration is usually secondary to some grave condition, and generally proves fatal.

Treatment.—The treatment is for the most part that of the primary disease. In fatty infiltration due to overfeeding, it is possible to reduce the size of the liver by regulating the diet, for the exclusion of all carbohydrates will stop to a great extent the formation and deposition of fat. Abdominal massage and an adequate amount of outdoor exercise are also beneficial in these cases.

TUBERCULOSIS OF THE LIVER.

Tuberculosis of the liver is not uncommon in children affected with general tuberculosis, but rarely causes any symptoms except enlargement of that organ, which is usually attributed to some other cause. Primary lesions in the liver need hardly be considered; but the infection may be carried into the blood stream from the lung and reach the liver through the hepatic arteries, or may be carried to the portal circulation in intestinal tuberculosis.

It is also possible for tuberculous infection to be conveyed to the liver by way of the bile ducts or lymphatics. In some cases in which the primary focus is in the lung, pulmonary symptoms may be latent until the disease is well established.

Pathology.—The miliary type of tuberculosis is the most common. Here the liver is usually enlarged, yellow in color, its surface studded with miliary tubercles, which are also scattered throughout the interior of the organ. They are most abundant within the lobules, and show a tendency to cluster about the bile ducts. In more advanced stages of the disease these tubercles form cyst-like cavities ranging in size

from a pin head to a large pea. Large tubercles are more frequently found in the liver during childhood than they are in adult life, but are extremely rare.

Diagnosis.—There is little opportunity to diagnose a tuberculous liver unless a large tubercle forms close to the surface and gives rise to a palpable tumor. Here the treatment is that of tuberculosis elsewhere in the body. Cirrhotic changes are occasionally attributed to chronic tuberculosis of the liver, and occur as its sequel. Tuberculosis of the bile ducts occurs in association with tuberculous infection of the peritoneum and intestines, the cases being relatively more numerous in children than in adults. It presents no characteristic symptoms, and is commonly diagnosed only at autopsy.

Treatment.—The treatment is that of general tuberculosis.

SYPHILIS OF THE LIVER.

Syphilitic infection of the liver is not uncommon. The majority of cases occur in children who suffer from congenital lues, although syphilis acquired during childhood may also result in syphilitic hepatitis. The acquired form differs from the congenital in its limited distribution, the infection being carried, as a rule, by the hepatic artery, while in congenital lues the syphilitic virus is transmitted by the umbilical veins, and diffused throughout the liver.

Most cases of syphilis of the liver are observed in newborn or premature infants and, at the latest, become apparent during the first few weeks of life. Exceptionally, cases are reported in which the disease did not manifest itself in the liver until after the fifth or sixth year, but these are uncommon.

Pathology.—In congenital cases, as a rule, the liver is enlarged at birth, and has a smooth but pale or mottled surface. It is much harder and firmer than the normal organ, and distinctly resists the knife when an attempt is made to section it. On section the cut surface is smooth and shining, and usually reveals many small gray areas, commonly called miliary gummata, which represent a diffused cell-growth throughout the organ. This diffuse infiltrative form is most common; but occasionally there may be scattered, throughout the liver, localized gummatous areas of much greater size, resembling the adult form of syphilitic hepatitis, although the gummata are usually smaller.

Adhesions also are frequently found about the capsule of the liver; occasionally the liver is adherent to adjacent organs or tissues. In the diffuse infiltrative form there is observed under the microscope a considerable growth of connective-tissue cells at the expense of functionating liver tissue. Later on this causes the cirrhotic changes not uncommon in syphilitic hepatitis. Enlargement of the spleen is also a common finding in syphilis of the liver.

Symptoms.—The infants affected are often premature or stillborn, and the enlarged liver can usually be outlined on examination of the

abdomen. If the child is alive at birth, the liver may then be found to be enlarged, or it may become larger within the first few weeks of life. Moreover, jaundice and the concomitant signs and symptoms of syphilis are usually present.

The nutrition of syphilitic infants is usually below par, and it is generally quite easy to feel the firm smooth edge of the enlarged liver, and to detect the spleen which is also increased in size. When congenital syphilis does not affect the liver until later childhood, ascites is not unusual, in addition to those symptoms observed in infancy.

Diagnosis.—In the majority of cases of congenital syphilis the diagnosis is easy. It is based upon enlargement of the liver in a child with inherited lues. The Wassermann reaction and the results of mercurial treatment may be confirmatory indications, but are scarcely necessary.

Prognosis.—This depends to a great extent upon the nature of the syphilitic process. It is less favorable in the diffuse infiltrative form which may go on to cirrhosis than in the gummatous type. From our observations it would seem that recovery is to be expected in the majority of cases.

Treatment.—Mercury should be administered from the time the diagnosis is first made until all evidence of syphilis has disappeared. After this it may be given at frequent intervals for two or three years. Inunctions are especially valuable in infants, and 15 to 30 grains of mercurial ointment should be rubbed into the skin each day, choosing a different area for the inunction from day to day, the abdominal wall, axillæ, and groins being preferable sites.

Calomel, in a dose of $\frac{1}{8}$ to $\frac{1}{4}$ of a grain, is the best form of mercury to give by mouth, although the gray powder is also sometimes given in 1- to 3-grain doses. Iodides are also beneficial when combined with mercurial treatment, and may be administered as potassium iodide, 1 to 5 drops of the saturated solution, or 5 to 10 drops of the syrup of ferrous iodide. If stomatitis (mercurial) is threatened, a 3 per cent. solution of potassium chlorate should be used as a mouth wash.

A syphilitic infant should be allowed to nurse from its syphilitic mother; its general health should be carefully attended to. Older children should be put on a full nourishing diet, take plenty of outdoor exercise, and have the advantage of all other hygienic measures for the improvement of the general health.

TUMORS OF THE LIVER.

New growths of the liver are extremely rare in children, but both malignant and benign tumors have been observed. Malignant growths are generally secondary, the primary focus being, as a rule, in the kidney. Both carcinoma and sarcoma are observed, adenocarcinomata being the most frequent.

Symptoms.—Jaundice and enlargement of the liver are the most common symptoms. On palpation the surface of the liver feels very irregular and uneven, and nodular tumors may be detected. In some

cases the tumor attains an enormous size, and may entirely fill the abdominal cavity.

Treatment.—This is of no avail. It consists mainly of attempts to relieve the discomfort, and to prolong the life of the patient. Death usually occurs in from two to six months.

The benign tumors of the liver are the adenoma, angioma, myxoma, lipoma, and fibroma. They are very rare in children.

CYSTS OF THE LIVER.

Three varieties of cyst may be found in the liver; *i. e.*, simple retention cysts caused by obstruction of a small bile duct, congenital cystic disease of the liver, and hydatid disease of the liver.

Hydatid cyst, caused by the invasion of the liver by the embryo or larva of the Tænia echinococcus, is quite rare in this country. These cysts, if small, produce no symptoms; but as they increase in size the liver slowly enlarges, perhaps considerably, and feels heavy and uncomfortable. Jaundice may or may not appear, and there is usually no ascites. Hydatid fremitus, which is a trembling felt by the finger when a superficial cyst is tapped, is characteristic.

Diagnosis.—The diagnosis may sometimes be made from these symptoms, but it may be confirmed by tapping a cyst, and searching for the hooklets of the embryo. Unilocular cysts are most common in children.

Treatment.—Aspiration may cause the cyst walls to collapse temporarily, but the cyst is apt to fill up again with serum; therefore laparotomy and removal of the cyst are recommended. In children rupture of these cysts is especially liable to occur, spreading their contents throughout the peritoneal cavity, and resulting in multiple cyst formation within the abdomen. If possible the cyst should be removed whole; if this can not be done, the cyst wall should be attached to the edge of the incision, and free drainage thus secured. All cases in which there is suppuration should also be freely drained.

DISEASES OF THE PANCREAS.

The pancreas is rarely diseased during childhood, but both acute and chronic pancreatitis are occasionally observed, and tuberculosis and syphilis of the pancreas have been reported.

Acute Pancreatitis.—Acute pancreatitis is most frequently a complication of mumps, the symptom appearing within a week after the onset of the parotitis, and usually subsiding within a week or ten days. There is epigastric pain, with vomiting and diarrhea. On palpating the abdomen tenderness may be perceptible in the epigastrium. This complication of mumps is, as a rule, not serious, and calls for no particular treatment.

Chronic Pancreatitis.—Chronic pancreatitis is, in most instances, a result of syphilis, but may be caused by tuberculosis, or accompany diabetes mellitus. The symptoms are by no means typical, although there is frequently an excess of fat in the stools.

Tuberculosis of the Pancreas.—Tuberculosis of the pancreas occurs less rarely in children than in adults, and is always considered as a secondary affection, which has either extended from adjacent lymph glands, or been carried to the pancreas in the blood stream from more distant parts of the body. It may be either acute or chronic; but the symptoms are not at all characteristic, and the diagnosis is rarely made.

CHAPTER XIV.

DISEASES OF THE RESPIRATORY TRACT.

ANOMALIES.

CONGENITAL absence or rudimentary conditions of the nose are rarely met with, but it is not uncommon for an infant to be born with its nasal passages considerably narrowed or occluded. If the latter defect is bilateral, it must be immediately corrected, or sudden death from asphyxia may result, since a baby never instinctively breathes through its mouth.

In later life, mouth-breathing is injurious because it interferes with Nature's provision for the filtration, warming, and proper moistening of the air during its passage through the nasal cavities, consequently the delicate structures of the pharynx, the larynx, the trachea, the bronchi, and the lungs suffer.

The narrow nasal passage may be still more contracted and respiration greatly obstructed by deformity of the turbinated bones, by a hypertrophied condition of the mucous membrane covering them, or by a deflected septum due to trauma or asymmetrical bony development of the base of the skull or upper maxilla, or to a high V-shaped palate.

Any difficulty in breathing interferes with proper nursing and feeding, and, no matter from what cause, may lead to faulty thoracic development. W. P. Parsons has called attention to the fact that an anterior nasal obstruction produces deformity of the maxilla, and a posterior obstruction causes depression and widening of the bridge of the nose.

DISEASES OF THE NASOPHARYNX.

ACUTE RHINITIS.

This affection, familiarly known as "a cold in the head," is very common in nurslings and throughout infancy. The swollen mucous membrane more or less completely blocks the nasal passages, while the labored breathing induces great fatigue, and interferes with sucking as well as with sleep. Moreover, the inflammation is liable to spread to the conjunctivæ, the nasopharynx, the tonsils, and, through the patulous opening of the Eustachian tube, to the middle ear. It

often gives rise to cough, which is sharp and irritating when the pharynx is affected, and croupy when the larynx is involved. By extension, the inflammation may produce tracheitis, bronchitis, or bronchopneumonia.

Etiology.—The exciting cause is, no doubt, of bacterial origin, the staphylococcus, pneumococcus, streptococcus, and the influenza bacillus having been found in the secretions, as well as the Diplococcus intracellularis.

Infectious diseases, such as measles, scarlet fever, whooping-cough, grippe, and influenza are often preceded or accompanied by coryza; the latter, even when exceedingly mild, may be diphtheritic. In view of these numerous possible inciting causes and in view of the fact that many varieties of bacilli may be and are actually found on the healthy nasal mucous membrane, it is difficult to point to a specific cause and to say definitely which of the microbes are only accidentally present. Under normal conditions the healthy nasal mucosa evidently resists the infection, and it is only when this natural immunity is reduced or destroyed by a depression of the local or the general resistance that rhinitis develops. Some factors that lead to such a lowered resistance are: exposure to cold and dampness, careless bathing, wet feet, the inhalation of irritating fumes or of dry, dust-laden, or superheated air, sudden changes in the weather—especially during the spring and winter months—and certainly malnutrition, anemia, and a generally run-down condition. The specific rhinitis of syphilis and of tuberculosis is closely associated with the symptoms of these diseases and is fully discussed under those subjects.

Symptoms.—The symptoms include the ordinary phenomena of mucomembranous inflammations, sneezing as an early symptom, swelling, redness, and increased secretion. After a period of stuffiness, the nasal discharge becomes profuse and watery, later thicker, mucopurulent, or purulent. It is always purulent in the gonococcic, scarlatinal, and diphtheritic varieties, and often irritates or erodes the nasal openings or the neighboring parts. In older children there is slight if any fever, but should the inflammation spread to the pharynx, the tonsils, or the middle ear, the temperature is high and continuous. The cervical lymph nodes may swell and become painful, with nervousness, sleeplessness, lassitude, and headache as the usual concomitants. Uncomplicated cases in older children run an uneventful course, recovery taking place in three to five days; but in young infants the difficult breathing and eventually the cough interfering with sleep and nutrition, their naturally weak resistance is still more reduced, and the risk of further spread and dangerous complications is increased.

Where there is a purulent or an occasionally blood-tinged discharge accompanied by fever, there is great danger of overlooking the possibility of diphtheria. The disastrous consequences of such an oversight are self-evident. Therefore, as long as a careful bacteriologic examination has not demonstrated the absence of the Klebs-Loeffler

bacillus, the patient should be given the benefit of the doubt and should receive a dose of antitoxin.

Treatment.—Considering the ease with which the infection spreads to the bronchi and the lungs of a delicate infant, thorough and early treatment seems especially important. Much can be done in the way of prophylaxis by providing proper and seasonable clothing—neither too much nor too little, a nutritious, easily digested diet, pure non-irritating air, and by judicious hardening begun in summer with an open-air life and the daily cold sponge. Coddling, the use of chest protectors, overheating the room or the bed during the colder season, should be avoided, and exposure to infection by contact with persons suffering with catarrh or colds carefully guarded against.

Active treatment in severe cases will naturally depend upon the condition of the patient and the character of the attack. As a rule children should be kept in the house in well-ventilated rooms, at a temperature of 60° to 70° F. A simple cathartic—castor oil or calomel and salts—causes depletion, rids the gastro-intestinal canal of the swallowed secretion, and prevents auto-intoxication from the bowels. Mild diaphoretics, fresh warm air, moistened occasionally with a spray, warm drinks of water or lemonade are useful and agreeable to the patient. During the early stage, $\frac{1}{8}$ to $\frac{1}{16}$ of a grain of sodium chloride crystals in each nostril relieves congestion by causing a copious discharge; for older children, we recommend an alkaline spray—saline, Dobell's solution, or one containing 1 per cent. of the chloride, bicarbonate, and biborate of sodium combined—to clear away the abnormal secretion, to be followed by a simple protective, such as camphor, gr. 1, menthol, gr. 1, liq. petrol. ℥ j, applied to the mucous membrane. In children under three years this is best done by instilling a few drops into each nostril several times a day. Vaseline or any mild ointment will protect the lips and nostrils from excoriation. Adrenalin, 1 to 1000 solution, is recommended where there is much obstruction from an acutely congested mucous membrane, but cocain hydrochloride, gr. 3, to the ounce, added to any of the above prescriptions will prove almost equally efficacious. In severe cases, painting or spraying with 25 per cent. argyrol after irrigation with warm saline, proves of value if done early enough. Generally speaking, little can be done once the secretion has become profuse, but should it continue so for any length of time, astringents are indicated—a 2 per cent. solution of formalin, although painful, often gives very good results. Kyle recommends fluidextract hamamelis 1 fluidram, fluidextract hydrastis, $\frac{1}{2}$ dram, water enough to make 2 ounces, as a cleansing solution and an antiseptic, a few drops to be instilled into each nostril morning and night.

A run-down condition, anemia, or malnutrition, predisposes the child to infection of any kind and should be treated. Repeated attacks of rhinitis may often be traced to carelessness in bathing the infant; or they may be caused by the presence of adenoids or of a foreign body, which, interfering with nasal drainage, produces a certain amount of irritation and congestion. No permanent results can, of course, be

expected unless the cause be removed. The treatment of the snuffles of syphilis, the coryza of tuberculosis, hay-fever, diphtheria, measles, etc., is discussed under these respective diseases.

CHRONIC RHINITIS.

This rather uncommon affection in infants and young children is usually spoken of either as hypertrophic or atrophic, depending upon the thickness of the nasal mucous membrane. Many observers consider the latter to be merely the natural consequence—the retrogressive stage—of the preceding hyperplasia. In fact, the Schneiderian membrane may show patches of both conditions in the same patient.

Hypertrophic rhinitis is characterized by a cushion-like swelling of the mucous membrane lining the nasal cavity, especially of the inferior turbinated bones. It rarely follows the acute forms of coryza, but accompanies scrofula and chronically enlarged tonsils and adenoids. The patients grow better and again worse with every change from warm to cold weather.

Symptoms.—There is usually some mucopurulent inoffensive discharge which gathers particularly in the inferior meatus and often flows backward into the pharynx causing a teasing cough or necessitating frequent clearing of the throat. Any slight exposure, or the onset of the cold season increases the trouble. The nose, alternately one or the other side, seems occluded; at all events, the swollen turbinals, together with the usually coexisting adenoids, cause mouth-breathing, especially at night. This in turn diminishes the appetite, disturbs the sleep, and creates a tendency to catarrh of the pharynx and the lower air tubes. A nasal voice, vertigo, migraine, unilateral headache, and ear trouble are perhaps the most frequent accompanying symptoms.

Treatment.—In addition to what has been said about treatment of the acute form, it is necessary to ascertain and remove the cause that directly or indirectly is affecting the secretions or the circulation. Adenoids or foreign bodies should be removed, and deformities of the nasal structure corrected by operation. Among other causative factors in children from ten to fifteen years of age are: engorgement of the turbinals at puberty, or occasionally mental excitement, sudden changes in temperature, automobiling, irritant vapors, and gastro-intestinal disturbances. In the scrofulous child the nasal condition will improve with the use of tonics, malt, cod-liver oil, iron, and nourishing food—while mountain or sea air will prove an additional advantage. The improvement of the general health—and the proper régime—often does more for recovery than local remedies, but the latter must frequently supplement the former.

For the very young child it is best to avoid nasal douches and inspection, but for older children a cleansing solution may be used—Dobell's, half strength—to be followed by a stimulating or astringent lotion, according to the special requirements of the case.

ATROPHIC RHINITIS.

In atrophic rhinitis, the nasal mucosa is thin and pale, and the cylindrical cell lining, especially of the inferior turbinals, undergoes retrogressive squamous epithelial proliferation. The air-passages of the nose thus become dilated, the secretion diminishes and changes its character, and a tenacious, greenish-yellow discharge, liable to form crusts, clings to the walls of the nasal cavities, where it decomposes and emits a very strong and peculiar fetor. Fortunately for the victim, his sense of smell is usually lost. An accompanying chronic, dry pharyngitis frequently adds to the distress of the patient.

As to the etiology, we lack definite knowledge. It seems certain that it is not due to syphilis although a specific microörganism may possibly be a causative factor, while some connection with tuberculosis has lately been emphasized. The disease occurs in families, preferably in girls, but rarely before the age of ten or twelve years.

Treatment.—When the mucous membrane is merely atrophied, it can be restored to normal, but the outlook is less hopeful when actual degeneration has taken place. The general health, especially an accompanying anemia, or any indication of scrofulosis, should be given prompt treatment. Locally, mild, antiseptic alkaline douches, or an oily solution, or cotton tampons saturated with a 3 per cent. hydrogen peroxide are useful in removing the crusts and stimulating secretion. Gentle massage of the mucous membrane with a cotton-tipped probe seems beneficial.

PURULENT RHINITIS.

A short time after birth a thick, yellowish pus is occasionally seen exuding from both nostrils of a baby whose mother has suffered from a purulent vaginal discharge at the time of delivery. Such an infection, usually limited to the anterior nasal cavities, can be avoided by proper care. Once it has occurred, hydrogen peroxide, 15 vol., or a mild alkaline antiseptic, should be used for thorough cleansing followed by an astringent lotion applied by atomizer or swab.

EPISTAXIS.

Nose-bleeding is rare in very young infants except in cases of sepsis or of syphilis. During childhood, however, epistaxis is quite common, apparently more so in boys, though girls are likely to suffer from it at puberty. The cause is not always obvious, but local and general factors together with mere delicacy of the tissues are responsible for most of the cases. Local causes include inflammatory changes, the presence of foreign bodies, tuberculosis, syphilis, erosions of the septal mucous membrane, and, more especially, trauma from falls, blows, or picking of the nose. Epistaxis may occur in general infections, such as typhoid fever, malaria, measles, influenza, scarlet fever,

whooping-cough, and nasal diphtheria; in diseases of the blood, such as hemophilia, grave forms of anemia, purpura, and scurvy; and in conditions that produce passive congestion, such as valvular heart affections, Bright's disease, adenoids, croupous pneumonia. Mental or physical excitement, or the slightest injury to the mucous membrane, may also excite epistaxis. It usually proceeds from a vessel in the anterior part of the septum, but occasionally the lesion is near the posterior nares. The blood then trickles down the throat, and may be either coughed up, arousing a suspicion of hemoptysis, or it may pass on into the stomach and be vomited up, or discharged through the rectum.

Treatment.—Any restriction around the neck should at once be loosened. Except in case of an underlying constitutional derangement the nasal hemorrhage will often stop spontaneously without treatment. However, cold applications over the nose, the forehead, or the nape of the neck can do no harm; while more efficient treatment consists of outside pressure, digital compression of the facial artery, the introduction of dry cotton compresses to the bleeding point, or the application of collodion to the bleeding area. Should any of these, or a simple astringent—a 5 per. cent. solution of zinc and copper sulphate and lead acetate—not suffice, a solution of adrenalin, 1 to 10,000, injected with a soft-rubber syringe, or applied on gauze strips over the bleeding point after the removal of the blood-clots, will prove effective. For the permanent relief of ulcers or erosions, usually found upon the anterior part of the septum, the parts should be thoroughly cleansed and dried, and touched once or twice with a 50 per cent. silver nitrate solution, or a 15 per cent. solution of chromic acid. All other means failing, the anterior and posterior nares may be packed with cotton, either plain or medicated, with 1 to 1000 adrenalin, hydrogen peroxide full strength, or 8 per cent. antipyrin. This packing should be applied with a moderate degree of firmness but should not be allowed to remain in longer than twenty-four to forty-eight hours in order to prevent a possible devitalization of the mucous membrane. Tincture of ergot and calcium lactate may be given but they are usually not very efficacious.

ADENOIDS.

Childhood, especially between the fourth and eighth years, is characterized by a peculiar liability to excessive development of lymphoid tissue, chiefly at the upper crossing of the respiratory and alimentary passages. There it forms the so-called "ring of Waldeyer," which in as many as 75 per cent. of all children who live in moist climates or near the seashore is prone to increase to tumor-like formations in three places—the pharyngeal and the two faucial tonsils. The fourth or lingual tonsil does not, as a rule, develop before puberty. The adenoid, also called Luschka's or the pharyngeal tonsil, is a reticular structure filled with lymph cells rich in bloodvessels, covered by

several layers of columnar epithelium, and differing from the faucial tonsils by the absence of follicles. It may be enlarged at any age, even in nurslings, especially in idiots; it is soft and friable in infants, and hard or fibrous in older children and adults. While the importance of this diseased condition in children is generally recognized, it does not seem to be sufficiently appreciated in infants, at least so far as efficient treatment is concerned. In some instances, no doubt, it is not merely a local condition, but coexists with congenital syphilis or a tuberculous constitution and gives rise to symptoms similar to those of syphilitic coryza; in fact, enlarged adenoid growth is, perhaps, the chief cause of chronic snuffles.

It is somewhat astonishing to find that a purely local condition may produce so many symptoms; their severity, of course, depends largely upon the size of the adenoid and the presence or absence of complicating inflammatory processes. That these symptoms need not necessarily be present in each individual case, and that the clinical picture of adenoids in a child of six to eight years differs considerably from that of the baby, is self-evident. It may not be superfluous, however, to point out the fallacy of believing an infant has no adenoids because it keeps its mouth closed. · If we bear in mind that at birth the posterior choana are just large enough to admit a medium-sized male catheter, and that the nasopharynx is very shallow indeed, $\frac{1}{4}$ to $\frac{1}{3}$ of an inch, we can readily see how even an apparently small adenoid growth may considerably constrict or practically obstruct the nasal passages. Such interference with normal breathing leads to all forms of disturbances, already discussed under the subject of rhinitis, and gives rise to chronic snuffles, often to mouth-breathing, especially at night, and its sequelæ—difficulty in feeding, dryness of the mouth, the habit of snoring, so distorting to the features, and a constant tendency to cold in the head. Sleep is interrupted and unrefreshing because the little one continually tosses about seeking to find a position in which he may be able to breathe more freely,·and because an irritating cough usually appears upon lying down. Attacks of pavor nocturnus are common; excited, no doubt, by the respiratory difficulty. Anyone who has been compelled to sleep for a night with his mouth open does not easily forget the feeling of exhaustion, the bad taste, and the want of appetite experienced on awakening, and will readily understand why such children have no relish for breakfast. Unable to masticate for any greater length of time than the period during which they can hold their breath, they eat only enough to satisfy the acute pangs of hunger and naturally bolt their food; through impeded respiration they inhale an amount of oxygen quite insufficient for intensive metabolism, and they get their sleep only by snatches. It is not astonishing then that the general nutrition suffers, with a consequent reduction in the power of resistance. This, in addition to a more or less pronounced catarrh of the nose, the nasopharynx, the tonsils, and the Eustachian tubes, accounts for the increased liability of these children to all forms of tonsillitis, to rheu-

matism, tuberculosis, scarlet fever, diphtheria, measles, etc., and the greater severity of their symptoms if attacked by any of these diseases.

The speech is peculiarly thick. In exceptional cases stuttering and stammering are reported to have been cured by adenectomy. The hearing also is often impaired; for the narrowing or obstruction of the Eustachian tube interferes with the proper ventilation of the middle ear, and thus causes retraction of the drum-head. Other disorders observed are those of taste and smell, irregular dental development, a high palatal arch, flat chest, diminished lung expansion, and, where there is a tendency to rickets, deformities of the bony thorax. While there is as yet no positive proof that adenoids cause asthma, chorea, or epilepsy, except in the presence of inherited neuropathic tendencies, yet it is a fact that enuresis has in some cases been cured by the removal of large adenoid vegetations. J. R. Clemens has recently pointed out that children with hypertrophied tonsils and adenoids show excessive general perspiration while asleep, but not during their waking hours. This abnormal sweating might well be mistaken for an early symptom of rickets were it not that it disappears after surgical removal of the excessive lymphatic growth.

Aprosexia, indifference to and diminished capacity for mental work, especially noticeable in school children with adenoid disease, is no doubt partly due to defective hearing which arises from the rarefaction of the air in the middle ear; there is, however, a probability that enlarged adenoids pressing upon the posterior and lateral walls of the nasal pharynx interfere with the return flow of lymph or blood from the brain, and cause mental disturbance by reflex action. Children with enlarged adenoids often run a slight temperature for days at a time, and, as already stated, a stubborn mucopurulent rhinitis is a common complaint in such cases. The discharge becomes more or less blocked up in the nose forming a favorable nidus for bacteria, and the resulting inflammation manifests itself by enlargement of the cervical lymph nodes and glands. The infection may reach the middle ear through the Eustachian tube and produce an otitis media; it may also spread downward and involve the pharynx, the larynx, the bronchi, and the lungs. A tenacious secretion collecting in the pharynx may be the cause of a spasmodic cough, retching, or vomiting, and may simulate either bronchitis or the early stage of whooping-cough. Many a long-standing case of so-called bronchitis disappears as if by magic on the surgical removal of previously unsuspected adenoids.

Treatment.—The lymphoid tissue is most labile and, in mild cases, the enlargement may possibly disappear under judicious topical applications of iodin and glycerin combined with treatment of the coexisting rhinitis, respiratory gymnastics, and last, but not least, a rational diet appropriate to the age and the digestive capacity of the patient. Large adenoids, however, which cause pronounced symptoms, cannot be thus melted away, and no time should be lost in resorting to the necessary surgical measures.

When should an operation be undertaken? As soon as the condition causes any noteworthy symptoms. With an infant it may be wise to wait until the fifth or sixth month of life. To delay longer simply because the adenoid vegetations may recur does not seem justifiable, especially in view of their deleterious influence upon development during this period of very active growth. No definite rule, however, can be laid down for cases in which the hypertrophy is only slight and the symptoms apparently absent; we say apparently, because close observation often reveals manifestations which had escaped attention before.

If spring or early summer can be chosen for the operation, the patient will have more time in which to recuperate before the onset of cold weather. The long-standing cases operated upon late in the fall may again exhibit the old susceptibility to colds during the winter that follows. While an acute inflammation, of course, necessitates a postponement of operative interference, this need not be delayed on account of a slight rise in temperature—100° to 101° F.—if it is due to the presence of the adenoid itself. In fact, this temperature usually disappears after the operation. Perhaps no operation is so often inefficiently done. Whenever possible, it should be left to the skilled hands of the specialist.

As a rule, after two years of age, the hyperplasia involves all the tonsils, pharyngeal as well as faucial; these should, therefore, not be treated as separate conditions. About 75 per cent. of all children above two years of age that have enlarged adenoids also have enlarged tonsils. The essential point is complete extirpation, though the details of the procedure itself may vary.

Since infants and weakly children cannot well endure hemorrhage, and since chronic cases are liable to bleed profusely, some practitioners give calcium lactate or chloride in 10-grain doses for two days prior to operation, the last dose being given on the morning of the operation. It is a safe principle to give a cathartic the night before and, of course, to withhold all food on the day of the operation, which should be performed in the early morning if possible. Infants under one year of age require no anesthetic for adenectomy. There is very little shock, and the whole procedure is over before the babe has had time to be seriously frightened. An assistant, holding the little patient with its arms alongside the body securely wrapped in a sheet, presses his right hand against its arm and his left hand against its forehead in such a way as to incline the head against his left shoulder. Perhaps a better plan is to have a nurse seated with the child on her lap, with its arms crossed and firmly held. An assistant standing back of the chair steadies the infant's head between his hands and at the same time keeps the mouth gag in place. The operator then presses down the tongue, passes the chosen instrument (a Gottstein or a Kirstein curette, or a Shutz adenectome) behind the soft palate, brings the blade forward as far as possible, and then, by making a stroke at first backward and then downward, removes the adenoid

in one piece. Any remaining particles can be quickly cleared out
with the forceps. The patient's head is then held forward over a
basin during the profuse bleeding which usually follows but which
lasts only a few minutes. If the hemorrhage is severe, it can easily
be checked by hot or cold water locally applied, or by any form of
cold application to the face. The child is put to bed on its side with-
out a pillow, and should be carefully watched for one or two nights
by a nurse, with instructions to use a 1 to 5000 adrenalin chloride solu-
tion as a spray in case of secondary hemorrhage.

DISEASES OF THE LARYNX.

ACUTE LARYNGITIS (FALSE CROUP—SPASMODIC CROUP).

Acute laryngitis, an inflammation of the laryngeal mucous mem-
brane, is also popularly known as false croup, or spasmodic croup,
because in young children of a certain predisposition it is often asso-
ciated with spasm of the glottis which gives rise to alarming symptoms.
When associated with the acute infectious diseases, especially measles,
acute laryngitis may become very serious. The occurrence of false
croup with influenza, whooping-cough, and scarlet fever, is discussed
under these respective headings. Acute laryngitis proper is nearly
always of microbic origin. While it may appear as a primary disease,
it is usually secondary to an infection spreading downward from the
upper respiratory passages, such as rhinitis, tonsillitis, or catarrh
of the nasal pharynx; in rare instances the pathological process may
extend upward from the bronchi and the trachea. It is a well-known
fact that bacteria capable of producing inflammation are found on
the normal mucous membrane, which has the power to destroy them.
Their pathogenicity may be due either to an enormous increase in
numbers, or to a lowered local resistance, the result of congestion
from one cause or another, or to an impaired general condition due to
some chronic ailment. Children under six years of age suffering from
rickets, anemia, malnutrition, chronically enlarged tonsils and
adenoids, or those having an acquired or an inherited spasmophilia,
are particularly liable to have serious as well as frequent attacks of
laryngitis associated with spasm of the glottis, one attack apparently
creating a tendency to another. In children without this predisposi-
tion or in those over six years of age, the disease is comparatively mild
in type and devoid of spasmodic manifestations.

Pathology.—The mucous membrane of the larynx, and often to a
slighter degree that of the trachea also, shows localized or general
hyperemia and a swelling, which seems limited to the mucosa in a
mild case, but which may involve the submucosa in the more severe
one. There is, however, very little tendency to edema in children as

compared with adults. The initial dryness is soon followed by an increased secretion of mucus which, clinging to the swollen mucosa, still further narrows the lumen of the windpipe, and interferes with the free passage of air. This at times produces a marked stenosis which may become almost complete, causing a spasm of the laryngeal muscles, especially of the adductors.

Symptoms.—In older children the ordinary case lasts from four to ten days. It is commonly preceded by nasal or pharyngeal catarrh, or it may be associated with influenza, measles, or typhoid. The clinical picture of simple infantile laryngitis closely resembles the adult type, except that the cough is more croupy and the dyspnea more marked. All the symptoms—slight fever, cough, dyspnea, and hoarseness—become more pronounced at night. Usually there is pain and tenderness over the larynx. Alarming symptoms are seen principally in infants and young children, for even a moderate swelling of the laryngeal mucous membrane in a very young child interferes with inspiration and may readily cause an obstruction, especially if there is a tendency to spasms, so usual in infants. In mild cases the hoarseness, the cough, and a high temperature, 100° to 103° F., may continue for a few days, and then pass off without more severe manifestations, but other and more alarming symptoms frequently make their appearance. After having played around all day apparently well except, perhaps, for a slight coryza, the child toward evening develops a slight hoarseness and a barking cough, which so increase in severity and frequency that sleep becomes restless. There is no evidence of serious illness, the temperature rise being only slightly above the normal, but respiration gradually becomes increasingly difficult, until suddenly the child awakens terrified, sits up, apparently suffocating, and struggles for breath. The alæ nasi vibrate rapidly, all the accessory respiratory muscles are brought into play, the lips and the finger tips become livid, and the labored breathing with its loud inspiratory stridor is only interrupted by a loud, ringing, metallic cough, which is sometimes hoarse and harsh. The distress may last from a few minutes to one-half or a full hour, often longer, but in a somewhat milder form, until finally the little patient falls back exhausted to continue his interrupted sleep. When he awakes the next morning the dyspnea has completely disappeared, and he is apparently as bright and well as ever except for a hoarse voice and some cough. However, unless adequate treatment is given, the cough, the hoarseness, and the dyspnea are liable to return at nightfall and to culminate in a second attack, perhaps even more severe than the first one. Usually these symptoms do not recur more than three nights in succession, but after exposure or other indiscretions some children suffer from slight laryngeal spasms every few weeks during the cold season.

Treatment.—Even mild cases must be treated with care on account of the possible development of spasms or of edema. The child should be purged with calomel given in divided doses, and should be put to

bed in a warm (70° F.), well-ventilated room.   Inhalations of steam, plain, or medicated with tincture of benzoin comp., oil of eucalyptus or creosote, repeated in severe cases every two hours for fifteen minutes, are very soothing to the inflamed mucous membrane.   If the adjacent structures, the faucial or lingual tonsils, or the naso-pharynx are inflamed they must be energetically treated.   Hot drinks, lemonade, but better still milk with lime-water, help to start the secretion; when the latter grows profuse, expectorants are indicated.

A teaspoonful of the syrup or the wine of ipecac relieves the spasm and the cough for a time by causing vomiting of the accumulated secretions, but such medication cannot be advised for weakened children.   In some cases antipyrin in small doses—1 grain for a child of two years—every two hours, may be necessary for relieving the spasm, and syrup of ipecac and squills, of each 5 drops, for soothing the irritating cough, while some exceptional cases may require heroin, $\frac{1}{70}$ grain, or Dover's powders in small doses.

When in connection with the dyspnea, the stridor, and the respiratory recession, there is marked frequency of respiration with extreme lividity or pallor and prostration, it may become necessary, especially in a very young child, to perform intubation or even tracheotomy. It goes without saying that a good prophylactic dose of diphtheria antitoxin should be given in every doubtful case.   During convalescence little treatment will be required beyond the use of the syrup of hydriodic acid and ferrous oxide as a tonic, and protecting the child against exposure.

Prophylaxis.—Proper clothing, an out-of-door life, a daily cold sponge followed by brisk friction, a simple nourishing diet supplemented by tonics—malt extract and cod-liver oil—these together with an annual stay at the seashore or the mountains, when possible, will go far to prevent the recurrence of croup; the predisposed child, however, must not be subjected to extreme changes of temperature, or to exposure to wet, and should be kept from contact with persons suffering from colds.

EDEMA OF THE LARYNX (GLOTTIS) AND THE SUB-MUCOUS MEMBRANE.

These conditions, both of them rare, may represent either a serous infiltration or an inflammatory edema of the larynx.

Serous Infiltration.—A true infiltration of the submucous cellular tissues of the larynx occasionally occurs in chronic nephritis; in fact it may be one of the earliest symptoms of the disease.   Other etiological factors may be glands or tumors which interfere with the normal laryngeal circulation, cardiac insufficiency, and local irritation, such as is produced by inhaling hot or irritating vapors, and by accidental swallowing of corrosive liquids.   In rare cases, the administration of potassium iodide has been known to produce a sudden edema, which disappeared readily when the drug was discontinued.

Inflammatory Edema.—Inflammatory edema, the result of a local inflammatory process, is more common. This is a true submucous laryngitis, which may be caused by direct infection, by trauma of the mucous membrane, by ulcers, impacted foreign bodies, or by an infection spreading from the surrounding tissues; it also occurs as a rare complication in syphilis, typhoid fever, smallpox, chicken-pox, scarlet fever, and diphtheria. The edema usually affects the epiglottis and the aryepiglottic folds, but it rarely attacks the vocal cords and interarytenoid folds; when associated with constitutional diseases, the swelling is apt to be bilateral.

Symptoms.—While expiration may be quite easy, inspiratory dyspnea is the most striking symptom of edema of the glottis. When the aryepiglottic folds are chiefly involved, the laryngeal orifice may become practically closed, producing most alarming dyspnea and signs of suffocation. Edema of the other parts of the larynx is not likely to embarrass the respiration to the same extent. The suddenness of the onset depends greatly upon the exciting cause, but, as a rule, the distressing symptoms develop with great rapidity and may prove fatal within a few hours. To this clinical picture may be added pain, cough, hoarseness, and dysphagia, if the condition is of inflammatory origin; and, naturally, also all the symptoms of a coexisting primary disease.

Diagnosis.—The examining finger, duly protected, detects a swelling around the base of the tongue and a more or less pronounced tumefaction at either side of it. When the tongue is drawn forward during inspection, or, better, when the laryngeal mirror is introduced, tumors are seen close together near the root of the tongue, their whitish color indicating a serous origin, or, if reddish, an inflammatory origin; in the latter case there is also tenderness over the larynx and the trachea. Careful examination of the heart and of the urine should never be omitted unless the history and the accompanying signs and symptoms leave no doubt as to the true cause of the condition.

Prognosis.—Prognosis depends upon the patient's general condition, the underlying cause, and the promptness with which the dyspnea can be relieved by medical means, or, in severe cases, by surgical measures.

Treatment.—While the ultimate success depends upon ascertaining the etiological factor and treating it effectively, local treatment at the time of the attack is of paramount importance. When there is no danger of suffocation, cold compresses, externally, and ice by mouth are useful. Topical applications of adrenalin may also be tried. In the inflammatory form leeches over the larynx, multiple punctures of the edematous tissues, deep incision where an abscess is suspected, each has its advocates. When the dyspnea is extreme, Heubner recommends intubation, followed, if necessary, by tracheotomy and subsequent local treatment. At all events, one should be prepared to perform tracheotomy if suffocation seems imminent.

LARYNGISMUS STRIDULUS (CHILD-CROWING).

This disease, peculiar to infancy and comparatively rare except in foundling institutions, is no doubt a neurosis, causing an incoördination of the laryngeal muscles.

Etiology.—The majority of cases occur chiefly in male infants from six to eighteen months of age, and usually during the winter months, when babies are more or less closely confined to overheated rooms. In children suffering from rickets, tetany, hereditary syphilis, or other diseases that produce profound malnutrition and excessive excitability of the nervous system, attacks of laryngismus stridulus are easily induced by nervous excitement, or by almost any kind of peripheral stimulation. A breath of cold air blowing across the sensitive skin of such a delicate neurotic child, or the reflex action of certain nasal conditions, or enlarged cervical or bronchial glands, the acute inflammation of adenoids and tonsils, postnasal dropping of mucus into the larynx, a tickling of the throat, or some gastric disturbance, as well as fits of temper, of fright, and of crying are sufficient to cause a spasm of the respiratory muscles, especially of the laryngeal abductors, resulting in sudden closure of the glottis and the partial or complete shutting off of air from the lungs.

Symptoms.—While the affection when mild may pass unnoticed in the beginning, on the other hand, the very first attack may awaken the child from its sleep and startle the family by its suddenness and severity, the latter depending almost entirely upon the degree of spasm. Just before the glottis closes the inrushing air makes a peculiar noise which frightens the child and causes it to struggle for breath; its chest remains fixed and its body stiffens, while the epigastrium bulges and feels as hard as a board. Respiration ceases, although the diaphragm appears to move convulsively, the face turns first pale, then livid. With its head thrown back and its eyeballs rolling, the life of the little patient seems to be in imminent danger. After some anxious moments, a sudden deep inspiration, usually but not always accompanied by a crowing sound, shows that the spasm is broken, and the danger past. The child breathes rapidly, its terror dissolves in tears, and after fretting and crying a little, it finally falls asleep, and on awaking seems as well as ever.

Although these attacks usually come on at night, and awaken the child out of a deep sleep, they may appear at any time, and may be repeated two or three times a day; in severe cases even as many as twelve attacks have occurred in twenty-four hours. The mild seizures pass off in ten to twenty-five seconds, leaving apparently no ill effects; but the more severe ones may be followed by general convulsions, loss of consciousness, and may even terminate fatally.

Carpopedal spasm is observed in about 50 per cent. of all cases, and, when present, becomes intensified during the attack. Laryngismus stridulus may last for a few days or for several months, recovery being

heralded by a gradual decline in the severity and the number of the paroxysms until they finally cease entirely.

Diagnosis.—The differential diagnosis is easy if the clinical picture and the history are kept in mind, especially the fact that the patient is comfortable during the intervals. In laryngismus stridulus a period of apnea precedes the crowing inspiration, while the whoop of pertussis immediately follows a series of short expirations. Catarrhal laryngeal spasm is always associated with symptoms of acute laryngitis; congenital laryngeal stridor dates from birth; in diphtheritic croup the dyspnea is lasting and increases in severity; spells of so-called "holding of the breath" are usually brought on by anger.

Prognosis.—The prognosis is good when the underlying cause can be treated successfully, and also favorable as far as the attack itself is concerned; although weak infants are known to have died from asphyxia or from subsequent convulsions.

Treatment.—During an attack the primary object is, of course, to break the spasm and to produce an inspiration. For this purpose, inverting the patient and lightly slapping him on the back often proves as effective as it is simple. Dashing cold water on the face and the chest, tickling the fauces, alternate hot and cold baths in rapid succession, a hot sponge over the larynx, or a whiff of chloroform administered by a competent person, have all been found useful. But, if these means should fail to give prompt relief, intubation or tracheotomy becomes necessary.

Following the paroxysm the child should be kept under the influence of chloral for twenty-four hours, and for a week or two sodium bromide should be given, 5 grains three times a day in cinnamon water. Considering that many of these cases suffer from disturbances of nutrition, antipyrin, 1 grain, may be given every four hours to a child one year old; antipyrin does not disturb the digestion as do the bromides, unless the bromides are given by rectum. The infant must be kept from all excitement, such as loud talking, the noisy play of other children, as well as the devoted attentions of relatives and friends. Of course, the underlying malnutrition, rickets, tetany, syphillis, tuberculosis, or the disease of the nose or throat should be treated in the most effectual way.

Congenital Laryngeal Stridor.—This is an inspiratory stridor peculiar to infants, but of rare occurrence, usually noticed very shortly after birth and disappearing spontaneously during the second year. Hutchinson calls the condition "a stammer of respiration." It may be due to incoördination of the laryngeal muscles or it may represent a neurosis like laryngismus stridulus. Some writers suggest a malformation of the larynx or, rather, a peculiar folding of the epiglottis upon itself along the median line and a flabbiness of the false vocal cords, which allow the larynx to fall upon itself on inspiration, thus producing a valve-like action.

Etiology.—As regards the etiology, we know little beyond the fact that the affection is congenital and that it may be increased by excitement as well as by exposure to cold.

Symptoms.—While expiration to all appearances is easy and noiseless, an inspiratory stridor in the form of a curious slight crowing or purring in the throat is heard. This sound varies in intensity and may at times cease altogether; it is rarely audible during sleep, but is apt to be louder when the child becomes excited. The voice remains unaffected, and, in spite of a slight cyanosis which accompanies the dyspnea, the child does not seem uncomfortable. The paroxysms of dyspnea usually increase in severity during the first few months of life and gradually subside in the course of the second year.

Diagnosis.—The disorder is readily differentiated from laryngeal spasm associated with enlarged adenoids, laryngismus stridulus, thymic asthma, papilloma and edema of the larynx, by its history, its onset immediately after birth, by its characteristic respiration accompanied by inspiratory dyspnea, and by the fact that the patient is not uncomfortable and the voice remains unaffected.

Prognosis.—Prognosis is good unless complicated by bronchopneumonia.

Treatment.—The condition has a tendency to disappear spontaneously, and therefore requires no special treatment; but the child should be protected from nervous excitement and undue exposure, especially to diseases of the respiratory tract. In infants these have a decided tendency to spread to the larynx and the bronchi; in such cases every preparation for performing tracheotomy or intubation should be made in time. Pure air, sunshine, careful feeding, and the usual hygienic measures for improving the general condition will help to shorten the course and to modify the severity of the disease.

NEW GROWTHS OF THE LARYNX.

Laryngeal tumors rarely occur in children and only exceptionally are they malignant. Of the benign growths, papillomata represent the most common variety; fibromata and myxomata are too rare to require consideration. Papillomata are probably congenital in about 25 per cent. of all cases, the majority of them making their appearance from the first to the fourth year, sometimes developing rapidly after laryngeal catarrh. Boys seem to be more frequently affected than girls. The growths, usually situated on the vocal cords or within them, may be single or multiple, sessile or pedunculated, and sometimes may attain the size of a large raspberry.

Symptoms.—The symptoms naturally depend to a certain extent upon the size and the location of the tumor, but they usually resemble those of chronic laryngitis. A change of voice, a gradually increasing hoarseness and occasional paroxysms of coughing are the early manifestations which may, indeed, date from birth, but which more infrequently appear during infancy and are progressive instead of passing off as in ordinary laryngitis. The increasing size of the growth eventually causes a certain degree of obstruction, the breathing becomes somewhat difficult, perhaps at first more noticeable during sleep, but

later also in the daytime, especially when the child exerts itself in any way. Usually no pain is experienced on swallowing and thus months may elapse before the slowly progressing symptoms, accentuated, perhaps, by repeated severe paroxysms of coughing or of suffocative attacks, compel serious attention.

Diagnosis.—In all cases of long-standing stubborn laryngitis, a papilloma should be suspected, but a positive diagnosis can, of course, only be made by the use of the laryngoscope.

Prognosis.—Papillomata sometimes disappear spontaneously if the larynx is given absolute rest for a few months. Surgical removal is followed by immediate and pronounced relief, but there is always a possibility of recurrence; indeed, removal seems to stimulate the growth of subsequent crops.

Treatment.—Treatment should be entrusted to a skilful laryngologist, who, if the child is tractable, may decide either on the endolaryngeal method of removal with the forceps or wire snare. Occasionally thyrotomy or laryngofissure with curettage may be resorted to. The latter, however, greatly endangers the quality of the voice and, aside from the difficulty of anesthetizing the patient, there is the risk of postoperative bronchial pneumonia. To prevent recurrence the long-continued use of Fowler's solution has been recommended as one of the best prophylactic measures.

FOREIGN BODIES IN THE LARYNX, THE TRACHEA, AND THE BRONCHI.

Particles of food, buttons, pins, tacks, seeds, pebbles, bullets, and coins, in fact, all objects which a child playfully puts into its mouth, can, if small enough, be aspirated into the larynx or the trachea during an attempt at crying or laughing, or a sudden inspiration occasioned by an attack of coughing, hiccoughing, or choking. The resultant symptoms will naturally depend upon the size, the shape, and the nature of the object swallowed and the age of the child; for even very small bodies may cause dangerous symptoms in the infant owing to the small size of its larynx. As a violent fit of coughing and of suffocative attacks usually follows the accident, this frequently helps to expel the foreign body or to make it slip along the trachea into one of the bronchi, usually on the right side, owing to local anatomical conditions. Sharp objects like fish bones, pins, shells, etc., instead of becoming dislodged often become imbedded in the larynx and produce edema or occlusion of the glottis, which may result in sudden death unless immediate relief is given. Or they may penetrate so deeply into the soft tissue thay they cannot easily be seen or felt, so that either blood-stained sputum, a spasm of the glottis, severe inflammation, edema, perichondritis, or an erosion of the bloodvessels may result. The severe lesions finally may lead to a certain degree of permanent stenosis owing to cicatrices that form during the process of healing. The object which has passed down the trachea may cause a great variety of other symptoms besides the paroxysmal cough,

the bloody sputum, and the localized pain in the chest. A whole lung or part of it may become atelectatic, depending on the size of the bronchus occluded, with absence of respiration and diminished resonance. Sometimes the inspired body acts as a valve and permits only inspiration, thus causing a rapidly developing emphysema with all its concomitant physical signs. If the foreign body is not removed it excites a local inflammation which, spreading to the surrounding lung tissue, may result in the development of pulmonary abscesses. These in turn may produce a high irregular septic fever that may end fatally with symptoms that somewhat resemble pulmonary tuberculosis.

Diagnosis.—The diagnosis can usually be made from the history of "something having been swallowed the wrong way," the suddenness of the attack and the violence of the early symptoms. Digital examination is somewhat dangerous, but Roentgen rays are a great help in many cases. Older children can be quieted down sufficiently for an examination by the laryngoscope or the bronchoscope, but this may prove rather difficult with a young child. It may be mentioned, in passing, that a lateral pharyngitis may be responsible for the sensation of a foreign body in the throat. On the other hand, a troublesome cough with some physical signs of bronchitis or of pneumonia in a young child that presents increased respiratory movements on one side, and diminished vesicular murmur on the other, will suggest the possibility of an obstructed bronchus.

Prognosis.—The prognosis is rather uncertain, much depending upon the nature of the foreign body and its location; for even when situated below the larynx it is by no means impossible for it to be expelled spontaneously. Suffocation is the most frequent cause of a fatal termination, especially when the object is large enough completely to obstruct the laryngeal opening.

Treatment.—Nothing should be done that may cause a sudden inspiration; emetics are therefore not advisable; nor should an instrument or the finger be rashly introduced into the larynx, while care should be taken not to push the obstructing body down into the trachea. Inversion of the patient, in the hope of thus assisting Nature's effort to expel the offending particles by coughing, may be tried. Immediate tracheotomy may become necessary, but intubation is contra-indicated. Urgent cases admit of no delay, and a skilled laryngologist should be called immediately since only a specialist should undertake to remove a foreign body from the trachea or the bronchi.

DISEASES OF THE BRONCHI.

ACUTE BRONCHITIS.

Acute bronchitis is an inflammation of the mucous membrane of the trachea and the bronchi with a tendency, more pronounced in infants, to involve the small bronchi as well.

Etiology.—It is often observed during the course of infectious diseases, especially measles, scarlet fever, pertussis, or epidemic grippe, but the essential etiological factor of simple bronchitis is, no doubt, an inflammation caused by the single or combined action of the staphylococcus, pneumococcus, influenza bacillus and Bacillus catarrhalis, or by the bacilli of typhoid and of diphtheria. Since the normal mucous membrane destroys the majority of invading germs, it is to be assumed that something diminishes this self-protective power and thus makes it easy for pathogenic bacteria to invade the deeper regions of the respiratory apparatus and there to multiply. In some children, especially fat, flabby, and pasty-looking ones, there may be an individual predisposition of the mucous membrane itself; this theory would at least explain why apparently the same cause will produce a simple catarrh in one child, and a diffuse bronchitis or an intestinal disturbance in another child of the same family.

Very young children (between three and six months of age) show a marked susceptibility which, however, rapidly decreases in the fourth year. Lowered vitality from any cause naturally weakens the local resistance in children even more so than in adults; hence, mouth-breathing, chronic nasopharyngeal affections, glandular and other forms of tuberculosis, anemia, syphilis, and similar conditions, favor the development of bronchitis. Lack of hygiene, uncleanliness, sunless dwellings, want of care (constant dorsal position), undue exposure either during the daily bath of the infant or by insufficient clothing, not to speak of bare legs during inclement weather (favored by a barbarous custom of endeavoring to "harden" the child), wet feet, raw winds, sudden atmospheric changes, are all frequently responsible for bronchial attacks. While it is true that a sudden chilling of the body surface may be harmful, on the other hand we can not too strongly condemn the excessive anxiety which, during the cold months, keeps children confined in close, overheated, ill-ventilated rooms, in school or at home, and compels them to breathe impure, germ-laden air.

Pathology.—Our concern here is not with a localized bronchitis secondary to pulmonary disease, but with an *acute* catarrhal process. This is usually bilateral, rarely appears in patches, and primarily affects the mucous membrane of the trachea and the bronchi. The mucous membrane is injected and swollen, its glands and its goblet cells are markedly increased in number and pour out an excess of secretion, which soon changes from serous to mucopurulent, and contains desquamated epithelium, many bacteria, and leukocytes. In acute attacks the lymph nodes at the root of the lung are slightly swollen, the swelling being more marked in protracted or recurrent attacks. The mucosa and submucosa are infiltrated with small round cells and pathological bacteria. The inflammation is limited to the bronchial walls. An extension to the peribronchial tissues would be bronchopneumonia, as is also the so-called capillary bronchitis of children which, postmortem, shows changes in the air vesicles surrounding the bronchioles. In *chronic* bronchitis the mucous mem-

brane is thickened and of a brownish-red color, while a certain amount of emphysema, dilatation of the smaller bronchi, and some degree of peribronchitis are quite frequently found, especially in infants.

Physical Signs.—Auscultation may be negative at first, but with increasing secretion bronchial fremitus is present. Percussion is also of little assistance except for the early detection of a possible pneumonia. Sometimes there is some hyperresonance due to a transitory emphysema over the border of the lungs.

Auscultation is usually of little value as long as the catarrh is dry and is limited to the trachea and the large bronchi, but sibilant, whistling rales appear early in the disease and are replaced within twelve to twenty-four hours by moist, bubbling rales; these are medium or coarse according to the size of the bronchi involved. They are most distinct between the scapula and in the intraclavicular spaces but can be heard all over the chest, changing frequently, and appearing and disappearing with the paroxysms of coughing. The vesicular murmur is feeble, expiration is often prolonged and rather harsh over the supra- and intrascapular region, simulating bronchial breathing.

The base of both lungs should be carefully and frequently examined for fine, crepitant rales, which would indicate an incipient pneumonia. The loud, wheezing sounds sometimes persist for two or three weeks following the acute stage. The physical signs sometimes very closely resemble those of asthma, especially in the presence of spasmodic or of asthmatic elements.

Symptoms.—The *milder form*, in which the affection is limited to the larger tubes, is very common indeed. It is not serious in older children, but in infants there is always danger of a complicating capillary bronchitis or bronchopneumonia. When a catarrh of the upper air passages extends to the bronchi—as in some children is the case after almost every coryza—it manifests itself by a cough, by slightly accelerated breathing, and by a further rise of temperature. It is usually the paroxysmal, tight, ineffectual, annoying cough which attracts attention; worse at night at first, it becomes looser and less irritating within a few days. The respirations, often accompanied by rattling in the late stage, may be accelerated to 40 or 50 per minute. The temperature, irregular in character, runs from 100° to 102° F. for two or three days, then sinks below 100° F., and usually returns to the normal within a week. There is no marked constitutional disturbance; in fact, there is very little danger except in young and delicate infants, older children being hardly sick enough for bed after the first or second day. There is, however, a moderate degree of restlessness, depending upon the severity of the cough, some substernal soreness in older children, also gastro-intestinal symptoms with vomiting and diarrhea in the very young patient.

Children under five years of age do not expectorate unless they are "experienced coughers," but they swallow the secretion as it is coughed up. In older patients the expectoration consists of a white, frothy

mucus during the first few days, which later on becomes more abundant and of a yellowish-green tinge.

In the *more severe types* of simple bronchitis all the symptoms are more pronounced, due to the involvement of the smaller tubes. The onset may be gradual, following a rhinitis or a pharyngitis, with prodromal fatigue, malaise and headache, but it is often abrupt, accompanied by chills, or by convulsions in infants, and by fever, pain in the head, the back and the chest, and a dry cough closely resembling that of incipient pneumonia. The cough, the most prominent symptom, is absent only in weak infants, and is of the same character as in the milder cases, except that it is considerably more severe and more troublesome, and at times, is associated with pain or even vomiting. It not infrequently lasts some time after all the physical signs have disappeared. In the early stages the expectoration is occasionally blood-streaked and later becomes profuse and mucopurulent, causing a persistent cough, especially in the morning on awakening.

The respirations are hurried, perhaps somewhat labored, though ordinarily there is no evidence of real dyspnea. But, as the inflammatory process extends deeper into the bronchial tree, respiration becomes more rapid and superficial. In children 40 or 50 respirations per minute are not necessarily serious, but with an increase to 60 or 80, accompanied by flaring of the alæ nasi and participation of the accessory respiratory muscles, together with inspiratory retraction, marked cyanosis and prostration, and unduly prolonged expiration, a bronchopneumonia, or so-called capillary bronchitis, may be apprehended. Weak and very young infants are the only exceptions to this rule, inasmuch as with them dyspnea and recession of the soft parts occur even in an uncomplicated bronchitis. Respiratory failure and suffocative attacks may develop quite suddenly, especially in infants under six months of age, who are unable to empty their tubes of secretion by coughing or by crying. The irregular, superficial breathing and the clammy, cyanotic skin produce dulness, apathy, and stupor, and unless promptly relieved convulsions and death may follow within a few hours. The temperature is not characteristic; during the first few days it runs up to 102° or 104° F., but if no complications arise it gradually falls to normal within a week. The constitutional symptoms abate with the fall of temperature and seldom cause anxiety except for the first twenty-four to thirty-six hours. Inspection of the bared chest shows nothing beyond rapid breathing and slight inspiratory retraction of the soft parts. When exaggerated (except in very young or weak infants), the latter sign, together with increasing dyspnea and cyanosis, becomes an important danger signal of incipient bronchopneumonia.

Prophylaxis.—All infants and children, especially those suffering from syphilis, rickets, and nutritional disturbances, should be carefully kept from close contact with persons afflicted with colds, rhinitis, tonsillitis, grippe, etc. If in spite of all precautions they become affected with catarrh of the upper respiratory passages, treatment

should be prompt and careful, and should be continued until convalescence is completely established, for tuberculosis may develop in the inflamed bronchial glands. Adenoids and diseased tonsils should be removed when necessary, preferably during the warm season of the year.

Treatment.—While fresh air is of the greatest importance, the advantage of inhaling a great deal of cold air has not yet been demonstrated. The sleeping apartments of susceptible children should be well ventilated but the temperature should not register less than 60° F. if possible. During cold weather the clothing should be sufficiently warm but not cumbrous, and the house should be kept at as equable a temperature as possible. A warm dry climate (sometimes mild sea-air does equally well) is a boon during the winter months, especially for the child with a tendency to pulmonary disease or one with tubercular antecedents. Generally speaking, the mild case requires little treatment beyond careful nursing and feeding, while the more severe type must be treated as bronchopneumonia. As long as there is fever, the patient should be confined to bed in a bright, large room, preferably with an open fireplace, to ensure thorough ventilation, and a screen as a protection against draughts. To prevent hypostatic congestion, delicate infants must be frequently taken up, or, at least, their position must be changed. They should be kept warm, though this does not mean bundling them up in blankets or warm shawls, as is frequently done.

The Diet.—The nursing infant has a much better chance of recovery than the bottle-fed baby. If its temperature rises above 100° F., and the little one is restless and irritable, it may be advisable to shorten the time of nursing to one-third or one-half the usual time, and give a few ounces of sweetened water before nursing. For bottle-fed babies it is well to reduce the strength of the milk formula and to give plain boiled or slightly alkaline water between feedings. Older children, while confined to bed, may be allowed a light diet consisting of toast, milk, cocoa, broths, gruels, and fruit juices.

While pure air, the importance of which has already been emphasized, usually contains enough moisture, inhalations of medicated steam for half an hour at a time and repeated at intervals of several hours are of great service in the treatment of acute bronchitis. Creosote, 10 drops to a quart of water, seems the best, but benzoin and eucalyptus have also proved useful. A tent may be improvised with sheets spread over the top and sides of the child's bed, and the nozzle of the croup-kettle, from which the steam flows, introduced within at a safe distance from the face and head of the child. Fresh air is admitted from time to time by raising the sheet.

During the first few days counter-irritation affords one of the most efficacious means of bringing the blood to the surface and thereby relieving pulmonary congestion. A paste made of one part of mustard and three parts of flour mixed with warm water and the white of an egg, and spread one-eighth of an inch thick on a piece of old linen,

cheesecloth, or muslin, is applied to the chest between linen cloths and left in place until the skin is well reddened. After its removal, vaseline, or, better, talcum powder gently applied will relieve the burning sensation. The hot mustard bath, $\frac{1}{2}$ ounce of mustard to 6 gallons of water at a temperature of 107° to 110° F., is especially useful when the rapid respirations and the cold extremities indicate an impending bronchopneumonia. The hot mustard pack (a sheet wrung out of hot mustard water) sometimes proves equally useful. These procedures can be repeated every four to eight hours; should the skin become very sensitive, the proportion of mustard may be reduced.

An old remedy of great value in some cases is dry-cupping over the chest and the back from five to ten minutes at a time; this can be repeated every few hours if necessary.

Drugs.—The treatment of bronchitis by drugs is entirely symptomatic and unimportant. Of course, an initial purge of castor oil, repeated every three to four days, is good in helping to relieve the intestinal canal of the swallowed mucopurulent materials. During the first stage, characterized by a dry cough and rough breathing, castor oil and syrup of ipecac, 2 to 3 drops each for infants, and 4 to 5 drops for children three years old, repeated every two to three hours for the first three days, will help to liquefy the secretions and lower the fever. It would be injudicious to suppress the cough; but, if very distressing, the following simple prescription may add to the patient's comfort:

```
℞—Pot. brom. .  .   .   .   .   .   .   .   .   .   .   .   .   .   .   gr. l
    Tinct. bellad.  .   .   .   .   .   .   .   .   .   .   .   .   .   ♏ xv
    Glycer. .  .   .   .   .   .   .   .   .   .   .   .   .   .   .   f℥ ij
    Elix. lact. peps.  .   .   .   .   .   .   .   .   .   .  q. s.  ℥ij
    One f℥ every three hours for a child one year old.
```

For older children, tinctura opii camphorata, 1 to 2 fluidrams, may be added. Or a tablet or a powder may be given as follows:

	At age of six months.	At age of one to three years.	After three years.
℞—Tartar emetic	$\frac{1}{150}$	$\frac{1}{100}$	$\frac{1}{80}$
Powdered ipecac	$\frac{1}{40}$	$\frac{1}{40}$	$\frac{1}{20}$
Ammon. chlor.	$\frac{1}{2}$	$\frac{1}{2}$	1
For a severe cough add Dover's powder	$\frac{1}{8}$	$\frac{1}{4}$	$\frac{1}{2}$

Two-hour intervals, eight doses in twenty-four hours.

Antipyretics are hardly ever needed, as the temperature can be controlled by warm tub or sponge baths, which have the additional advantage of quieting the nervous system and stimulating the action of the skin. Emetics, $\frac{1}{2}$ to 1 dram of either the wine or the syrup of ipecac, were formerly used extensively for clearing out the tubes, but they are very depressing, and only in exceptional cases should they be given to children under two years of age. Guaiacol in a 10 per cent. ointment is easily absorbed by the delicate skin of an infant, and when so used does not cause any digestive disturbance. Quinine, still believed by many to be anticatarrhal, can be given by rectum in suppositories.

DISEASES OF THE LUNGS.

PNEUMONIA.

In early life the lungs, more frequently than any other organs, are attacked by acute or subacute inflammation, either as a primary affection or secondary to acute infections. The pathology and symptoms of the inflammatory process vary according to the pathogenic organisms present and the extent of the lesions. The two leading types are: (1) lobar, fibrinous or croupous pneumonia, a sharply circumscribed, inflammatory process involving an entire lobe or the greater portion of it, without affecting the walls of the bronchioles and the alveolar septa. Both the onset and the termination are rather sudden, the disease running its course within a few days; (2) the lobular type—bronchopneumonia—involving the walls of the bronchi as well as the individual lobules of the lungs, and forming small irregularly distributed areas of consolidation. The exudate is composed mainly of cells with very little fibrin. The duration and course of this type are more or less indefinite and the termination takes place by lysis. In typical cases, these two types are quite distinct, but intermediate forms may occur, especially during the second and third years of life, more or less resembling both types.

Lobar Pneumonia.—This form, though rare in infants, is not at all uncommon after the first year. It is an acute infectious disease, running a well-defined course, and differing in no essential details from the adult type. In typical cases pneumococci (diplococci pneumoniæ of Fränkel or of Weichselbaum) are often found in the blood and in great numbers in the lungs, either as pure cultures, or predominating over the associated streptococci and staphylococci.

Pneumococci are normally present in the saliva and on the mucous membrane of the respiratory passages of healthy children; it is, therefore, self-evident that they incite disease only when the local or the general powers of resistance are lowered. Traumatic and other predisposing influences naturally also play an etiological role; a blow on the chest, a fall, fatigue, the inhalation of irritating vapors, and, above all, undue exposure to raw winds and inclement weather, are no doubt responsible for precipitating many an attack. Fully two-thirds of all cases occur during the winter and early spring months, especially after a sudden fall of temperature accompanied by wind and rain that follows a period of depressing heat—conditions that account for lobar pneumonia being spoken of as a "house disease." The disorder occasionally follows grippe, typhoid fever, or whooping-cough. As a rule it affects children previously healthy; that is to say, if a healthy child contracts pneumonia it is usually of the lobar type. As in adults, repeated attacks within a year may be attributed to a family tendency to the disease. Boys are more often affected than girls, probably because they are more likely to be exposed to weather conditions.

Morbid Anatomy.—The anatomical changes, seldom seen postmortem on account of the low mortality of the disease in children, closely resemble those of the adult type, except that the line of demarcation is less well defined, because the pneumococcus may produce either pneumonia or bronchopneumonia or both at one time, together with a more or less pronounced pleurisy. Generally an entire lobe or the great part of it is affected, but several lobes, both apices, or a whole lung may be involved.

During the first stage—that of congestion—which may last from a few hours to several days, the epithelial lining of the alveoli becomes turbid and swollen owing to the rich fibrinous exudate that pours into them. The second stage is that of red hepatization, so called because the affected portion of the lung resembles the liver, being red, heavy, tough, and granular on section due to fibrin blocks in the alveoli. It may last anywhere from a few days to one or two weeks. This is followed by a shorter third stage—that of gray hepatization. The lung then appears bulky, soft, yellow and bloodless, with costal imprints. The contents of the alveoli gradually disappear by absorption rather than by expectoration. Resolution sets in with the fall of the temperature or soon after, and is complete in about a week. In addition to these pulmonary changes the mucous membrane of the neighboring bronchi as well as of the pleura shows signs of inflammation. Pathological changes, due to complications, such as fibrinous or purulent pericarditis, especially in left-sided pneumonia with pleurisy, or, more rarely, pneumococcic meningitis, peritonitis, arthritis, may become manifest before, during, or after the attack of pneumonia.

Physical Signs.—The little patient is apt to be restless and irritable, and examination is not always easy. Nevertheless it should be repeated at frequent intervals, because consolidation with all its concomitant signs may appear within a few hours after a negative finding. Sometimes the only indications in an otherwise typical attack are a brief impairment of resonance and a few rales, and again flushed cheeks, herpes of the lips and the nose, short, rapid, superficial respiration, the flaring alæ nasi, and possibly a deficient expansion of the affected side may give a clue as to the correct diagnosis, when for days and even up to the crisis the characteristic physical signs are delayed. Usually, however, as in the adult type, the three stages of congestion, consolidation, and resolution also occur in children.

Percussion.—Percussion of the bases is, of course, a routine procedure, but it is very important not to omit an examination high up in the axillary and the infraclavicular regions. Impaired resonance or slight dulness may often be elicited over the affected area, and a somewhat increased resonance over the rest of the lung, according to the diminished or increased amount of air entering the lungs. While dulness is likely to be absent in the infant, tympany may be obtained even over a consolidated lung when it overlies a distended viscus, the stomach, for instance. The tactile vocal fremitus is of little diagnostic

value in children, as it would be altogether absent over the area of an occluded bronchus on account of their thin, high-pitched voices.

Auscultation.—The vesicular murmur is very early diminished over the affected portion, and breathing over the opposite lung becomes harsher. This puerile respiration, due to vicarious emphysema, must not be mistaken for bronchial breathing, which, as is well known, lacks the vesicular element and is more intensive on expiration. With the inverted type of breathing, so often present in pneumonia, it may, however, give the impression of being louder on inspiration than on expiration. Bronchial breathing and bronchophony over a sharply defined consolidated portion are most valuable signs; they may often be heard as early as the second day in basal pneumonia, and by the fourth or fifth day are usually audible over the spine of the scapula in apical cases.

In order to bring out these signs more clearly it may be necessary to cause deep breathing by inducing coughing or crying, or by having the child lie on one side while examining the other, thus allowing the air to enter more freely. In this way a high-pitched, tubular breathing and crepitation may be revealed which at once clinch the diagnosis. This crepitation is best heard at the end of inspiration, either as a crepitus indux disappearing with consolidation, or as a crepitus redux reappearing with resolution; but medium or coarse rales are much more common in children. Very often when one lung is affected, these signs are also audible over the other side. This is merely due to transmission; the faint character of the abnormal sounds as well as the absence of impaired resonance should be sufficient to prevent a mistaken diagnosis of double pneumonia. Occlusion of a bronchus may produce such complete absence of all breath sounds as to suggest the presence of a fluid. Rales and a pleuritic friction rub are usually absent during the stage of consolidation, while during resolution the signs of consolidation disappear gradually. The tubular quality of the expirations and all kinds of moist rales are gradually replaced by a vesicular murmur, and friction sounds are also sometimes heard. The physical signs clear up with remarkable rapidity, as a rule within a week, but occasionally a slightly diminished resonance and a feeble vesicular murmur may persist for several weeks.

Symptoms.—By the second or third day herpes of the lips or the nose usually appears, the tongue shows a whitish or yellowish coat, the temperature and respiration continue high and rapid, and the bowels are usually constipated. About this time the physical signs also set in, although they may be delayed for a few days. Toward the seventh day all the symptoms are aggravated and alarming, perhaps with severe delirium, when the crisis occurs with its sudden change. The fever abates, the body becomes moist with a warm perspiration, the cough grows looser, respiration less rapid, and after a quiet sleep the child awakens refreshed, with a slow pulse and often a subnormal temperature. Unless the inflammatory process spreads to another lobe or a complication arises, the tongue clears and the appetite

returns. The child now wants to sit up in bed, and at the end of the second week recovery seems established except, perhaps, for a few rales and a slightly impaired resonance over the affected area.

In children from seven to eight years of age the clinical picture closely resembles that of the adult, but younger patients differ in the ease with which the immature organism, especially the nervous system, is upset. The constitutional symptoms seem to depend not so much on the extent or the seat of the local affection as on the toxemia produced. This may be meningeal, gastro-intestinal, migratory, or nephritic. As in other infectious diseases, abortive cases are occasionally observed. At the onset these present all the signs of the first stage of pneumonia, but clear up so quickly that even the keenest observer may well doubt the correctness of his diagnosis. Some mild but indubitable cases may last only three or four days, but typical lobar pneumonia in childhood runs a definite course of at least five to nine days. After a few hours, sometimes even days, of prodromal lassitude, discomfort, or a slight cold, profuse and sometimes repeated vomiting suddenly takes place. This may be accompanied by diarrhea, chilly sensations, epistaxis, or even convulsions. The temperature rises sharply to 103° or 104° F., the child looks ill, appears heavy and dull, refuses food, and is easily persuaded to go to bed. It may complain of headache or of earache, and also of severe abdominal pain suggesting appendicitis; sharp costal pain, however, is a rare symptom in children under six years of age. The next day the fever continues high, the cheeks are flushed, the skin is burning hot, the eyes sparkle, the alæ nasi vibrate; the child, though willing to stay in bed, is rather restless; and during the night it may be drowsy or delirious, breathing rapidly, with a short expiratory moan or grunt. The short, dry cough, if present, is often very slight at first, though occasionally it may be so painful as to induce crying, but it is not followed by expectoration. Whatever sputum can be obtained with a swab shows the typical, characteristic, tenaciousness and rusty color.

The facial expression appears neither anxious nor distressed in spite of the rapid respiration. The cheeks are not always flushed—in fact, sometimes there is marked pallor. Herpes of the lips and the nose is frequently absent in infants under one year of age. As a rule, there is no true dyspnea, the accessory muscles remain inactive, but the breathing is shallow and mainly abdominal, sometimes irregular, and always disproportionally rapid (40 to 60 or more respirations per minute) compared to the pulse rate and the temperature. The cardiorespiratory ratio is, however, not so significant as in adults owing to the ease with which the pulse in children is accelerated for any slight cause. An expiratory grunt or moan with the pause after inspiration is often observed. The range of temperature is irregular in infants, oscillating one to three degrees or more, but in robust and older children it often shows the adult type—sudden rise to 103° to 105° F., with slight diurnal variations, finally ending by crisis on the fifth, seventh or ninth day, unless the inflammatory process spreads or

27

complications arise. In such cases it may not end before the four-teenth day, and then by lysis.

Pseudocrises are a good omen, and are much more common in chil-dren than in adults. The inflammation of the lung itself does not seem to be the direct cause of the fever because the temperature often reaches its maximum and falls before there is any evidence of change in the lungs.

The Pulse.—The pulse, at first full, bounding, and slightly accel-erated, becomes small, rapid, irregular, and of low tension, as the toxemia increases; this together with the cyanosis and the venous pulsation indicating a weakened heart.

Gastro-intestinal Symptoms.—Besides the furred tongue, the failing appetite, the diarrhea or constipation, jaundice and vomiting may be early symptoms. Meteorism, or abdominal distention may last throughout the course of the attack and are particularly distressing because they embarrass cardiac and pulmonary action.

Cerebral Symptoms.—These seem to be dependent more on the degree of fever and the virulence of infection than upon the par-ticular portion of the lung affected. They may vary from slight wandering to active delirium with incontinence of the feces and the urine. In infants and young children unable to formulate their complaints, restlessness, muscular twitching, and a retraction of the head may sometimes be so severe as to simulate cerebrospinal menin-gitis, while again restlessness, apathy, and, later, drowsiness are the only cerebral manifestations.

The Crisis.—The crisis usually occurs between the fifth and the eighth day of the disease, or earlier in exceptional cases, though some-times it sets in as late as the ninth or tenth day. Should the fever continue beyond that time it is likely to end by lysis. After the criti-cal fall the temperature may be subnormal for a few days, and although the danger of collapse in children is less than in the adult, the patient should be watched with special care during this time.

The Blood.—From the time of the first chill the leukocytes increase rapidly up to 40,000 or 70,000. The count returns to the normal number a few days after the crisis, but it may persist high for weeks in complicated cases or when resolution is delayed. While a low leuko-cytosis indicates reduced resistance and augurs ill, a high white cell count is not necessarily a favorable prognostic sign. Pneumococci are found in the blood cultures in about 50 per cent. of all cases even before the appearance of the characteristic physical signs in the lungs.

The Urine.—The urine, as in all fevers, is scant and dark, with a high specific gravity and increased acidity, and contains casts and a trace of albumin. The chlorides are reduced, or even absent, but reappear at the time of the crisis, when the total quantity of urine is also greatly increased.

Diagnosis.—Diagnosis is clear in the typical case; but in the atypical case, the late appearance of the characteristic physical signs and the great variety of general symptoms may puzzle the physician for several

days, unless the diagnosis is definitely established by a bacteriological examination of the sputum.

The most common error is to mistake pneumonia for some other disease rather than the reverse. Considering how frequently this mistake is made in children, a continuously high temperature, with rapid respirations and marked leukocytosis, should arouse a suspicion of pneumonia, no matter what the other symptoms may be. A careful search for the physical signs high up in the axilla may often be of use in establishing the diagnosis.

The onset of scarlet fever, tonsillitis, gastro-intestinal disturbances, and malaria may resemble that of pneumonia; but the appearance of the characteristic rash on the second day in scarlet fever, the aggravation of local symptoms in tonsillitis, the rapid decline of fever, as well as the improvement in the constitutional symptoms produced by laxatives and proper feeding in gastro-enteritis, and, finally, the fact that chills followed by a sharp rise in temperature and marked leukocytosis are rarely seen in malaria, make the differentiation comparatively easy. On the other hand, it is often impossible during the first few days clearly to distinguish a bronchitis with high fever from pneumonia. A rapid course with severe dyspnea and cyanosis without corresponding physical signs in the lungs, especially in the infant, may suggest miliary tuberculosis until the subsequent course of the disease, the temperature range, and an examination of the sputum, together with the history of the onset, make the diagnosis clear. The constitutional symptoms of influenza, if the catarrh of the conjunctiva and the upper air passages is not marked, may at first simulate pneumonia, but the appearance of marked leukocytosis and of the physical signs, as well as the course of the disease and the bacteriological findings in the sputum, soon dispel all doubt. Initial vomiting, abdominal pain, tenderness, and distention, especially when associated with slight resistance on the right side, so strongly suggest appendicitis, that operation has actually been resorted to in such cases.

It is therefore important to make a careful examination in every case. The sudden rise of temperature to 103° or 104° F., the continuous type of the fever, the disproportionately rapid respirations, the relaxation of the abdominal walls between respirations, the tenderness diminishing or disappearing on deep pressure with the flat hand, and the possible cough, will put the careful observer on his guard. Pneumonia is sometimes masked by intense cerebral symptoms—vomiting, convulsions, delirium or stupor, rigidity of the muscles of the neck and opisthotonos—and may be mistaken for cerebrospinal meningitis; in the absence of the physical signs in the lungs, it may be several days before a definite diagnosis can be made. The difference, however, may be noted in the absence of the slow, intermittent pulse and the slow irregular respirations as well as of the dilated pupils of meningitis and the signs of paralysis. Another differentiating factor is the presence of the nervous symptoms at the onset of pneumonia, while in meningitis they come on so slowly that

by the time the resemblance between the two ailments is closest the physical signs in the lungs will have made their appearance.

When typhoid fever with a sudden onset and high temperature is associated with symptoms of pulmonary involvement, as is the case with children, the diagnosis may be doubtful until the presence of leukopenia, or of roseola, or a diazo, or a positive Widal reaction leave no room for doubt. The physical signs of atypical cases of pleural exudation may so closely resemble lobar pneumonia that the diagnosis can be made only by exploratory puncture. In pleurisy the area of dulness shifts somewhat with a change of position, and is usually greater, while the flatness is more marked behind and below, and gradually increases instead of disappearing with the decline of temperature. The greater resistance noticed on percussion over the intercostal spaces, their markedly unilateral fulness or bulging, the limitations of respiratory excursion, the displacement of the heart and perhaps the liver, and, finally, egophony, when present, all favor effusion. While the bronchial voice is distant and the intensity of the respiratory sounds is diminished in the majority of young patients, they are not altogether abolished as in the adult. The bronchial breathing is feeble and most marked at the upper border of dulness; while in pneumonia it is louder and most pronounced in the area of maximum dulness.

Prognosis.—Unless complications arise, pneumonia is rarely fatal in children, especially after the second year (mortality being 2 to 5 per cent.); although death may occur from otitis, meningitis, cerebral abscess, pericarditis, pleurisy, or toxemia—the toxins acting upon the vasomotor centres in the medulla and on the cardiac muscle fibers. As a rule, the heart stands the strain well, but fatal syncope has been known to occur during convalescence. In girls and undernourished children resistance is diminished; while rickets, heart lesions, and previous lung affections distinctly reduce the chances of recovery. Convulsions are comparatively rare in children over two years of age; but should they occur toward the end of the disease a fatal issue may be expected. Neither a violent onset nor a high temperature, except when over 106° F., nor the severity of the initial nervous symptoms has much prognostic value.

Treatment.—A large, sunny, quiet, well-heated sick-room, with an abundance of fresh air flowing in through windows, open both day and night, and an intelligent nurse who can be relied upon tactfully to keep out visitors, and to watch the child with special care at the time of the crisis, are important in the general management of the case. The hours for feeding and medication should be so arranged that the patient can enjoy intervals of three to four hours of perfect rest. Milk, whey, meat juice, albumen-water, fruit juices and broths, will give the required amount of nutriment and fluid. If given at proper times, they will not overtax the weakened digestion, manifested in most cases by anorexia, vomiting, and diarrhea. While a very weak patient may require gavage feeding, overfeeding should be

avoided, as it is apt to induce gastro-intestinal disturbances, fermentation, and abdominal distention, which may considerably increase the respiratory difficulty. The well-nourished child when attacked by pneumonia can, in the beginning, very well go without food for one or two days provided it is given plenty of water.

If an ice-bag is used over the chest, the feet must be kept warm; the bag should be removed whenever the temperature falls below 100° F., and not be replaced until it has again risen to 102° F. However, a child usually dislikes extremely cold applications, and as their superiority has not been established warmth in some form seems preferable. This is applied by wringing a piece of flannel out of hot water, wrapping it around the thorax, and covering it snugly with several layers of flannel. The procedure can be repeated at intervals of a few hours without disturbing the child. Poultices are cumbersome and are rarely of any benefit. If applied over the thorax they, by their weight, tend to increase the respiratory difficulty, although this disadvantage is obviated by allowing the child to lie upon the poultice. They are occasionally useful in relieving pain of an associated pleurisy. A mustard paste—left in place only long enough to produce redness but no blistering—and intermittent stupes will relieve the pain and combat pulmonary congestion by reflex action; although dry cups applied over the base of the lungs are more efficacious.

Notwithstanding the fact that lobar pneumonia is a self-limited disease with a strong tendency to recovery, and that we know of no specific for it, and that overmedication certainly may do harm, there is no doubt that skilful treatment not infrequently saves life and always relieves the suffering of the little patient. In the ordinary, uncomplicated case an initial cathartic is indicated; this may consist of one or more teaspoonfuls of castor oil, or, if nausea and vomiting prohibit this, of small doses of calomel followed by citrate of magnesia. The bowels should be kept open throughout the entire course of the disease, especially in the very young, in order to prevent abdominal distention. A simple mixture of camphorated water containing potassium citrate and sweet spirits of nitre, given every three to six hours, may be of advantage in getting rid of the sputum that may have been swallowed. Expectorants such as ammonia, ipecac, or squills can usually be dispensed with as they seem to be a frequent cause of gastro-intestinal disturbance. Alcohol has lost much of its former popularity, being now chiefly reserved as a stimulant during the crisis; but weakly, undernourished children need it from the onset. Children under two years of age should be given 20 to 30 drops, sweetened and diluted in 6 to 8 parts of water, three times a day; it is especially indicated when the toxemia and the prostration are marked. Respiratory and circulatory stimulants may be needed, especially during or directly after the crisis, the most valuable being strychnine, gr. $\frac{1}{300}$ and atropine, gr. $\frac{1}{400}$, aromatic spirits of ammonia, gtt. x, camphor monobromate, grain $\frac{1}{2}$, caffein citrate, gr. $\frac{1}{4}$, and soda benzoate or salicylate in 3-grain doses. These doses are suitable

to a child five years of age and may be repeated every three hours as long as may be necessary. Oxygen inhalations do good service when there is cyanosis with very rapid respirations.

Other symptoms are treated as they arise. Convulsions can be controlled by chloral hydrate or sodium bromide administered by mouth or by rectum; for sleeplessness, the same drugs, with the addition of cold applications to the head are preferable to opiates. A dry, irritating cough, which does not yield to wine of ipecac, syrup of tolu, or tincture of belladonna, may call for small doses of heroin, paregoric, or codein. Fever exceeding 105° F., demands hydrothera-peutic measures, which if judiciously applied not only reduce the temperature but also quiet the nervous system and stimulate nutri-tion. All children do not react equally to baths; as a rule, tepid or moderately cool water is the choice, but a cool pack, or a sponge with alcohol and water may be better in certain cases. The coal tar derivatives are, of course, out of place as antipyretics, but for the relief of pain, headache, nervousness, and sleeplessness in children over two years of age, phenacetin in small doses is certainly helpful and is much used. When the nervous symptoms are pronounced, repeated warm baths at a temperature of 95° F. will usually prove efficacious. The child should not be immersed above the lower part of the ribs when sitting in the tub. In addition to this, for a child of two, a sedative, 4 to 5 grains of bromide with 1 minim of tincture of belladonna in elixir of lactated pepsin, will be beneficial. Opiates, besides being dangerous, are rarely necessary; although codeine, $\frac{1}{10}$ grain, or camphorated tincture of opium, 5 to 20 drops, is occasionally permissible for children three to five years of age, in order to relieve the excessive pain produced by the cough or the pleurisy.

During the last few years, laboratory methods of treatment have gained some prominence. While leukocyte extract has not as yet been tried in a sufficient number of cases to allow of any definite con-clusions, it appears that there is a fairly constant reduction of tempera-ture after two to four injections. As for pneumococcus serum, Rowland G. Freeman regards a dosage of 100 to 150 c.c. a safe method for combating pneumonia in children. Very soon after the injection the majority of them seem to brighten up, the appetite and color appear improved, while the temperature is reduced, sometimes markedly so. Although the condition of the lung itself does not change much, as a rule, the average duration of the disease as well as the mortality, are slightly lowered. The reports as to the value of vaccine, especially the autogenous, treatment continue to be contradictory. Morton Illman gives 400 to 500 millions of pneumococcic stock vaccine in every case of lobar pneumonia as soon as it has been diagnosed, and follows it with a second, slightly smaller, injection in two or three days. In some of the cases there is a prompt return to normal tem-perature and a marked amelioration of all the toxic symptoms; in others, a pseudocrisis takes place with a subsequent slight rise of temperature ending within twenty-four to forty-eight hours by lysis.

Finally, there are cases which to all appearances are not favorably influenced and which terminate in the usual way. When streptococci or the bacilli of Friedländer are found in cultures of the sputum, it is, of course, necessary to add proportionate amounts of these to the vaccine in order to obtain good results.

Convalescence.—A few days after the crisis, when the physical signs are clearing up, the child may be allowed out of bed, and after the lapse of a week may be taken out of doors, provided the weather be favorable and there is no danger of complications. During convalescence, warm clothes, a carefully selected, nourishing diet, and plenty of fresh air are ordinarily all that is needed. If the general improvement does not progress steadily, some tonic may be required, such as malt extract and iron, the syrup of ferrous iodide, or the like.

BRONCHOPNEUMONIA.

The pathological condition described under this name is really a syndrome rather than a clinical entity *per se*, following more or less closely upon an infection of the upper air passages, or arising in the course of certain infectious diseases. Next to gastro-intestinal disturbances, it is the most common and the most serious trouble of infancy; its characteristic feature being an inflammation of the respiratory passages which extends to the minute bronchi and adjacent alveoli. The fact that the latter are not fully developed before the third or fourth year may partially explain the striking susceptibility of infants to this affection. While rare during the first six months, about 50 per cent. of all cases occur during the first year, 30 per cent. during the second, and, approximately, 10 to 15 per cent. in the third year of life. It does not often appear as a primary disease after the fourth year, but it may occur throughout childhood as a complication of the acute infectious diseases.

In only about one-third of all cases of bronchopneumonia is the disease primary; as such it is most commonly due to the pneumococcus, and in older children it probably manifests itself as lobar pneumonia. In the remaining two-thirds, nearly always due to mixed infection, it is secondary to simple bronchitis, measles, whooping-cough, or to influenza, and in delicate and in older children to scarlet fever or to diphtheria, especially in the laryngeal form after intubation or tracheotomy. No doubt a good many of these cases may be traced to the attitude of well-meaning but uninformed parents who, in their fear of colds, keep their children confined in close, overheated and poorly ventilated rooms.

Poorly nourished, rachitic, or syphilitic children, especially when in institutions, are particularly predisposed to the disease, and should, therefore, be guarded from undue exposure during the winter months, in which bronchitis and infectious diseases are prevalent. Bronchopneumonia is probably always due to the action of pneumococci but the bacilli of Friedländer, and the influenza bacilli, and more rarely

those of diphtheria may be found in all possible combinations of predominance. Its severity is, however, not so much dependent on the particular organism as on the soil in which the organism flourishes.

Pathology.—In older children, typical lobar and bronchopneumonia may at times be quite as distinctly differentiated as in adults, although there are cases, especially in children under four years of age, which cannot be definitely classified even with the aid of a microscopic examination. Again, both types may be found in different parts of the lungs in the same patient.

Bilateral bronchopneumonia affects the posterior portion of the lower lobe about five times as frequently as it does other parts. The inflammatory process spreads through the walls of the bronchioles to the surrounding tissues, which become markedly infiltrated with lymphocytes. Pus oozes from the bronchioles on pressure, and their adjacent alveoli are more or less distended with epithelial débris as well as with white and perhaps some red blood corpuscles, serum, and a small amount of fibrin. At first these areas of peribronchitis are small—the size of a pea or smaller—and are surrounded by normal or gray emphysematous lung tissue appearing in the surface as purplish foci; later they become yellowish-gray and may increase to the size of a nut, or by coalescence they may sometimes affect the greater part of the whole lobe. The bronchial and tracheal lymph glands are also swollen.

Symptoms.—There is no regular course of symptoms. As most cases of bronchopneumonia are secondary conditions, they are preceded by febrile or afebrile manifestations of catarrh of the nose, the larynx, the trachea, or the larger bronchi, or as complicating symptoms of infectious disease. The onset is therefore usually gradual with unmistakable pulmonary signs, thus contrasting with lobar pneumonia with its often misleading initial abdominal or cerebral symptoms. However, a sudden onset with a sharp rise of temperature, vomiting, and anorexia is not rare; and in the breast-fed infant the refusal to nurse is frequently one of the first signs of the disturbance of respiration. The breathing becomes more rapid and difficult, and the cough more constant. The child appears very ill and prostrated, and as the dyspnea progresses, the cyanosis and drowsiness deepen and the pulse and the cough grow weaker; finally, convulsions and coma may lead to a fatal termination after an illness of a week or two.

Fortunately, more often the outcome is a happier one. After one or two weeks a more or less rapid abatement of all the symptoms is followed by a short pyrexial period, and then uncomplicated recovery, provided, of course, there is no relapse with its signs of involvement of fresh areas. This may, naturally, delay recovery for several weeks. An increasing cyanosis and respiratory distress, a feeble cough, consequent increased large moist rales in the trachea and the bronchi, a weakened pulse, together with a cold, clammy skin, a dull, listless, drowsy attitude—all indicate that the disease is progressing unfavorably. On the other hand, the fact that the symptoms do not grow

worse is a favorable indication. Improvement sets in as the cyanosis and dyspnea become less intense, the temperature gradually falls, while the now effectual cough helps to clear the air passages; the child awakens from its lethargy and takes its food more readily.

The Cough.—The cough is usually intermittent, though sometimes it is continuous, dry, harsh, and irritating, loosening as the mucous secretion increases. Or it may be entirely wanting for a time, after which it is feeble and accompanied by pronounced prostration and delirium. As already noted, young children do not expectorate but swallow the purulent sputum and consequently often suffer from gastrointestinal disturbances. It is difficult and usually unnecessary to obtain any sputum for examination. Vomiting, although rare in the beginning, is quite common in the later stage. Diarrhea and tympanites also appear at this time, the one exhausting the patient's strength, and the other interfering with the unhampered action of the diaphragm and thus aggravating the dyspnea.

The Urine.—The urine as in all fevers contains a trace of albumin, a few hyaline casts, and now and then a granular cast, but it clears up with the general improvement.

Respiration.—As restlessness, crying, and coughing completely alter the respirations, their real character can be judged only when the patient is quiet. They are irregular and frequent, 40 to 60, or more, accompanied by a short expiratory grunt or moan. Extreme counts of 100 or 120 are probably due to the action of toxins on the respiratory centre. Dyspnea exists from the onset, but varies in intensity according to the amount of obstruction offered to the free entrance of air into the bronchioles and the alveoli, as well as to the pulmonary circulation. It may become so marked, and, with the activity of the auxiliary muscles of the soft parts above the clavicle and below the ribs, so resemble laryngeal stenosis or diphtheria, that, except for the absence of the stridor, one might be tempted to perform intubation or tracheotomy.

Circulatory Organs.—The pulse rate is increased from 120 to 160 and more, depending upon the height of the fever, the extent of the inflammation, the amount of toxemia, and the condition of the heart. An affection of the latter is a most serious complication. As long as the pulse continues full and strong, even though it be very rapid, there is very little cause for alarm, but when it becomes thready, easily compressible, and irregular the outlook grows serious. A marked obstruction to the pulmonary circulation causes overdistention of the right heart, and the resulting venous stasis manifests itself in cyanosis, and swelling of the eyelids, the hands and the feet.

Temperature.—Except in very debilitated (marantic) children, who may have little or no fever, the temperature ranges from 101° to 105° F., attaining its maximum in a week or ten days and running an irregular, markedly remittent, even intermittent course. As a rule, it terminates by lysis; a crisis is probable in those cases which are due chiefly to the pneumococcus. A relapse causes the tempera-

ture to rise again for a few days, after which it gradually returns to normal. Though it is true that diagnosis is largely made from the clinical picture, nevertheless the physical signs are important confirmatory evidence.

Inspection.—The character of the breathing alone is frequently sufficient for a provisional diagnosis. Symptoms of dyspnea, abnormal activity of the auxiliary respiratory muscles, inspiratory retraction of the soft parts, the peripneumonic groove, progressive cyanosis with cold, blue extremities and, finally, signs of more or less prostration cannot fail to be recognized.

Auscultation is often puzzling on account of the frequent changes noticed from day to day. Usually both lungs are involved but by no means equally so. During the early stage, the breathing is often harsh and accompanied by medium or coarse rales. After some time, fine crepitations of the bronchioles and the alveoli become audible, disappearing in one place, reappearing in another, and being irregularly distributed in patches over both lungs but principally over the posterior bases. Consolidation of larger areas is evidenced by bronchial breathing and distinct bronchophony, which are brought out more clearly on coughing or crying. It is therefore equally important to auscult the chest both while the child is breathing quietly and while it is crying. Often the heart sounds are heard very distinctly over large consolidated patches of the left lung.

Percussion.—Early in the disease the resonance is not impaired; on the contrary the percussion note may be somewhat tympanitic. It may, however, be slightly diminished over patches, especially in the lower posterior lobes, this impairment becoming more marked later when the scattered small areas of consolidation tend to coalesce. Real dulness is rare except when larger areas are involved; these then give rise to signs similar to those of lobar pneumonia, except that they appear later and clear up more slowly. When the right heart is embarrassed by obstruction to the pulmonary circulation, its dulness extends beyond the sternum to the right, and with increasing difficulty in breathing the inferior border of liver dulness may be found to be very much lowered.

Diagnosis.—The cough and the changed character of the respiration indicate the lungs as the seat of the disease. During the early stage it is often not easy to distinguish bronchopneumonia from severe bronchitis, since the latter may also begin with a fever of 103° to 104° F. This, however, in bronchitis, falls to 100° or 101° F. within twenty-four to forty-eight hours and, in addition, only coarse rales are heard over the whole chest, while the prostration and all the other symptoms, with the exception of the cough, are less severe than in bronchopneumonia. On the other hand, the appearance of fine crepitations, areas of consolidation, the bronchial breathing, and bronchophony indicate the more serious character of the trouble. A localized bronchitis, especially in children under three years of age, and when accompanying or following measles, whooping-cough, or pronounced rachitis,

always arouses a suspicion of bronchopneumonia, provided that acute pulmonary tuberculosis (particularly the caseous form which affects the lower lobe in children) can be excluded. The latter affection is rare, but may give rise to exactly the same symptoms and signs except that the areas do not shift as in the other forms of prolonged bronchopneumonia; it is, however, definitely determined by the finding of tubercle bacilli in the sputum and a family history of tuberculosis.

It is not difficult to differentiate bronchopneumonia from lobar pneumonia in typical cases. A large area of consolidation limited to one lobe with definite dulness, bronchial breathing, and subcrepitant rales usually indicates lobar pneumonia. In bronchopneumonia, while the dulness may be wanting, coarse rales are found over the larger bronchi and crepitant or subcrepitant rales appear and disappear over different parts of both lungs. In the many atypical cases, where the physical signs are not thus clearly defined, the clinical history as to the onset, the course, and the termination of the symptoms, may help in the differential diagnosis. In fact, in the very young child the diagnosis often rests entirely upon the rapid respirations, the cyanosis, and the prostration. When following either simple bronchitis or that complicating the infectious fevers, an increase in these symptoms together with a rise of temperature takes place at least twenty-four hours before the appearance of the physical signs. The occurrence of bronchopneumonia in weakly infants is not surprising, but we must look for a special reason when it attacks apparently-healthy children. With them it may be preliminary to measles, or it may be masking a whooping-cough of pneumococcal or tubercular origin.

In cases with a markedly remittent temperature and a very slight cough, a combination which occurs not infrequently in infants, the possibility of primary or secondary malaria can easily be excluded by an examination of the blood. Atelectasis is uncommon after the fourth month, and congenital atelectasis is often impossible to diagnose *in vivo*. It is seen most commonly in delicate infants who were with difficulty resuscitated at birth, and the cyanosis which is a common symptom is out of proportion to the findings in the lungs.

Complications.—Thrush, in delicate infants, and stomatitis in older children are quite common, but true gastro-enteritis is not a frequent complication of bronchopneumonia. Vomiting and diarrhea, so often seen at the onset, are, no doubt, chiefly functional. There is a certain amount of emphysema usually present, not marked enough, however, to produce any physical signs. An irregular, remittent, or intermittent temperature following bronchopneumonia usually indicates some complication. Pleurisy, usually the purulent type, does not occur as frequently as in lobar pneumonia; otitis media is rather common and should be looked for if the child shows increased restlessness, irritability and sleeplessness in addition to the usual symptoms. Meningitis, arthritis, and purulent pericarditis occur but rarely.

Prophylaxis.—Children should be kept away from persons suffering from a cold or a sore throat, since a coryza or bronchitis is easily

acquired by such contact, and may, in a delicate child, lead to a fatal bronchopneumonia. If catarrh of the respiratory passages occurs in spite of all precautions, it must not be neglected. It is of great advantage to protect children, at least up to the fourth year, from contracting measles or whooping-cough; after this period there is less risk of the dreaded complication of bronchopneumonia. No doubt it could often be prevented if children convalescing from measles or whooping-cough were not allowed out of doors before the physician feels satisfied that there is no further danger of pulmonary complication. There should, however, be no lack of fresh air. More cases of bronchopneumonia formerly followed the infectious fevers, when the patients were kept in overheated rooms and in beds loaded with blankets, than at present when plenty of fresh air is the rule. While primary cases of bronchopneumonia need not necessarily be isolated, isolation is certainly advisable when the disease is secondary to infectious fevers (measles and whooping-cough); these seem to lend it a special virulence or infectivity as evidenced during epidemics in measles wards, for example.

Treatment.—There is no specific remedy and the general treatment follows the line of other febrile conditions. It requires special care because, the disease being protracted and not self-limited, it is a matter of the greatest importance to conserve every bit of strength. Restlessness, loss of sleep, indigestion, and an increased strain on the heart cause so much waste of vitality that it is most essential to make the child comfortable, not by drugs, however, but by a good sick-room régime. This will influence the course of the disease considerably and save many lives.

The room, bared of all unnecessary furniture, should have, first and foremost, a constant supply of fresh—not necessarily cold—air. For the very young a temperature under 60° F. is hardly of advantage, while 70° F. should be the upper limit. As a dry atmosphere is irritating, it must be moistened from time to time, by means of a spray, a croup-kettle, or wet cloths hung up in the room. There is a tendency to overclothe the patient; nothing more than a flannel shirt and a simple night-dress is required even in winter. When older children are given the open-air treatment, under which the sleep, the appetite, and the cough often show striking improvement in prolonged cases, they of course require more clothing. The patient should not be allowed to lie on his back constantly, but should be turned on the side or the abdomen several times a day to encourage aëration of the posterior parts of the lungs. It is of the greatest importance to avoid overfeeding, or anything (including many of the time-honored expectorants) that might disturb the appetite and the digestion. Food should be given in small quantities and in an easily digested form. Nurslings should have some water before feedings, and the intervals between the feedings should be shortened, and for the bottle-fed the milk formula should be reduced in strength. Children, between two and five years of age, with poor appetites, are given albumin

water, whey, meat or vegetable broths, expressed beef juice, or pep-
tonized milk, and when the appetite improves diluted milk, gruels,
light puddings, etc., may be added to the list. Under all circum-
stances boiled water or sweetened lemonade should be freely offered
and the bowels kept open at least once daily. In order to disturb
the patient as little as possible, food, medicine, and local treatment
should be given at one time, with intervals of two and a half hours of
complete rest. Of all the various remedies used, the most important
are baths, counterirritants, stimulants, and inhalations of oxygen,
and of moistened air. The oiled silk and cotton jacket seems super-
fluous. Although the old method of using the tent and the croup-kettle
may have been overdone at times, for there is no doubt that a con-
tinuously moist atmosphere has a depressing influence, still, when
judiciously used (10 drops of creosote added to 1 quart of water) for
about thirty minutes every three or four hours, it certainly relieves
the acute symptoms, especially the irritating cough and the bronchial
or the laryngeal spasm.

Counterirritation over the chest is useful in relieving pulmonary
congestion and marked bronchial catarrh by causing depletion into
the peripheral bloodvessels. For this purpose the mustard paste
applied over the chest or the mustard bath, both continued until the
skin is thoroughly reddened, are equally useful, but a mustard pack
is more lasting in its effects. This consists of steeping a cloth in one
quart of water (105° F.) containing 1 ounce of mustard flour, wringing
it out and wrapping it snugly around the patient so as to cover the
entire skin surface except the head and the neck. A woolen blanket
serves as an outer covering and is so adjusted around the neck that
the irritant vapors of the mustard oil are not inhaled by the patient.
As soon as the skin is thoroughly reddened—after ten to thirty
minutes—the child should be washed with plain warm water and put
into a moist pack for one to two hours and, finally, given a cold
sponge. In this way the hyperemia of the skin is kept up for a long
time afterward. The present-day more intelligent use of hydrotherapy
yields good results, especially when given with as little disturbance
to the patient as possible. Sponging with cool water, followed by
light friction with a dry towel, or a chest compress, the water used
being at room temperature (70° F., or less, for older children), and
renewed every one to two hours, is always agreeable to a fever patient.

For infants and delicate children we prefer to induce deep respira-
tions by applying warm compresses to the chest followed by a very
short application of cool water. A similar effect can be obtained by
spraying a little cool water on the shoulders and the back of the
patient during a warm bath (93° to 95° F.). Hot baths (100° to 104°
F.), even in the presence of fever, but more especially with cyanosis
and a cold skin and feeble pulse, are valuable in quieting the patient
by reducing pulmonary congestion and equalizing the general circula-
tion. The use of drugs cannot be justified unless clearly indicated
for the relief of severe symptoms; they are directly injurious if they

upset digestion. Usually the temperature does not run so high as to require special treatment, but in exceptional cases where it rises above 105° F., hydrotherapeutic measures prove superior to quinine and the coal-tar products; if used at all the latter should only be employed to allay irritation and restlessness.

Inhalations of steam, as in bronchitis, for ten to fifteen minutes at a time, hot drinks, etc., promote secretion and relieve the cough; but when the latter is painful or incessant a sedative will be required in order to secure sleep and to avoid any unnecessary strain on the heart. We recommend codeine, $\frac{1}{20}$ gr., or morphine, $\frac{1}{40}$ gr., or a Dover's powder 1 gr., every three or four hours, for a child over one year of age. When the secretion grows excessive, it seems better to check it by administering five to ten drops of belladonna than to try to get rid of it by emetics. Sooner or later the heart suffers from the pulmonary obstruction, as well as from the prolonged fever and the general toxemia, and should be stimulated as soon as a very rapid or a soft, irregular pulse shows that such assistance is needed. Tincture of strophanthus retards the pulse, while strychnine is indicated with an irregular, soft, compressible pulse; both are useful drugs because they are easily digested. Caffein, or a 10 per cent. solution of camphorated oil, or a $\frac{1}{1000}$ solution of adrenalin in 5- to 10-minim doses, intramuscularly, is especially valuable for hypodermic medication, in spite of the evanescent effect. While the use of alcohol has been abused in bronchopneumonia, it is sometimes serviceable when all other means fail, and should be kept in reserve for such emergencies. The milder stimulants such as broth, beef-tea, tea, or coffee mixed with milk can be given quite early and may suffice in the less severe cases. Marked cyanosis is temporarily relieved by inhalations of oxygen, which may be combined with menthol, turpentine, or bubbled through alcohol. Atropine, $\frac{1}{100}$ of a grain, administered hypodermically, every four hours to a child of three years, is useful when the dyspnea and the rapid respiration are due to toxic influences on the respiratory centre.

Convalescence.—Convalescence is often slow. Open-air treatment should be kept up during the warm months. If the catarrh does not fully clear up, a stay in the country, or removal to a warmer climate during the winter is usually of great benefit. Where these privileges cannot be enjoyed, iron, arsenic, quinine, or malt extract with cod-liver oil should be given as required.

DISEASES OF THE PLEURA.

PLEURISY—SEROUS AND PURULENT.

Inflammation of the pleura occurs in children, as in adults, either in the dry, fibrinous form or with an effusion which may be either serofibrinous or purulent (empyema). Of these the purulent variety

demands our special interest because of its comparative frequency in children, especially those under five years of age, and because it may lead to a fatal issue unless prompt diagnosis—by no means always easy—points the way for proper treatment.

Although pleurisy may be caused by an inflammation spreading from the ribs, the vertebræ, and the peritoneum, it is not merely a local affection but is almost always a sign of a general morbid state. In the rare examples in which it apparently results from a chill or from exposure, it is, no doubt, really tuberculous or rheumatic in origin. It has been observed in sepsis of the newborn, during and following the infectious fevers, such as scarlet fever, measles, grippe, whooping-cough, diphtheria, and typhoid fever, but the majority of all cases occur secondarily to pneumonia and bronchopneumonia. For this reason it prevails more often during the cold, damp season, and is more apt to affect boys than girls on account of their greater liability to contract pneumonia. Pleurisy is not frequent in infants less than six months old, but is quite common between the sixth month and the sixth year, and, as a rule, the younger the patient the greater the likelihood of its being purulent in character.

Pathological Anatomy.—Postmortem the pleura may merely show a lack of luster, some adhesions either localized or spreading over more or less extensive areas, and deposits of fibrin varying from simple roughening to a felt-like layer sometimes $\frac{1}{4}$ to 1 inch thick. In these extreme cases, a shrinking of the lung and a corresponding retraction of the affected side naturally follow. The effusions differ greatly in quantity and also in character in different cases; they may be unilateral, bilateral, or sacculated, *i. e.*, localized, and shut off from the rest of the pleural cavity by adhesions. A large amount of fluid in the pleural cavity will push the lung upward, thus assisting its natural tendency to recoil and allowing very little possibility for expansion. A serous effusion has a light greenish-yellow color; it is clear, or slightly cloudy, and contains fibrinous shreds; in favorable circumstances, it is capable of being completely absorbed with little diminution of the respiratory capacity. Large purulent effusions, however, are not absorbed, and an empyema if not properly treated results in pyemia or cachexia, unless the pus finds a natural outlet by ulcerating through the thoracic wall, or by breaking into a bronchus. While it is true that a serous effusion may exceptionally become purulent by secondary infection, an acute pleuritis, as a rule, must necessarily be either serous or purulent, according to the predominating microörganism. Generally speaking, a thin, yellowish pus indicates a streptococcic infection and a thick, pale green discharge a pneumococcic infection, though, of course, the naked eye appearances are not very reliable. The consistency depends upon the relative proportion of serum and pus cells present.

Bacteriology.—In pleurisy, pneumococci are usually present; streptococci and staphylococci are less frequent, while typhoid, colon, and influenza bacilli are only occasionally found. Empyema, in 70 to 90

per cent. of cases is due to pneumococci, either in pure culture, or combined with other microörganisms; streptococci predominate in 5 to 15 per cent., while tubercle bacilli are responsible for only about 5 per cent. of all cases of empyema in children.

The amount of fluid varies according to the age and the size of the child and the duration as well as the character of the effusion; it is greater when the fluid is thin. In children under three years it rarely amounts to more than $\frac{1}{2}$ to 1 pint while in older patients as much as 3 or 4 pints is not uncommon.

Symptoms.—These vary considerably in the different types of pleurisy. In a well-marked case, more or less fever, cough, and pain, together with respiratory and circulatory disturbances, are present, and are not infrequently accompanied by headache, vomiting, and constipation. Generally speaking, a baby sick with pleurisy is very sick indeed and looks it. As mentioned before, most cases occur secondarily to pneumonia or to bronchopneumonia, either during their course, thereby greatly prolonging the duration of the fever, or—more commonly—within a few days after the crisis or the lysis. In these cases the onset is marked by a new rise of temperature up to 103° or 104° F., an aggravation of the dyspnea, and the cough, often also of the pain, and a pronounced increase in the pulse rate. A sudden onset, somewhat resembling that of a milder pneumonia, characterizes these comparatively rare cases of primary pleurisy, which begin with headache, vomiting, chills and fever, 102° to 103° F., and are soon followed by a hacking cough, pain in the chest, and shallow, rapid breathing. Occasionally the onset is more lingering or gradual in type. The child is ailing for a week or two, with failing appetite and a low fever, increasing weakness, anemia, and a slight cough.

When the pleuritic effusion has taken place, the clinical picture changes, the symptoms naturally varying with the amount of fluid and the rapidity of its accumulation. The patient often lies on the affected side, avoids loud crying, is pallid, as a rule, but becomes cyanotic when the heart is embarrassed by the fluid. The cough may disappear, though sometimes it becomes spasmodic. Breathing is rapid, superficial, and increased on the sound side, while the dyspnea becomes very apparent on the least exertion in speaking or moving. Often there is neither pain nor cough to indicate the seat of the trouble; such cases of malaise, feverishness, accompanied by poor appetite, coated tongue and restlessness are quite likely to be designated "dentition fever."

$$\text{Individual symptoms} \begin{cases} \text{Fever.} \\ \text{Pain.} \\ \text{Cough.} \\ \text{Respiration: dyspnea.} \\ \text{Circulation: cyanosis.} \end{cases}$$

Fever.—Too much reliance should not be placed on the temperature charts, as the fever often runs an irregular and exceedingly variable

course. It is usually high during the first few days, ranging between 101°, 104°, or 105° F., and in serofibrinous effusion is apt to become remittent; during the second or third week it abates as the absorption of the fluid progresses. In empyema, the temperature rise is usually not quite so high at first, it may be irregular, intermittent, or hectic, with night-sweats and rapid emaciation, especially when following the exanthemata. In a pneumococcic pleuritis, the temperature variations do not seem so marked. Unless properly treated, the fever may last many weeks; but, on the other hand, it may be entirely absent in the later stage.

Pain.—Pain in the side, aggravated by deep breathing and coughing and not infrequently referred to the abdomen or the shoulder, is an early symptom, and usually disappears with an effusion sufficient to separate the pleural surfaces. The affected side may be tender to pressure; pain in the right side is due to pressure of a right-sided effusion on the liver.

The Cough.—The short, dry, painful cough, which the child makes every effort to suppress, may become less frequent and may sometimes, but not always, disappear when the effusion is large.

Respirations.—Respirations are frequent, shallow, often irregular, and more or less painful, especially in diaphragmatic pleurisy. Often the child lies on the affected side in order to limit its movements and allow more freedom of expansion to the sound lung. The pain usually diminishes as the extravasation increases, but the shortness of breath (dyspnea) becomes more noticeable the more the lung recoils (favored by mechanical compression). When the effusion advances rapidly, the distress is very marked indeed, while it may escape attention when its progress is slow. In empyema the respirations are always increased to 40 to 70 per minute. The dyspnea corresponds to the amount of fluid, but often manifests itself only on exertion or by a feeling of fatigue.

The Circulation.—The pulse is always rapid, 100 to 150 per minute, even where there is little or no fever; it becomes still more accelerated during the febrile period, or when a large or rapidly increasing effusion embarrasses the heart. It then becomes feeble, irregular, and very rapid upon the slightest exertion. In addition, venous engorgement of the face and the neck may become noticeable. All cases of pleural effusion lead to a more or less pronounced anemia; empyema, furthermore, is usually attended with loss of flesh and prostration, sometimes with night-sweats, and, in more chronic cases, a rapidly developing clubbing of the fingers accompanied, not with cyanosis, but with a peculiar yellowish tint of the skin.

The Course.—Dry pleurisy clears up within a week, unless due to tuberculosis or to pneumonia. Very large effusions may cause cyanosis, pulmonary edema, and death if not relieved in time. The acute stage of empyema, with loss of appetite, flesh, and strength, and sometimes accompanied by hectic fever and night-sweats, lasts about two to four weeks. It may be followed by apparent convalescence, during

28

which the patient has little or no fever and seems to be regaining his color and strength, but the cough and the rapid respiration and the dyspnea reappear on the slightest provocation and give fair warning of a false security. If empyema is allowed to become chronic the symptoms resemble those of tuberculosis.

Physical Signs.—The chest signs are essentially the same whether caused by a serous or by a purulent effusion. While an appreciable amount of free fluid in the pleural cavity of one side is quite easily detected, detection is more difficult when the effusion is localized or bilateral, or when there has been a previous thickening of the pleura. The upper border of the lung, as a rule, remains unchanged, but the lower resonant border, retracting from the gradually increasing fluid, is highest in the axilla, sloping downward and inward to the sternum in front and toward the spine posteriorly. The lower limit of the fluid is not identical with the lower border of the normal lung because the diaphragm is often elevated even with a comparatively large effusion, and becomes depressed only in the late stage when extravasation is excessive.

Inspection.—In addition to the shallow, accelerated, jerky, and painful breathing in effusion of appreciable degree, the affected side appears larger, the respiratory excursions are diminished and the depression of the intercostal spaces is less noticeable.

In a very marked effusion the signs of severe dyspnea, of expiratory distention of the large veins of the neck, the increased measurement of the affected side, the displacement of the heart, and possibly an elevation of the shoulder, are so obvious as hardly to be overlooked.

Palpation.—Vocal fremitus, quite apart from the fact that it is not easily elicited in young children, is lost or lessened over the fluid; respiratory movements are diminished over the affected side and increased over the sound one. There may be sensitiveness to pressure between the ribs. With a large left-sided effusion the apex beat may be palpable at the epigastrium or even further to the right, but in a right-sided effusion the apex may be found displaced to the left as far as the midaxillary line.

Percussion.—Percussion is often painful over the affected pleura, and often gives much more valuable information than auscultation. It should be done lightly as, otherwise, a thin layer of fluid over the spongy lung tissue or over the stomach would cause no appreciable impairment of the note. With the child in an upright sitting posture a dull rather than a flat note is elicited over the fluid, which commonly gathers in the lower and posterior part of the chest, and when encapsulated occasionally in other parts of the chest. Owing to the gravitation of the fluid, the upper line of dulness often shifts with a change of position; resonance amounting to subclavicular tympany in very large effusions is always found directly above the fluid. When the latter almost fills the entire side, the dulness may extend beyond its border some distance to the sound side—beyond the sternum anteriorly and the spine posteriorly.

A very valuable sign appreciated by the percussion finger is a peculiar board-like resistance, doubly striking in children on account of their naturally thin and elastic chest walls. Displacement of the liver downward encroaching upon the stomach, but especially on the heart, as mentioned under palpation, is almost pathognomonic. Dulness on percussion and a board-like resistance are alone sufficient to justify exploratory puncture in order to determine the character of the suspected pleural effusion.

Auscultation.—As a whole, auscultatory findings are very variable, and therefore unsatisfactory and even misleading in infants and children. Early in pleurisy, and again when the fluid is almost absorbed, a friction rub may perhaps be heard with inspiration or expiration or with both; it is superficial in character and unchanged by coughing. Only in rare cases where a large effusion leads to compression of the lungs is there sometimes complete absence of voice and respiratory sounds. They frequently remain almost normal but are usually diminished, distant, and indistinct. Bronchial breathing, although usually more feeble and distant over the fluid on deep breathing, is sometimes as marked as it is in pneumonia. It is often heard also along the spine and the sternum—an important fact in differentiating the condition from pneumonia. Whatever the character of the breath sounds on the affected side they contrast markedly with those of the healthy lung where they are puerile and harsh (exaggerated).

X-rays are helpful in locating sacculated empyemas; in a general effusion they show the diminished movement of the pleura on the affected side and the displacement of various organs. In the interest of exact diagnosis and prognosis an exploratory puncture is advisable, even though there may be bronchial breath sounds or crepitations audible over the area of increasing dulness. The point of choice for the puncture is the posterior axillary line—the sixth interspace on the left side, the fifth on the right side. When the fluid is localized the puncture should be made over the point of most marked impaired resonance. With the proper antiseptic precautions, a sterile needle is introduced slowly for about an inch, gentle suction being kept up all the time. If the needle becomes plugged up on account of its small calibre, it may happen that no pus can be obtained; this may also occur if the needle is pushed beyond the visceral pleural layer into the lung tissue, or if it penetrates an adhesion, or a mass of fibrin, etc. The fluid when obtained should be submitted to a bacteriological examination.

Differential Diagnosis.—The diagnosis of pleural effusion in the presence of dulness or flatness, and of more or less displacement of the heart or the liver, is definitely determined by exploratory puncture. This makes it possible to exclude allied conditions, such as unresolved pneumonia, pulmonary tuberculosis with extensive caseation, and the rare cases of abscess of the lung. The differentiation is more difficult in case of subphrenic abscess, of large pericardial effusions, and of localized empyema. Again, typhoid fever and malaria may simulate

the constitutional symptoms of empyema, but the different physical findings and the blood examination hardly leave room for doubt.

Prognosis.—Spontaneous recovery of empyema cannot be expected, although Nature does make an attempt to get rid of the effusion. The pus may break through into a bronchus and be coughed up, thus giving some temporary relief; or it may be directed externally and continue to discharge for many months, the patient finally dying from exhaustion, amyloid degeneration, or tuberculosis. In the absence of serious complications, such as double empyema, purulent pericarditis, meningitis, pyemia, or pulmonary tuberculosis, prompt and efficient treatment is certainly very satisfactory; but the final outcome will naturally depend upon several other factors. The disease is very grave and the mortality is high, especially in hospital children under one year of age. After the second year the prognosis is very good, for with early surgical treatment recovery is almost the general rule. A delayed operation cannot give as good results on account of the greater weakness of the patient and the increasing difficulty of proper reëxpansion of the lung. In general, the outlook is best when the disease is of pneumococcic origin, less so when staphylococcic, worst of all when streptococcic.

Treatment.—Fresh air, sunshine, avoidance of all exertion, are as essential here as in pneumonia. Fixation of the affected side by flannel bandages and the application of heat in the form of fomentations or turpentine stupes, or of mustard paste, usually suffice for the relief of pain. However, sometimes opiates (codeine, morphine, or paregoric) are necessary to prevent loss of sleep from excessive coughing.

During the febrile stage, dry and serofibrinous pleurisy are treated alike by a fever diet and rest in bed. As soon as there is a temperature or there are signs of an effusion, the child must neither sit up nor exercise its arms and its lungs more than is absolutely necessary. When a serous effusion has occurred, the diet adapted to the diminished digestive capacity is (for children over two years of age) usually restricted principally to solids—bread and butter, eggs, puddings, beef-juice, malt extracts, and very little milk; although it is very doubtful whether a diminution of liquids influences the absorption of the fluid. This may be facilitated to some extent by external applications of heat, counter-irritants, ointments containing 10 to 16 per cent. of guaiacol, hydrargyrum or iodine, and by purgatives and diuretics. It is always wise to prescribe some heart stimulant, either digitalis or strophanthus. Sodium salicylate, or aspirin (5 grains 4 times a day) in sugar water, is best given in the early stage, and potassium iodide later on when absorption has begun.

Evacuation of the effusions during the acute stage is contraindicated, except when breathlessness, cyanosis, and cardiac embarrassment make it imperative; but if the fluid does not diminish within two to three weeks it should be removed (except in tuberculosis) by siphonage or aspiration. Drainage can be secured either by simple

incision after the site of the pus has been explored with the needle or by resection of a portion of one or of several ribs. Simple incision is more quickly done. It requires only local anesthesia and usually proves successful, especially in babies under two years old. An incision about two inches long is made in the fifth or sixth interspace in the middle axillary line, close to the lower rib through the intercostal muscles. After opening the pleura, the pus is allowed to escape slowly, large coagula being removed with forceps, and a short drainage tube of large calibre (or two smaller ones side by side if the ribs are close together) is inserted and fastened with large safety pins. The whole is covered by a thick aseptic dressing which must be renewed or reinforced when it becomes saturated. The drainage tube should be removed as soon as the discharge grows serous. The wound then closes in a short time, and is usually completely healed within three to seven weeks after operation.

Resection of a part of one or of several ribs is the best means of ensuring thorough evacuation and also rapid recovery. Only light, general narcosis is necessary. An incision, two to three inches long, is made directly over the eighth or ninth rib, in the posterior axillary line; the periosteum being stripped back, about two inches of the rib are cut away with bone forceps or with special rib-shears. On opening the pleura, pus flows out with each respiration, and more rapidly with coughing. Some surgeons insert the finger to remove thick masses, or break down adhesions in order to empty any existing pockets of pus, and even advise irrigation of the pleura with saline solution. However, it is not necessary to empty the cavity completely, for what remains is often rapidly absorbed, but it is essential that the withdrawal should be gradual, and that it be stopped with the appearance of violent coughing, weakness, fainting, or bleeding.

If the effusion were allowed to stay from six to eight weeks, thickening of the pleura, adhesions, and impeded expansion of the lungs would probably result. Therefore, when repeated tapping in serous effusions effects no cure, incision and drainage become necessary. The point of choice for paracentesis is in the sixth or seventh interspace near the middle or posterior axillary line or at the place of greatest flatness. If there is any uncertainty as to the exact site of the fluid, this should be ascertained by puncture. With the proper antiseptic precautions and with the child in a sitting posture, a trocar of $\frac{1}{12}$-inch calibre is quickly introduced for about an inch close to the upper edge of the rib. While an effusion of recent date evacuates itself, unless fibrin shreds obstruct the opening, very little or nothing at all will flow out spontaneously in a chronic case on account of the insufficient pressure, and removal by siphon or aspiration may become necessary. The small opening is subsequently closed with collodion.

The fluid in young children is usually purulent or seropurulent. The only satisfactory treatment for this is surgical evacuation of the pus as soon as diagnosis has been made. A short delay may be permissible only in a small empyema following pneumonia when con-

valescence is otherwise satisfactory. Aspiration is insufficient except as a temporary measure to relieve distress, and as a preliminary reduction of excessive effusion.

During convalescence the patient should live in the open air but should avoid any excessive use of the chest and the arms. The diet should be carefully regulated, and malt extract with iron, cod-liver oil, and arsenic administered in proper moderation. Later, breathing exercises, varied so as to take the fancy of the child, such as blowing a trumpet, or soap-bubbles, or fluids from one bottle to another, are advisable as aids in successfully reëstablishing the expansion of the chest. After six months the orthopedic surgeon should be consulted for any chest and spinal deformity, although breathing and gymnastic exercises properly directed and intelligently carried out may do much to diminish such deformities.

Bilateral empyema is rare. If general, on both sides, it is wise to relieve strain on the heart by preliminary aspiration followed in a day or two by draining the left, and later the right side. In chronic empyema more extensive operations, such as advised by Estlander Schedes, may become necessary, or even decortication of the thickened visceral pleura may have to be resorted to. Summing up, one would say that for the successful management of empyema it is essential to diagnose it early, to establish prompt and efficient drainage, and to pay particular attention to the after treatment. Failure is due to either delay, to thick adhesions, to inefficient drainage, or to the formation of a persistent sinus.

REFLEX COUGH.

A cough, strictly speaking, is always reflex in origin; but, for the sake of convenience, we will group under this heading all those clinical varieties of cough which, because they furnish no obvious sign of inflammation of the larynx, trachea, bronchi, lungs, or pleura, are supposed to be due either to vague stimulation in the mouth, throat, stomach, or ear, or to irritation of other organs. It is not an affection of early infancy, but quite frequently attacks older children. Cough is most common in that form of postnasal catarrh which is associated with enlarged tonsils and adenoids. Mucus or mucopus accumulations in the nasopharynx excite a dry, tickling cough which usually comes on as soon as the child lies down (and sometimes only then), and is thus apt to disturb the sleep for hours. It may last for many months, especially in winter.

Similar symptoms may be caused by an elongated uvula, or in rare cases, by cerumen impacted in the external auditory meatus. In mitral disease, probably because of the resulting pulmonary congestion, and in pericarditis, a dry hard cough often makes the patient very uncomfortable.

About the time of puberty, and frequently associated with anemia, chorea, or extreme nervous irritability, a cough may spring up which

may be either of the ordinary type or a curiously monotonous one; it is increased by exercise, and ceases during sleep. There is neither pain, nor expectoration; the onset and the termination may be either gradual or sudden, while relapses are frequent.

Periodic attacks of a brassy cough may occur regularly every night for months, the paroxysms sometimes becoming so severe as to resemble whooping-cough, except for the almost complete absence of vomiting. These paroxysms are ascribed to an irritation of the pneumogastric nerve or its branches, due to the pressure of enlarged lymph nodes surrounding the trachea and the large bronchi. This glandular enlargement may be present in Hodgkin's disease, and in lymphatic leukemia, or it may follow various pulmonary affections, but in many cases it is really the primary focus of tuberculosis.

The patients sometimes seem the very picture of health; but ordinarily long-continued loss of sleep tells on the general health, and when the enlargement is part of a tuberculous process the child is apt to be delicate, irritable, and easily fatigued, while the appetite is failing or capricious. The patient complains of interscapular backache, or ill-defined pain within the thorax, or sharp and lancinating pain brought on by deep inspiration or by violent exertion. The superficial thoracic veins may be dilated. Usually there is some expiratory dyspnea, and not infrequently an increased sensitiveness to cold, especially in the region of the shoulder. A similar cough has been observed in connection with abscesses of the posterior mediastinum, and in Pott's disease.

Symptoms.—In all of these cases it is chiefly the cough which excites concern. This grows worse or occurs only at night, and is liable to become paroxysmal, the attacks coming on rather regularly. The general health may not be affected except for the disturbed sleep.

Diagnosis.—A reflex cough should not be diagnosed lightly. By a thorough examination of the ears, the nose, and the throat, as well as the heart, the lungs, and the stomach, in conjunction with careful observation of the general condition, it is often possible to ascertain its precise cause. X-rays are of great service in cases where enlarged lymph nodes are suspected, as after measles, pertussis, repeated attacks of bronchitis, or in patients with tubercular antecedents.

Treatment.—It is obvious that neither opium, nor inhalations, nor expectorants will effect a cure. The underlying cause must be treated. If this cannot be detected and the condition seems purely nervous, small doses of the bromides and of antipyrin may be given at bedtime to relieve the cough.

ASTHMA.

Excluding cases of mere dyspnea due to cardiac or renal diseases, two chief types of asthma are met with in children; the first, the spasmodic or true bronchial variety, resembles that of adults, and is characterized by paroxysms of expiratory difficulty, the respiratory system remaining apparently normal during the intervals. It is only

exceptionally observed in children under four years of age, but seems
not so uncommon from the seventh or eighth year up to puberty.
The second, the catarrhal type or so-called asthmatic bronchitis,
differs from the former variety in the increased amount of bron-
chial secretion and in the character of the dyspnea, the paroxysms
being preceded, associated, or followed by bronchitis. It is not quite
so rare in infants as the first type, and is also rather commoner in
older children. They may suffer from one or several attacks during
the year, and are liable to be somewhat short of breath during the
intervals. It may be said, in passing, that undoubted cases of *hay
asthma*, occurring especially during hot and protracted summers, have
been observed as early as the fourth year in children of neuro-arthritic
families.

Etiology.—The pathogenesis in the child is no doubt essentially
the same as in the adult; that is, there exists a neurosis of the respira-
tory system, manifested by paroxysmal spasms of the respiratory
muscles, combined with hypersensitiveness, temporary vasomotor
paresis, and, perhaps, an abnormal secretion of the respiratory mucous
membrane. These diverse factors probably cause a temporary nar-
rowing of the lumen of the bronchial tubes. Heredity plays an impor-
tant role, there being usually a family history of various neuroses,
gout, eczema, and neuro-arthritis. The poorer classes are relatively
seldom affected. Dry climates yield the smallest percentage of cases.
When an attack has once occurred, other paroxysms are readily induced
by central or by peripheral reflexes. While psychic factors, such as
fright, emotion, and excitement, are only occasionally responsible for
an attack, one may be easily induced by any local irritation of the
nose, the pharynx, or the bronchial mucous membrane, due to inflam-
mation caused either by the inhalation of irritants, such as dust, pollen,
gases, certain animal emanations, or to sudden changes of climate or
of weather conditions. Furthermore, disturbances of the gastro-
intestinal tract and enlarged bronchial glands also act as exciting
factors in susceptible children. These are usually delicate, nervous,
irritable, and anemic, suffering from eczema, from chronic urticaria,
and hypertrophy of the tonsils and adenoids, though the overfed
child is by no means exempt. The symptoms of *spasmodic or true
bronchial asthma* in children do not differ essentially from those in
adults. There is a periodic, urgent, and usually sudden dyspnea.
The child, apparently well, awakens in a fright a few hours after
retiring, it has a sense of impending suffocation, looks anxious and
pale, is more or less cyanotic, restless and rigid, or sits up in bed with
staring eyes, head thrown back, shoulders raised, and its little hands
clinched, to all appearances fighting against a distressing dyspnea.

The breathing is slow and labored, the shallow inspirations are fol-
lowed by a pause and prolonged expirations, while both are accom-
panied by wheezing sounds, often audible over the entire room. The
skin and the extremities feel cold and clammy, the pulse is feeble,
very rapid, and often irregular. In a typical case there is no hoarse-

ness, or fever—the temperature may even be subnormal in prolonged paroxysms—and the cough, if present, is infrequent, dry, and short in character, bringing up a mucous expectoration. This contains Charcot-Leyden crystals, Curschmann's spirals, and many eosinophiles. The paroxysms last several hours, occasionally several days; they subside slowly, as a rule, but sometimes subsidence is abrupt, ending with a fit of coughing or vomiting. Considerable exhaustion usually follows but the little patient seems healthy in the intervals, which may last for weeks or months, depending upon the exciting cause. On the other hand, the paroxysms may recur at the same hour for several successive days; if at all severe, they naturally impair the health. Their severity varies just as do their duration and frequency, some children suffering from periodic attacks of musical rales for a few days at a time without any apparent dyspnea.

The type of asthma called *spasmodic bronchitis* differs from the above mainly in the amount of bronchial secretion and the presence of fever. It develops during and after an attack of nasal or bronchial catarrh, or follows measles or whooping-cough. The paroxysms usually come on at night, they vary in frequency, and are characterized by an increasing dyspnea, a feeble pulse, and dry rales. After two or three days the child appears perfectly well except for a few rales, and perhaps a slight cough. During an attack the thorax is held in the position of full inspiration with the diaphragm depressed and the respiratory muscles contracted. Only a slight recession of the soft parts is visible during inspiration. Diffused loud wheezing and rales, which at first are dry, then moist, and later mostly expiratory, obscure the vesicular murmur. In prolonged cases a hyperresonant percussion note reveals emphysema, which in children rapidly becomes extreme, but quickly subsides unless protracted by frequent and prolonged paroxysms. In asthmatic bronchitis there is practically no hyperresonance but inspiratory recession of the soft parts and lowering of the ribs become marked. Many fine rales are heard at the bases.

Diagnosis.—The special diagnostic features of asthma are: sudden onset, recovery when the symptoms are apparently most alarming, the absence of fever, and recurrent attacks of inspiratory dyspnea out of proportion to the physical signs. With these in mind, it should not prove difficult to differentiate the condition from cardiac, renal, or diabetic dyspnea. Enlargement of the thymus or the bronchial glands, as an etiological factor, can best be excluded by an x-ray examination. The dyspnea of retropharyngeal abscess, or of laryngeal obstruction, and of inflammatory affections, is decidedly inspiratory, while the difficult breathing of hysteria, though it may occur in paroxysms, does not cause distress.

Prognosis.—The prognosis is the more favorable the shorter the duration, the younger the patient, and the less pronounced the hereditary taint. A cure may be effected by the removal of the local exciting cause, or by a change of climate. Towards puberty an abatement and even the disappearance of the attacks is not infrequently observed. With a poor heredity, a predisposition to true bronchial asthma may

last for a lifetime, and may gradually lead to emphysema and embarrassment of the right heart.

Treatment.—The importance of preventing an attack by proper hygienic measures, rather than by relying upon means simply to relieve or to abort it, hardly needs emphasis. Everything should be done to remove the exciting cause and to improve the general health and the underlying nervous condition. Systematic breathing exercises are of great value. Sometimes a radical change of climate may be necessary. The diet must be carefully regulated; special attention should be paid to the evening meal, which should be light and taken sufficiently early to insure an empty stomach on retiring. While no drug has proven a specific for all cases, 3 to 5 minims of a 1 to 1000 solution of adrenalin chloride given intramuscularly often affords prompt relief; inhalations of nascent oxygen, or internal doses of cocain hydrochlorate and apomorphin sometimes cut short the paroxysms. Expiratory pressure on the thorax is also helpful. Potassium iodide is of real value when administered regularly for periods of from four to six weeks, and interrupted by a course of Fowler's solution for a fortnight. In certain cases hypodermics of morphine and atropine may be necessary during an acute attack.

ABSCESSES OF THE LUNG.

In fatal cases of pyemia and of bronchopneumonia, multiple small abscesses are found postmortem, while tuberculous cavities, varying greatly in size, are either single or multiple. A larger, single, non-tuberculous abscess is of rare occurrence in children and is mostly due to staphylococcic or to streptococcic infection; it may follow influenza, or pneumonia, or it may result from the aspiration of a foreign body, or occasionally from the breaking down of a caseous bronchial gland. The physical findings are rather confusing, resembling those of effusion as well as of consolidation; the exploring needle may, or may not, demonstrate the presence of pus.

Symptoms.—The symptoms in a large pulmonary abscess are similar to those of empyema—an irregular hectic temperature, ranging between 99° to 102° F., sweats, progressive emaciation, and marked leukocytosis—so that a differential diagnosis of encapsulated empyema, or of gangrene, or of a bronchiectatic cavity is often difficult. The abscess may cause an empyema either by breaking into the pleural sac or by opening into a bronchus and thus lead to a spontaneous cure.

Treatment.—The treatment is practically that of empyema—incision after exploratory puncture, drainage, and the very best after-care. The pleura is usually adherent, but, if not, an adhesion may be artificially produced by packing the wound.

GANGRENE OF THE LUNG.

This disease, due to anaërobic bacteria, is rare in children and seldom diagnosed during lifetime. It affects only weakly children in the

course of bronchopneumonia, measles, typhoid fever, tuberculosis of the lungs, and of the bronchial glands, or it may follow aspiration of a foreign body (especially after laryngeal diphtheria), or septic embolism or thrombosis originating in distant parts of the body.

Pathological Anatomy.—The lower lobes are usually affected. The process is generally a diffuse one, with small, grayish-green scattered areas; only exceptionally does it involve a whole lobe or an entire lung. These foci are often wedge-shaped with their bases directed towards the outer surface of the lung, indicating a thrombotic or embolic origin. They may soften later on when they emit a characteristic gangrenous odor, and often produce large cavities with ragged necrotic walls, partially filled with fetid pus.

Symptoms.—The constitutional symptoms depend to a certain extent upon the disease of which the gangrene is a complication; they usually resemble those of a typhoid state. The peculiar gangrenous odor of the breath if associated with a dirty green, or a sanguineous expectoration that contains necrotic lung tissue separable into three layers, is an unfailing diagnostic indication, but death often ensues before active decomposition and sloughing in the lungs has taken place. The physical signs are those of bronchopneumonia.

Prognosis.—A fatal termination is the rule, although modern surgery has several cures to its credit.

Treatment.—Medicinal treatment should be directed toward maintaining the strength of the patient by stimulants and proper feeding, and toward arresting the process by inhalations of antiseptics and the administration of the oil of creosote.

ACQUIRED ATELECTASIS, OR PULMONARY COLLAPSE.

Pathology.—This is distinct from the congenital form, inasmuch as the lung had expanded after birth. It is not infrequently seen in young children. It may result from pleuritic or pericardial effusions so compressing portions of the lung that, although the bronchi remain open, the alveoli collapse; after a certain time tissue alterations occur which make their reëxpansion difficult, or even impossible.

Similar changes take place when a bronchus is blocked, for instance, by a foreign body; in time the air beyond the obstruction becomes absorbed, and that part of the lung collapses; finally, atelectasis may be acquired by marantic, or greatly debilitated children with feeble inspiratory force; this is especially the case in rickets, where weak musculature and very flexible ribs interfere with thorough aeration of the lungs.

Symptoms.—The symptoms are more or less marked, and resemble those of the congenital form. Rapid superficial respiration, inspiratory dyspnea, and cyanosis of varying degree, with a feeble vesicular murmur, and normal or even subnormal temperature point strongly to atelectasis.

Treatment.—The treatment follows the principles laid down for the congenital form of the disease. As a prophylactic measure, all young

infants should be taken up, or turned over on the abdomen, several times a day. This is especially necessary if the child is suffering from rachitis.

EMPHYSEMA.

The peculiar structure of the lung favors the occurrence of acute emphysema in young children, and especially so if the patient is rachitic, but most cases of emphysema subside quickly after the termination of the primary disease. Usually three forms are differentiated.

In one, called compensatory, the alveoli of certain portions of the lung become overdistended from an effort to compensate for deficient aeration of other parts; as, for example, in pneumonia, tuberculosis, and when there is diminished expansion caused by adhesions, thickened pleura, or external pressure.

The second form causes the same pathological lesions, but depends more upon an obstruction to expiration or a prolonged cough—the vesicles becoming distended because the air cannot readily escape—as seen in pertussis, bronchitis, and all forms of laryngeal stenosis.

A third kind, so-called interstitial emphysema, in which air from ruptured air vesicles escapes into the interstitial and even subcutaneous tissues, is very rare indeed.

Pathological Anatomy.—The adult form of more or less general emphysema with permanently enlarged alveoli and dilated right heart is hardly ever seen in young children. The thorax is not barrel-shaped, but may be abnormally full just beneath the clavicle. On opening the chest, the lung does not readily collapse. Certain localized areas, principally around the apices and anterior borders, are raised, feel velvety, look whitish-yellow, and crepitate under the finger. Usually only a few septa are ruptured, but in more severe cases (pertussis) blebs of fairly large size may be seen.

Symptoms.—The signs in cases of emphysema which occurs in acute pulmonary disease are not very distinctive. There may be hyperresonance over the emphysematous areas, prolonged expiration, and, perhaps, diminished tactile and vocal fremitus. The ordinary dulness of heart, liver, spleen, and consolidated lung is not infrequently masked or diminished by overlying emphysematous lung tissue. The symptoms are principally those of the underlying disease, and on its subsidence disappear in a comparatively short time. Fat, pasty-looking children with flabby musculature are especially prone to the affection.

Treatment.—The treatment resolves itself chiefly into that of the primary disease; *i. e.*, bronchitis, asthma, pertussis, etc. Outdoor life is essential, but undue exposure must be avoided. Both country and mountain air offer undoubted advantages. During convalescence, a course of arsenic, interrupted after a few weeks by the administration of small doses of the iodides, certainly does good. In the rare subcutaneous forms of emphysema, the skin is punctured, the air pressed out, and mechanical means—that is, compression of the chest during expiration—may be used to relieve the dyspnea.

CHAPTER XV.

DISEASES OF THE HEART.

CARDIOVASCULAR diseases are more common in children than is generally assumed, often beginning in early childhood, especially among the poorer classes. They are less complex and appear in fewer forms than in the adult, but are by no means less severe. In order to understand and to differentiate diseases of the heart, the physician must have accurate knowledge of the anatomy and physical peculiarities of the circulatory apparatus, both in infancy and childhood, and of its mode of development.

As the primary cardiac tube develops, it is gradually transformed by constriction, sigmoid twisting, and the formation of internal septa, into the fetal heart. Fetal circulation differs chiefly from the circulation after birth in the fact that, with the exception of the umbilical veins, which carry pure arterial blood, most of the bloodvessels contain a mixture of arterial and venous blood; that is, the systemic and the pulmonary circulations have not as yet become distinctly established.

The Heart.—In children the organ is relatively larger than in adults, as are also the lumina of the bloodvessels. As a natural consequence, the blood-pressure in early childhood is lower than in adults, and increases toward puberty as the relative narrowness of the arterial system increases.

At birth the heart weighs from one-half to one ounce, doubles this weight by the end of the second year, increases to two and one-half ounces by the fifth year, and to about five ounces by the fourteenth year; thus the most rapid increase is in the first year and toward puberty.

In this connection it is interesting to note that, while the total body weight of the adult is twenty times as great as at birth, the adult heart is only fifteen times its original weight; also that, while the walls of the right and left ventricles are in early infancy almost equal in thickness, the left is twice as thick as the right toward the end of the sixth year.

The circumference of the heart increases but little during the first five years, but the heart muscle grows stronger; therefore there is no corresponding increase in the size of the cavities. After the fifth year, however, the increase in the size of the cavities corresponds more nearly to the increase in the size of the organ. While the total size of the heart becomes twelve times as great between infancy and adolescence, the aortic orifice becomes only three times as large as at birth.

In fact, the obstacles which the cardiac muscle has to overcome find expression in a gradual rise of the blood-pressure which, in the first and second year, reaches 80 to 85 mm.; at the seventh year is between 90 and 95 mm.; from the eighth to the tenth year is 95 to 100 mm.; and thence to puberty is 100 to 110 mm.

The Pulse.—In childhood the pulse is very rapid, the number of beats varying from 120 to 140 at birth to 100 to 120 at the end of the first year, then diminishing by about five beats a year until the tenth year, when the average is about 80 for boys and 90 for girls. Both its rate and regularity are influenced by even trivial causes, such as crying, excitement, accelerated respiration, and sleep.

The Apex Beat.—The location of the apex beat varies not only in different children, but also in the same child at different times, following no definite rule. Generally speaking, it is a little higher in infancy than it is later. Up to the fourth year it is found just outside the nipple line; from the fourth to the seventh year at the nipple line; subsequently a little lower, but well within the mammary line, according to the shape and development of the chest, and the position the child is in. It moves laterally as the child turns, and sinks when it assumes the erect or horizontal posture. Up to the fifth year it is usually found in the fourth interspace, and later sinks to the fifth.

In recent years great progress has been made in the study of functional disorders and diseases of the heart in childhood. But, in order to investigate these with an open mind, one must disregard experience gained from the study of the adult heart, upon which degenerated arteries, chronic bronchitis, emphysema, chronic renal disease, and poisons (alcohol and syphilis) exert such a powerful etiological action, these factors being practically non-existent in childhood, many forms of cardiac disturbance at this early age being non-organic in nature.

This period of life has its own problems and peculiarities. Not only is the vasomotor tone of the arteries unstable, but the nerve-controlling mechanism, central as well as peripheral, and even the heart itself, show great instability. They are readily disturbed because not yet fully developed. We must therefore allow ourselves considerable latitude when considering physiological cardiac manifestations, and be cautious in regarding them as pathological.

Even real lesions which affect the auricular and ventricular muscular tissue may, if limited in extent, be completely compensated by normal growth. In discriminating between those signs which are of no significance and those which point to actual disease, it is extremely important to consider what the heart is capable of doing when the child is at rest, and also when exercising, rather than the sounds produced. While in later life the changes produced by heart disease are degenerative and fibrotic in nature, in the early stages of heart disease in children these changes are inflammatory in nature, and do not, as a rule, produce dyspnea, cough, or edema.

General Symptomatology and Diagnosis.—The symptoms of cardiac disease depend chiefly upon the anatomical structure and physio-

logical function of the organ, and the nature of the morbid process affecting it. Although the symptoms are less complex than in adults, since secondary conditions in remote regions (such as dropsy or congestion of parenchymatous organs) are usually lacking in early childhood, yet the physical examination calls for greater precision, since everything depends upon accurate observation, and the chief difficulties are in deduction, not in method. In regard to the mode of examining the heart it may be well, however, to state that in the case of struggling children it is best to use the ear, and, instead of a stethoscope, a phonendoscope which, being flat, can easily be placed in the axilla or under the back without raising the child from the bed.

The pulse gives us little information, there being neither arterial degeneration, marked irregularity in rate, nor variability in strength; but the continuous rapidity and progressive diminution in volume in acute pericarditis and the arterial spasm and heightened blood-pressure in renal disease are quite significant. Disturbance of rhythm is not at all uncommon; indeed, arrhythmia may be said to be almost physiological in infancy, especially during sleep. It is also apparent after infectious diseases, and in the early stages of tuberculous meningitis may be marked, but is of significance only in diphtheria. In older children the sole assignable cause for it is a neurotic disposition. In contradistinction to arrhythmia due to myocarditis, the harmless arrhythmia of childhood disappears with increase in the pulse rate due to fever or exertion.

Transitory tachycardia, which appears during the course of fevers and is readily produced by excitement, is most marked in neurotic children. Even in the older neurotic child, excitement and exertion often make the pulse extremely rapid and compressible; this, however, is of no significance, even if accompanied by a diffuse apex beat. Accelerated pulse rate induced by moderate exertion, and not abating after three minutes of rest, suggests weakness of the heart muscle.

Paroxysmal tachycardia, in some cases hereditary, has been observed in older children. Although not amenable to treatment, it usually subsides under hygienic management and judicious exercise. Hochsinger mentions a permanent tachycardia due to the pressure of enlarged bronchial glands upon the pneumogastric nerve.

Bradycardia is common in infants. In older children it is frequently associated with arrhythmia, and is most likely to occur in diphtheria, but may be pronounced in infectious myocarditis. In rare cases, it accompanies appendicitis.

MURMURS.

There are cardiac disturbances which produce no changes that can be detected by percussion, these being apparent only on auscultation. In children, as a rule, cardiac sounds are loud; only in very young infants are they dull, or even impure.

Bruits, or murmurs, are frequently found, and during the first two years of life they usually indicate congenital disease of the heart. At

this early age acquired as well as accidental murmurs are rare. Albuminuria does not always signify renal disease; neither does a cardiac murmur in itself necessarily mean heart disease. The important thing is to decide whether the working power of the heart is, or is likely to be, affected, and to decide this question a clear distinction must be made between organic disease in childhood (congenital or acquired valvular, or acquired pericardial) and so-called functional or accidental (extracardial or cardiopulmonary) disorder. Functional murmurs are very common in childhood, especially during school age. In school children one often finds a soft systolic murmur over the pulmonic area. Some observers with modern sensitive stethoscopes may find them in even a great number. When faint they are, however, more interesting to the observer theoretically than of practical importance to the patient. While there is no doubt that distinct murmurs of this type have been found even in children under two years of age, they are, as a rule, rare before the fourth year.

Three types of functional murmurs may appear between early life and puberty; (1) a pulmonary systolic murmur, so common that it may be considered almost physiological; (2) a cardiopulmonary murmur; (3) a systolic murmur of cardiac atonicity which, although produced at the valve, does not signify cardiac disease. In the other types, accidental murmurs are probably caused by the more rapid circulation together with decreased viscosity of the blood. ·

1. **Pulmonary Systolic Murmurs.**—In this type of murmur the maximum intensity is between the second and third costal cartilages close to the sternum, and is either limited to this area, or, in exceptional cases, may extend a few inches to the left, sometimes being faintly audible even at the apex. This bruit with an accentuated second pulmonic sound is almost physiologic in children, and usually disappears at puberty. Its origin is, no doubt, at the pulmonary valve, and it is probably due to the rush of the blood through a narrow ostium into the relatively wide pulmonary artery. The murmur, itself, may be very distinct, but it is always soft, blowing, and short, and follows closely upon the first sound. It is not accompanied by any symptoms of heart disease, and has no bearing on the future health of the child. It should be differentiated from a congenital pulmonary murmur, which is usually rougher, more rasping, and of longer duration, and is often accompanied by dilatation of the right ventricle and weakening of the second pulmonic sound.

2. **Cardiopulmonary Murmurs.**—These murmurs are extracardiac in origin, and are caused by changes in the lungs which occur during the systolic contraction of the heart. Whether the murmurs are the result of aspiration of air, friction of the lungs and the heart, or the simultaneous vibration of the lungs and heart, has not been fully explained. The bruit varies during the respiratory movements, being intensified at inspiration, diminished at expiration, and disappearing when breathing is suspended. It is usually heard between the apex and the ensiform cartilage, but may vary with the child's position and the rate of

the heart; it is always systolic in time, soft and superficial in character, and in rare cases only is accompanied by a whiffing sound during the diastole. It not infrequently diminishes or disappears altogether under firm pressure of the stethoscope on the elastic chest wall. Fortunately, neither the patient nor his heart is affected, no symptoms are produced, and it is of no prognostic significance.

3. **Intracardiac Murmurs.**—Presumably valvular in origin, these murmurs may occur without the existence of any organic lesion, and are probably due, in part, to temporary insufficiency of either the cardiac muscle or the papillary muscles of the mitral valve, this causing reflux of blood with or without a systolic murmur, the mitral valves themselves remaining perfectly intact. A similar condition is often found during and after specific fevers, and disappears during convalescence. Both of these murmurs are probably due to atonicity of the cardiac muscle. The former appears in overgrown, neurotic girls, and passes away after rest or, in some children, on taking active exercise.

Anemia, in itself, cannot be considered the sole cause, because many anemic children never present a murmur; but in neurotic and neurasthenic children a disturbed or enfeebled condition of the central nervous system may manifest itself by debility which leads to cardiac murmurs. It must be understood that whenever general debility is present, whatever its origin, this is shared by the heart and causes a tendency to murmurs. Therefore, loss of tonicity of the heart muscle prevents the prompt and complete closure of the valve segments—in other words, the murmur is not valvular, but myocardial.

How can we differentiate these from organic murmurs? By considering the murmur itself, and by noting the presence or absence of constitutional local disease. A functional cardiac murmur is always systolic in time, usually soft, blowing, and short, and involves almost exclusively the left heart—that is, the pulmonic, rarely the mitral area. The heart sounds may be altered, but not absent; they vary more in time than do organic murmurs, being present at one time and absent at another, and also vary with the posture, as when the child lies down or sits up, and after exercise.

When cyanosis, dilatation of the right heart, and clubbing of the fingers and toes are marked, and associated with a loud rasping murmur, it is not difficult to recognize a congenital origin; but when the murmur is the only evidence it is sometimes hard to differentiate congenital from functional murmurs. However, for practical purposes, this is of little importance when other signs and symptoms of heart disease are lacking. Only long-continued observation will enable us definitely to determine the underlying lesion, and make an accurate diagnosis. Summarizing, we would say it is most important to realize that functional murmurs not only exist but are common in children, and that the mere presence of a murmur does not necessarily indicate disease of the heart.

Reduplication of the second pulmonary sound may occur in healthy

29

little children as the result of crying or excitement, the closure of the pulmonary valve preceding that of the aorta. A faint venous hum in older children is not infrequently heard at both sides of the sternum, diminishing when the child assumes the horizontal position. A similar, but long and almost continuous, bruit is sometimes heard to the right of the sternum at about the third intercostal space, and is increased during the systole; it apparently originates in the superior vena cava, but varies considerably. This occurs chiefly in the anemic child; but when dealing with the tuberculous patient it may suggest the possibility of compression by enlarged bronchial glands.

In deciding whether organic heart disease is acquired or congenital, the following points are worthy of consideration: loud, rough, musical murmurs without increase in cardiac dulness, or, in the first and second years, such bruits with a weak apex beat and increased cardiac dulness, as well as bruits preponderating in the pulmonary area, all indicate congenital heart disease; very loud mumurs all over the heart, without a thrill, indicate a patent septum. A systolic murmur with a thrill, its maximum intensity over the upper part of the sternum, and no cardiac hypertrophy, indicates in all probability an open ductus arteriosus. Arteriosclerosis, chronic myocarditis, poisoning by tobacco or alcohol, or a fatty heart is only exceptionally found when studying diseases of the heart in children.

CONGENITAL DISEASES OF THE HEART.

The complicated normal process by which the simple tube of the primordial heart becomes transformed into a system of contractile spaces is a masterpiece of art that even Nature does not always succeed in carrying out to perfection. It can readily be understood that deviations of the simple parts from the normal in size, direction, or position, due to disease or other adverse factors which must be in operation early in fetal life, should leave some defect in the heart's otherwise perfect structure. Another element, fetal endocarditis, or an inflammation set up soon after birth, may in itself cause cardiac disease. However, not all congenital cardiac diseases can be attributed to either mechanical or embryonic causes.

Most of these conditions are due, not to disease, but to faulty development. In 15 per cent. of the cases they form only one of several deformities, such as cleft-palate, undescended testicle, supernumerary or web-fingers, talipes, or microcephaly, and are evidently the expression of a general tendency to maldevelopment or, as in Mongolian imbecility, exhaustion products. Depressing influences which impair the mother's perfect power of reproduction (syphilis, for example), or poisons which act upon the embryo, such as alcohol, tobacco, lead, or mercury, are undoubtedly important etiologic factors, and give evidence of their effect sometimes in two or three members of one family. Most cases, however, are due to faulty development, and only very few to so-called fetal endocarditis.

In the order of their frequency lesions take place as follows: A defect in the ventricular septum; a defect in the auricular septum; pulmonary stenosis; patent ductus arteriosus; abnormal origin of the great vessels. In the great majority of these cases there are several lesions, consequently a correct diagnosis is difficult, and sometimes practically impossible.

Clinical Symptoms.—The most striking symptom is cyanosis, which is present in many severe cases, but absent in about 60 per cent. It may manifest itself at birth or soon after, giving the skin and mucous membrane a dark leaden color, if intense, and when slight a bluish
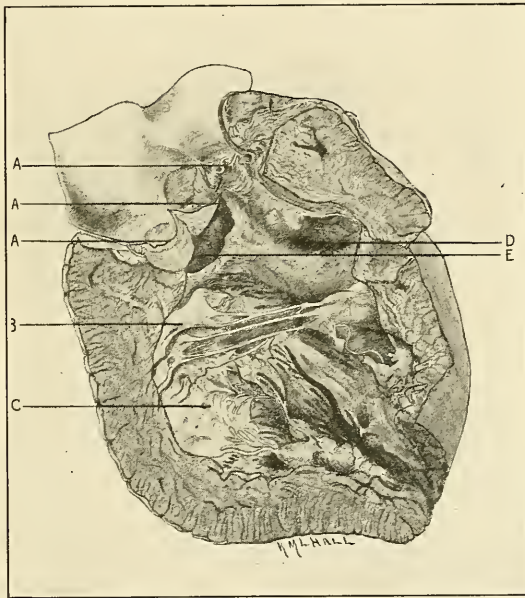
Fig. 32.—Infant's heart, showing congenital lesions. The right ventricle laid open, incision extending outward through the aorta, which communicates with both ventricles. The aorta is open and its valve leaflets are shown at *A A A*. Between *D* and *E* is the incision which extends outward through the pulmonary artery. *B* and *C* are leaflets of the tricuspid valve. *D*, the sinus that communicates with the pulmonary artery and constitutes the opening of the vessel in the right ventricle. *E*, the semilunar communication between the two ventricles just below the aortic orifice.

tint on coughing or crying; although healthy infants also may turn cyanotic during a prolonged crying spell. Some children show a slight bluish color only in the finger tips and toes. Marked cyanosis may not appear before puberty, and in other cases it becomes more obvious year by year. It has a certain value in prognosis, inasmuch as cases in which the cyanosis is severe usually do not live long, while in the less severe, or those in which it appears only in later life, the prognosis is more favorable.

This cyanosis seems to be partly due to obstruction of the lungs, deficient oxygenation, and to polycythemia—that is, 6,000,000 to

8,000,000 of erythrocytes, with an increased amount of hemoglobin. Cyanosis is usually accompanied by clubbed fingers and toes, probably owing to venous obstruction. The respiration in cyanotic cases is generally accelerated, sometimes difficult, and increases the tendency to acute bronchitis or bronchopneumonia,—the usual termination of congenital heart disease.

Edema of the face and lower extremities, dropsy, and epistaxis are comparatively rare. The pulse is usually small, rapid, and easily compressible; sometimes it is irregular in rate and variable in strength. These children are often inclined to diarrhea, and especially to vomiting which recurs for months; they develop slowly, are undersized, and are living problems of nutritional difficulty. Irritable or apathetic, easily fatigued and short of breath, complaining, perhaps, of pain over the heart, these children find walking increasingly difficult and climbing impossible. They finally cease to walk, and take to their beds, there to end their short lives, dying from the disease itself or some complication, usually pulmonary. Some, however, have lived for 15 to 25 years, and even beyond this.

Clinically, we distinguish three distinct groups of congenital diseases of the heart, as follows:

    I. Abnormal persistence of fetal conditions.
        (*a*) Patent ventricular septum.
        (*b*) Patent foramen ovale.
        (*c*) Patent ductus arteriosus.
    II. Deformities of valves.
        (*a*) Pulmonary stenosis or atresia.
        (*b*) Aortic stenosis.
        (*c*) Tricuspid stenosis.
    III. Abnormalities of vessels and of the cavities of the heart.
        (*a*) Abnormally large or small pulmonary artery.
        (*b*) Transposition of vessels.
        (*c*) Single ventricle with single or double auricle. (The latter is of no practical importance, because such children die within a few hours after birth.)

I. Abnormal Persistence of Fetal Conditions. (*a*) *Defect in the Ventricular Septum.*—This is the most common congenital lesion of the heart; and, as it is always due to a defect in development, it is frequently associated either with pulmonary stenosis or other anomaly of development, such as hare-lip, etc. The opening is usually located in the upper membranous part of the septum, and is about one-fourth to one-half inch in diameter; but the septum may be almost wholly wanting, in which case there may be scarcely any bruit present.

In the majority of cases, however, the defect manifests itself principally by a loud, rough, systolic murmur which, although heard all over the heart, is of maximum intensity in the second or third interspace close to the sternum, and is transmitted to the back, but not to the carotids. The pulmonary second sound is almost always accentuated, except in the cases where the opening is minute.

In the later stages of the disease a certain amount of hypertrophy and dilatation of the right ventricle becomes clearly distinguishable. There is usually no cyanosis, and the general health is so little affected that the lesion may be found accidentally rather than by intent, and the patient live to old age.

Diagnosis in early infancy of a defect in the ventricular septum is often not difficult if a diffuse systolic murmur is detected, with its maximum intensity close to the left side of the sternum, with no change in cardiac dulness, with an accentuated second pulmonic sound, and absence of cyanosis. In later childhood similar signs on percussion and auscultation may be found in diseases of the mitral valve; but the maximum intensity is at the apex, and the less rough and more localized character of the murmur, with, possibly, a history of rheumatic infection, will help in the differentiation. When the lesion is associated with open ductus arteriosus, pulmonary stenosis, or both, a positive diagnosis is often difficult, and the prognosis is poor.

(*b*) *Patent Foramen Ovale.*—This is of very frequent occurrence, but is not always recognized during life because it may give rise to no symptoms whatever.

(*c*) *Persistent Ductus Arteriosus.*—The ductus arteriosus should close during the first four weeks after birth by overgrowth of the cells in its inner wall. Under abnormal conditions, such as atelectasis or cardiac defects, these cells break down and the duct remains open. It seldom occurs alone, being usually associated with defects of the septum or the pulmonary valve.

During early life a systolic murmur is heard, its maximum intensity being at the pulmonary area, and probably caused by the whirl produced by the meeting of blood streams from the pulmonary artery and the aorta. The bruit is transmitted to the carotids. In later life a distinct thrill is felt over the base of the heart. The second pulmonary sound is accentuated; the upper portion of the sternum may be bulging. Hypertrophy and dilatation of the right ventricle as well as dilatation of the pulmonary artery rapidly take place, and may be demonstrated by dulness in the second intercostal space.

In uncomplicated cases the general health may be undisturbed for a long time. There is rarely dyspnea or cyanosis; on the contrary a deathly pallor is often seen. Subsequently there is a disposition to catarrh. The diagnosis can be definitely made only in cases uncomplicated by other congenital cardiac defects.

II. Deformities of Valves. *Pulmonary Stenosis.*—Of much greater clinical importance is pulmonary stenosis, which is one of the most common and serious congenital lesions due either to malformation or fetal endocarditis. The stenosis may be slight or grow to almost complete atresia, and may be situated at the pulmonary orifice in the conus arteriosus or in the pulmonary artery just beyond the valve. It is compatible with long life when association with other defects compensates for it; for instance, when there is a patent ductus arteriosus which permits the blood to flow to the lungs from the aorta, and

a patent ventricular septum which allows blood to flow to the left ventricle that cannot be forced through the stenotic pulmonary valve to the lungs.

Cyanosis more constantly accompanies pulmonary stenosis than any other congenital heart lesion. It is usually marked at birth, and increases to a dark slate color on crying or any other exertion. Clubbing of the fingers and toes is especially marked. There is a decided tendency to lung affections, dyspnea, dizziness, vertigo and attacks of suffocation.

A loud systolic murmur in the left second interspace and a lacking or weakened second pulmonary sound are characteristic. The murmur is not transmitted to the vessels of the neck except when due to a defective interventricular septum; the blood rushes directly to the left ventricle, and thence to the aorta. When associated with an open ductus arteriosus the second pulmonic sound may be accentuated, and the murmur together with a thrill be transmitted to the carotids. After a lapse of years dilatation and hypertrophy of the right ventricle become marked, and in many cases death occurs from pulmonary tuberculosis.

(b) *Congenital Stenosis of the Aorta.*—This condition may be found at the point of origin, at the entrance of the ductus Botalli, or throughout the entire aorta. If pronounced, life is sustained but a short time while the blood continues to circulate through the ductus arteriosus. Stenosis of the isthmus, *i. e.*, near the entrance of the ductus arteriosus, if not too extreme, can be successfully overcome by hypertrophy of the left ventricle. A systolic murmur is heard over the upper part of the sternum and the second pulmonic sound is normal. If the patient lives, collateral circulation is established through the intercostals and the internal mammary arteries, and the parts below the isthmus are better supplied.

III. Abnormalities of Vessels and of the Cavities of the Heart.—(a) Is not a common malformation. (b) Transposition of vessels is not uncommon, and is always found to be associated with some other congenital heart defect. The aorta may arise from the right ventricle, or the pulmonary artery from the left ventricle. Occasionally a common trunk may serve as the origin of both the aorta and the pulmonary artery. Dextrocardia, or transposition of the heart, is very rare. (c) This form of congenital heart disease is of little clinical importance, as the condition is usually incompatible with life.

PHYSICAL EXAMINATION IN DISEASES OF THE HEART.

Physical examination of the heart in children calls for the greatest care, everything depending upon accurate observation, since we get little or no information from the child. Generally speaking, the situation of the apex beat depends upon the development and shape of the chest, but it changes also with the position of the patient. As a rule, before the seventh year the impulse is found outside of the median

line, later at about the nipple line. Moderate displacement to the left occurs with cardiac hypertrophy, especially of the left ventricle, while if to the right it suggests hypertrophy chiefly of the right ventricle. Extreme displacement points to an outside cause, such as pleurisy or empyema.

As mentioned before, the pulse gives us no assistance except in acute pericarditis where we find continuous rapidity with progressive diminution in volume, and in renal disease where arterial spasm finds its expression in a hard pulse with high blood-pressure.

Inspection, also, is very unsatisfactory in children. The child must be stripped and examined in a good light. In infants the impulse is comparatively weak, while in the well-fed the chest walls are so well covered that the apex beat is invisible. Precordial prominence soon occurs in young children with rachitis, or after this disease; sometimes also in hypertrophy, and when there is a large pericardial effusion. Valvular disease or adherent pericardium produces a wide area of visible pulsation.

Palpation.—In *palpation* the whole hand should be pressed gently but firmly over the precordial area while the child is sitting with its body bent slightly forward; the finger will then quickly find the point of greatest impulse. Thrills resulting from congenital or acquired heart disease are easily located, and the force of the systole gauged.

Percussion.—Percussion is best performed in infants while recumbent, and in older children in the upright posture. It should be done very lightly, bearing in mind the extreme thinness and elasticity of the chest wall. The area of relative cardiac dulness is proportionately larger in children than in adults, and is the greater the younger the child. Failure to allow for this might lead to an erroneous diagnosis of hypertrophy. As regards the relative and absolute cardiac dulness, we would say (avoiding too great refinement) that for most clinical purposes it is sufficient to remember the following points concerning their location:

RELATIVE CARDIAC DULNESS.

At one year.	At six years.	At twelve years.
Upper limit: Second costal cartilage.	Second intercostal space.	Third cartilage.
Right margin: Right parasternal line.	Slightly more to the left.	About midway between the parasternal and the right border of the sternum.
Left border: Slightly beyond the apex beat.	Slightly beyond the apex beat.	Slightly beyond the apex beat.

ABSOLUTE CARDIAC DULNESS.

At one year.	At six years.	At twelve years.
Upper limit: Lower border of third rib.	Upper border of fourth rib.	Lower border of fourth rib.
Right margin: Left border of the sternum.	Left border of the sternum.	Left border of the sternum.
Left border: Does not quite reach the mammary line.	Does not quite reach the mammary line.	Does not quite reach the mammary line.

Method of Percussion.—The best results seem to be obtained by percussing on the left side from the anterior axillary line toward the heart in lines parallel with the 2d, 3d, 4th, and 5th interspaces, and on the right side from the right mammary line along the 4th interspace toward the sternum. For clinical purposes, it is quite sufficient to make out this most projecting part, which corresponds to the curve of the right auricle to the right of and behind the sternum. The younger the child, the more difficult it is to mark out the area of absolute cardiac dulness, and it is certainly more important to determine the apparent size of the heart than the area not covered by the lungs.

Auscultation.—All sounds are comparatively louder and are diffused over a greater area in children than in adults. In infants the muscular quality of the first sound is rather of the type of the fetal *tic tac.* In children the second aortic sound is normally weaker than the second pulmonic. Reduplication of the sounds is not infrequently heard in children who are excited, and may mean nothing more than that the valves of the two sides do not close at exactly the same time. A diminution or absence of the second pulmonic sound is invariably found in pulmonary stenosis; an increased apex beat with a rather diffuse, dull, first sound, strongly points to cardiac hypertrophy; while a weak and distant apex beat is highly suggestive of either dilatation of the heart or pericardial effusion. The latter, which is comparatively rare, is confirmed by a friction rub and pain on pressure over the precordial area.

THE GENERAL SYMPTOMS OF HEART DISEASE.

The general symptoms of heart disease are no indication of the severity of the affection, and even in children with serious lesions they may be slight or often totally absent. As already mentioned, cyanosis, although a frequent accompaniment of congenital heart disease, may be absent, as evidenced by the pallor usually seen in those who suffer from aortic disease. In mitral disease congestion is the rule; while in malignant endocarditis the grayish color and pinched expression often tell the story. Attacks of fainting, sometimes difficult to differentiate from *petit mal* and the functional disturbances of puberty, are not rare.

Muscular weakness, anemia, malnutrition, and shortness of breath on exertion, all call for an examination of the heart. Marked edema, digestive disturbances, and angina are seldom met with in childhood; but restlessness, vomiting, subnormal temperature, and pallor are grave symptoms in children suffering from cardiac disease.

Although the recuperative power of a child's heart is considerable, yet in various diseases there is an increased tendency to heart complications, and the child is more liable to an aggravation of an already existing cardiac affection than is the adult; valvular lesions are apt to be multiple, pericardial adhesions and cardiac hypertrophy frequently occur, and when circulatory disturbance is of severe type and begins early in life, growth and development are considerably retarded.

ACUTE ENDOCARDITIS.

The usual clinical classification of endocarditis, myocarditis, and pericarditis is based on the most striking symptoms of each of these diseases; but it is well to emphasize the fact that in any of these conditions the child's entire heart is more or less involved. Simple endocarditis may, in rare cases, be intra-uterine in origin, and is then usually localized in the right heart. Postnatal endocarditis, in which group the majority of cases belong, seldom occurs under three or four years of age, because at least 75 per cent. of these cases are due to acute rheumatism and chorea, which are both rare before the fifth year. It appears most frequently between the sixth and twelfth years, being less common in children of the well-to-do, because to some extent preventable.

While the chief cause in all of these cases is rheumatism or chorea, yet it not infrequently follows the infectious fevers, especially scarlet fever, influenza, and pneumonia, also pleurisy, bronchitis, and pyemic conditions. A cold, damp season, overexertion in anemic or rapidly growing children, shock, fright, bad habits, severe digestive disturbances, all favor the infection, especially when rapid growth and development at puberty severely tax an already weakened heart.

Summarizing, we would say that endocarditis almost invariably appears as a secondary infection. Even when apparently primary, thorough investigation will prove that either rheumatism (of which a cardiac affection may be the first manifestation) or, in rare cases, tuberculosis is the real cause. The Streptococcus viridens is the principal offender in the smouldering type of endocarditis (malignant); but staphylococci, pneumococci, gonocci, the Bacillus typhosus, and the Bacillus coli communis are also not infrequently found.

Pathology.—The pathological lesions are mostly confined to the left side of the heart, involving the mitral, and much less frequently the aortic valves. The hyperemia, swelling, inflammation of the cells, and wart-like excrescences on the free margins of the thickened valves do not essentially differ from those in the adult. There is, however, the difference that, when the lesions are slight, apparently complete healing takes place, and the ulcerative form is extremely rare in children.

Symptoms.—These are often remarkably latent, the insidious onset being expecially noteworthy. There may be vague pains, the so-called growing pains, or repeated attacks of angina and sore throat; but in many cases nothing draws attention to the diseased heart until breathlessness disproportionate to the exertion becomes noticeable.

Unless the first symptoms are masked by an underlying disease, weakness, pallor, failing appetite, and slight irregular fever ensue, the latter disappearing when the child rests in bed, and reappearing when he gets up. After an interval, the pulse gradually grows rapid, irregular, and low in tension; subsequently a characteristic blowing murmur makes its appearance, at first gradually increasing, later,

possibly, diminishing, being systolic in time and frequently accompanied by a thrill.

Sometimes a child is brought to the physician for symptoms directly traceable to the heart, *i. e.*, palpitation, precordial pain, and shortness of breath; but whenever a stiff neck, fleeting pains in the joints, and so-called growing pains are complained of, it is advisable to examine the heart.

In the earliest stages there may be no bruit at all, only very slight increase in the area of cardiac dulness, and sometimes only a rapid and irregular pulse. A deep dulness should be carefully noted, because dilatation occurs easily and early; in fact, it may be the first or only sign of an affection of the heart.

Careful estimation of the size of the heart is often more enlightening than the auscultation of a murmur, but it requires considerable training. In the early stages, more or less dilatation of the left side is often caused by toxic or inflammatory changes in the cardiac muscle itself; but in advanced cases special attention should be paid to the right border, because dilatation of the right heart, unless quickly relieved by bleeding, leads to lividity, ascites, enlarged liver, and extreme dyspnea.

Hypertrophy of the heart following increased work against higher pressure in the arterial system (especially in nephritis) causes bulging of the precordia more readily in children, because the chest walls are so pliable. In auscultating, certain differences between the signs in children and in adults must be taken into consideration. Aortic bruits are very rare in children, but do occur after mitral disease, the murmur being systolic in time; later, when the valves shrink, a diastolic bruit may be added. These murmurs are usually audible in the second or third interspace close to the sternum or over the middle of the sternum.

The vast majority of cases are due to lesions of the mitral valve, manifesting themselves by a weakened first sound at the apex, which becomes impure, and later may be replaced by a bruit which is blowing and soft, and may be transmitted, The second sound is at first clear and well defined, but soon becomes less distinct and is reduplicated. After a time a short puff develops; this is less loud and shorter than the systolic bruit; thus we have the well-known double murmur at the apex.

On account of its splendid reserve power the left heart easily overcomes the obstruction, so that for a long time there may be no accentuation of the second pulmonic sound, and no enlargement is demonstrable by percussion or the *x*-rays. Hypertrophy and dilatation occur much later in children than in the adult. It must be borne in mind that the clinical picture in infants differs considerably, and a diagnosis can seldom be made, chiefly because the infant or child has always had a congenital murmur, due to a congenital lesion, and the endocardial murmur having developed subsequently and in addition to the congenital one it was, therefore, not noticeable.

The course of the disease shows great variations, and a fatal issue

usually occurs in infants when myocarditis and pericarditis complicate matters, or malignant endocarditis supervenes. Acute endocarditis ends in chronic valvular disease except in the few cases in which, within a few weeks, or months, the murmur gradually disappears and the slightly affected valves to all appearances return to the normal. Malignant endocarditis, which is very rare in children, is usually secondary to simple endocarditis, or may follow erysipelas, scarlet fever, septicemia, and infection of the oral cavity or of the bladder.

Diagnosis.—As a rule the diagnosis cannot be made until the emboli lodging in the kidney, brain, intestine, or lungs lead to infarcts or multiple abscesses; this is chiefly because cardiac murmurs and dilatation often do not appear until shortly before death, which usually occurs within a few weeks. Clinically, it presents symptoms of typhoid fever or pyemia; *i. e.*, fever, prostration, sometimes delirium, an enlarged spleen, and meningeal symptoms.

The diagnosis sometimes presents considerable difficulty. First, accidental murmurs must be excluded. Only when a daily careful examination reveals a bruit of gradual onset, which is persistent and more or less uniform in character, may we feel sure of existing endocarditis. Sometimes the symptoms themselves may be so marked as to suggest the disease; but it is only by carefully eliciting the physical signs that the suspicion may be verified and the diagnosis confirmed. On inspection a rapid diffuse apex beat, with sometimes an undulating motion over the precordia, may indicate cardiac overactivity. Palpation confirms the strong, irregular heart action. On percussion, even in the early stages, increased cardiac dulness, especially on the left side, is not infrequently found.

But it is chiefly by auscultation that we are enabled to decide as to the character of the murmur, this depending on the valve involved and the nature of the valvular lesion. Examination of the heart in children is greatly simplified by the fact that in them acute endocarditis is almost invariably limited to the left side. The murmur may be single or multiple, systolic, diastolic, or presystolic in time, and soft, blowing, harsh, grating, or rough in character. As mentioned before, in the majority of cases the mitral valves are involved.

With mitral regurgitation we find a soft, blowing, systolic murmur the greatest intensity of which is toward the apex; it is usually transmitted outward to the axilla, and is often distinctly audible posteriorly between the scapula and the spine.

Mital stenosis occasions a presystolic bruit which is not transmitted, but is usually accompanied by a thrill, and is loudest between the mitral area and the apex.

More often than with a single murmur we have to deal with a double, sharp, presystolic mitral murmur, followed immediately by a systolic murmur which almost drowns the first sound. The systolic murmur of aortic stenosis is loudest over the aortic area, is transmitted to the carotids, and, especially in younger children, is audible over the middle of the sternum. Aortic regurgitation is characterized by a diastolic

murmur, heard principally between the second and third intercostal spaces.

Differential Diagnosis.—In children pericardial murmurs are to be distinguished, as in the adult, chiefly by the triangular area of cardiac dulness, the base of the triangle corresponding with the apex beat, the apex pointing toward the great vessels, and gradually so increasing that the cardiohepatic angle and Traube's area below the heart tend to disappear. Difficulty in differentiating functional murmurs is really experienced only when we are dealing with a soft, blowing, mitral lesion. The accidental murmur in these cases is inconstant, changes more or less, may even disappear on change of position, and, as a rule, is not distinctly transmitted. Under proper care it usually passes away after a short time. There should be no accompanying cardiac hypertrophy.

Prognosis.—The prognosis of endocarditis is always grave. The disease is usually fatal in infants, the danger to life in older children being rather remote. Some may remain in a more or less invalid condition, but the case does not necessarily go from bad to worse; in fact, the greater number recover more or less completely if treated efficiently from the beginning.

But in some cases, even with the best of care, the valves are impaired for life, and as long as evidence of rheumatism persists there is danger of further cardiac mischief. Considering that the tendency to rheumatism and chorea is greatest between the fifth and twelfth years, the earlier the first attack of heart disease occurs, the greater is the probability that the condition will be aggravated by subsequent attacks; consequently, after puberty the chances for comparative comfort steadily improve.

Pericarditis is the one complication most to be feared; many cases end fatally within a month or two, and all lead to adhesions which gradually cause hypertrophy and dilatation, and end in ruptured compensation. On the other hand, it cannot be too strongly emphasized that loud murmurs, unquestionably of rheumatic origin, may disappear completely, and thus they warrant a hopeful outlook, especially when of recent origin; even in advanced cases we may hope for considerable improvement. Ulcerative endocarditis, fortunately extremely rare in children, probably in all cases ends fatally within a few weeks.

Treatment.—It is the mild case that presents the most serious problem, because, owing to the lack of objective symptoms, the parents are not easily convinced of the gravity of the disease. The remedy *par excellence* is rest, and rest in bed. Whether we are dealing with a mild or a severe case, our chief aim is to tax the heart as little as possible. No strain or stress of any kind should be allowed; even the use of the arms and legs had better be restricted, especially in the first stage, which is the time when most damage is done. This should be kept up for weeks until the heart becomes more regular, less excitable, and can endure slight exercise tests, and the treatment must be insisted upon in spite of the protests of both parents and children.

In this connection it may not be amiss to emphasize the importance of sleep as the best means of securing rest. It may even be necessary to resort to drugs, as codein or bromide, for this purpose, especially in the more advanced stages of the disease. Ordinarily a comfortable bed, a quiet, darkened room, and a tactful nurse are sufficient; in order to avoid too frequent disturbance of the child, it is advisable to give medicine and food at the same time.

An ice-bag, half-filled with shaved ice and applied over the precordium usually quiets and slows the heart. Children, however, do not always take kindly to this measure, and if it makes them irritable or restless it had better be removed for a short time, and then applied again. Of course, it cannot be used on the infant.

Diet.—No tea or coffee should be given. In the beginning the child's food should be mainly liquid, varied by the addition of gruels, broths, and custards—not a strictly milk diet, of which the child soon becomes surfeited, and which it refuses, consequently is apt to suffer from undernourishment.

With progressive improvement, eggs, bread and butter, and stewed fruits may be added; later poultry, fish, and simple puddings provided they do not distend the stomach. Saline laxatives may be necessary to insure a daily bowel movement.

Drugs.—Salicylates combined with double their amount of alkali, or aspirin, which is less irritable to the stomach, should be given whenever there is evidence of rheumatism or the least suspicion of endocarditis, unless the latter can be traced to diphtheria or an infectious fever. This medication should be kept up until the fever and rapid cardiac action abate, and even then small doses may be given at short intervals.

It is advisable to continue this medication for at least a month, and afterward to repeat it for one week in every month to ward off any possibility of a recurrence, especially when there are signs of tonsillitis or other manifestations of a rheumatic tendency. Ordinarily digitalis can be dispensed with; when, in severe cases, stimulation seems imperative, camphor and caffeine are preferable, while for excitement and restlessness five to ten grain doses of sodium bromide and $\frac{1}{8}$ grain of codeine, p. r. n., are permissible and even advisable.

If, during convalescence, the pulse is well below 100, the child may gradually be propped up in bed for an hour a day and, if the heart can stand it, very cautiously gotten up in a reclining chair. So, little by little, and chiefly guided by the heart action, the child may increase its exercise every day, and gradually resume its normal life. Diseased tonsils and adenoids should be removed.

CHRONIC ENDOCARDITIS OR ACQUIRED VALVULAR DISEASE.

Valvular defects usually follow an endocardial infection which, in the majority of cases, is rheumatic in origin; therefore they rarely originate before the third year, but almost always appear after the fifth. There is, of course, no definite time limit for the acute condition,

which, indeed, may gradually run into the chronic. It is true that the preceding acute endocarditis is often either not suspected or not diagnosed; nevertheless, the most important etiological factor is usually a rheumatic affection; arteriosclerosis and poisons, such as alcohol or syphilis, being rarely the cause of valvular disease before puberty.

Symptoms.—These may make their appearance very insidiously, and depend in some degree upon the character and the site of the lesion. They may be latent for years, the child looking well, appearing vigorous and showing good powers of endurance up to puberty, the cardiac defect often being discovered accidentally during a routine examination of the heart. Some patients are rather irritable, easily tired, pale at times, and complain of headache after mental or physical exertion. Other subjective signs are slightly disturbed compensation, or nosebleed which may be repeated and violent and accompanied or followed by headache, ringing of the ears, dizziness which is increased on stooping, or shortness of breath on climbing or running upstairs, and persisting for some time after exertion.

Children very rarely complain of cardiac palpitation; but a stubborn catarrh of the respiratory passages, an irritating little cough, coryza, and swollen turbinals, diminished appetite, vague pains in the back and nape of the neck, may all indicate an undiscovered cardiac affection.

With the advent of disturbed compensation these symptoms become exaggerated and, in addition, are associated with a consequent mechanical impairment of the circulation as shown by cyanosis of the lips, the fingers, and the toes, enlarged veins of the neck, scalp, and thorax, enlarged and, possibly, tender liver, edema of the limbs and of the peritoneal, pleural, and pericardial cavities. Dyspeptic symptoms are conspicuous in all of these cases.

Physical Signs.—In valvular disease of the heart in children the physical signs are for the most part identical with those in the adult, although children do not usually complain of palpitation, the cardiac impulse being visibly and palpably strong. When the lesions are severe the bulging of the precordia is the more marked the younger the child. Often no such bulging is found; for years there may be no perceptible difference on percussion; the area of dulness may be only slightly extended to the left, as revealed by *x*-ray examination.

Murmurs.—Organic murmurs are usually distinct, often loud and rough, especially over the apex, and they are heard not only over their respective areas, but very often in older children, always in younger ones, also in the back between the shoulder blades and at the angle of the scapula. Only when the compensation is ruptured is the area of cardiac dulness greatly extended on the right side, this suggesting pericardial effusion for which, in fact, it is often mistaken.

The Pulse.—If the cardiac muscle is involved, we may occasionally have arrhythmia. The special symptoms of disease of the different valves are, generally speaking, similar to those in the adult; but it must be emphasized that the most common clinical variety is mitral

disease, which before puberty occurs about twenty times more frequently than do aortic valvular defects. Mitral insufficiency often appears alone, and represents the common form in early life. The murmur is more or less synchronous with the first sound, is best heard over the apex, and is transmitted to the left. The murmur is more or less diffuse, accompanied by an accentuation of the second pulmonic sound, and the signs of hypertrophy.

Although the two latter points are important in differentiating organic from merely functional murmurs, it must be stated that they may not appear for quite a little time. Mitral stenosis usually complicates mitral insufficiency, but may not be detected until years after the appearance of the first lesion, while as an isolated or primary lesion it is hardly ever found before puberty. The bruit may be almost inaudible or may become noticeable only after exertion; as a rule it is rough, terminates sharply with the first sound (presystolic), is loudest at the apex, and strictly localized.

Aortic disease is rarely observed before the tenth or twelfth year, insufficiency and stenosis appearing at about the same time. It is usually of serious import, and may cause sudden death.

Stenosis is characterized by a systolic murmur, heard chiefly at the right border of the sternum and second interspace, and transmitted upward into the carotids. The second aortic sound is weak. With aortic stenosis there is necessarily present hypertrophy of the heart, although this is not as marked as in aortic insufficiency. If there is no hypertrophy the murmur is probably an accidental (hemic) one, therefore it would necessitate a revision of the diagnosis. Aortic insufficiency causes a prolonged diastolic murmur, coincident with or replacing the second sound. It is loudest at the left side, and is transmitted along the sternum toward the apex; associated with it is most marked cardiac hypertrophy which, of course, is especially noticeable when beginning failure of compensation causes dilatation of the left ventricle. There is also intense throbbing of the carotids.

Tricuspid insufficiency is usually caused by dilatation of the right ventricle after a serious mitral lesion. It gives rise to a systolic murmur, the greatest intensity of which is over the lower part of the sternum. The jugular veins stand out prominently, and may show systolic pulsation.

As regards the future course of cardiac disease, there is no doubt that children may recover from mitral insufficiency, even though we have been careful to classify as such only those cases in which the bruit appears gradually after an acute rheumatic infection, and persists for months, or even for years. In the majority of cases, however, the disease remains more or less latent until puberty. There are cases in which serious manifestations appear after years of apparent latency, this probably being due to the fact that the cardiac muscle gradually becomes weaker and can no longer continue to compensate, *i. e.*, because of the necessarily increased amount of work, or because a fresh endocarditis is superimposed upon the old lesion.

In the rare cases where death occurs in a comparatively short time

(within a few months or years) we, no doubt, are dealing with more than a valvular defect; in all probability with pericarditis or myocarditis. In a general way, we may say that in children a purely valvular defect can be far better compensated and for a longer time than in the adult. On the other hand, when failure of compensation appears the end is close at hand.

Diagnosis.—In making a diagnosis of organic heart disease in children one cannot be too cautious. The same underlying principles as in the adult must be our guide. But there are added difficulties to be overcome in eliminating the different varieties of accidental murmurs, and it is sometimes almost impossible correctly to diagnose mitral insufficiency in children between five and fifteen years of age. On the one hand, in organic mitral insufficiency, enlargement of the left ventricle and accentuation of the second pulmonic sound may be absent for a long time; yet when the systolic murmur is not permanent, is loudest in the mitral area, is equally audible whether the patient is lying down or sitting up, and is associated with a heaving apex beat due to hypertrophy of the left ventricle, it cannot be clearly distinguished from that due to mitral regurgitation.

Furthermore, it must be differentiated in those cases of defective intraventricular septum when only the upper membranous part of the septum is patent. In these cases there is a loud systolic murmur, audible over the whole heart (which may or may not be hypertrophied), the murmur being most intense in the third left interspace, is transmitted to the back but not to the carotids, and may or may not be accompanied by an accentuated second pulmonic sound. Finally, it is well not to overlook the fact that in anemic school children absolute cardiac dulness is not infrequently increased on account of the insufficient expansion of the margin of the lungs overlying the heart. If, in such a case, a hemic murmur is audible, the affection can easily be mistaken for organic disease.

Treatment.—Prophylaxis practically resolves itself into the most careful treatment of any manifestation of rheumatism, including sore throat or chorea, also the observance of cardiac hygiene, with rest, and the use of the salicylates. Considering the fact that these rheumatic affections have a tendency to recur, a weekly course of salicylates and alkalies once a month, or once in two months, seems a wise precaution. Whether we adopt passive treatment—that is, restriction of the ordinary routine of life—or active measures by drugs, etc., nothing should be done until the diagnosis is definitely settled.

As long as compensation is maintained satisfactorily, no special treatment is necessary. The child need not be frightened, but the parents should be told of the condition so that they may guard against any excessive physical exercise, cycling, football, baseball, rowing, etc.; alcohol, tea, and coffee should be prohibited. While the whole life should thus be regulated, and the child carefully watched, there is no reason why it should be condemned to a life of invalidism; and the less interference the better for the patient.

Careful regulation of the exercise taken by the child is necessary from time to time, the physician being guided by the symptoms and physical signs of cardiac disease. This will enable him to decide upon the proper management of the individual case. All possibility of reinfection must, as far as possible, be prevented by avoiding exposure to cold and wet, and by having diseased tonsils, the teeth, the kidneys, or any general disturbances of the alimentary tract, promptly treated.

Country life, especially in a warm and dry climate, is very beneficial, and a gradual hardening of the body against changes of temperature is to be recommended. Woolen undergarments should be worn, especially in the cold season. As soon as cardiac failure appears, rest becomes imperative, and such other measures or treatment should be applied as will improve the functional power of the left ventricle. All nervous strain should be removed, and rest for the heart and refreshing sleep secured by the judicious use of the bromides, morphine, or codein, and a carefully regulated diet; for the first eight days a somewhat restricted milk diet is probably the best. If these measures do not avail, it is then time, just as in the adult, to use digitalis, or any of the other heart stimulants, such as camphor, caffeine, adrenaline, or morphine.

ACUTE PERICARDITIS.

There is a mild form of pericarditis marked by a slight deposition of fibrin or a slightly increased amount of pericardial fluid. This may occur in the course of infectious diseases, and, as a rule, is not diagnosed unless it goes on to suppuration, as sometimes happens in scarlet fever and measles. But the more severe form of pericarditis is of real clinical importance. It occurs in general sepsis of the newborn and in young infants, and is usually purulent in character. Between the second and the seventh years the infection spreads from an adjacent focus in the lungs and pleuræ, and is then usually caused by the pneumococcus. After this age most pericardial effusions are due either to tuberculosis or to repeated rheumatic infections. In these cases the functional capacity of the heart is threatened by adhesions which are liable to form when the effusion becomes absorbed.

Summarizing, we would say that rheumatic infection is by far the commonest cause of pericarditis in children; but we must not lose sight of the fact that this pericarditis is always associated with endocarditis and more or less involvement of the cardiac muscle. While often obscured by the primary disease, that is, by sepsis, pleurisy, or pneumonia, the clinical picture of rheumatic endocarditis is clearcut except when another underlying etiological factor (such as endocarditis or arthritis, and in older children chorea) produces additional symptoms.

Symptoms.—Acute pericarditis often begins with vomiting, fever, headache, loss of appetite, and restlessness, soon followed by pallor of the face, and later by lividity of the mucous membrane. The patient is short of breath, and a frequent, dry, irritating, little cough often

30

interferes with sleep. Pain over the precordium, or a sense of oppression, is not always complained of, even when well-marked friction sounds are heard.

The pulse usually becomes more rapid and weak. Upon examination we may hear slight friction sounds which in the beginning may resemble a soft, blowing, endocardial bruit; but these sounds are variable, usually localized at the base, and sometimes increased by pressure with the stethoscope. The well-known triangular area of increased cardiac dulness with its apex toward the aortic area bears witness to the progressive accumulation of pericardial fluid. Traube's space is obliterated, and the cardiohepatic angle as well, because the exudate collects somewhat more to the right and left of the apex.

The impulse often persists in palpable form for a long time, and the heart sounds become more and more distant. The greater the amount of exudate the greater the tax on the heart, which is manifested by a pulse rate of 140 to 180, or even 190. Cyanosis, the fulness of the large cervical veins, marked swelling of the liver, and general edema are all due to congestion.

When no absorption takes place, the feeling of oppression and the dyspnea continue to increase, and lead to cardiac weakness which ends fatally within a short time. Even absorption of the fluid does not necessarily indicate a cure, because the resulting adhesions of the pericardium considerably embarrass the heart, and quickly lead to hypertrophy. Death may be postponed for only a few years, especially when, as is often the case, there is a complicating endocarditis.

Diagnosis.—As in the adult, the diagnosis rests upon the gradual increase of the triangular area of dulness, which obliterates the cardiohepatic angle and Traube's semilunar space. The fact that the area of absolute cardiac dulness increases or extends more rapidly than that of relative dulness is a decided help. While a large effusion can hardly be overlooked unless there be an accompanying left-sided pleural effusion, it is often difficult definitely to diagnose a small effusion, especially in children; often the x-rays alone can decide this question. The x-ray picture enables one also to differentiate these cases from cardiac dilatation. When it is a question in a given case whether the exudate is serous or purulent in character, a distinct increase in leukocytes favors the latter.

Prognosis and Course.—These vary according to the individual conditions. Purulent cases, unless saved by surgical intervention, are likely to end fatally, while the non-purulent, which are chiefly of rheumatic origin, offer a good prognosis as to life, but not as to complete recovery; since, even though the fluid be absorbed, adhesions between the two layers and complete obliteration of the pericardial sac occur much more often in children than in adults, and, by crippling the heart permanently, mark the beginning of a life of invalidism which may be more or less prolonged.

In mild cases, the outcome depends largely upon the management. As regards the duration of the acute symptoms, one week is the

minimum in mild cases, while in severe ones they may last much longer; the average time being about three weeks. Recovery at best is slow, and only partial.

Treatment.—The prophylactic measures discussed under endocarditis are applicable here also, especially precautions against recurrent attacks of rheumatic fever, the removal of diseased tonsils, etc. The first principle in the management of the patient during an attack is absolute rest in bed. The child should be allowed to do nothing that others can do for him, and should not get up until he can do so without any symptoms of heart strain.

In cases of rheumatic origin, sodium salicylate should be administered, combined with a double quantity of bicarbonate of soda— 5 grains of the salicylate and 10 grains of the soda every three hours to a child of five years. For delicate children and those who cannot tolerate the salicylates, aspirin is preferable, in doses of 8 to 10 grains, three times a day, for a child ten years of age.

The early and intermittent use of an ice-bag seems to quiet the heart action and reduce the fever. It should be continued only until the acute symptoms begin to subside; later, warmth seems preferable. In my experience counterirritants and blisters are of little use, therefore not recommended.

The diet should be simple, nutritious, light, and somewhat restricted during the acute stage, consisting principally of cereals, milk, eggs, custards, and toast.

Extreme rapidity of the pulse may be counteracted by the judicious use of tincture of strophanthus, which is superior to digitalis inasmuch as it does not so easily derange the stomach. When the patient is restless or distressed, morphia, in $\frac{1}{10}$ to $\frac{1}{50}$ of a grain doses, relieves pain and, at the same time, acts as a cardiac sedative. In the very young, sodium bromide should first be tried; if it has no effect, paregoric should be given in doses of 10 to 20 drops at three hour intervals, p. r. n., and in children above two years of age $\frac{1}{6}$ to $\frac{1}{10}$ of a grain of codein (not more than three doses in the twenty-four hours).

Excessive exudate may call for surgical intervention. This, in fact, offers the only chance in an otherwise hopeless case of purulent effusion. When we are dealing with a serous effusion, a mere puncture of the pericardial sac may be sufficient. This puncture is usually made by means of a small aspirating trocar, either in the fifth right interspace, one and one-half inches to the right of the right sternal border, or in the fourth or fifth left interspace, at the same distance from the left sternal border, thus avoiding the internal mammary artery. After the trocar is introduced, the fluid is allowed to drain out slowly.

ADHERENT PERICARDITIS.

Adherent pericarditis is not unusual after the eighth year, and may follow a single or repeated attacks of pericarditis (usually rheumatic in origin), although the preceding disease may not have been recog-

nized. The adhesions left after absorption of the exudate greatly hamper the heart, and thus give rise to hypertrophy and to dilatation of both ventricles.

Morbid Anatomy.—The pericardium may be greatly thickened, or both of its layers, the parietal and visceral, may become adherent, thus partially or completely obliterating the pericardial sac. Sometimes even external adhesions form and bind the heart to the chest wall, the pleuræ, the mediastinal structures, or the diaphragm. When tuberculosis is the etiological factor, caseous deposits and tubercles are found. Other changes are usually also present, especially a low-grade myocarditis which causes increasing dilatation and weakening of the cardiac muscles.

Symptoms.—These are often latent, and the condition may remain unrecognized in those cases where murmurs of valvular lesions or the symptoms of an underlying constitutional disease obscure the clinical picture. As a rule the symptoms are those of gradually increasing insufficiency, especially a rapid, weak, irregular pulse, easily affected by exertion, which also causes dyspnea and cardiac distress, finally leading to gradual failure or to sudden death. The apex beat is diffuse, often feeble, or even absent.

The characteristic sign of adhesions between the pericardium and the chest wall is a systolic retraction of the latter at or near the apex of the heart. This must, however, be carefully differentiated from the systolic sinking of the intercostal spaces with marked apex beat which is found in cardiac hypertrophy.

Almost equally characteristic is a diastolic rebound of the intercostal spaces over the greater parts of the pericardium, associated with diastolic collapse of the jugular vein, and immobility of the heart—the position of the apex and the cardiac dulness in general not changing with the position of the child.

All other symptoms, such as dyspnea, precordial distress, cyanosis, small pulse, and signs of hypertrophy and dilatation, may also be due to endocarditis or myocarditis, and can be regarded as suspicious of obliterative pericarditis only when there is a history of a preceding attack of pericarditis, from which the patient has not recovered.

Percussion proves that the cardiac dulness is increased in all directions. The children are sickly; they do fairly well as long as they stay in bed, although the small, rapid pulse, and difficult respiration on the least exertion, show their true condition. As soon as they get up, however, they are easily fatigued, become cyanotic and dyspneic, and willingly take to bed. Sometimes the child may show considerable improvement for a while, then the heart again breaks down, with accompanying fever, bronchitis, pleurisy, and subsequently edema. The liver and spleen enlarge, ascites develops, and sooner or later death ensues.

Diagnosis.—The diagnosis is often merely a surmise when neither the friction sound nor the exudate of preceding acute pericarditis has been observed. It may, however, be correctly made when

symptoms of myocardial insufficiency follow an acute attack of pericarditis. Although x-ray examination may be a valuable aid, diagnosis is often very difficult, especially in the absence of the characteristic sign of systolic chest retraction. Adherent pericarditis in its later stages, characterized by lost liver and ascites, can be differentiated from cirrhosis of the liver by the concomitant cyanosis, the small, rapid pulse, and extreme dyspnea.

Prognosis.—This is unfavorable in all but the mildest cases, death invariably occurring after a few months or years. For this reason all treatment is unsatisfactory, and must be symptomatic, as in myocardial insufficiency. On account of the accompanying myocardial degeneration, even digitalis is unavailing. Embarrassing pleural or abdominal effusions should be removed by puncture. Placing the child on a nourishing diet, giving it good general care, and, perhaps, injections of fibrolysin, are all that can be done to insure the patient's temporary comfort. Operation with a view to freeing the external adhesions of the heart can hardly be considered in childhood.

MYOCARDITIS.

Myocarditis is rarely a primary disease. In most cases designated as such it is probable that the underlying affection has escaped detection. The acute form, usually of mild degree, and due to the action of bacterial toxins, frequently accompanies the acute infectious diseases, especially diphtheria, scarlet fever, and sepsis, also typhoid fever, influenza, whooping-cough, and pneumonia.

These toxins appear to affect chiefly the nerves and muscle tissues, giving rise to parenchymatous degeneration, while bacteria, such as streptococci, staphylococci, and pneumococci, which invade the heart by way of the blood stream, seem to attack the interstitial tissues. As emphasized elsewhere (page 457), the inflammation of endocarditis or pericarditis usually extends more or less to, and affects, the myocardium, thus producing a real carditis, which affects the whole organ. The chronic form is much more rare in children than in adults, and causes no arterial degeneration.

Pathology.—The cardiac muscle is pale in color, soft, and somewhat friable. Seen under the microscope, the changes are very distinct, round-celled infiltration and granular hyaline fatty degeneration of the muscle fiber being apparent. This explains why the weakened heart muscle so readily undergoes dilatation.

Symptoms.—There may be no appreciable symptoms, and death may occur very suddenly. When the affection develops during the course of an infectious disease, the symptoms are usually indefinite, and become more distinct only during convalescence. At this period the clinical picture of the acute infectious disease masks wholly or in part the symptoms and physical signs of the associated myocarditis. The first and most characteristic sign is a persistently irregular pulse, either more rapid or slower than normal. This is especially significant

during or after diphtheria, scarlet fever, or pneumonia. Later a systolic murmur is heard over the apex, the apex beat becomes more feeble, the pulse rapid and flickering.

A child in this condition must be watched most closely, as dyspnea, cyanosis, coldness of the extremities, pallor, and restlessness may become urgent at any moment, and require the most prompt treatment. Usually there is no edema, and pain in the chest and precordial distress are comparatively rare. Diminished blood-pressure, weakness of the cardiac impulse and heart sounds (the second sound may almost entirely disappear and the pulse at the wrist be lost) are danger signals of impending cardiac dilatation.

In fulminant cases diphtheritic myocarditis, purely toxic in origin, may cause sudden death during the first few days, or in the second or third week, or during convalescence, and as late as the sixth to the eighth week. Pallor, cold extremities, cyanosis, vomiting, and syncope are ominous signs. In mild cases an irregular pulse on even slight exertion may persist for months after recovery.

The rapid pulse of the first week, and bradycardia during the second week of scarlet fever, are in all probability also due to myocarditis. Typhoid fever is much less apt to affect the cardiac muscle in children than in adults, manifesting itself chiefly by arrhythmia, and rarely leads to heart failure.

Chronic myocarditis sometimes follows the acute form of myocarditis, but develops more frequently after diphtheria. There may be tachycardia, more rarely bradycardia, often arrhythmia, and, upon slight exertion, dilatation of the heart. The most important symptom is increasing muscular insufficiency, just as in the later stages of chronic valvular disease.

Diagnosis.—This is doubtful, especially during the febrile period of infectious diseases, in which case the abnormal pulse is often due to vasomotor disturbance or an affection of the nervous system. Increased cardiac dulness with acute nephritis, which follows scarlet fever, for example, might be mistaken for that of myocarditis if it were not for the hardened pulse. Diagnosis is mainly based upon an irregular and weak pulse following infectious disease, upon sudden attacks of cyanosis and collapse, and upon a weak and impure first sound while the second may be almost lost.

Prognosis.—This is always doubtful; an advanced case seldom permanently improves. Early and sudden death is almost invariably to be expected, especially in diphtheria, unless the most careful treatment can be instituted early; this should be continued for months or for years.

Treatment.—The treatment of chronic myocarditis is purely symptomatic, and is directed to lightening the work of the heart and overcoming the disturbed circulation. When it follows even a mild attack of an infectious disease, absolute rest in bed is necessary. The child should not be allowed to lift its head from the pillow, a trained nurse should be constantly on the watch to prevent any physical exertion or

mental excitement. Needless to say, that even the child's struggles against certain forms of treatment may be detrimental.

A warm sponge bath should be given daily, and a daily movement of the bowels without straining be secured. No alcohol or coal-tar derivatives are allowable, for fear of further damaging the already hampered heart. In older children an ice-bag applied over the heart, if not objected to, may give relief. Easily digested food should be given in small quantities at frequent intervals, so that there will be no distention of the stomach by either food or gas; a liquid or semi-liquid diet, containing not too much fluid, answers best.

For the cardiac weakness digitalis is usually of little use. When the pulse becomes weak, and dyspnea and cyanosis appear, camphorated oil may be given subcutaneously in doses of ten to twenty minims. Caffein sodium salicylate or caffein sodium benzoate may be administered hypodermically in 1-grain doses, three times a day, to a child three to five years old; strychnine, $\frac{1}{200}$ of a grain for a child one year old, and double this dose at three years, can be repeated three or four times daily, and may be pushed even to its physiological effect—slight twitching of the muscles of the face and extremities; adrenaline—30 to 40 minims of a $\frac{1}{1000}$ solution in normal saline—may be given three to six times daily subcutaneously. It is probably best to give a combination of several of these drugs, although in somewhat smaller doses.

How long should a convalescing child be kept under strict observation? It should stay in bed as long as the pulse is irregular, or more rapid than normal. When the heart seems to be regular, the head may be raised by the addition of an extra pillow, subsequently the shoulders and head may be elevated until, guided by the cardiac action, the child may be allowed to sit up, then to stand up, and finally to walk.

THE BLOOD IN INFANCY AND CHILDHOOD.

THE blood of an infant or child shares the instability of the pulse, temperature, and respiration which is characteristic of childhood. Just as some slight disturbance will give a hyperpyrexia with very rapid pulse in a child, so are grave anemias and high leukocyte counts resultant from causes which would scarcely alter the blood of an adult. This exaggerated response, which occurs whether the cause be pathological or physiological, is due to the as yet incomplete establishment of the function of producing blood, further shown by the slow regeneration of the cellular elements after hemorrhages and the prompt appearance of abnormal red and white cells whenever the blood-forming organs are taxed. The fact that certain toxins are acting on the child's blood for the first time, must also be taken into consideration. With this knowledge in mind, we consider the blood findings in early childhood far less significant than in adult life.

Physical Properties.—Color.—The infant's blood is darker for the first few days, but quickly assumes the normal color.

Reaction.—The reaction is always alkaline.

Specific Gravity.—Specific gravity is about 1.065 at birth and fluctuates till the end of the first year when it reaches the normal level of about 1.055. The specific gravity is uninfluenced by the number of cells or other causes, such as food, rest or exercise, but is in direct proportion to the amount of hemoglobin.

Hemoglobin.—There is a greater amount of hemoglobin at birth than in adult life due to a higher percentage in the individual red cell, the amount ranging from 104 to 110 per cent. of that found in adult life. This higher percentage sinks quickly after birth and at six weeks 55 to 65 per cent. is normal with proportionate color index. From the sixth month to the second year, it is lower than in adult life, and, in the normal child during this time, it may go as low as 60 per cent. and is rarely above 80 per cent.; then there is a steady rise till, at six years, it reaches normal adult percentage. As a general rule, the percentage of hemoglobin is higher in boys than in girls. In all the various forms of anemia, the amount of hemoglobin is reduced.

Red Cells.—The number of erythrocytes is increased at birth and counts run as high as 8,000,000. They remain high for about twenty-four hours, but begin to diminish the second day. Throughout childhood the number of oscillations in twenty-four hours is greater than in the adult. A few nucleated red cells may be seen at birth, but these disappear in the course of a few days; they are rarely found

in the blood of normal children but are common in premature infants. Erythrocytes vary greatly in size, shape, and staining reaction; they are particularly susceptible to structural damage from trivial causes.

Normoblasts are normal-sized erythrocytes with a dark staining nucleus. They are found in both mild and severe anemias, in disease of the bone marrow and in severe leukocytosis.

Megaloblasts are very large red cells, 10 to 20 micromillimeters (μ) in diameter, with nuclei of various shapes and polychromatic. They occur in very young infants and in pernicious anemia, and indicate blood regeneration.

Microcytes are small red cells from 4 to 10 μ in diameter. They are seen in chlorosis and severe anemias.

Poikilocytes are irregular shaped cells occurring in severe anemias.

The number of red blood cells is diminished in both primary and secondary anemias. The red cells are increased during cyanosis, at the sea coast and in high altitudes.

Leukocytes.—There is a physiological leukocytosis at birth averaging 20,000 to 30,000, followed by a rapid reduction to about 12,000 during the next ten days. At the end of the first year, they average 9000 in number, and vary between 6000 and 12,000 during childhood.

Normal Varieties.—The following types are seen normally in the child's blood:

Small Mononuclears.—These comprise 40 to 60 per cent. of the leukocytes in the blood of children. They are the size of a red corpuscle and have a large deeply staining nucleus.

Large Mononuclears.—This variety is two or three times larger than the small lymphocyte and has an oval nucleus not exactly centrally placed, which stains faintly. It comprises from 4 to 18 per cent. of leukocytes in children.

Polynuclears.—These cells are smaller than the large lymphocytes, and the nuclei stain deeply with basic dyes. The nucleus has the appearance of being composed of several parts joined together. The protoplasm of the cell contains small granules which stain with neutral dyes. While these cells make up 60 to 70 per cent. of the total number of leukocytes in the adult, in childhood they average from 20 to 40 per cent. of the white cells.

Esinophiles.—These resemble polynuclears but the granules are larger and stain deeply with acid dyes. They average 2 to 4 per cent. of the leukocytes in childhood.

Mast Cells.—This name is given to leukocytic cells whose granules stain only with basic dyes, not at all with triacid. They may be either mononuclear or polynuclear, and are never seen in early childhood. They are differentiated from the finely granular basophiles as follows: The mast cell has a pale nucleus which is usually single and may be indented and takes basic stain very faintly, while finely granular basophiles are darker and lobed or polynuclear, the nuclei taking a moderate amount of basic stain. The finely granular basophile is the size of a polyneutrophile, while the mast cell is much larger.

Pathologic Forms.—Myelocytes have a single rounded nucleus and contain neutrophilic granules. There is also a non-granular type observed in severe anemias.

Mononuclear eosinophiles appear in the pathological blood. They resemble polynuclear eosinophiles and are called eosinophilic myelocytes.

Leukocytosis.—In adults, leukocytosis is practically always caused by an increase in the polynuclear cells, but in childhood there is a greater tendency for the lymphocytes to increase, as is seen in pertussis, hereditary syphilis, scurvy, and rachitis.

A polynuclear increase in children indicates the presence of actual pus formation, septicemia, or severe intoxication. It also occurs in pneumonia and diphtheria. A physiological increase follows cold bathing, eating, exercise, and massage. An eosinophilia is noted in certain cases of chronic skin diseases and is also seen when there is infestation by intestinal parasites.

Leukocytes may be practically unchanged as to number in measles, mumps, German measles, and most forms of tuberculosis in children. Actual lymphocytosis in acute infections of childhood sometimes resembles the blood picture of acute lymphatic leukemia.

A leukopenia is a diminution in the number of leukocytes and is frequently observed in cases of malnutrition, the latter weeks of typhoid fever, certain forms of pure tubercular infection, and various anemias.

A normal leukocyte count, or a leukopenia in a disease which usually produces a leukocytosis, indicates an overwhelming infection with no reaction on the part of the patient, and justifies an unfavorable prognosis.

The diagnostic value of leukocytosis, in a few of the commoner diseases of children, is emphasized below:

Diphtheria.—There is a marked leukocytosis in most cases, and, although there is no direct ratio between the degree of leukocytosis and the clinical findings, the most severe cases are accompanied by the highest leukocyte counts. The presence of myelocytes is regarded by some observers as a fatal prognostic sign.

Empyema.—A rise in the leukocyte count during the course of, or following a pneumonia, with no changes in the lungs to warrant it, is strongly suggestive of a beginning empyema.

Meningitis.—Septic meningitis is always accompanied by a leukocytosis. Cerebrospinal meningitis causes a leukocytosis in most cases, but in the tubercular form it is by no means constant, and, if present, only to a slight degree.

Pertussis.—From the very onset of this disease, there is usually a high leukocytosis, especially under five years.

Pneumonia.—There is a marked leukocytosis in pneumonia which increases from the onset of the disease to the crisis, when it falls rapidly. The usual count is from 20,000 to 30,000, although it frequently reaches 50,000 and may go above 100,000. A low leukocyte count is against a pneumonia, but when it occurs it is an ominous sign.

Tuberculosis.—There is no increase in the number of leukocytes unless a mixed infection exists.

Scarlet Fever.—A leukocytosis is present which reaches its maximum on the second or third day of the disease, and is of value in differentiating this disease from measles.

Blood Platelets.—These are small colorless cells, usually found in clumps, and have no known significance, except that thrombi are more apt to form when they are increased.

Blood Dust.—This is the title given to the colorless granules ranging up to 1 μ in diameter, found in normal blood. They are highly refractile and possess a dancing motion, but their function has not been discovered.

ANEMIA.

In childhood, as in adult life, we have two forms of anemia, the primary and secondary.

Secondary anemias are more common and much more easily produced in children, frequently reaching an alarming degree. Primary anemias are rare, and the distinct types are less clearly defined.

Secondary or Simple Anemia.—Secondary anemia is that condition of the blood in which the changes found are the result of disease, poor hygiene, or other causes acting on the general system and causing an impoverishment of the blood, the blood-forming organs being involved.

Etiology.—Hemorrhage is very apt to produce a well-marked anemia in children, if at all severe or prolonged. Malnutrition is the common cause of anemia in infancy. Anemia follows many of the acute infections, the severity depending on the previous condition of the child and the intensity of the disease. Syphilis, tuberculosis, rachitis, malaria, and scurvy all produce a secondary anemia. Poor food, lack of fresh air, prolonged grief or worry and severe mental strain are other important factors. The poor physical condition of a mother during gestation may be responsible for an anemic child. The administration of certain drugs and the action of certain toxins generated within the body cause anemia. Children with chronic heart disease or nephritis are usually anemic.

Symptoms.—Pallor of the skin and mucous membranes and pearly white conjunctiva are the common visible signs. The child may or may not be emaciated, but is listless, languid, fatigues easily, has a poor and capricious appetite, and is apt to be irritable and restless. Headache, fainting spells and hemorrhages from the nose or other mucous membranes are frequent. Enuresis is common and edema may be present in the dependent tissues with no albuminuria. This is due to the poor circulation, which also causes the child always to feel cold, and to become cyanotic and dyspneic on exertion. The heart sounds are weak and, after the third year, hemic murmurs are usually present. Fever is not constant, but the child is subject to repeated and prolonged chills. The spleen is usually enlarged—the liver may be.

Blood Picture.—The blood changes are much more marked in the secondary anemia of children than in adult life. If the anemia be a mild one, there may simply be a reduction of the hemoglobin and red cells, generally with a leukocytosis. The blood of the infant or very young child easily reverts to the embryonal type and the presence of microcytes, megalocytes, normoblasts, and megaloblasts, is noted. Poikilocytosis and polychromatophilia are frequent. The hemoglobin may fall to 20 per cent. or even below, and the red cells be less than 1,000,000 in severe cases. The average anemic child has a hemoglobin content of from 40 per cent. to 50 per cent., with the red cells about half their normal number.

Diagnosis.—The diagnosis is quite simple if the etiological factor is apparent, but in the absence of a known cause it is difficult. The blood picture may reveal many of the embryonal types of cells with an indifferent cell count, and render the differentiation between a severe secondary anemia and one of the primary anemias impossible. Under treatment, however, the secondary anemia usually improves, while the primary anemia progresses in spite of all remedial measures.

Prognosis.—The prognosis depends on the severity of the anemia and the cause. Leukocytosis is regarded as a bad sign. The presence of pathological red cells, a high color index, and very low hemoglobin content or red cell count indicate a severe grade of anemia in which the outcome is apt to be unfavorable.

Treatment.—Unless the case be very severe, the removal of the cause soon results in a marked improvement and return to normal. Fresh air, good hygiene and nourishing food are absolutely necessary. Iron is perhaps the one best drug, and is given to young children and infants, preferably, in the form of the saccharated carbonate, albuminate or peptomanganate. Iron citrate is far more effective when given intramuscularly than when given by mouth. One to 2 grains may be given daily and will cause a rapid and lasting increase in the hemoglobin and number of erythrocytes. No untoward effects occur if the injection is given carefully and all forms of anemia respond very well to this treatment. Older children may be safely given the same preparations as adults. Arsenic, in the form of Fowler's solution, may be given advantageously, where iron is not tolerated, or in conjunction with the iron.

The Primary Anemias.—By the term primary anemia, we mean those conditions of the blood due to changes in the blood-forming and blood-destroying organs and tissues of the body. Primary anemias are very rare during childhood.

Chlorosis.—Chlorosis is a primary anemia of gradual onset and runs a chronic course in which relapses are common. It is characterized by a reduction of hemoglobin wholly disproportionate to the diminution of the red cell count.

Etiology.—This is a disease of late childhood, occurring about puberty. Although a few cases have been reported before the fifth year, these are probably cases of chlorotic blood and not true chlorosis.

It is usually seen in girls, particularly of the blonde type; rarely in boys. Heredity is regarded as a possible factor. The exciting cause is unknown, but overwork, close confinement, undernourishment, and poor hygienic conditions predispose to it. Psychical elements, as shock, grief, care, fright and excessive mental strain should not be overlooked. Clark's view, that auto-intoxication is largely responsible, is supported by the frequent history of constipation elicited in these cases.

Pathology.—Postmortem findings have shown a hypoplasia of the heart and large vessels, and occasionally of the uterus and ovaries. There is usually a fatty degeneration of the heart muscle, and the right heart may be dilated with hypertrophy of the left ventricle. Death, if it occurs, is generally due to a complicating tuberculosis or round ulcer of the stomach.

Symptoms.—The symptoms are those of a simple anemia. Dyspnea, palpitation, attacks of vertigo, and syncope are common. The appetite is poor and frequently perverted. Constipation is the rule. The menstrual periods may be very painful and amenorrhea is generally present, probably as a result rather than a cause of the disease. Enteroptosis and hyperacidity are often found. The heart may show evidence of right-sided dilatation and hypertrophy of the left ventricle. A hemic murmur, systolic in time, is heard over the mitral and pulmonic areas. There is a venous hum in the neck with palpitation in the jugular veins. Thrombosis is more apt to occur in the femorals. The pulse is soft and weak. Headache, neuralgia, hysteria and chorea are among the nervous manifestations. There may be edema of the feet, puffiness of the face, and occasionally albuminuria. This disease is also called "green sickness" because of the yellowish-green tinge of the skin. Pigmentation is occasionally noted. There is apparently no emaciation and the patient may look plump and healthy.

Blood Picture.—The gross changes in the blood are the pale color, rapid coagulability, and low specific gravity. Under the microscope, the blood shows a moderate decrease in the number of red cells, the count ranging from 4,000,000 down to 1,000,000—rarely below. Nucleated red cells are common, normoblasts predominating; megaloblasts never occur. Each individual red cell is pale, smaller, and one is occasionally deformed. There is no leukocytosis, but a lymphocytosis with absolute diminution of polynuclears. The hemoglobin is greatly reduced, frequently as low as 35 per cent. to 40 per cent. The color index is low. The blood platelets are markedly increased, perhaps accounting for the shortened coagulation time.

Diagnosis.—The color of the skin and the blood picture are characteristic. Age, sex, and the functional derangement of the heart are of additional help.

Prognosis.—The prognosis in uncomplicated cases is uniformly good; death, if it occurs, is usually due to a complicating tuberculosis or gastric ulcer. The average case lasts a year; exceptionally, cases will go on for several years.

Pernicious Anemia.—This is the most severe form of anemia known. It pursues a progressive course, accompanied by constant blood changes, and rapidly goes on to a fatal termination.

Etiology.—It is not a common disease in early life, and the greatest number of the cases in children occur in later child life. No specific cause has been discovered, and the nature of this disease is as yet unknown. Syphilis, rachitis, and infestation by intestinal parasites have been observed in some cases. Others have followed a severe simple anemia or a grave nutritional disturbance. There is unquestionably a red cell destruction and that this is a toxic hemolysis is very probable. It is believed that these toxins are absorbed from the intestinal tract, and the peculiar deposits of iron in the hepatic cells have led to the theory that most of this red cell destruction occurs in the liver.

Pathology.—There is a severe anemia of all the viscera. Fatty degeneration is particularly marked in the heart, bloodvessels, liver, and kidneys. Capillary hemorrhages of the viscera and skin are common. The heart is larger than normal, and very flabby. The liver is enlarged, and histological findings of iron pigments in the liver cells are characteristic. This deposit of iron is sometimes noted in the intestinal mucosa. The mucous lining of the stomach is apt to atrophy. The spleen is enlarged, as it is commonly found to be in all blood diseases of children. The bone-marrow is darker and softer and simulates the embryonic state, the changes being due to reversion to an embryonal type and similar type of hemogenesis. It contains numerous nucleated red cells. The lymph nodes are enlarged and congested. Loss of flesh is not constant.

Symptoms.—The patient, while not emaciated, is very weak and frequently prostrated. The skin and mucous membranes are pale. The onset is very gradual and may be accompanied by no symptoms until the disease has progressed fairly well. There are disturbances of digestion, causing nausea and vomiting. The heart may be dilated, hemic murmurs are common, and the pulse is soft and full. Dyspnea follows exertion and there is restlessness and insomnia. Fever is generally present, but it is irregular. The urine is scanty with low specific gravity and contains no albumin. Dropsy may appear in the dependent tissues, and exceptionally in the serous cavities. Late symptoms include epistaxis and hemorrhages from other mucous membranes and ecchymoses of the skin. The duration of the disease is shorter in children than in adults, usually terminating in less than a year.

Blood Picture.—The drop is very pale, its fluidity is increased, and the coagulation time delayed. The specific gravity is low. The hemoglobin usually falls below 30 per cent., and at the time objective symptoms appear, the red cell count is 1,000,000 or less. During remissions, the number of red cells may tend to approach normal, but rapidly decreases again in the relapses, so that in the later stages a count below 500,000 is not uncommon. Abnormal red cells are abun-

dant and the megaloblasts exceed the normoblasts in number. Micro-blasts are rarely met with. Karyokinesis is common, and the non-nucleated red cells are deformed, variable in size, and show poly-chromatophilia. Diffuse basophilia, punctate basic degeneration, and basic nuclear remains are very common. Rouleaux formation is absent. The white blood cells are diminished as a whole, but in infants they may be increased. Lymphocytosis, which occurs in the severe cases, is accompanied by a diminution in the number of polynuclears. Myelocytes, if present at all, are very few in number, and eosinophilia is observed where there is infestation by intestinal parasites.

Diagnosis.—This is based almost entirely on the blood picture, of which the following are the distinguishing features: An extremely low red cell count, high color index, marked poikilocytosis, the predomi-nance of megaloblasts, and lymphocytosis with diminution of poly-nuclears. In early infancy, a severe secondary anemia will so closely simulate a pernicious anemia that it can be ruled out only by the absence of any causative factor.

Prognosis.—The great majority of cases end fatally. A few cases have recovered in which intestinal parasites or rachitis were associated and effectively treated. High color index, few normoblasts, and many megaloblasts foretell a fatal termination, while an increasing number of normoblasts indicates efforts at regeneration and is regarded favorably.

LEUKEMIA.

This is a disease in which there is a steady and persistent increase in the number of white blood cells. The red cells are but moderately reduced, although abnormal forms are frequently present. There are gross changes in the spleen, lymph glands, and bone marrow. Three forms are met with which differ as to the blood picture and pathological findings. They are the splenomyelogenous type and the lymphatic, which may be acute or chronic.

Etiology.—Children are sometimes the subject of this disease, but it rarely occurs in the purely myelogenous form. The lymphatic variety is more common, and congenital cases have been reported together with several in early infancy. Males are more liable to suffer from this disease than females. It may follow the acute infections and has been observed in cases of congenital lues, rachitis and malaria, but the relation is very obscure. Simple anemia has been followed by leukemia and in some cases it occurs primarily in a healthy child. Various observers have believed in its infectious nature, or that it may be a disease of the lymphatic system, or a later sequence of any anemia under certain conditions.

Pathology.—The spleen, lymphatic glands and bone marrow show distinct changes. In the lymphatic form there is a general glandular enlargement with slight enlargement of the spleen and liver, and little or no apparent change in the bone-marrow. The lymphoid structures of the alimentary tract may also be affected. In the splenomyelog-

enous type, there are marked changes in the spleen and bone marrow, and enlargement of the liver. The spleen is greatly enlarged, due to a chronic hyperplasia, and may occupy half of the abdominal cavity. In the early stages, it is soft and pulpy, and becomes firm and hard as the disease progresses, and a perisplenitis usually develops. Lymphoid masses are scattered throughout the body of the spleen, and microscopic lymphoid deposits are found in close proximity to the arteries. There is a great increase in the number of leukocytes in the spleen. The bone-marrow contains a greater number of red and white marrow cells, the fat content being reduced. Myelocytes and various other cells, including red cells in all stages of development, replace the fatty portion of the marrow. The enlarged liver is due to infiltration and formation of lymphomata, which cause a marked increase in size in either form of leukemia. Other organs are occasionally the site of lymphoid infiltration.

Symptoms.—The onset is gradual, but the progress is rapid and the course is apt to be more acute in childhood, although chronic forms occur. General weakness with pallor, dyspnea and digestive disturbances are first noticed. Sight, hearing and the nervous system are frequently deranged. Abdominal enlargement or glandular swellings may precede any of the above symptoms. Hemorrhages of the mucous membranes occur in the nose, stomach and intestines, and there may be hemorrhage from the kidney or into the skin. The heart shows no symptoms, although the pulse is weak and rapid, and attacks of vertigo are common. There is tenderness over the shafts of the long bones. Albuminuria occasionally occurs, and there may be a varying amount of fever.

Blood Picture.—The blood is opaque and flows sluggishly, but the coagulation time is normal, or but slightly increased. The hemoglobin is usually much reduced. The average red cell count is about 3,000,000. Nucleated red cells are abundant in the myelogenous variety, but rare in the lymphatic. As the disease progresses, the red cells diminish, and occasionally fall below 2,000,000. The leukocyte count varies in the two forms, being about 50 per cent. greater in the myelogenous type where the average count is 400,000. Each variety has a distinct blood picture, which enables us to distinguish the three forms. Characteristic of the myelogenous type is the high number of myelocytes, ranging from 30 to 60 per cent. of the white cells. All the intermediate forms of leukocytes between the ordinary varieties are present. The lymphatic type, if chronic, shows a lymphocytosis made up of about 90 per cent. small lymphocytes with few myelocytes or eosinophiles. Acute lymphatic leukemia is characterized by an increase in the large lymphocytes with polynuclears and eosinophiles in very small numbers. The other forms of leukocytes are all increased absolutely, but relatively decreased. The proportions of white cells to reds may run from 1 to 15 up to 1 to 3 in the very severe cases. Mixed forms of leukemia are met with in children which are neither myeloid nor lymphatic, but intermediate between leukemia and splenic anemia of infants.

Diagnosis.—The diagnosis is based solely on the blood picture. The high leukocyte count rules out Hodgkin's disease. The presence of pathological forms of leukocytes and red cells differentiates it from an ordinary leukocytosis.

Prognosis.—The disease runs a chronic course. There may be remissions of symptoms, but the termination is practically always fatal.

Pseudoleukemia of Infants (von Jaksch).—In the year 1889, Von Jaksch first described this disease which is a severe lymphatic anemia occurring only in infants. It is characterized by a leukocytosis with a decrease in red cells and hemoglobin, and an enormously enlarged spleen.

Etiology.—Practically all cases occur between the sixth and eighteenth months. Most of the cases observed have been associated with rachitis, and a few with syphilis. Gastro-intestinal disturbances have preceded this disease. It has been suggested that it may develop from a severe anemia.

Pathology.—The spleen is greatly enlarged, so that it is often apparent to the eye on inspection of the abdomen. On palpation, we find it hard and firm due to a hyperplasia. The liver is somewhat enlarged, and is firmer than normally. The lymph nodes show moderate enlargement. The cellular content of the bone marrow is increased. The heart and lungs are negative, the kidneys may reveal a degeneration of the parenchyma.

Symptoms.—Emaciation may or may not be present, although anorexia and other gastro-intestinal disturbances are common. The skin is pale and waxy and occasionally has a yellowish tinge. Dentition and closure of the fontanelles may be delayed. Jaundice may be present, though not marked. Very often the parents will bring a child for treatment of the enlarged spleen, which they themselves have noticed. Enlargement of the liver and lymph nodes is generally to such a degree that it is noted in clinical examination.

Blood Picture.—The specific gravity varies between 1035 and 1045. The hemoglobin is reduced to below 50 per cent., as a rule, and may go down to 20 per cent. The red cell count commonly falls below 2,000,000, but in some cases it is 3,000,000 and above. Various types of red cells are present, of which the nucleated reds are abundant. Megaloblasts, microcytes and megalocytes occur, and there is poikilocytosis and polychromatophilia. Both polynuclear and mononuclear leukocytes are increased and eosinophilia may occur. There are occasionally a few myelocytes. The total leukocyte count is not as high as in true leukemia, and varies between 20,000 and 50,000. In but few instances do these cases present definite blood pictures, and the summary above merely represents the characteristic findings in the few cases so far observed. Polymorphism of leukocytes is the distinctive change and a high white cell count suggests complicating factors.

Diagnosis.—The diagnosis rests chiefly upon the blood picture, the enormous size of the spleen, and severe gastro-intestinal disturbances

31

in a syphilitic or rachitic infant. The lower leukocyte count with paucity of myelocytes and the ultimate recovery exclude leukemia. Pernicious anemia has a high color index, the red cells are fewer, as a rule, and myelocytes do not occur so frequently as in pseudoleukemia of infants. The greatly enlarged spleen and presence of myelocytes in the blood signify more than a mere secondary anemia.

Prognosis.—Recovery is the usual outcome; those cases which are said to have passed on to a true leukemia were leukemia from the onset. With the inception of an intercurrent disease, the prognosis should be guarded, as this may cause a fatal termination.

Treatment of Anemia and Leukemia.—The general management of all cases of anemia is the same, since there are certain measures which are beneficial to all of the various forms. Fresh air in abundance with plenty of sunshine is of distinct importance. Regulation of diet, proper hygienic surroundings, and a change of scene and environment are of great value. The amount of exercise is governed by the severity of the anemia and the strength of the patient. If the anemia be of a mild grade, moderate exercise should be advised, but care must be taken to avoid fatigue. The severe grades of anemia require rest in bed, but this should not exclude fresh air and sunshine. The food should contain a maximum of nutrition, but must be given in small quantities at frequent intervals, since the gastro-intestinal tract is easily disturbed. Due regard to this susceptibility should be given in the administration of drugs, for, with digestion and assimilation below par or deranged, the treatment of a given case is rendered much more difficult. Cod-liver oil, beef extracts, phosphorus and strychnine are used for their tonic effect. Iron and arsenic each has a distinct influence in the treatment of anemia, and are given separately or together. Iron in the form of the citrate, saccharated carbonate, albuminate, or peptomanganate, is borne best by infants, and very young children; the chloride is recommended for later childhood life. Arsenic is indicated at times, and should also be given when iron is not borne well. Iron cacodylate given by needle is the best form of iron in all severe anemias, dose $\frac{1}{10}$ to $\frac{1}{4}$ grain. Arsenic may be given with iron, or may alternate with it. Fowler's solution is perhaps the best preparation and should be administered in ascending doses to the point of tolerance. In all cases of anemia, although it is very difficult at times, an earnest endeavor should be made to ascertain the cause, for, if this be found, and removed before the anemia has reached a fatal degree, prompt recovery will usually ensue.

Secondary Anemia.—If this is due to hemorrhage, it must be met promptly by stimulation of the patient, warm applications, autotransfusion and salt solution intravenously, subcutaneously, or by rectal irrigation. When due to disease or other conditions, the cause should be removed as quickly as possible, and the general treatment, as outlined, should be carried out.

Chlorosis.—Iron is perhaps the best drug for this condition and should be given whenever tolerated. Arsenic may be resorted to if

the patient cannot take iron, or may be used with iron. The general management is essentially the same for all anemias.

Pernicious Anemia.—In addition to the general management, other measures, such as continued inhalations of oxygen and removal to the seashore or mountains, should be resorted to. Arsenic is of more value in this disease than iron. Glycerinized extract of red bone-marrow has been given, but with indifferent results. Stimulants must frequently be resorted to because of the extreme exhaustion and weakness present. Intestinal parasites should be looked for and disposed of. Recently salvarsan has been given with good results, and splenectomy has been resorted to in severe cases.

Leukemia.—The treatment of leukemia is largely symptomatic. The general management should be carried out. Here again arsenic is the best drug, and should be given up to the point of tolerance. X-ray treatment of the spleen has been abandoned, and splenectomy in this disease is fatal. Recently the use of the x-rays upon the epiphyses of the long bones has met with promising results, and this procedure should be tried. German investigators report good results from the use of benzol in leukemia.

Pseudoleukemia of Infants.—Strict supervision of the diet and careful attention to the bowels are necessary. The underlying cause should be ascertained and treated. Iron alone or iron and arsenic alternating are indicated. Phosphorus must be given cautiously and is of doubtful value.

SPLENIC ANEMIA (BANTI'S DISEASE).

This is a severe primary anemia accompanied by enlargement of the spleen and a cirrhotic condition of the liver. Both red and white cells are diminished in number.

Etiology.—Males are more prone to this disease than females and the majority of all cases occur in children and young adults. The exact cause has not been discovered. Syphilis is suspected in some cases. There exists a theory that the greatly enlarged spleen generates a toxin which causes the disease. Most cases occur spontaneously in otherwise healthy infants.

Pathology.—The spleen may attain a considerable size. The liver is smaller and cirrhotic. Edema may be present in the dependent tissues with dropsical fluid in the serous cavities. Purpura is occasionally found.

Symptoms.—Contrary to the belief of Banti, whose name this disease bears, we find the first symptoms to be those of a severe anemia. Asthenia is marked and prostration is common. Enlargement of the spleen soon becomes apparent, and the organ itself is apt to be painful. The heart sounds are soft and weak and there is a hemic murmur. Edema and ascites are evident as the disease progresses, and hemorrhage may occur in the stomach, intestines, or kidneys.

Blood Picture.—Hemoglobin is markedly reduced to 50 per cent. or below. The red cell count rarely falls lower than 3,000,000. Poikilo-

cytosis and the presence of normoblasts may be noted. There is a lymphocytosis, but an actual leukopenia.

Diagnosis.—The enlarged spleen is suggestive of leukemia, but this is ruled out by the blood findings, which show a leukopenia. Splenic anemia, because of its rarity, should be considered only after careful study of the case excludes a chronic suppuration, syphilis, or tuberculosis.

Prognosis.—The prognosis, in cases not properly treated, is grave, and the disease runs a more rapid course in children than in adults. The treatment is radical, and for this reason quite a few cases are lost.

Fig. 33.—Banti's disease in a child aged seven years.

Treatment.—Splenectomy is, at present, the only treatment that gives results. This is often a dangerous undertaking owing to the severe grade of anemia which is present, but should not be deferred, and transfusion, preferably direct, should precede the operation.

LYMPHATIC ANEMIA (PSEUDOLEUKEMIA: HODGKIN'S DISEASE).

Primarily, this is a disease of the lymph structures, but it is discussed with blood diseases to distinguish it from true lymphatic leukemia. It is characterized by persistent enlargement of the lymph glands and spleen, and the formation of lymph nodules in the spleen, liver, and kidneys. As the disease progresses, a secondary anemia is induced.

Etiology.—It is a disease of early life, although few cases occur before the tenth year. It is more common in boys than in girls. The exact cause is unknown, but syphilis, malaria and rachitis are regarded

as predisposing factors. Tuberculous involvement of the affected structures is a complication rather than the primary cause. No acute infectious agent, which gives rise to Hodgkin's disease, has been found, although this probability is often advanced. The patient is frequently in apparent good health at the onset of the disease.

Pathology.—There is hyperplasia of all the lymphatic structures of the body. Both the superficial and deep lymphatic glands are enlarged, and, quite commonly, one set of glands in particular will be greatly hypertrophied. This occurs most often in the neck. In the absence of secondary infection, these enlarged glands do not break down and are not adherent to the surrounding tissues. The microscopic examination of sections from these glands reveals marked changes which distinguish them from the enlarged glands of lymphatic leukemia, tuberculous adenitis, and lymphosarcoma. There is no leukocytic infiltration, although eosinophiles may be numerous. There is a proliferation of endothelial cells, with the formation of lymphoid and giant cells. An increase of the connective-tissue stroma is observed in the more advanced cases, which accounts for the hardening of the glands. The spleen is enlarged, sometimes to a great extent, and contains lymphatic nodules. The liver and kidneys are also increased in size, and the site of lymphoid deposits. The bone-marrow may show changes, and lymphoid tissue may be present. Skin tumors are occasionally seen. No specific organism has been isolated from the enlarged glands of Hodgkin's disease, but many observers have found a diphtheroid bacillus.

Symptoms.—Enlargement of the superficial lymph glands, usually in the neck, is commonly the first complaint. Other glands quickly show enlargement, but they are not painful. With the progress of the disease, enlargement of the spleen and liver, with an increasing anemia, occurs. The patient grows weaker rapidly and complains of headaches, vertigo, palpitation, and dyspnea. The circulation becomes poor, and edema occurs in the dependent portions of the body. Gastro-intestinal disturbances follow with constipation, as a rule, and sometimes nausea and vomiting. Ecchymoses of the skin and hemorrhages from the mucous membranes occur. Fever is irregular and subsides during remissions. Cachexia develops late, and, commonly with it, various symptoms from pressure of the enlarged glands on nerves and vessels (Figs. 34 and 35).

Blood Picture.—Early in the disease the blood may show no change, but with the onset of the secondary anemia there is a steady fall in hemoglobin. The color index is low since the red cells are not decreased in proportion to the hemoglobin. There is no increase in white cells in uncomplicated cases, but there may be a lymphocytosis.

In the event of a complicating tuberculous infection of the glands there is a tendency toward high leukocyte counts which, if the process be mixed, as it often is, affects chiefly the polynucleated types of cells.

Diagnosis.—The diagnosis is based upon the enlargement of the lymph glands, their histological structure, and the accompanying blood

picture.   The structure of the enlarged glands and their movability
eliminate lymphosarcoma.   Tuberculous adenitis generally goes on to

Fig. 34.—Hodgkin's disease.

Fig. 35.—Hodgkin's disease.

suppuration, and there are usually other foci of tuberculosis present.
Leukemia is excluded by the blood findings.   No positive conclusion is

justified without a careful microscopic study of an incised section of a gland.

Prognosis.—Since there is no successful remedial procedure known, the prognosis is uniformly bad. The disease is fatal in from a few months to three or four years at the longest. Death finally comes on from exhaustion with general edema, hastened sometimes by a complicating tuberculosis.

Treatment.—Few measures give any relief and those which have any appreciable effect are only temporary in their results. Hygienic surroundings, fresh air and good food exercise their beneficial influence. Arsenic, in the form of Fowler's solution, is most widely used. Tonics containing iron, strychnine, cod-liver oil, and quinine are recommended. Phosphorus may be given cautiously in doses from $\frac{1}{200}$ to $\frac{1}{100}$ of a grain. Glandular extracts and bone marrow have been given with indifferent results. Local applications bring temporary relief. It is claimed that the Roentgen rays influence certain selected cases. Surgery is of no avail except that it may be resorted to early for the removal of a chain of enlarged cervical glands.

HEMORRHAGIC DISEASES.

In this group of blood diseases we include those with hemorrhage as the dominating feature, and caused solely by changes in the blood or circulatory apparatus. Hemorrhage, especially in infants, may be merely symptomatic of an anemia, syphilis, scurvy, or the various infectious diseases. The etiology of the true hemorrhagic diseases, however, is obscure, and their infectious nature or endogenous toxic origin is not yet proven. An overabundance of blood and a lessened coagulability of the blood have also been suggested as possible causes. These diseases are classified as hemophilia, which has a persistent tendency to hemorrhage and a distinct hereditary basis; and as purpuras, in which the tendency to hemorrhage is only transitory.

Hemophilia (**Bleeder's Disease**).—This is an inherited disease in which the individual shows a disposition to alarming and persistent hemorrhage from trivial abrasions or lacerations and spontaneous bleeding from no demonstrable cause.

Etiology.—The hereditary nature of this disease is well established and very rarely does a case present itself where this may not be brought out. The transmission of this disease is, moreover, along definite lines. In a family of bleeders, the female members transmit the disease and the males manifest it. Thus, a girl whose mother is a bleeder will not show signs of the disease, but will transmit it to her children; while her brother will be a bleeder himself, yet his children by a healthy wife will not be bleeders, although his grandsons, through his daughters, may suffer from the disease. The tendency to transmit hemophilia is no stronger in a woman from a family of bleeders, who herself is a bleeder, than in her sister, who may not be a bleeder. Marriage, to individuals who have not the disease, does not check its transmission,

and more than 50 per cent. of the children in the affected families inherit hemophilia. Large families have been the rule in the cases studied and traced through generations. It is rarely noticed at birth, but most cases are seen during childhood, generally before the second year. They usually die of hemorrhage before the tenth year; after the twentieth year this disease is exceedingly rare. It is observed more often in boys since girls are not, as a rule, actual bleeders. Most of the cases reported have been in Germans and Jews. This disease is unknown in the tropics, and occurs most frequently in temperate climates.

Pathology.—The findings at postmortem are vague and indefinite. The cause of the fatal hemorrhage may not be found. There may be an endarteritis, fatty degeneration of the intima, and thinning of the vessel walls. A secondary anemia may be present as a result of the hemorrhages, but the only blood change is a lessened coagulability.

Symptoms.—The most significant symptom is bleeding of a serious nature from slight injury or no apparent cause. This may be a severe sudden hemorrhage, or a constant oozing of blood which resists all attempts to check it. Such trivial injuries as the extraction of a tooth, or even dentition, may give rise to prolonged bleeding of a serious aspect; but it is a curious fact that menstruation and childbirth are not, as a rule, accompanied by great loss of blood. Spontaneous hemorrhages occur and may be preceded by buzzing in the ears, great excitement or even convulsions. These hemorrhages, usually of mucous membranes, take place in the gums, nose, throat, or bowel. Effusion of blood into the joints may be of an acute nature with or without fever, or it may be chronic with some limitation of motion and even ankylosis resulting. Following these hemorrhages, we have symptoms common to hemorrhage from any cause, and if it be a fatal one the patient dies from exhaustion. Sometimes death is preceded by, or occurs during, a convulsion.

Diagnosis.—A distinct hereditary history with a personal history of unusual loss of blood from trivial accidents makes the diagnosis certain. This is practically all the data obtainable in the average case, and the lack of other clinical findings excludes other diseases. Blood examination rules out anemias and leukemias. Hemophilic blood-clots in from two and one-half to five hours, the delay being attributed to an excessive antithrombin blood content, due to actual reduction of prothrombin, the ultimate factor of which is referable to some altered function of the platelets. Scurvy may be suggested, but diet will quickly relieve that condition and has no effect on hemophilia. Hemorrhages of the newborn are different in nature, and purpuras are accompanied by systemic manifestations.

Prognosis.—The final outcome is death in over 85 per cent. of cases. The longer the patient survives, the better the prognosis, and after puberty it is good. The death-rate is higher in males than in females. Each hemorrhage may be the last, and the patient recovers from one only to have another, until the final one, which is suddenly overwhelming or absolutely uncontrollable.

Treatment.—Prophylaxis is essential, and, with this view, some observers have claimed that the women of affected families should not marry. The patient should be guarded from birth and carefully protected to prevent any abrasions of the skin and mucous membrane. Removal to the tropics should always be suggested. Operative procedures are contraindicated and only surgical operations to save life should be allowed. If operation is imperative, a careful study of the coagulability of the blood should precede it, and an attempt made to have the coagulation period as near normal as possible at the time of operation. This may be accomplished by intravenous or subcutaneous injections of human blood serum, or, if time permits, thyroid extract may be given over a short period. In the event of hemorrhage, styptics may be used locally, of which tannic acid and the perchloride of iron are perhaps most serviceable. Adrenalin chloride may be given internally and applied locally. I have obtained good results from the administration of calcium lactate in the dose of 15 grains three times daily in a case of persistent epistaxis. Other drugs which should be given are: tincture of chloride of iron, Monsel's solution, perchloride of iron, ergot, and sodium sulphate in small but frequent doses. Gelatin has been given for hemorrhage of the stomach, but the results are very unsatisfactory. Rectal injections of lead acetate have been resorted to in intestinal hemorrhage. Sterile gelatin in a 5 per cent. solution has been given subcutaneously. Blood transfusion should be tried and the father's blood be used, if practicable. Human serum is preferable, but animal serum may be used, of which normal horse serum is perhaps the best.

Purpura.—Purpura, although a blood disease, is sometimes classified under diseases of the skin, because of its chief clinical feature, which is the appearance of hemorrhages in the skin. The areas of hemorrhage vary in size from that of a flea bite to large ecchymotic spots. At times they may be found in the mucous membranes and also in the viscera.

Etiology.—Purpura is thought to be caused by the action of toxins on the blood or circulating apparatus. It is usually secondary, sometimes merely symptomatic, but other cases are apparently primary. Hemorrhagic disease of the newborn, ileocolitis, jaundice, leukemia, primary and secondary anemias, nephritis, scurvy and syphilis are largely responsible for this condition. In older children it appears with measles, scarlet fever, smallpox, diphtheria and cerebrospinal fever, and renders the prognosis bad. It is common in septicemia, pyemia, and malignant endocarditis. The administration of such drugs as antipyrin, benzoic acid, chloral, chloroform, ergot, the iodides and mercurials, mineral acids, potassium chlorate, arsenic, belladonna, copaiba, phosphorus, quinine and the salicylates, may produce purpuric outbreaks. An attack of purpura may be brought on by bites of snakes and insects, and the toxins liberated in ptomain poisoning and jaundice have a similar action. The paroxysms of whooping-cough, the removal of splints, or even epilepsy, may cause it. There

is not the same relation between neurotic conditions and purpura in children as in adults, and it is rarely seen in association with such a condition before puberty. Purpura occurs in the cachectic states of malignancy and atrophy of infancy. It is said to be a disease of young life, most cases occurring before the age of fourteen. In a certain number of cases no cause is assignable, and these we term primary, but the existence of primary purpura is not proven.

Pathology.—There is either a hemorrhagic exudate, an escape of blood by diapedesis or merely a transudation of blood pigment into the tissues. Lesions of the surrounding vessels, such as endarteritis, and fatty and hyaline degeneration have been found, but they may be caused by the underlying factor of the purpura and it is doubtful whether they belong to the pathology of purpura. Stasis and embolism have been observed in the surrounding vessels, near hemorrhagic areas. Visceral changes include enlarged spleen, gastric ulcers, and hemorrhage into the adrenals. The blood picture is not characteristic, but usually shows a secondary anemia with or without a leukocytosis. There may be evidence of hemorrhage into the joint cavities.

Diagnosis.—The existence of purpura is easily proven, but in children the various forms are not clearly defined and a positive diagnosis of any one form is not always possible. Scurvy is sometimes difficult to differentiate from an attack of purpura. The following forms are met with in children; purpura simplex, purpura hemorrhagica, purpura rheumatica, and Henoch's purpura. The fulminating form, purpura fulminans, is only a rapidly fatal case of the hemorrhagic type.

Purpura Simplex.—This form is the mildest variety met with and may not be accompanied by any prodromal systemic symptoms. The hemorrhages are confined to the skin.

Symptoms.—If there be any prodromal symptoms, they are slight and consist of general malaise, anorexia, headache, and constipation or diarrhea. With the appearance of the purpura, which usually consists of small lesions varying in size from that of a flea bite to the diameter of a lead pencil, there may be nausea and vomiting, and a slight rise in temperature. The characteristic feature of the purpura spot is its change of color, first to purple, then to brown, and finally by degrees to yellow. Successive crops may appear, the first of which is usually seen on the legs; the arms are next involved, and the lesions are most abundant on the extensor surfaces. The face and trunk are apt to be free from the lesions, and if the mucous membranes are involved bleeding externally does not occur.

Diagnosis.—The diagnosis is easily made by the appearance of the purpuric spots, with little or no systemic symptoms. Symptomatic purpura is eliminated by the absence of any apparent cause, and the more severe forms of purpura are excluded by the absence of their particular systemic symptoms.

Prognosis.—This is guardedly favorable. The attack lasts from one to four weeks, but relapses are common.

Treatment.—The patient must remain in bed as long as fresh crops of purpuric spots tend to appear, for the duration of the disease can be prolonged by exercise and even walking about at this period. The diet should be light but nutritious, and contain much of fruit acids. Mineral acids, astringents such as gallic acid, and hamamelis are indicated. Adrenalin chloride and calcium lactate are also used.

Purpura Hemorrhagica.—This is a more severe form of purpura, in which the skin, mucous membranes, and even the viscera are involved, and systemic symptoms are marked.

Symptoms.—Prodromal symptoms are common, and include chilliness, fever, vertigo, diarrhea, and anorexia. The hemorrhages vary in size from petechia to ecchymotic spots $\frac{1}{2}$ inch in diameter. These may be light or dark red, do not disappear on pressure, and are either painful or itch. Free bleeding may occur from any mucous membrane, and hemorrhage from the nose, mouth, throat, stomach, bowels, lungs, or kidneys has been observed. A hemiplegia has occurred from an intercranial hemorrhage in this disease. A secondary anemia, often of severe degree, is produced by the loss of blood, and, as a result, the patient complains of dyspnea, palpitation, headache, backache, and may have abdominal pains. A typhoid state may ensue with prostration, dry tongue and mouth, insomnia, carphologia, muttering delirium, or even coma. This is apt to result fatally. When the symptoms come on rapidly and the patient quickly succumbs, the attack is known as purpura fulminans (Fig. 36).

Fig. 36.—Purpura hemorrhagica.

Diagnosis.—The diagnosis is based upon the presence of purpuric hemorrhages from the skin and mucous membranes, and severe constitutional symptoms which may or may not indicate visceral hemorrhage. Joint pains are not marked, if present at all, and this, with the absence of urticaria, excludes the rheumatic type. A possible hemophiliac phase is eliminated by the lack of a family history characteristic of the bleeder and the freedom from previous hemorrhage. Abdominal crises of Henoch's purpura do not occur.

Prognosis.—If the case be mild a favorable prognosis may be given guardedly. The weak and young usually succumb and the typhoid state generally bespeaks a fatal outcome. High temperature and copious hemorrhages are unfavorable signs.

Treatment.—Rest in bed, good hygienic surroundings, fresh air, and a light nutritious diet containing an abundance of fruit acids are essential. If there is gastric or intestinal hemorrhage, orange juice, peptonized milk and gelatin may be given. Fowler's solution is used

in severe cases and a tonic containing iron is best for the secondary anemia during convalescence.

Purpura Rheumatica.—Children rarely suffer from this type of purpura, which is characterized by the eruption of purpuric spots especially about the large joints, by polyarthritis and exudative erythemata. A possible relation between this disease and rheumatic fever is often claimed because of the arthritis and occasional cardiac involvement, but, in my opinion, several infective or toxic agents may be capable of producing it.

Symptoms.—Preceding the appearance of purpura, there is usually slight fever and often sore throat. A polyarthritis follows, which is usually in the joints of the lower extremities, and rarely involves those of the arms. Urticaria and erythema nodosum are frequently associated with the arthritis, and there may be marked edema of the lower extremities. The joints are usually swollen and boggy, and are painful and tender. The spleen is sometimes enlarged. Albuminuria may occur, but the nephritis is mild. Abdominal pain and tenderness are occasionally observed.

Diagnosis.—This is easy when a polyarthritis and urticaria accompany a purpura with but slight fever and a sore throat.

Prognosis.—After two weeks a recovery may be expected, but relapses may prolong the attack for months. The ultimate outcome is good, although the heart may be permanently affected if cardiac involvement occurs.

Treatment.—The patient should be kept in bed and the same hygienic and dietetic measures as for purpura simplex should be carried out. The polyarthritis should be treated by the internal administration of salicylates and by local applications to the affected joints.

Henoch's Purpura.—Henoch's purpura or abdominal purpura is most frequent in children and the symptoms are referable to the skin, joints, and abdomen. The skin lesions are usually purpuric, but may also consist of circumscribed areas of edema and exudative erythemata. The joints of both upper and lower extremities are painful and swollen. The abdominal symptoms are gastro-enteric crises of colic, vomiting, tympanites, and tenesmus with melena. There may be hematuria and albuminuria, with a fatal nephritis. Occasionally there are cerebral and pulmonary symptoms.

Diagnosis.—The abdominal crises occurring in a case of purpura, with joint lesions, are indicative of Henoch's purpura.

Prognosis.—The prognosis is grave where recurrence is frequent; the mortality-rate in Osler's series was 25 per cent.

Treatment.—Rest in bed with the same hygienic and dietetic treatment as suggested in the other forms of purpura, should be carried out. Further treatment is symptomatic. The calcium salts have been used where hemorrhages were severe, and atropin is recommended for the abdominal pain. Local applications may give relief to abdominal and joint pains, and, if the arthritis is marked, salicylates should be administered internally. During convalescence iron and arsenic in tonic doses are of special value.

CHAPTER XVII.

DISEASES OF THE DUCTLESS GLANDS.

THE spleen is very easily affected by disease during childhood, and is then more readily palpable, which makes it of greater diagnostic value in children than in adults. Its length ranges from 2 to 4 inches, according to the age of the child, it is half as wide as it is long, and about ¼ of an inch thick. The size of the spleen varies greatly with the state of the nutrition. As a rule, a poorly nourished infant will have a very small spleen, but when malnutrition is caused by syphilis or rachitis the spleen is enlarged. There is a physiological increase in the size of the spleen during digestion, owing to the increased amount of blood in the organ at this time.

The position of the spleen may be made out by percussion with the child lying on its back and percussing above downward in the midaxillary line. Splenic dulness will be found to extend from the ninth to the eleventh rib and from the posterior axillary line to a little beyond the midaxillary line. In infants the area of splenic dulness may not be distinguishable, may sometimes be obliterated by gastric or colonic tympany, and may be simulated by fecal masses in the colon.

In order to palpate the spleen the patient should lie on his back with the thighs flexed to afford good relaxation of the abdominal muscles. The physician's hand should be warmed, and should first be gently placed over the splenic area, just at the lower border of the ribs, until abdominal rigidity is overcome. The right hand is preferable, the examiner standing on the right side of the patient. The finger tips are pressed up under the costal margin between the posterior and midaxillary lines, when the spleen should be felt touching the finger tips with each respiration. The edge of the spleen should be felt just under the lower border of the ribs. If it extends below the costal margin it is enlarged.

Enlargement of the spleen may be acute or chronic; in children acute enlargement is much more frequent than in adults, since many of the acute infectious diseases cause an increase in its size. A chronically enlarged spleen is seen in malaria, cirrhosis of the liver, the chronic infectious diseases, particularly syphilis and tuberculosis, and in chronic congestion caused by cardiac and pulmonary disease. The spleen is also enlarged in the various forms of anemia, in Hodgkin's disease, gastro-enteritis, acute catarrhal jaundice, and amyloid degeneration.

SPLENITIS.

Inflammation of the spleen is rare, and when it occurs is generally caused by extension from surrounding structures, such as the stomach,

diaphragm, lungs, or the perinephric tissue. It may be due to trauma.
In splenitis the spleen is found to be enlarged, percussion in the splenic
region is painful, and palpation reveals acute tenderness. The diagnosis
is based on the symptoms outlined above, with a history of trauma
or inflammation of surrounding structures.

PERISPLENITIS.

Perisplenitis is an inflammation of the serous covering of the spleen.
It occurs in general peritonitis, and may also be a result of extension
from an ulcer of the stomach, a left diaphragmatic pleurisy, peri-
nephric inflammation, or chronic colitis. Trauma, hemorrhagic in-
farcts, syphilis, and tuberculosis are among the rare causes of peri-
splenitis.

The diagnosis is made by detecting the friction rub of the roughened
capsule. On auscultation the friction rub is heard better at the lower
margin of the rib than over the chest wall, and this helps to differentiate
it from a pleural friction rub. In some cases the spleen may be immov-
able owing to adhesions.

ABSCESS OF THE SPLEEN.

This is a rare condition in children, but may arise from trauma or
during the course of malaria, typhoid fever, or pyemia. It may also
be due to extension from a suppurative process in adjacent tissues.
When the suppurative lesion reaches the surface perisplenitis results,
and rupture usually follows, the contents pouring into the free peri-
toneal cavity or some adjacent viscus.

WANDERING SPLEEN.

As the result of elongation of the gastrosplenic ligament and the
splenic artery and vein, the spleen may attain a wide range of mova-
bility. When this condition exists there is tympany over the area of
normal splenic dulness, and abdominal palpation reveals the spleen
low down in the abdomen, but usually on the left side. As a rule it
can be replaced, but when the patient assumes the upright position
it will again fall out of place.

The most constant symptom is a dragging sensation in the abdomen.
The spleen is enlarged, and may produce symptoms by pressure on the
ureter, bladder, or bowel, and there are usually nervous symptoms like
those accompanying nephroptosis. The presence of a fecal tumor or a
floating kidney must be considered and excluded before a positive
conclusion is reached. The treatment consists of mechanical measures
to keep the spleen in its proper position, but they are only moderately
successful.

PRIMARY SPLENOMEGALY.

This is a rare form of enlargement of the spleen, which is most common in children. The cause is unknown. There is hyperplasia of the endothelial cells of the spleen, with changes in the mesenteric lymph nodes. The liver is enlarged secondarily, and contains an increased amount of connective tissue. There are abdominal pains and gastro-intestinal disturbances. Simple anemia with subcutaneous hemorrhages and bleeding from the gums and nose may be present. Dyspnea and dysuria have been noted. The spleen may attain such a size as practically to fill the abdominal cavity.

NEW GROWTHS OF THE SPLEEN.

In children the spleen is rarely the site of new growths, but occasionally the roughened or nodular surface of the spleen will warrant their consideration. Tuberculosis is the most common cause of nodular spleen in children. Gummata of the spleen are rare, although the enlarged spleen is the most constant sign of hereditary syphilis. Leprosy, actinomycosis, and parasitic cysts have been observed. Benign splenic tumors are very rare. Of the malignant tumors, sarcomas are the most frequently found, and are usually secondary. Cancer is extremely rare.

DISEASES OF THE LYMPH GLANDS.

Enlarged lymph glands are much more common in children than in adults, and of all the lymph nodes in the body the cervical glands are the most frequently enlarged. Pediculosis capitis, or disease of the scalp, such as an eczematous condition, causes enlargement of the posterior cervical glands. The anterior cervical glands become enlarged with disease of the nose and throat, and are also subject to infection by the tubercle bacillus. The axillary glands are involved by infection of the arm and outer side of the upper chest wall.

Enlargement of the epithrochlears is seen in syphilis. The inguinal glands, which are the only set normally palpable, are enlarged when there is a lesion of the genitalia or infection of the leg. General glandular enlargement is observed in syphilis, tuberculosis, lymphatism, the various forms of anemia, and following acute infectious diseases. Children of lymphatic diathesis rarely exhibit it after adolescence.

SIMPLE ACUTE ADENITIS.

Children are very susceptible to simple adenitis, which is an acute inflammation of the lymph nodes. The cervical glands are the ones affected in over 80 per cent. of the cases; they become inflamed by the

draining of an infected area, such as a diseased tonsil or a decayed tooth. Acute adenitis may complicate the acute infectious diseases, and usually appears during tonsillitis or any throat infection. Eczema and stomatitis may also give rise to acute adenitis.

The deep lymphatic glands may become inflamed from draining an infected source, but they are not demonstrable, hence only the superficial glands will be considered. The axillary and inguinal glands enlarge when there is infection of those areas of the body which they drain. Most cases of simple adenitis occur before two years of age. The exciting or direct cause is the entrance of pyogenic organisms into the lymph glands.

Pathology.—The enlargement of the gland is due to hyperplasia of lymphoid cells, with acute congestion of the glands. The infection may go this far, and then stop, or may proceed to suppuration according to the virulence of the organism and the resistance of the patient.

The streptococcus is usually isolated in those cases which suppurate, although the staphylococcus, pneumococcus, gonococcus, and typhoid bacillus have also been found. As suppuration takes place the nodes become softer and the surrounding tissue infiltrated which causes cellulitis. Frequently only one node in a chain of several diseased ones will suppurate.

Symptoms.—Swelling just below the angle of the jaw is, perhaps, the first sign of cervical adenitis. The degree of tenderness varies; in some cases there is very little pain, while in others merely moving the jaw is painful. There is moderate fever, with symptoms of the underlying disease if adenitis is caused by one of the acute infections. Suppuration takes place between the first and fourth weeks; after the fourth week, if there has been no suppuration, resolution may be expected to follow the stage of hyperplasia. The glands become smaller and harder, and slowly return to their normal size. After repeated infections the gland may become chronically enlarged, and remains as a palpable nodule throughout life.

Upon inspection only one or two glands in the chain will appear to be affected, but palpation shows the whole chain to be involved. When suppuration begins the gland softens, the overlying skin becomes red, and tenderness and pain are increased. If not lanced there is pointing and rupture through the skin, with the discharge of a creamy pus. This relieves the symptoms, and there is slow recovery, with an ugly scar at the site of rupture.

Diagnosis.—Acute adenitis may simulate mumps, but in mumps the swelling is in the parotid region, the lobe of the ear forming the centre of the swelling. In mumps there is usually a history of contact, and in adenitis a history of some preceding nose or throat condition. After two years of age tuberculous adenitis is quite frequent, but may be ruled out by its chronicity.

Treatment.—The cause should be ascertained and removed if possible. Careful attention to the throat, especially to the tonsils and any adenoids, will prevent most cases of cervical adenitis. Locally,

cold applications will give relief, and are indicated if there is merely congestion and swelling with no suppuration. Cold compresses are better than an ice-bag, but should be renewed every fifteen to twenty minutes.

When suppuration becomes inevitable, heat and ichthyol ointment should be applied. If there is fluctuation the abscess should be opened and drained. A course of calomel should be followed by magnesium sulphate, and then syrupi ferri iodidi given in full doses for a period of weeks. For those glands which do not suppurate, but which remain large and later offer but poor resistance to the tubercle bacillus, the iodides, electricity, Bier's hyperemia, or surgical removal may be resorted to.

SIMPLE CHRONIC ADENITIS.

This is usually a mild degree of enlargement of the lymph glands, which persists in consequence of repeated attacks of acute adenitis or because the source of infection is still acute. Chronic nose and throat conditions and chronic skin or scalp diseases are frequently responsible for the persistent enlargement of these glands. As a rule, hypertrophied tonsils and adenoids are present, and, possibly, that condition known as status lymphaticus.

Symptoms.—Enlargement of the glands is the main clinical feature. Pain and tenderness are absent, and there are no constitutional symptoms. The glands may remain enlarged for months, or even years, or may never return to normal, those affected remaining as small hard nodules throughout life. Suppuration does not take place in these glands, but the centre of the gland may be found to consist of broken-down tissue resembling tuberculous caseation.

Diagnosis.—Simple chronic adenitis must be differentiated from tuberculous adenitis and Hodgkin's disease. The absence of any clinical features other than enlargement of the glands, the age of the individual, and the slow progress of the process usually favor simple chronic adenitis.

If we suspect some specific factor rather than simple chronic enlargement of the glands, an incised section examined under the microscope will reveal the true nature of the disease. The importance of diagnosing a possible tuberculous adenitis early is so great that a section of the suspected gland should be made in every case where doubt exists as to whether or not surgery should be resorted to.

Treatment.—Removal of the cause of the enlargement is essential before improvement can be expected. Potassium iodide and the syrup of the iodide of iron should be given in full doses. Cod-liver oil and arsenic in the form of Fowler's solution are both beneficial for their tonic effect. An outdoor life, with plenty of exercise and good nourishing food, will materially hasten the ultimate recovery. In view of the fact that these glands, if they remain enlarged, frequently become tuberculous, surgical removal should be advised when other treatment is of no avail.

32

TUBERCULOUS ADENITIS.

The term "tuberculous adenitis" is awarded by common usage to tuberculosis of the cervical lymph glands. The infecting organism gains access to the glands through abrasions or the extension of inflammation in the mucous membrane of the nose and · throat. Diseased tonsils, adenoids, and chronic inflammation of the naso-pharynx predispose to the tuberculous infection.

It is now believed that the bacilli do not penetrate the normal mucous membrane, but are carried in with food and air, and obtain access through the point of least resistance, which is usually the site of some chronic inflammation. In uncomplicated cases the infecting organism is often difficult to find. It is usually the human type of the tubercle bacillus, although the bovine form has been isolated.

Suppuration is generally caused by mixed infection with the strepto-coccus. Merely one chain of glands or the lymphatics on both sides of the neck may be affected. In some cases all of the glands of the body may be involved—the cervical first, then the axillary, mediastinal, retroperitoneal, mesenteric, and inguinal glands.

The disease may progress in such a manner as to bear a resemblance to Hodgkin's disease. The glands may appear as separate and discrete nodes, varying in size from that of a bean to a walnut, or they may coalesce. They may be quite hard or almost gelatinous in consistency. An incised gland will be found to contain an increased amount of connective tissue, with many small areas of necrosis throughout its substance. Microscopically, giant cells containing several nuclei may be seen; the tubercle bacillus is hard to demonstrate. Caseation does not occur in this generalized adenitis.

Symptoms.—As a rule, there are no constitutional symptoms and the health may apparently be unaffected; usually, however, there is a gradual loss of weight with mild secondary anemia. If there is second-ary infection of the glands, slight fever is noted and the glands are painful. Those glands situated at the angle of the jaw first become enlarged, and this swelling may be the only noticeable sign of tuber-culous adenitis. The swelling is usually unilateral, but may involve both sides.

As the disease progresses the glands become immovable from the periadenitis which develops, and adhesions to the surrounding tissues form. There is practically no pain in uncomplicated cases. With secondary infection and pus formation the usual symptoms of abscess are present.

If the gland is not incised at this stage, the abscess may rupture through the skin, or rupture beneath it and the pus burrow down through the fascia of the neck, collecting at the most dependent part, and there escape through the skin. When this occurs there is a fistula between the infected gland and the point where it opens through the skin. The whole process is essentially chronic, only the neglected cases resulting in ruptured abscesses and fistula formations.

Diagnosis.—Tuberculous adenitis must be differentiated, first, from simple chronic adentitis. The age of the child, the tuberculin reaction, and the presence or absence of other tuberculous lesions must be taken into consideration in determining with which of these forms of adenitis we are dealing. If lymphosarcoma or pseudoleukemia is suspected, a section of the enlarged gland should be examined microscopically. Examination of the blood will rule out the possibility of leukemia as a cause of the glandular enlargement.

Prognosis.—The prognosis of tuberculous adenitis is better than for any other form of tuberculosis, since the infected tissue is easy of access, and the disease tends to remain localized. Systemic infection from foci in the glands of the neck is rare, hence the mortality-rate of tuberculous adenitis is extremely low. If the case is diagnosed early, and its surgical removal undertaken, recovery with complete eradication of the disease may be expected.

Treatment.—Early and complete removal of all the infected glands is unquestionably the only safe treatment of tuberculous adenitis. In the hands of a skilful surgeon there need be no appreciable scar after the operation, although through fear of disfigurement many parents will object to operative procedure. Following the operation the patient should have the advatage of the routine treatment for tuberculosis with, possibly, a change of climate.

The diet should be wholesome and nutritious, with an abundance of eggs and milk. Cod-liver oil is of great benefit, but care should be taken that it does not impair the appetite. The syrup of the iodide of iron should be given in full doses throughout convalescence, and arsenic in the form of Fowler's solution is indicated for the secondary anemia which is usually present.

DISEASES OF THE THYMUS GLAND.

The thymus gland is a lymphoid structure which exists only during childhood, the period of its greatest growth being reached at the end of the second year. Atrophy of the lymphoid tissue with an increase in the fibrous and adipose tissue takes place gradually from the second year until puberty. From puberty on to early adult life there is rapid atrophy of the gland, until at adult life there exists merely a fibro-fatty body with but faint traces of thymus lymphoid tissue. During childhood two distinct lobes may be defined, lying in close contact with the pericardium and the trachea. The two lobes are unequal in size, but may be said to extend from the lower border of the thyroid gland to the level of the fourth costal cartilage. The gland lies just beneath the sternum in the chest, and farther up is found under the sternohyoid and sternothyroid muscles. The two lobes may be united, or there may be a third lobe. The average weight of the thymus,

as given by various observers, offers a wide range, but to the best of
our knowledge the normal thymus gland should weigh from 7 to 10
grams (100 to 150 grains) between birth and two years of age. It is
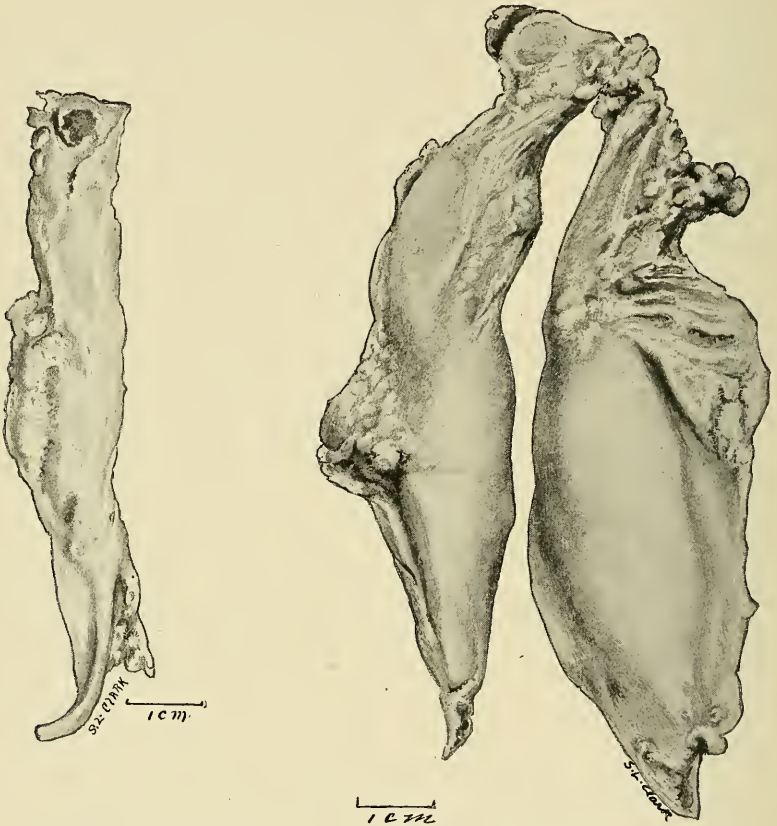
<center>Fig. 37   Fig. 38</center>

Fig. 37.[1]—Unilobar thoracic thymus. Newborn infant, death from causes in no
way referable to thymus.   Single-lobed thymus extended from just below interclavicular
space to diaphragm along right border of heart.   Weight 3.75 grams.   No other thymic
tissue found.   No evidence of pressure on trachea; position such that trachea could not
have been compressed.   (Coplin.)

Fig. 38.—Bilobar thoracic thymus. Status lymphaticus thymic death. Male,
aged fourteen years; asthma of long duration; otherwise in good health.   Received
immunizing dose of antitoxin; death in a few minutes.   Notable hyperplasia of many
lymph nodes.   Thymus consists of two tongue-like lateral lobes extending downward
over heart to diaphragm; no median lobe; weight 17 grams.   Upper margin behind
sternum more than 2 cm. below notch.   No evidence of tracheal compression.   Upper
third of each lobe shows wrinkled capsule, fat infiltration and shrinkage—evidences
of regression.   The absence of any isthmus or mass at point of junction precludes
tracheal pressure.   (Coplin.)

about 12 cm. (5 inches) long and from $\frac{3}{4}$ to $1\frac{1}{4}$ inches (2 to 3 cm.) wide when it has attained its largest size.

FIG. 39   FIG. 40

FIG. 39.—Trilobar cervicothoracic thymus. Infant; death a few hours after birth, not thought to have been due to thymus. Long median lobe extending above sternal notch; two imperfectly distinguishable lateral lobes, the inferior margin of which extended over anterior part of auricles but not reaching the ventricles. No evidence of pressure on vessels or trachea. Weight of thymus 4.14 grams. (Coplin.)

FIG. 40.—Trilobar cervicothoracic thymus (persistent). Male, colored, aged thirty-six years. Hemiplegia, epilepsy, aphasia. Death from cerebral softening. Middle or superior lobe abnormally large; lateral lobes about normal for childhood, extended from above suprasternal notch to diaphragm. Weight of thymus 7.82 grams. No evidence of pressure. Were the superior lobe large, or greatly thickened antero-posteriorly, pressure on trachea would be possible. (Coplin.)

Physiology.—The function of the thymus is not yet understood, and we are still in doubt as to whether it should be classed as one of the ductless glands or with the other lymphoid structures of the body.

It is thought to be connected with the lymph system and the thyroid and parathyroid glands. That there is a relation between the thymus and testes is shown by the delayed involution of the thymus following castration, and the rapid growth of the testes in thymectomized animals. Extirpation of the thymus is also followed by softening of the bones
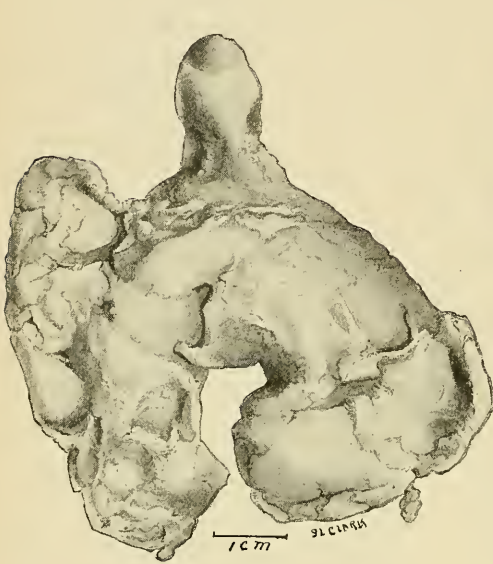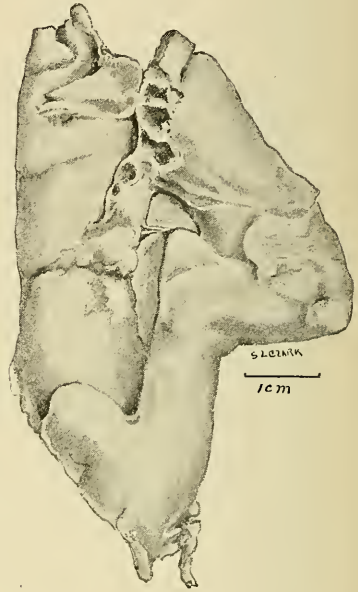
Fig. 41.—Trilobar cervicothoracic thymus. Male, white, aged thirteen years. Splenic anemia, greatly enlarged spleen weighing 1100 grams. Splenectomy; recovery. Two years later admitted to hospital in convulsions, probably unconscious, face flushed, dyspnea, respirations 40, slight cyanosis, rapid heart (100). Temperature 104°; extremities cold. History of dog bite. Clinical diagnosis: Status lymphaticus. Postmortem: Status lymphaticus; congestion of lungs; adhesive pleuritis; congestion of liver and kidneys; hyperplasia of lymphoid tissue of intestine, colon and stomach; enlarged lymph nodes; chronic adhesive peritonitis in splenic area; adhesions probably due to splenectomy. Organ of quite unusual shape, resting upon and overlapping heart and extending upward behind sternum just above notch. Weight of thymus 23.61 grams. Such an organ might press upon trachea in critical space or beneath sternum. There is sufficient evidence to justify the recognition of lobes; otherwise it might be called a conglomerate thymus which it in part resembles. (Coplin.)

Fig. 42.—Conglomerate thoracic thymus. Female, colored, aged twenty months. Clinical note: Bronchopneumonia, pleurisy; dyspnea; cyanosis; rapid pulse of low tension. Postmortem: Bronchopneumonia, bilateral pleural effusion, partial atelectasis, congestion of liver and spleen; fatty infiltration of liver. Thymus lies over and anterior to heart, is thin, of rather flat type, lobes imperfectly and irregularly joined above and below. Weight 9 grams. (Coplin.)

and cessation of their growth, by an increased electrical excitability of the peripheral nervous system, increased fat absorption, malnutrition, and cachexia. Enlargement of the spleen is followed by shrinking of the thymus. Bourneville found the thymus in only 27 per cent. of idiots at postmortem. In addition to this relation of the thymus to
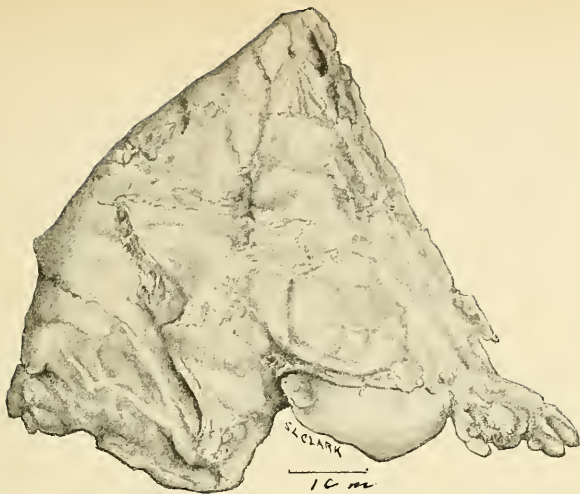
FIG. 43.—Conglomerate thoracic thymus. Female, white, aged one year. Clinical diagnosis: Bronchopneumonia. Postmortem: Bronchopneumonia. Patent intraventricular septum; chronic mitral endocarditis; hypertrophy and dilatation of left ventricle. Congestion of liver, spleen and kidneys. Pyramidal or conic thymus; lobulation imperfect, somewhat flattened anteroposteriorly; pale, some edema; organ firm, pressure phenomena improbable. Weight of thymus 16.3 grams. (Coplin.)
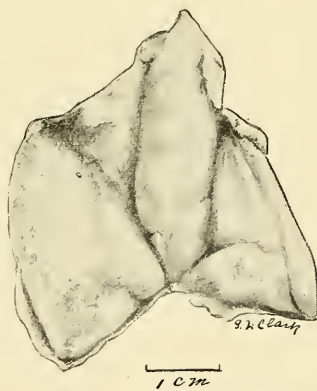
Fig. 44   Fig. 45

FIG. 44.—Conglomerate cervicothoracic thymus. Eclamptic mother died suddenly following therapeutic administration of horse serum. Infant (seven months?) obtained postmortem; length 46 cm., weight 2020 grams. Subpericardial hemorrhages; congested kidneys; congested, soft, friable liver. Thymus: Two (possibly three) imperfectly differentiated lobes with a prolongation extending through critical space to 1.9 cm. above superior sternal border; the flattened inferior aspect was just over cardiac base upon which, when the auricles were distended, it no doubt rested; left lobe extended to near middle of left ventricle. Weight of thymus 8.11 grams. No gross lesion. (Coplin.)

FIG. 45.—Conglomerate thoracic thymus. Female, aged twenty-four hours. Low attachment and partial separation of placenta; Cesarean section. Weight of infant $6\frac{1}{2}$ pounds. At birth infant cried naturally, did well for twenty-four hours, died in spasm with dyspnea. Postmortem: Aside from enlarged thymus and changes referable thereto, nothing abnormal found. Thymus greatly enlarged, intensely edematous, bulges in front and above heart. Body opened and fixed in formalin; thymus dissected from bed. Weight of thymus 45 grams. Histologically, richly cellular thymic tissue, intensely edematous. Precava and trachea manifestly compressed; trachea collapsed. Irregular areas of pulmonary aeration; all anterior margins of both lungs airless; bases imperfectly expanded, many lobules atelectatic. (Coplin.)

other organs of the body, it has been proven that lymphocytes and eosinophiles are found in the lymphoid tissue of this gland.

Percussion.—The outline of the thymus may be elicited by light percussion over the upper part of the sternum in young children, but thymic dulness disappears after the second year, after which it is pathological. The area of dulness produced by the thymus in children is continuous with cardiac dulness, and should not extend more than $\frac{1}{2}$ inch (1 cm.) beyond the margin of the sternum on either side.

ATROPHY OF THYMUS.

The thymus gland is very susceptible to changes in the general state of nutrition of the body, and is found to be very small in cases of malnutrition. Infants in whom the thymus was so small as to be considered absent, and in whom the gland did not weigh over 30 grains, have been fatal cases of marasmus due to improper feeding, imperfect assimilation, congenital lues, rachitis, and chronic tuberculosis. If the case be one of chronic toxemia the lymphoid tissue is practically altogether replaced by fibrous tissue, while in children dying of starvation there is simply an atrophy of all the tissue elements with marked reduction in weight.

ENLARGEMENT OF THE THYMUS.

Acute enlargement of a normal-sized thymus gland may occur from congestion due to cardiac disease or after goitre operations, or as a result of hyperemia or edema. This enlargement is occasionally sufficient to cause death from pressure on the underlying structure. True hyperplasia of the thymus has been observed in cases of congenital lues, rachitis, anemia, Hodgkin's disease, chlorosis, leukemia, Addison's disease, and exophthalmic goitre. The theory has been advanced that enlargement of the thymus is a secondary compensation measure occurring in infections, auto-intoxications or disturbances of metabolism in which there is lymphoid exhaustion. Enlargement of the thymus is a part of that rare condition known as status lymphaticus, in which there is a general hypertrophy of the whole lymphatic system.

Pathology.—Postmortem findings in a few cases have shown merely congestion or edema to have been the cause of fatal enlargement of the thymus. In the majority of cases there is a true hyperplasia of the organ, particularly of the lymphoid tissue. Although there is much discussion over the weight of the normal-sized thymus, a thymus over $\frac{1}{2}$ ounce (15 gm.) may be considered as enlarged. Of the dimensions, increased thickness is of greatest importance, since it results in pressure on the underlying structures, particularly the trachea which has been found to be flattened, and even stenosed to a considerable degree. Pressure on the great vessels may cause hypertrophy and dilatation of the heart. Thrombosis of the jugular vein has been found at autopsy.

Symptoms.—Enlarged thymus has been observed after death which caused no symptoms during life, but, as a rule, there is evidence of respiratory difficulty. This interference with respiration may vary from mild stridor to severe dyspnea which terminates fatally. Thymic stridor is believed to be due to compression of the trachea and is usually noticeable at birth, although it may not develop until some time after. It is chiefly inspiratory, but expiration is also impeded. Attacks of thymic stridor may be induced during one of the acute
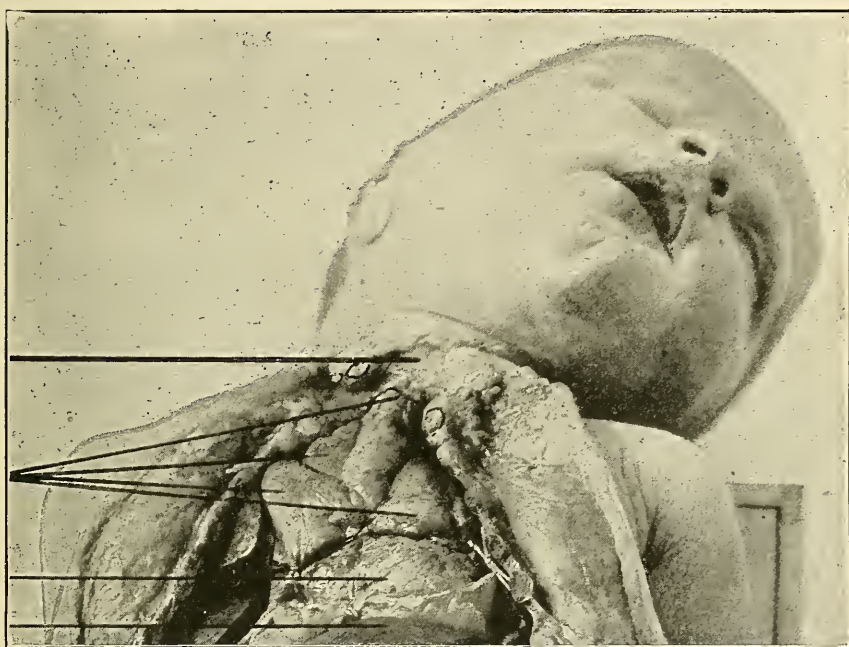
Fig. 46.—An infant with an enlarged conglomerate thoracic thymus *in situ*. *A* thyroid, slightly below normal position. *B*, lobes of greatly enlarged thymus. Incomplete separation is into lobes, upper, right and left. The upper is partly divided by an incompletely formed vertical depression. The fissures separating the lobes are nowhere complete. Near the left inferior anterior margin of the right lobe is a small partly detached mass of thymic tissue that might be called an accessory lobe. *C*, heart covered by intact pericardium; auricles concealed and compressed by enlarged thymus which extends a little lower on the right than on the left side, thereby conforming to the base of the ventricles. *D*, summit of liver. (Case of Dr. E. P. Davis.)

infectious diseases, or may be precipitated by a fit of crying or screaming in which the head is thrown backward. These acute attacks, which may subside entirely, are probably due to congestion of an already enlarged gland. In other cases thymic stridor is constantly present and there is an audible respiratory sound, both on inspiration and expiration, the voice remaining clear. The intensity of this sound is increased during crying or coughing, and is diminished during sleep.

Aside from the presence of this stridor the child may be practically

well. The child subject to thymic stridor may suffer from acute exacerbations in which the difficulty of respiration is markedly increased, and these attacks are known as thymic asthma. At such a time the child appears to be suffocating, the head is thrown back, and inspiration is accompanied by retraction of the intercostal spaces and the suprasternal notch. The face wears an anxious expression and becomes cyanotic and then pale. The extremities are rigid and the hands clenched. The pupils dilate, the heart sounds become weak, the pulse is lost at the wrist, and the child may die. Recovery from the attack may be complete with disappearance of all symptoms, or the
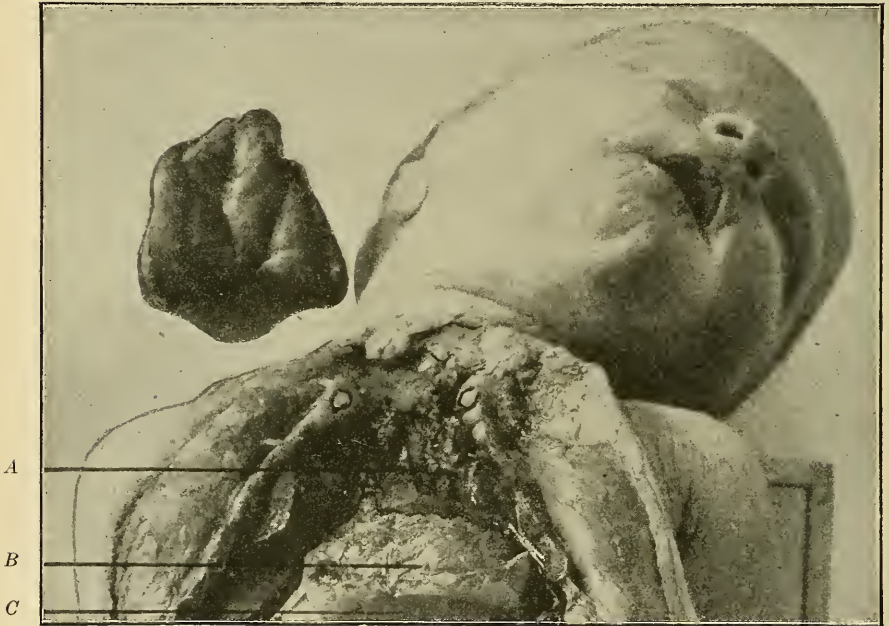
Fig. 47.—An infant with an enlarged conglomerate thoracic thymus, removed (shown just over right shoulder of infant), disclosing cavity occupied. *A*, compressed cava. *B*, heart, the flattening of which is well shown. *C*, summit of liver. (Case of Dr. E. P. Davis.)

stridor may persist. As a rule, repeated attacks occur, growing progressively worse until death. Cases of thymic asthma occur in which there has been no previous thymic stridor, and thymic death is not always preceded by thymic asthma. The term, thymic death, is applied particularly to those cases of sudden death attributed to enlargement of the thymus in which there has been no previous stridor or asthma.

The question as to whether or not the enlargement of the gland itself is the essential pathological basis for thymic death is still in dispute, but the preponderance of opinion seems to justify this con-

clusion. The manner in which death is caused by enlargement of the thymus has not been proven to the satisfaction of all, but most authorities agree that the pressure exerted by the enlarged gland causes tracheal stenosis and secondary laryngeal spasm. In some cases which have presented no clinical evidence of stenosis of the trachea or spasm of the larynx, death is presumed to have resulted from cardiac paralysis, since there is also pressure upon the heart, great vessels, and vagi and recurrent nerves. Pressure upon the great vessels is held responsible for the general edema usually present in these cases, and pulmonary edema which frequently precedes death may be due to pressure on the pulmonary arteries and veins. Death from enlarged thymus is sudden, and a previously healthy child may be found dead in bed. Frequently death follows some trivial accident or occurrence, such as fright, or a fall into water, a slight burn, the prick of a hypodermic needle, or the inhalation of a few drops of an anesthetic. Trivial operations, such as the extraction of a tooth or removal of the tonsils and adenoids, have been fatal. Thymic death may occur during an acute infection, and is particularly frequent in diphtheria.

STATUS LYMPHATICUS.

Status lymphaticus is a rare condition in which there is found an enlarged thymus and general hypertrophy of the lymphatic system. The tonsils and adenoids are enlarged, and there is a hyperplasia of both superficial and deep lymph nodes. There is a tendency to hyperplasia of the vascular system, seen especially in the aorta. From a clinical standpoint there is a lowered vitality, and an unstable equilibrium of vital forces which results in sudden death of the individual from cardiac and respiratory failure, brought on by trivial incidents.

Symptoms.—The child may be apparently well, but looks anemic, and frequently shows signs of rachitis. It is subject to frequent attacks of tonsillitis and catarrhal conditions of the nose and throat. Gastro-intestinal disturbances are common. Cyanosis, dizziness, and syncope indicate an unstable circulation. A prominence of the upper part of the sternum and the suprasternal notch may be visible on inspection. The upper part of the gland may be palpable at the root of the neck, and the superficial lymph glands and spleen are found to be enlarged on palpation. Thymic dulness is increased as is shown by a wider area of impaired resonance on both sides of the upper part of the sternum. The stridor is frequently audible some distance from the patient, and is always heard distinctly all over the chest.

Diagnosis.—The diagnosis of enlarged thymus is based upon the occurrence of chronic stridor, usually congenital, which at times becomes severe, resembling asthma, and may require intubation or tracheotomy for the relief of dyspnea. Careful examination of the throat should be made to exclude adenoids or malformations or obstruction of the larynx as a cause of the stridor. Enlarged bronchial glands may produce stridor by compression, but the enlargement

usually follows pertussis or bronchopneumonia, while thymic stridor is apt to be congenital. The *x*-ray is probably the most valuable aid in diagnosis of enlarged thymus, although variation in size, within reasonable limits, may be expected under normal conditions.

Prognosis.—The prognosis of thymic enlargement is unfavorable. Recovery is quite common in those cases with mild stridor, but when more severe symptoms develop, a fatal outcome is to be expected. Intubation or operations on the thymus are often successful in relief of symptoms, or as curative measures, but are very dangerous procedures, since death frequently occurs during the administration of an anesthetic or on the operating table. Intercurrent infections are often fatal in a child with an enlarged thymus.

Treatment.—The child with an enlarged thymus should live as quiet a life as possible, and be carefully guarded from all excitement. The general health must be improved by careful hygiene, outdoor life, and well-regulated diet. Bathing should be carried out most carefully and always in lukewarm water, as either extreme of temperature may be fatal. Acute infections of the upper respiratory tract are especially liable to precipitate an attack of thymic asthma, and should be carefully avoided. During an acute attack of stridor or asthma hot or cold applications may be made to the neck or sternum. Cardiac stimulants with oxygen may be necessary to prevent suffocation. If dyspnea is severe, intubation or tracheotomy may be necessary. The parents should be told of the possibility of sudden death before anesthetization, which should be proceeded with most cautiously. Tracheotomy may be done under local anesthesia, and chloroform given through the tracheal cannula is preferred for thymectomy. Intubation may terminate a severe attack of thymic asthma, but if there be a stenosis of the trachea, the withdrawal of the tube causes another attack. The Roentgen ray brings about a decrease in size of the thymus, lymph nodes, and spleen when applied to the thymus in status lymphaticus, and the results are so satisfactory that this plan of treatment should always be tried. In children Roentgen irradiation is preferred to thymectomy because of the effect of removal of the thymus on the bones and sexual organs. For this reason partial thymectomy is the usual operation, or the organ may be stitched to the under surface of the sternum.

DISEASES OF THE THYROID GLAND.

GOITRE.

Acquired goitre occurs most frequently in children at or before puberty, while congenital goitre is very rare and is only seen in goitrous districts where the parents are also goitrous. The infectious

diseases are occasionally followed by goitre. Exophthalmic goitre is rare in childhood, but may occur in the first year.

Exophthalmic Goitre (Graves's Disease).—This disease, which was first described by Graves in 1835, is characterized by enlargement of the thyroid gland, tachycardia, exophthalmos, and muscular tremors.

Etiology.—Emotional shocks, such as fright, grief, worry, are said to be important factors in the production of Graves's disease. During childhood it is most apt to occur in neurotic girls, at or about puberty. Infection can only be indirectly associated with exophthalmic goitre. The children of epileptic and alcoholic parents are said to be predisposed to this disease. It is more than twice as frequent in girls as in boys, but care must be taken not to confuse the hyperemic goitre which occurs in young girls with exophthalmic goitre. The hyperemic goitre is not accompanied by exophthalmos or tremors, and the condition disappears with menstruation.

Symptoms.—The symptoms of exophthalmic goitre in the child are practically the same as in the adult. There is enlargement of the thyroid gland, tachycardia, exophthalmos, and muscular tremors. The symptoms may resemble chorea. The child is irritable, easily excited, and becomes depressed if left alone. There may be nausea and vomiting at the sight of food. In early childhood there is usually profuse diarrhea. The general health of the child is poor, sleep is disturbed, and there may be attacks several times a day in which there is a breaking out into cold perspiration. The skin may be pigmented. The eyes are staring and may bulge out of the orbital cavity so that there may be hesitation in the descent of the upper lid when the eyes are turned down. The enlargement of the thyroid is usually bilateral. There is a systolic murmur at the base of the heart, the blood-pressure is increased, and hemorrhages may arise from the nose, stomach, or intestines.

Prognosis.—Graves's disease is rarely fatal in children but tends to run a chronic course with remissions of symptoms. The younger the child, the better the prognosis, as a rule.

Treatment.—The patient should be put to bed and kept there until the active symptoms subside. The diet should be nutritious, but light, in order to avoid gastro-intestinal disturbances. The tachycardia may be so severe as to demand attention, and for this the child may be given spartein sulphate, strophanthus or digitalis. Cold applications to the heart are also beneficial. Belladonna combined with sodium iodide is beneficial in some cases. In conjunction with this treatment, the x-rays, galvanic current, and injection of serum are sometimes used. Thyroidectin, a product from thyroidectomized sheep, has proven of distinct value in many cases and should be administered. The use of thyroid extract or the gland substance is contraindicated. Adrenalin extract and thymus gland have been used. When medical treatment fails, the case should be treated surgically, a partial thyroidectomy being the usual operative procedure.

CRETINISM.

Cretinism is a form of idiocy associated with myxedematous cachexia and defective growth of the bony skeleton, and is due to deficient secretion of the thyroid gland. There are two forms, the sporadic and endemic, but the latter type does not occur in the United States.

Etiology.—The endemic form of cretinism is seen in many of the geographical locations of endemic goitre. It is common in the goitrous districts of Europe, and particularly in Styria, the Tyrol, Savoy, Piedmont, and in Switzerland, but does not exist in those areas of North America where goitre is of frequent occurrence. Cretins in goitre districts have goitrous mothers, or both parents may be mild cretins, but cretins, as a rule, cannot conceive or bear a living child. The thyroid gland usually presents a goitre, and shows extensive degeneration in this form of cretinism, but the symptoms are practically the same as in the sporadic type. Sporadic cretinism is related to endemic cretinism, but the etiology and pathology differ. The parents of sporadic cretins are not goitrous, as a rule, but tuberculosis, alcoholism, and consanguinity in the parents are said to have a predisposing influence. Psychical disturbances in the mother during pregnancy, such as grief, worry, and fright have been noted in connection with cretinism. Thyroiditis, arising from trauma or during measles, enteritis, or typhoid fever may give rise to cretinism. It is rare in tropical climates, and extremely unusual in the negro. Male cretins are more common than females.

Pathology.—The most important changes in the body of the cretin are related to the thyroid gland. The gland may be congenitally absent. In other cases it is goitrous, and in most cases it is atrophied. It may be so small as to be overlooked after careful search, and considered absent. The bony skeleton shows arrested and retarded development. The bones of the skull are thickened, the sutures remain open, and the fontanelles do not close until late. The base of the skull may be altered in shape, and the posterior clinoid processes are higher than the anterior. The foramina are narrowed. The bones of the extremities and the ribs may be altered in shape. The pelvis is narrow. Bony changes vary in degree according to the age at which the lack of thyroid secretion was felt. Adipose tissue is very abundant in the omentum, and also beneath the skin which is thick, with scanty development of hair and sweat glands. The brain shows no gross abnormality, but the pituitary is occasionally found to be atrophied or hypertrophied.

Symptoms.—Evidences of cretinism are rarely noticed by the parents until after the first year. In those cases observed before the first year the infant is dull and passive, mentally inactive, and takes little or no interest in its surroundings, or as to how it is manipulated. It is usually of normal length and weight at birth, but fails to gain in height or weight. A child of eight years will simulate one of three, and full-grown cretins are never over three to five feet tall. The skin is thick,

coarse, and dry, but does not pit on pressure. The hair is dry and coarse and grows poorly. The fontanelles remain open late, but there are no signs of rachitis. The teeth may not appear, or if they do are late and very imperfect. The eyelids are wide apart. The nose is flattened, with the nostrils dilated, and the bridge sunken. The ears are large, and have a waxy appearance. The lips are thick, and the tongue which is thickened and broad, protrudes from the mouth. The neck is short and thick, and the head appears to be placed directly on the chest which is short and hollow. The abdomen is usually large, protuberant, and there is invariably an umbilical hernia. The arms and legs are short and stubby. The hands are spade-like,
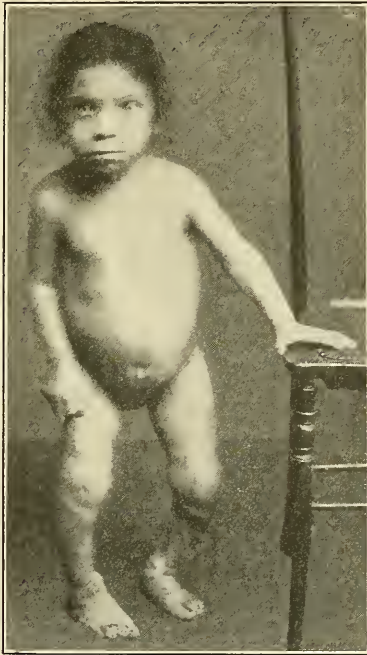
Fig. 48.—Cretinism.

and the fingers are blunt. The genitals are large, and the skin of the scrotum is thick. Mental dulness may be noticed as early as the sixth month. The child has a vacant stare, a meaningless smile, and does not play. Deafness caused by adenoids or middle-ear disease, which is common in cretins, adds to the deficient mental state. Cretins may be trained just as animals are, and may even learn easy sentences, but further mental development is impossible. Both mental and physical exertions are difficult. The habits are uncleanly, and the disposition is usually pleasant, but may be vicious. The temperature is subnormal. The appetite is good but there is apt to be a dislike for meat. Constipation is usually met with, is obstinate, and

may persist for a long time. Blood examination reveals an anemia with marked diminution in red cells and hemoglobin. The thyroid gland is not palpable. The arrest in mental and physical development varies according to the age at which the deficiency of thyroid secretion became effective.

Diagnosis.—The diagnosis of a typical case, well advanced, is easily made, but early diagnosis, which is so extremely important, may be very difficult in infancy. The coarse, dry skin, short stubby extremities, subnormal temperature, and slow mental responses form a combination which points strongly to cretinism. It must be differentiated from rachitis, Mongolian idiocy, achondroplasia, and dwarfism.

Fig. 49.—A cretin, eighteen years old.

Rickets.—The mentality in rickets is normal, and the characteristic bony changes are easily demonstrated by the skiagraph.

Dwarfism.—Even if associated with idiocy, dwarfism may be differentiated by the absence of the facial expression of the cretin and the skin changes. The body of the dwarf shows symmetry, which the cretin lacks.

Mongolian Idiocy.—Mongolian idiots present the characteristic slanting eyes, but lack the peculiar faces of the cretin. The body is more symmetrical, and instead of the apathy of the cretin undue restlessness is the rule.

Achrondroplasia.—This disease may be differentiated from cretinism by the fairly well-developed intellect, and the more marked shortness of the extremities. The size of the thyroid gland may be of service in clearing up a puzzling case, and a trial administration of thyroid extract will show definitely whether cretinism be present or not.

Prognosis.—The prognosis depends largely upon the stage of the disease at which treatment was instituted. The earlier it is given, the greater the improvement, but if the condition is neglected and allowed to continue too long the case may go beyond medical aid and fail to show even temporary improvement under treatment. When treatment is carried out thoroughly from an early stage of the disease, the improvement is miraculous. The child grows taller, gaining several inches each year. It loses its dull, apathetic appearance, and the mind becomes alert and receptive. Cretins rarely live over thirty years, although exceptional cases, attaining the age of fifty and sixty years, have been reported. Death is usually due to an intercurrent infection, to which they are very susceptible.

Treatment.—The cretin must be treated for years if permanent results are to be obtained. The fact that the most beneficial results of treatment are noticed where it is instituted early in life, has already been emphasized. After treatment is once begun, a neglect of it causes a relapse and tendency toward the original condition. The administration of the desiccated extract of the thyroids of sheep is the accepted treatment for cretinism. The dose should commence with $\frac{1}{2}$ grain t. i. d. for an infant, and proportionately larger doses for children. The amount of thyroid taken daily should be increased until the maximum effect is obtained, and then kept at that level until the de-

FIG. 50.—A cretin, twelve years old.

sired improvement has taken place. Recession of the tongue, loss of adipose tisue, change in the facial expression, and mental changes are the first signs of improvement. The skin becomes moist with restoration of activity in the sweat glands, the hair grows more abundantly, and is fine and glossy. The body becomes shapely, and there is a rapid increase in height. The mental improvement is even greater than the physical change. These results are usually obtained without increasing the dose over 15 to 30 grains daily, according to age. After definite improvement is noticed the dose may be gradually cut down, and even stopped at intervals, until the actual amount of thyroid required by the patient is ascertained. Grafting the thyroid gland has not met with definite success, and the old method of feeding the fresh gland,

33

either cooked or uncooked, has been discarded. Overdoses of the thyroid extract may be quite serious, and too large doses cause headache, faintness, rapid pulse, nausea, and fever. Exaggerated pulse rate is an indication to cut down the dosage. In addition to the administration of thyroid extract, the child should have the benefit of the best hygienic measures. The diet should be carefully watched, and the protein intake increased. Fresh air is essential, and massage and exercise are of distinct value.

DISEASES OF THE ADRENAL GLANDS.

The adrenals rarely become diseased before the tenth year, but during early childhood may be the seat of hemorrhages or tumors. Hemorrhages into the adrenals may be capillary or punctate. They occur in the newborn, during the course of gastro-enteric infection, chronic cardiac or pulmonary disease, and in septicemia and pyemia. Some writers attempt to classify the symptoms into three groups: asthenic, nervous, and peritoneal. There is usually associated with hemorrhage of the adrenals a severe acute illness and purpura. The onset is sudden, with fever, violent pain in the hypochondrium, convulsions, vomiting, diarrhea, tympanites, collapse and death in forty-eight hours. The symptoms, together with the purpura, simulate a fulminating type of one of the exanthemata, and unvaccinated cases are not infrequently mistaken for smallpox. The pathology is unknown, and the treatment ineffectual, because of the rapid progress and peculiar nature of the affection.

Tumors of the adrenals, arising during childhood, may cause marked disturbances in development with regard to growth, and sexual development particularly. In some cases the symptoms are thought to be due to hypersecretion, and in other cases to toxic products from a breaking-down process in the tumors. It occurs much more frequently in girls than in boys, and if the girl be very young she tends to acquire the male sex characteristics. The reverse is not true in boys, but their development is also precocious. There is a marked tendency to take on fat, and an excessive growth of body hair. The outlook in these cases is unfavorable, for diagnosis is difficult, the growth rapid, and metastasis early and frequent.

ADDISON'S DISEASE.

Addison's disease is rare in children, and when it does occur usually comes on after the tenth year. It is characterized by pigmentation, muscular and vascular weakness, and nervous and gastro-intestinal disturbances.

Etiology.—The exciting cause and predisposing factors of Addison's disease are unknown.

Pathology.—In the majority of cases there is a lesion of the adrenals which shows caseation and calcification. Tuberculosis also may be demonstrated in other parts of the body. The tubercle bacilli may be present in the adrenals at death. Some few cases are not tuberculous and the gland may show simple atrophy, resulting from a chronic interstitial inflammation. Sarcoma, cancer, and hypernephroma of the adrenals have also been found associated with Addison's disease.

Symptoms.—The skin is pigmented and becomes a deep yellow or bronze; the discoloration beginning at the nipples, axillary regions, hands and face. The mucous membranes of the mouth and vagina are also pigmented. White areas of skin may be observed scattered over the body. Pigmentation of the mucous membranes is said to be pathognomonic of Addison's disease. The child very gradually becomes weak and emaciated and listless. This weakness progresses steadily to exhaustion, and the emaciation grows worse, with a rapidly developing secondary anemia. There are vomiting, diarrhea, and other gastro-intestinal disturbances. Nervous symptoms are marked, and convulsions may occur. The heart is weak, and dyspnea and palpitation follow the slightest exertion. There may be abdominal pain, with rigidity of the walls of the abdomen, suggesting peritonitis.

Diagnosis.—Although Addison's disease is rarely seen, the diagnosis may be made easily if there is pigmentation with gradual asthenia, uncontrollable diarrhea, vomiting, and abdominal pain. The pigmentation must be differentiated from that caused by metallic poisons, such as silver, lead, and arsenic. Other symptoms of Addison's disease may suggest a primary anemia, but this may be excluded by careful study of the blood. Tuberculosis of the peritoneum, with melanodermic and abdominal crisis may resemble Addison's disease, but there is no pigmentation of the mucous membranes in tuberculous peritonitis.

Prognosis.—The course of the disease is much more rapid, and death comes on sooner in children than in adults. Practically every case is fatal, and doubt exists as to the true nature of the disease in those cases with recoveries reported, although recovery is possible. If there is uncomplicated tuberculosis of the gland, the disease runs a slow course and may last years, but cases due to atrophy of the adrenals are rapidly fatal. Death may come on gradually from exhaustion, or suddenly with diarrhea, vomiting, fever, syncope, toxic symptoms, and paralysis of the cardiac muscles.

Treatment.—The treatment of Addison's disease is largely symptomatic. The child should have plenty of rest, a light nutritious diet, and be kept warm at all times. Tonics containing arsenic or strychnine may be given. Hematinics and roborants are sometimes used. Glandular extracts from the parathyroids, pituitary, and suprarenals have been tried in these cases with indifferent success. The adrenal gland of the sheep may be given raw or cooked; the dose varies up to

one-half gland, according to the age of the child. The dose of the dried gland in tablet form is from $\frac{1}{4}$ to $\frac{1}{2}$ grain t. i. d. There have been cases reported where adults were benefited by operative treatment, and this may, at times, be advisable in the child.

THE PINEAL GLAND.

The pineal body which lies under the posterior end of the corpus callosum is occasionally during childhood the site of tumors which cause characteristic changes in development. The growth of the child is markedly increased and there is psychic as well as physical precocity. The sexual organs grow very rapidly and functionate early. In boys, especially, there is early and profuse growth of the beard and body hair. The child is obese, the voice changes early and cachexia gradually develops. As in tumors of the pituitary gland, the progress varies according to whether the tumor be malignant or benign. In either case the prognosis is fatal, and treatment, at the best, will only bring temporary relief of symptoms.

THE PITUITARY GLAND.

The pituitary body is sometimes during childhood the site of both benign and malignant tumors, which, in addition to the usual symptoms of cerebral tumor, also cause precocious development. There is a marked increase in adipose tissue and a lack of sexual development, the diminutive size of the organs being made more striking by the oversized child. The penis may be infantile after the age of puberty. Symptoms of cerebral tumor are usually present, and include headache, vertigo, vomiting, somnolence, and epilepsy, with disorders of the taste, smell, and vision. The course depends upon the character of the growth, and is rapid if there be malignancy, but may be very gradual, with remissions, if the tumor is benign. The treatment is surgical if the symptoms become severe, but the ultimate result is fatal. The headache is often severe and hard to relieve. Pituitary extract has been used upon the basis of hyposecretion, and thyroid extract is sometimes given for the adiposity.

DISEASES OF THE BONES AND JOINTS.

ACUTE INFECTIOUS OSTEOMYELITIS.

Definition.—Acute infectious osteomyelitis is most common during childhood and signifies an acute infectious inflammation of the bones.

Etiology.—The infection is hematogenous, the organisms gaining access to the blood through ulceration of the mucous membranes, lesions of the skin, and intestines. The Staphylococcus pyogenes aureus is the invading organism in most cases, but the Bacillus influenzæ, Bacillus coli, and Bacillus typhosus have also been isolated. Streptococci may be found in cultures from cases following scarlet fever, measles, or pneumonia. The pneumococcus has been isolated in a few cases, and may complicate or occur independently of pneumonia. Injury to a bone predisposes to an acute osteomyelitis, and compound fractures are a common cause. This disease occurs with equal frequency in boys and girls.

Pathology.—The infection may begin as a periostitis and extend into the marrow cavity through the Haversian canals or juxta-epiphyseal disks, or it may arise in the marrow cavity and infiltrate the cancellous bone, giving rise to necrosis with a periostitis following. After the deposition of the infecting organism in the tissues, there follows hyperemia, swelling, and rapid formation of pus which may fill the medullary cavity. The pus may infiltrate the epiphysis and the joint may become involved, or it may, after involving the periosteum, rupture through and burrow along the line of least resistance to the skin.

As a rule the infection spreads rapidly, but it may become circumscribed in a bone, forming a bone abscess. The long bones are most frequently involved, but acute osteomyelitis has been observed in the bones of the hands, feet, and skull.

Symptoms.—The onset is sudden in children. There is high fever, preceded by a chill and accompanied frequently by vomiting. Pain is acute and severe, and there is exquisite tenderness in the affected part. This may be hard to demonstrate in very young children, but the part soon becomes red and swollen, with an increase in the local temperature. Bacteria may be found in the blood stream.

Diagnosis.—The diagnosis is not usually attended with difficulty and is made on the acute onset, with localized symptoms usually referable to one of the long bones, and accompanied by severe constitutional disturbances.

Prognosis.—The prognosis is serious in very young children, and the mortality is over 50 per cent. This is caused by the development of

secondary foci of suppuration. In older children the outlook is much better, and recovery may be expected in over 80 per cent. of cases.

Treatment.—The treatment of acute infectious osteomyelitis is surgical.

TUBERCULOSIS OF BONES.

Bone tuberculosis may, as a part of miliary tuberculosis, give rise to an acute tuberculous osteomyelitis, but the chronic form of bone tuberculosis is by far the most common type. The bacilli are carried to the bone through the blood stream, and may set up a destructive inflammatory process in any part of the bone, but the infection usually

FIG. 51.—Tuberculosis of foot.

begins in the epiphysis. The process shows a tendency to extend, and, as a result, the neighboring joint and surrounding tissues become involved, with the formation of a "cold abscess."

Bone tuberculosis is usually secondary to some foci in the body. The human type of tubercle bacillus is practically always present. During the first three months of life, tuberculosis of bone is rare. The most frequent sites are the neighborhood of joints of the vertebræ and the long bones, while the bones of the hands and fingers are very rarely involved.

Symptoms.—Pain is constant in bone tuberculosis, but is often indefinite and obscure. It is rarely present in the onset, and when

present is almost invariably referred to some other part. The surrounding tissues are gradually involved, causing swelling of the part and frequently, suppuration. The general health is impaired, and emaciation and cachexia develop in the later stages.

Diagnosis.—The question as to whether a bone lesion is syphilitic or tuberculous will arise in a great many cases. If tuberculous the pain is less severe, suppuration is more frequent, and the tissues surrounding the infected bone are involved. Other foci of tuberculosis may sometimes be found, and a tuberculin test should be made. The constitutional symptoms are more marked in tuberculosis of the bones. In acute inflammation the process is always rapid, while tuberculosis is slow.

Prognosis.—The prognosis is good if the case is treated early and upon rational principles that have been proven efficacious in lung tuberculosis.

Treatment.—The treatment of bone tuberculosis is both general and local. Outdoor life at the seashore where the climate is equable is ideal for these children. The diet should consist largely of milk, eggs, oatmeal, and other high protein cereals. Tonics, such as cod-liver oil, malt, tincture of nux vomica, iron, and quinine are indicated if they do not interfere with digestion and the appetite.

Local treatment consists in extreme conservation, which is more successful in children than in adults, probably because the after-treatment is carried out to a greater degree. Avoidance and, later, correction of deformity are aimed at, and strict immobilization is maintained in the most favorable position by means of carefully applied plaster-of-Paris splints.

Abscesses should rarely be opened, as they always become absorbed when the patient's general health has received proper attention. Injection treatment in bone tuberculosis is useless. The evacuation of a tuberculous abscess always tends toward a perpetual sinus, with accompanying mixed infection.

SYPHILIS OF BONES.

The bone changes in delayed hereditary syphilis usually come on at about the fifth year. The long bones are most frequently involved, particularly the tibia. The greatest changes are seen at or near the epiphysis, causing an irregular broadening of the epiphyseal line. There are proliferative changes in the periosteum from a chronic or subacute periostitis, and this may often be detected by running the finger down along the crest of the tibia. The epiphyseal junction is enlarged and swollen, and in exceptional cases the epiphysis may become detached. Gummata are infrequent in bones.

Symptoms.—Swelling and enlargement of the epiphysis, with roughening along the shafts of the long bones, is characteristic of syphilis. Pain is acute and worse at night. Suppuration rarely occurs as a result of bone syphilis, and there is no cachexia or other marked constitutional symptom.

Diagnosis.—Bone syphilis must be differentiated from tuberculosis of the bones, and from rachitis. In addition to the points of differentiation from tuberculosis, mentioned in the diagnosis of bone tuberculosis, a positive Wassermann reaction and the concomitant signs of syphilis may be present. In rachitis the bones are not so painful as in syphilis, and there is no roughening, although they may be thickened at the ends.

Prognosis.—The prognosis is good if the child comes under observation early, and treatment is carried out thoroughly.

Treatment.—Syphilitic bone lesions in children respond strikingly to mercury and the iodides. A child five years of age should be given from $\frac{1}{30}$ to $\frac{1}{20}$ of a grain of bichloride of mercury daily, with from 12 to 20 grains of potassium iodide daily, the drugs being given in separate mixtures. If the bichloride of mercury given by the stomach disagrees, from 20 to 30 grains of mercurial ointment may be well rubbed into the skin daily. In order to prevent gastric disturbance, the administration of potassium iodide should be interrupted for a few days every month; later these drugs may be given alternately. The part should be protected locally, and placed at rest. Suppuration calls for surgical intervention with removal of the dead bones.

Fig. 52.—Tuberculous dactylitis of ring finger.

DACTYLITIS.

Dactylitis is a disease of the phalanges in children, causing a fusiform swelling, and may be syphilitic or, rarely, tuberculous. It may also be due to streptococcic infection or trauma.

Pathology.—In both tuberculous and syphilitic forms the process, which is a rarefying osteomyelitis, begins in the centre of the bone,

resulting in an enlargement of the medullary canal, while at the same time there is a proliferative periostitis, causing a fusiform enlargement. Suppuration and necrosis occur, and a finger or toe may be lost. An acute dactylitis, arising from streptococcic infection or trauma, presents the symptoms of acute osteomyelitis.

Diagnosis.—Syphilitic dactylitis is more common than the tuberculous form. It occurs most frequently during the first two years and usually involves several bones. The proximal phalanges are apt to be involved in syphilis, and there is not the tendency to affect the metacarpals, seen in tuberculosis. There is rarely suppuration, and the surrounding tissues are usually not involved if the type be syphilitic.

In the tuberculous form metacarpals and phalanges are involved. There is tumefaction, due mostly to swelling of the soft tissues, and the
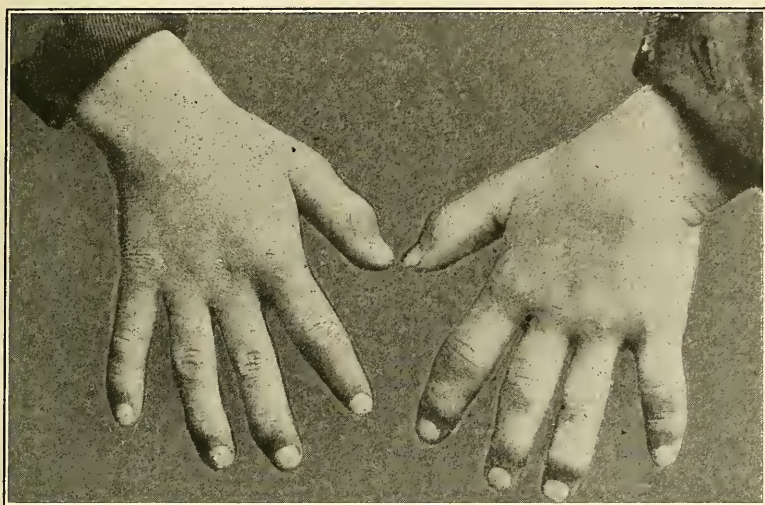
Fig. 53.—Syphilitic dactylitis involving all fingers and thumb of left hand and thumb of right hand.

part is tender. The tuberculous lesions are less apt to be multiple. The history of the case, the presence of concomitant symptoms of either disease, and the Wassermann and von Pirquet tests are additional aids to diagnosis.

Prognosis.—The underlying constitutional disease should be treated at once. Locally the part should be put at rest and kept immobile by splints for months. Abscess formation and necrosis, if they occur, require surgical intervention.

CRANIOTABES.

Craniotabes is a condition characterized by the presence of soft spots of thinning in the cranial bones. It may be due to several causes. Syphilis is found in over 50 per cent. of the cases. Rachitis is also

present quite frequently, and the most marked cases are seen in children with both syphilis and rachitis. It may occur in hydrocephalus of the chronic type.

In a certain percentage of the cases, no underlying condition can be found to account for the craniotabes. These areas are found in the parietal and occipital bones. They are caused by bony absorption which begins on the inner table of the skull, and is supposed to be due to pressure of the brain internally, and the pillow externally.

BOSSING OF THE SKULL.

Bosses on the bones of the vault of the skull are caused most frequently by rachitis or syphilis. The most marked cases are seen in children where both these etiological factors are present. This condition is due to exuberant formation of bone around the centres of ossification.

Bosses are usually found on the frontal and parietal bones, but may occur in the occipital and temporal regions. They may be confused with the osteoperiostitis of the bones of the skull seen in tuberculosis. Among the rarer causes of bossing are achondroplasia, hereditary cleidocranial dystosis and tumors.

ACUTE ARTHRITIS OF INFANTS.

This disease is also known as acute purulent synovitis and acute epiphysitis. It is a form of pyemia resulting in an acute inflammation of the joints with suppuration. The majority of cases occur during the first year, and most of these are seen during the first six months of life. The disease may begin in the epiphysis or medullary canal, but the joint is soon involved, the nature of the arthritis depending upon the infecting organism. The gonococcus causes an inflammation of the joint proper, and the synovial membrane is involved, but there are no destructive changes in the cartilage, ligaments, or bone.

The cases due to the staphylococcus or streptococcus are more severe, and the joint may be destroyed. The suppurative process shows a tendency to spread, and may result in diffuse osteomyelitis, subperiosteal abscess, or even a separation of the epiphysis. The final outcome is either a flail joint or bony ankylosis.

Etiology.—The infection is hematogenous, and may occur very soon after birth from an infected umbilicus. During the later months of infancy the organisms may enter the blood stream through abrasions of the skin or from the conjunctiva, genital tract or mouth. In some cases the portal of entrance cannot be demonstrated. The staphylococcus, streptococcus, gonococcus, and pneumococcus have been isolated.

Symptoms.—The constitutional symptoms are marked, and usually precede local signs. There is high fever, malaise, anorexia, and vomiting. Following this, one or several joints become swollen,

painful and tender. The overlying skin is reddened, and the local temperature increased. Suppuration occurs rapidly, and fluctuation may be present.

In severe cases, a general pyemia may develop, with visceral complications, such as pneumonia, pericarditis, or meningitis. In milder cases, the abscesses continue to grow larger and suppuration is confined to the joints. In the gonococcic form there may be no suppuration.

Diagnosis.—Some cases resemble acute rheumatic fever, but there is no endocarditis, and the joint lesions are much more severe. Syphilitic and tuberculous epiphysitis are very much more chronic in nature, the symptoms are not as severe, and other evidences of these diseases are present.

Treatment.—Cold compresses should be applied locally for relief of pain. When fluctuation is apparent, free evacuation of the pus should be obtained by an incision into the joint cavity. This should be followed by fixation of the joint. Life is often saved by prompt evacuation of pus, but the function of the joint is usually impaired.

TUBERCULOSIS OF JOINTS.

Tuberculosis is the most frequent infection of joints during childhood, and is usually secondary to tuberculosis of the epiphysis. In some cases there may be no demonstrable foci of tuberculosis anywhere else in the body. The process is essentially chronic, but an acute tuberculous osteomyelitis may sometimes be observed in miliary tuberculosis.

Symptoms.—The onset is insidious, and the earliest symptoms may not be noticeable to the parent. Pain is indefinite, quite obscure, intermittent, and referred to more or less remote parts. Impairment of function and atrophy of the muscles connected with the joint together with characteristic night cries are among the earlier symptoms. The joint may become swollen, and there is tenderness on pressure.

Spasm of the muscles surrounding an affected joint is one of the most constant symptoms. Rigidity is not present until the later stages of the disease, and fluctuation cannot be detected until the destructive process is well advanced. The constitutional symptoms are night sweats, night cries, anemia, anorexia, and a slight afternoon temperature.

Diagnosis.—Intermittency of symptoms often causes delay in arriving at a correct diagnosis. The chronic nature of a tuberculous arthritis distinguishes it from acute inflammatory rheumatism and acute infectious arthritis. Syphilitic arthritis is very rare, and is usually manifested by a bilateral effusion of the knee-joints. It responds quickly to the mercurials and iodides, so that the therapeutic test is of value in the differentiation. In syphilis the Wassermann reaction will be found positive, and concomitant signs may be in evidence. A positive von Pirquet reaction, and the presence of other foci of tuberculosis may be demonstrated in tuberculous arthritis.

Prognosis.—The prognosis is good if treatment is instituted early, and the destructive process stopped; otherwise, the condition grows steadily worse, and complete disorganization of the joint may result. Complete recovery with little or no impairment of motion is often obtained under careful management.

Treatment.—The treatment of uncomplicated cases of joint tuberculosis is conservative. This conservative treatment of bone tuberculosis in the child is extremely important. The tuberculous process in children almost invariably begins in the epiphysis or the juxta-epiphyseal region. Active operative interference on the epiphysis interferes with the subsequent growth in the length of the bone, which is a matter of vital importance.

Local pain, swelling, effusion into the joint, and fever do not necessarily point to operative interference unless pyogenic bacteria are present, with a resulting formation of pus. Of course, if pyogenic pus—not tuberculous pus—is present, an opening must be made and the pus removed.

Absolute rest to the joint, life in the open air, good food, and appropriate tonics, with plenty of time, will accomplish a great deal in children. This element of time is less important in the child than in the adult; usually the adult is a wage-earner, and the loss of time is a more serious matter. The joint should be put at rest in a plaster-of-Paris cast, but weight bearing has been proven not to be injurious when proper fixation of the joint is secured. Passive hyperemia, if used carefully and systematically, is of benefit in the earlier stages of the disease. Tuberculin is often administered with good results.

In the later stages, when the joint becomes destroyed and systemic symptoms are marked, arthrectomy may be considered but is rarely advisable. Cold abscesses are complications in about 50 per cent. of the cases of tuberculous arthritis, and when they occur they should not be opened except when spontaneous rupture is inevitable. The child with a tuberculous lesion in a joint should be put under the most hygienic living conditions, and everything possible should be done to improve the general health. Life at the seashore is of special benefit to these children.

CHAPTER XIX.

DISEASES OF THE GENITO-URINARY SYSTEM.

DISEASES OF THE KIDNEY.

THE URINE.

The infant usually micturates within twelve hours after birth, and in many cases micturition occurs spontaneously with birth. Certain constituents of urine are found in the liquor amnii, indicating intra-uterine activity of the kidneys, if not actual urination. The bladder normally contains urine at birth. The amount of urine passed daily and the specific gravity and percentage of the different constituents vary greatly during infancy, as well as during the greater part of childhood.

Quantity.—The quantity of urine passed for the first three days corresponds to the amount of liquids ingested. At each evacuation of the bladder from ½ to ¾ of an ounce is passed, with a total quantity of from 2 to 3 ounces in twenty-four hours. With the establishment of breast-feeding, the amount of urine increases with each succeeding day, and at the end of the first week the daily output is about 8 ounces. At six months, the average daily quantity of urine passed is about 12 ounces, and at two years 16 ounces. From the second year onward the daily output of urine increases 1½ ounces each year until the child is twelve years of age. There is an increased quantity of urine in diabetes mellitus and chronic interstitial nephritis, and a decrease in renal congestion and acute nephritis.

Children pass relatively larger amounts of urine than adults. Normally micturition occurs often during infancy, and a baby may urinate from ten to fifteen times a day under ordinary circumstances. There is great difficulty in collecting a twenty-four-hour specimen in children because of this frequency in urination, and the trouble experienced in securing a retainer to the child or infant. Male infants may have a condom attached to the penis, or a wide-mouthed bottle. Absorbent cotton may be used to collect the urine of female infants. These methods, however, all expose the specimen to contamination, and render it useless for bacteriological study, though perfectly reliable for ordinary chemical tests. To obtain an unadulterated specimen, a No. 6 American gauge catheter should be passed, and suprapubic pressure made, preferably after the child has been sleeping for a long time.

Specific Gravity.—For the first twenty-four hours the specific gravity averages 1005 to 1010, for the child does not ingest much liquid. When nursing becomes regularly established, the specific gravity falls to 1003 or 1004, and remains at this level during breast-feeding. From the second year the specific gravity steadily increases, until at puberty it ranges from 1010 to 1015. The specific gravity is increased by diarrhea, fever, and sweating.

Reaction.—The urine at birth is acid, and remains faintly acid, normally, throughout life. The ingestion of an alkaline diet will change the reaction of the child's urine, and a diet containing too much fat may cause it to become ammoniacal.

Color.—The color of the urine is a fair indication of its specific gravity, a pale yellow urine usually having a low specific gravity, and a high-colored urine, being concentrated, having a high specific gravity. The urine of infancy is much paler than during childhood. In fevers the urine becomes high-colored and turbid. Milky-white urine suggests pus, while urine of a reddish hue should lead one to suspect the presence of blood. Bile is sometimes found in the child's urine, and imparts to it a greenish-yellow color.

Hematuria.—Hematuria is the term applied to urine that contains blood corpuscles and blood pigment. In every case of suspected hematuria a microscopic study of the urine should be made to determine the presence of blood corpuscles, which differentiate this condition from hemoglobinuria. While the causes of hematuria are many and varied, idiopathic hematuria is quite common in children. These cases are unassociated with any appreciable constitutional disturbance, and no organic lesion can be demonstrated to account for the appearance of the blood. They usually clear up in a day or two, and are sometimes referred to as renal epistaxis. In some instances large quantities of blood may appear in the urine with no other symptoms than a slightly increased frequency of urination. The best treatment for these cases is rest in bed, mild catharsis, and a soft diet.

Hematuria immediately following birth may be due to the passage of uric acid crystals and infarcts, to hemorrhagic disease of the newborn, or to septic infection. After infancy it is seen most frequently in scarlet fever, typhoid fever, malaria, variola, scurvy, purpura, hemophilia, and leukemia. The ingestion of certain drugs, such as cantharides, turpentine, and other poisonous substances may also give rise to hematuria. Blood appearing in the urine may have come from the kidney, ureter, bladder, urethra, or from the genital tract. When hematuria has its origin in the kidney, urine is usually abnormal in color, the blood imparting to it a smoky hue. The appearance of blood casts in the urine is significant of renal hematuria.

The most common causes of hemorrhage from the kidney are the acute infectious diseases, hyperemia of the kidney, and acute nephritis. It may occur during chronic nephritis, and is also seen in association with infarcts, tuberculosis of the kidney, neoplasms, calculi, parasites, angiomata, abscesses, embolism, and cysts.

Hemorrhage from the ureter is usually caused by the passage of a stone or by neoplasms. Blood which comes from a lesion of the bladder is usually normal in color, and sometimes very abundant. It is generally due to either tuberculosis of the bladder, vesical calculi, or neoplasms. The blood from a hemorrhage of the urethra is normal, and may be uncontaminated with urine. It may be due to traumatism from stone or catheter, and in some cases is caused by gonorrhea. The treatment of hematuria consists, for the most part, in tracing the source of the hemorrhage, and removing its cause. Rest in bed, mild purgation, and a light diet are also beneficial.

Hemoglobinuria.—Hemoglobinuria is that condition in which blood pigment only is found in the urine, no blood corpuscles being present. Hemoglobinuria indicates either that the blood cells are being destroyed by some process and hemoglobin is being set free in the circulation, or that the hemoglobin is being dissolved out of the blood cells and passes into the circulation. Hemoglobinuria in epidemic form occurs in the newborn, being known as Winckel's disease. It may be produced by the ingestion of poisons such as potassium chlorate or carbolic acid. It is occasionally observed in the course of yellow fever, typhoid fever, malaria, and scarlatina, and occurs in children affected with scurvy, inherited syphilis, or purpura.

There is a paroxysmal form of hemoglobinuria in which the cause is unknown. The attacks are accompanied by chills, dyspnea, palpitation, and cyanosis. It is thought that the individual carries the hemolysin in his own blood, but, in addition, cold and exertion are necessary to precipitate an attack. Syphilis is regarded as a factor in this type of hemoglobinuria, having been found associated in 50 per cent. of the cases of the disease.

The treatment of hemoglobinuria is to remove the cause and support the child's strength by plenty of rest and a good nourishing diet. Syphilis should be suspected in every case, and upon its discovery antisyphilitic measures at once instituted.

Functional Albuminuria (*Postural Albuminuria, Cyclic Albuminuria, Orthotic Albuminuria*).—Functional albuminuria occurs principally in children, and is characterized by the presence of sero-albumin in the urine in the latter part of the forenoon and afternoon only. It disappears after a night's rest. The term "cyclic albuminuria" designates its regular appearance at certain hours of the day, and "postural albuminuria" that the erect posture on arising is a factor in its production. Albuminuria has been observed frequently in children with lordosis of the lumbar vertebræ, and is sometimes referred to as lordotic albuminuria.

Symptoms.—Children who give evidence of functional albuminuria are usually neurasthenic, and have an anemic appearance. They complain of vague pains, headache, and sometimes nausea. There is a lack of tone in the involuntary muscles. Dilatation of the stomach and heart are common. The pulse tension is low; the hands frequently become cyanosed. Many of these little ones suffer from epistaxis.

On the other hand, functional albuminuria may be observed in children apparently enjoying good health.

Diagnosis.—A diagnosis of functional albuminuria should be made only after careful consideration and the exclusion of any signs confirmative of pathological albuminuria. Its most distinguishing features are the entire absence of symptoms of nephritis and of casts in the urine, also the effects of rest on this condition.

Treatment.—Recovery from functional albuminuria is often slow, but is the rule. It may be hastened by a change of residence to a healthful, invigorating climate, and careful regulation of the diet to increase nutrition. Strychnine sulphate, in tonic doses of $\frac{1}{200}$ to $\frac{1}{100}$ of a grain, taken twice daily, is of recognized value in this condition.

Paroxysmal Albuminuria.—Paroxysmal albuminuria may be recognized as one of the forms of functional albuminuria. It is most frequently observed in connection with paroxysmal hemoglobinuria. There are no associated symptoms of nephritis during an attack, and the urine contains no casts. Between attacks, which usually last two or three days, the child enjoys good health.

Lithuria.—An excess of uric acid bodies in the urine is termed lithuria. It is common in children from birth to puberty, and indicates an increased amount of uric acid in the blood and tissues. Since the deposition of amorphous sediments of urates as the urine cools is a common event, and, in view of the fact that such trivial causes as mere concentration of the urine, or an increased acidity, or the temperature of the air, may precipitate these uric acid bodies, the amount of the sediment must not be interpreted as indicative of the amount of uric acid output.

Symptoms.—During infancy the urine contains a comparatively small amount of urea, but the uric acid output is high; in some cases the minute crystals coalesce and form calcareous masses. The passage of these masses and of uric acid infarcts may cause considerable abdominal colic, with tenderness over one kidney. The irritation produced by these bodies is evinced by the appearance of blood, of albumin, and even of casts in the urine. In infants there may be slight hemorrhage, giving rise to actual hematuria for a week or so during the excretion of crystalline uric acid.

A mild inflammation of any part or of the entire urinary tract is sometimes shown by the presence of large numbers of epithelial cells, leukocytes, and red blood corpuscles, also of mucus. After infancy an excess of urates in the urine is not so frequent; but in nervous, irritable, anemic children there may be recurrent attacks throughout childhood. It is sometimes observed in chorea, chronic dyspepsia, malnutrition, rheumatism, and scurvy, and always indicates severe nutritional disturbance. The microscopic features of the urinary sediment in lithuria are large numbers of uric acid crystals and a moderate collection of calcium oxalate crystals. A simple test is the application of heat to a specimen of urine, whereupon the tur-

bidity will disappear and the sediment, if composed of uric acid or urates, will be dissolved.

Treatment.—Regulation of the diet is impossible during infancy as the child should be kept on the breast; but older children may be placed on a diet that is poor in purin substances. Alkaline diuretics are indicated, and, to a child of three years, 5 grains of potassium bicarbonate, or 3 grains of potassium citrate, may be given four times a day to advantage.

Acetonuria.—Acetone is found in minute quantities in normal urine, but may be considerably increased in many pathological conditions. In children acetonuria is induced by slight causes, of which changes in the diet are, perhaps, most common. The chief cause of the formation of acetone is, apparently, the withdrawal of carbohydrates from the food, or inability to utilize carbohydrates. That carbohydrates alone are not the only factors in the production of acetonuria is demonstrated by the increased acetonuria caused by a diet rich in fats; and, in view of most recent experimental research, there seems to be no doubt that the proteins of the food and tissues must be looked upon as contributing to the total yield of acetone.

The frequency of acetonuria in diabetics need not be emphasized; but it also occurs in malignancy, prolonged fevers, starvation, digestive disturbances (especially when associated with persistent vomiting), and is frequently associated with bronchopneumonia. The appearance of acetone in the urine of epileptics has no bearing on the convulsive seizures, and though the association of acetonuria with cyclic vomiting is as yet not clearly understood, it is thought to be the result of the persistent vomiting, rather than the cause.

Symptoms.—The most important symptoms of acetonemic acidosis are dyspnea, or air-hunger, rapid pulse, and in fatal cases coma. The diagnosis, however, is based on an examination of the urine which reveals the presence of acetone and, perhaps, its kindred bodies—diacetic acid and oxybutyric acid. The presence of acetone is indicated by the well-known iron reaction; *i. e.*, the development of a deep red-brown color on the addition of a solution of ferric chloride to a specimen of urine.

When patients are taking salicylates or aspirin this reaction is, of course, masked by the iron reaction due to the drug. If doubt exists as to the cause of the reaction, the urine should be boiled for a few minutes, and the test repeated after it has cooled. As a result of such treatment acetoacetic acid will be broken up into acetone and carbon dioxide, and the reaction will no longer be obtained.

Treatment.—The first thing to be done in a severe case of acetonuria combined with great acidosis is to give a purge of calomel, 2 grains to a child of three years, followed by a saline cathartic; magnesium sulphate, 1 dram every two hours until free catharsis is produced, would, perhaps, be better. In addition to this, two measures are clearly indicated—the administration of alkalies to neutralize the acidosis and the giving of easily assimilable carbohydrates, such as levulose

34

or glucose. Sodium bicarbonate is probably the most efficient alkali, and should be given in 30-grain doses with glucose by the mouth, unless there be persistent vomiting, when both of these drugs may be given in solution by rectum. Fresh air, outdoor exercise, and massage are all beneficial, and prophylactic measures to promote convalescence.

Indicanuria.—Indican is found in minute quantities in the normal urine when a mixed diet is being ingested, and may be much increased by a protein diet, since it is derived from the proteins of the food which are in part decomposed in the intestine by bacteria. It may also be produced by conditions in which the cavities of the body contain fetid pus, as in fetid empyema and pulmonary abscess. With this exception, indicanuria may be considered a sign of bacterial disintegration of proteins in the intestinal canal, and the amount of indican in the urine as an index of the extent of this putrefaction.

Although indicanuria is, in most cases, due to either acute or chronic intestinal derangement, it is also frequently observed in chorea, typhoid fever, peritonitis, epilepsy, and malignancy. It is especially common in tuberculous enteritis, and has also been found in association with urticaria and other skin diseases. Indican is usually absent in the urine of normal breast-fed babies, but appears soon after infancy; it is much increased by constipation or masturbation, so that a strong indican reaction in children is always of pathological significance.

In testing for indican it is essential that a freshly voided specimen of urine be used, as indican rapidly disappears on standing, and renders an accurate estimate of the quantity of indican impossible. If tested accurately, the quantity of indican may serve as a guide to the severity of the associated disease and the effects of treatment. The presence of indican is detected by adding to 10 c.c. of normal urine an equal volume of hydrochloric acid and 1 or 2 drops of liquor sodæ chlorinatæ, or 3 drops of aqua hydrogenii dioxide, or a small pinch of sodium perborate. On standing several hours, the color changes to a bluish hue from the formation of indigo blue. By shaking with 1 c.c. of chloroform, the indigo dissolves and settles as a blue bottom layer, when the amount of indican may be estimated by the depth of the color.

Treatment.—The indications for treatment are to control the putrefactive process going on in the intestinal tract by the same measures referred to in the treatment of chronic intestinal indigestion and constipation.

Glycosuria.—Normal urine contains such a minute quantity of glucose that a positive reaction can not be obtained by the usual tests. Occasionally an appreciable amount may be detected in the urine of apparently healthy children; but this is usually a physiological glycosuria, the result of the ingestion of an excessive quantity of carbohydrates. A pathological glycosuria, which is temporary, sometimes occurs in affections of the heart, lungs, liver, brain, and spinal cord; but when sugar is present in the urine in appreciable amount we are dealing with diabetes mellitus. The urine of infants fed on artificial

preparations frequently gives a positive reaction for sugar; and in some breast-fed babies the urine responds to Fehling's test, but not to the fermentation test, showing, perhaps, the presence of lactose and not glucose.

Fehling's Test.—Fehling's solution, used for this test, deteriorates on standing, hence, for preserving, it is best made in two parts, and put in separate bottles; in this way it may be kept indefinitely. In one bottle, marked No. 1, keep the following:

Pure copper sulphate	17.32 grams
Distilled water	250.00 c.c.

In another bottle, marked No. 2:

FEHLING'S ALKALINE SOLUTION.

Rochelle salt	87.0 grams
Caustic soda	25.0 "
Distilled water	250.0 c.c.

To make this test, mix in a test-tube equal volumes of Nos. 1 and 2, dilute with an equal quantity of water, and boil. No precipitate should be noticed. Now, slowly add to this mixture half its volume of urine, and again boil. The presence of glucose will be indicated by an orange or red precipitate.

Pyuria.—Pyuria is that condition in which pus, from any cause whatsoever, is found in the urine. In children its origin is most frequently the bladder or pelvis of the kidney, but it may come from any part of the genito-urinary tract, or, in exceptional cases, from extraneous sources. This latter instance is best demonstrated by the rupture of a perinephric or appendiceal abscess into some portion of the genito-urinary tract.

The appearance of urine containing pus depends upon the quantity present and upon its reaction. In acid urine, in which pus is usually of renal origin or due to the Bacillus coli or a tuberculous infection, the pus tends to settle as a dense deposit of a yellowish or greenish hue. In alkaline urine the pus is stringy and less circumscribed, and tends to cling to the sides of the containing vessel; it usually comes from the bladder. In pyonephrosis the discharge of pus is apt to be intermittent; in cystitis it is fairly constant; and when due to the rupture of a neighboring abscess into the urinary tract there is usually a copious discharge of pus for a time, followed by more or less rapid cessation of the pyuria. Rarely, during childhood, does pus in the urine come from the genital tract; but, exceptionally, it may be due to purulent urethritis or vulvovaginitis, and when this is the source the pus appears in flakes and is mixed with mucus.

The only positive method of determining the presence of pus in the urine is by means of the centrifuge and microscope. The ropy condition produced by the addition of caustic alkali is often significant. The nature of the infection is of considerable importance, but can be demonstrated only by a culture from a catheterized specimen.

Treatment.—The treatment of pyuria depends chiefly upon the cause. Because of its elimination in the urine, hexamethylenamin is of value in every case, regardless of the etiology. A child two years old should be given 2 grains every four hours.

Dysuria.—Dysuria, or painful urination, is not uncommon in children and infants, and may be due to a variety of causes. Hyperacidity and the passage of large quantities of oxalates or uric acid crystals frequently give rise to dysuria during infancy, and in childhood the concentrated urine voided during fevers may be just as irritating. Small calculi are often passed by children; in these cases not only is there dysuria but severe colic attends the passage of the stone down the ureter. These calculi are frequently found on examination to be lodged in the anterior urethra. Dysuria may also be caused by cystitis, urethritis, vulvovaginitis, and phimosis.

In rare cases one meets with congenital anomalies which may render urination difficult and painful. In female infants there sometimes exists at birth a membrane which covers the vaginal orifice and extends over the urethral meatus with but a minute opening through which the urine may filter out drop by drop. In these cases, urination is accompanied by great straining and pain, but the membrane can easily be divided with a blunt dissector, which procedure at once relieves the condition. Urethral caruncles are extremely rare in little girls; but occasionally in little boys painful micturition is caused by a condition of the urinary meatus in which the lips of the urethra are swollen and slightly everted, and on close examination is seen a small bead of granulations, which is very sensitive. The best treatment is to remove the granulations and stitch up the wound.

In male infants there is occasionally a cellular adhesion of the prepuce and glans penis, or a thin membrane over the urethral meatus, which causes difficult, and often painful micturition. In these cases the prepuce should be freed from the glans, and the membrane removed from the urethral orifice. Following circumcision, a painful crack or vesicle sometimes develops near the meatus and makes urination agonizing. A bland antiseptic ointment will protect the raw surfaces from the urine, and effect a speedy cure.

Treatment.—The treatment of dysuria from other conditions is directed to the cause. If due to inflammatory processes along the genito-urinary tract, dysuria will ameliorate with improvement in the local condition; when due to hyperacidity, dysuria is relieved by the administration of alkaline diuretics and an increased intake of water. Potassium citrate, in a dose of 4 grains, t. i. d., in plenty of water, to a child of five years, is perhaps the most efficacious therapeutic measure to be employed in hyperacidity.

Anuria.—Anuria is that condition in which the kidneys fail to secrete any urine; it should be carefully distinguished from retention of urine, in which affection urine is normally secreted by the kidneys, but is retained in the bladder. When anuria occurs in the course of nephritis, it is referred to as "suppression of urine." In rare cases it

is due to congenital malformation of some portion of the urinary tract, but more commonly is a result of traumatism, of operative procedure, of the passage of a catheter, or the administration of ether or chloroform. Complete cessation of secretion by the kidneys has been observed in cases where children have in some way taken phenol, phosphorus, turpentine, cantharides, lead, bichloride of mercury, or other violent irritants to the kidneys.

Many cases of anuria during infancy cannot be accounted for, although it is believed that a considerable number are caused by uric acid infarcts of the kidney. Occasionally mere chilling of the body, a gastro-intestinal disturbance, or an acute infectious disease will be the only demonstrable cause of anuria. Uremic symptoms appear very late in children with anuria; and in some instances in which there has been no secretion of urine for ten days recovery has occurred. Unless due to malformation or organic disease of the kidneys, anuria rarely persists over twenty-four hours, and in that time nothing abnormal may be noticed in the infant's general condition. When urine is not voided, it is important to ascertain whether we are dealing with retention of urine or anuria before attempting to treat the condition. This distinction can easily be made by the passage of a catheter, which will reveal a full bladder in retention, and an empty one in anuria.

Treatment.—The general measures for the relief of anuria are hot fomentations to the loins, hot baths, and free purgation. To a child four years old 1 dram of magnesium sulphate in concentrated solution is, perhaps, the best saline cathartic to use. Sweet spirits of nitre, 5 drops every hour, and potassium citrate, 3 grains hourly, should be administered until the urinary secretion begins. Colonic flushing, using normal saline solution at a temperature of 110° F., is often a valuable aid to this medication. Care must be taken not to inject too much fluid into the colon or it will not be retained, and the use of one pint at not less than six-hour intervals is, perhaps, the most efficacious mode of employing it.

Retention of Urine.—Retention of urine to a mild degree is not uncommon in young children, but rarely does a case come under observation which requires catheterization for relief. It may be the result of lodgment of a stone in the urethra, of urethritis, or of vaginitis. Phimosis, when extreme and due to edema or local infection, also reflex painful conditions of the anus and rectum, may cause retention; still other cases are due to myelitis, injury to the spinal cord, or hysteria. Simple retention of urine not due to organic causes is rarely productive of any symptoms, and may not cause the child the slightest inconvenience, although the distended bladder may be noticeable on inspection of the abdomen, and on percussion may be found to extend to the umbilicus.

Treatment.—Immediate relief of retention is obtained by catheterization, but this is not always necessary, and quite frequently a small catheter may not be available. A No. 6 American scale catheter may

be used for infants, but only after the application of heat to the suprapubic region and genitals has proven ineffectual. In every case the exciting cause should be ascertained and treated. In myelitis, nothing can be done but to catheterize. Hysterical children cease to have retention when their nervous systems are gotten under control, and those cases caused reflexly by painful conditions of the rectum and anus disappear when proper treatment is directed to the under-lying factor. Not infrequently investigation may reveal calculi in the anterior urethra of children which will clear up a puzzling case of retention for which no other cause could be demonstrated.

Polyuria.—Polyuria is a temporary condition in which excessive quantities of urine are passed. It is a symptom of diabetes mellitus and chronic interstitial nephritis, and when chronic is called diabetes insipidus. Under ordinary conditions an increased consumption of fluid, or the decreased elimination of water by the sweat glands or bowels, will naturally result in polyuria, and certain drugs, among which are caffein, diuretin, and digitalis, materially increase the amount of urinary secretion. Polyuria is frequently observed in children with neurotic tendencies, and is often induced by exposure to cold or sudden fright in normal children.

The absorption of dropsical fluids is accompanied by polyuria, and during convalescence from fevers children are apt to void a larger quantity of urine than usual. An interesting case is reported by Fowler, of a child who merely contracted the habit of drinking large quantities of water, and who had polyuria as a natural sequence. Chronic poly-uria, or diabetes insipidus, is usually due to organic lesions, and this is a clinical entity quite distinct from polyuria.

Enuresis.—Enuresis is the term applied to incontinence of urine occurring in childhood, and is due to involuntary emptying of the bladder. Incontinence in the infant is normal; but, with careful training, continence at night should be established between the second and third years, and incontinence after the third year should be considered abnormal, and so treated. Continence during the day should be effected even earlier if the child receive proper training; after it is eighteen months old it should not wet itself during the day, and after the second year, if awakened and placed on the toilet twice each night, there should be no bed wetting.

The term "nocturnal enuresis" is applied to bed wetting, and incon-tinence during the day is known as "diurnal enuresis." A child is most apt to wet the bed during the first few hours of sleep, for at this time sleep is most profound, and it is not awakened by the desire to empty the bladder. Diurnal enuresis may complicate bed wetting, and these cases are the most persistent and obstinate to treat; but rarely does incontinence during the day exist alone. Nocturnal enuresis is usually associated with pollakiuria (frequent urination) during the day, and diurnal enuresis when not associated with bed wetting may be accompanied by incontinence of feces.

Active incontinence is said to be present when sufficient urine

accumulates in the bladder to cause irritation of the sensory nerves, with contraction of the bladder walls from dilatation of the sphincter, resulting in a full stream of urine which is rapidly passed. When passive incontinence exists there is weakness or paralysis of the sphincter vesicæ, causing a continuous dribbling of urine.

Etiology.—By far the greater number of cases of enuresis have a neurotic basis, and it is only exceptionally that an organic lesion, such as malformation of the urinary tract or paralysis of the sphincter vesicæ, is found to explain this condition. Of the various malformations which cause enuresis little need be said; the more common ones are congenital vesicovaginal fistula, persistence of the urachus, extroversion of the bladder, and a congenitally small bladder. Paralysis and deficient nerve innervation of the sphincter of the bladder are observed in spina bifida, idiocy, meningitis, brain tumor, myelitis, and injury to the spinal cord.

Aside from these comparatively rare cases, due to organic disease of the nervous system, there is, in the vast majority of cases of enuresis, an unstable nervous system which is the direct result of age and heredity. During childhood the spinal cord and especially the motor nerves are so active, and the brain as yet so comparatively undeveloped, not having inhibitory control over the lower centres, that it remains only for some slight condition which will increase the irritability of the spinal centre or terminal filaments of the vesical nerves or interfere with the cerebral control over this centre to cause enuresis.

The importance of heredity as a predisposing factor in enuresis is emphasized by the number of children with enuresis who display various other neurotic symptoms which indicate inherited neuropathic tendencies. In other instances the central nervous system has been weakened by chronic malnutrition, due either to improper feeding, unhygienic surroundings, enteritis, tuberculosis, or syphilis. The exciting cause of an initial attack is often an acute debilitating illness, and there is usually a recurrence of enuresis after such an illness, but there are various other exciting causes of enuresis.

Whether there be a predisposition to enuresis of nervous origin or not, one will often find one of the following conditions to account for the affection. The urine itself may be at fault, examination revealing a highly acid or alkaline reaction, or there may be an excess of oxalates or uric acid crystals. Occasionally there is inflammation somewhere along the genito-urinary tract, either pyelitis, cystitis, or urethritis; less frequently one may discover calculi, tumors, or polypi, as a cause of irritation. Atony of the bladder, although hard to demonstrate, unquestionably exists in delicate, undernourished children; and in atony as well as in abnormal irritability of the bladder from infection along the urinary tract or irritation of nervous origin, may lie the explanation of many obscure cases of enuresis.

Various conditions are recognized factors in producing incontinence by reflex irritation; they comprise balanitis, vulvovaginitis, rectal

polypi, fissure in ano, intestinal parasites, and constipation. In some cases, conditions even more remote, such as enlarged tonsils and adenoids, or thyroid insufficiency, may be considered etiological factors. Enuresis dependent upon diabetes mellitus or diabetes insipidus, should, perhaps, be attributed to the excessive quantity of urine excreted. In the home there is often a tendency to consider enuresis as purely a habit. This view may be erroneous, yet it is quite possible that enuresis may be the result of faulty training. There is no doubt that once the condition is established, its continuance is largely due to habit, a fact clearly demonstrated by its persistence after the recognized cause has been removed. Frequently, after careful study and investigation no cause can be found. Enuresis occurs with equal frequency in boys and girls, and is observed at all ages up to puberty, although the majority of cases are seen before the fifth year.

Symptoms.—Ordinary enuresis is characterized by the complete evacuation of the bladder in a full, rapid stream; when there is constant dribbling an organic cause may be strongly suspected. Nocturnal incontinence is by far the most common form, and may or may not be accompanied by pollakiuria, while in diurnal enuresis pollakiuria is generally present. An attack of enuresis may be chronic, persisting from infancy until the seventh year; or it may be recurrent, each attack being due to some slight disturbance. Temporary attacks are thought to be due to occasional hypersensitiveness of the bladder. The act of bed wetting usually occurs in the early evening when sleep is most profound, and in the morning when there is an accumulation of urine in the bladder.

Diagnosis.—The diagnosis is self-evident, and is made by the child's parents.

Prognosis.—The prognosis in enuresis depends on the underlying cause; but the age of the child and the duration of the symptoms have a marked influence on the rapidity of the cure. When there is organic disease of the brain or cord, or when malformations exist which are beyond surgical intervention, the outlook is, of course, hopeless. In the absence of these conditions the prognosis as to ultimate recovery is good.

If some cause is discovered and removed, the case may clear up in a comparatively short time; but, as a rule, months of systematic, thorough treatment are required to bring about a cure, and no case should be pronounced cured until a year has elapsed without a return of the condition. Enuresis shows a strong tendency to cease spontaneously without treatment at about the seventh year; for, by this time, the balance of the nervous system is fairly well established. Very rarely does it persist after puberty, although an occasional instance has been recorded in a neurotic girl.

Treatment.—Prophylaxis with regard to enuresis consists in early training of the child and careful supervision of its habits of micturition and defecation. If taught to control its bladder and make known its desire to empty it, the child should have the mechanism of micturi-

tion under cerebral control by the second year. Given a fully developed case of enuresis to treat, the physician should first make a careful, systematic search for any condition which might possibly cause it, inasmuch as treatment directed to the symptom alone is, in most cases, a failure.

The coöperation and confidence of the parents should be secured by telling them in the beginning of the chronicity of enuresis and its stubborn resistance to treatment. It is obvious that any predisposing cause should be removed; among these are phimosis, adherent prepuce, adherent clitoris, vaginitis, vulvovaginitis, urethritis, pyelitis, pinworms, rectal polypi, fissures of the anus, and other reflex conditions.

The urine should always be examined, and, if highly acid, the diet should be regulated, and the amount of proteins decreased; in addition, potassium citrate in 3-grain doses may be given to a child of three years, and the amount of liquids increased if the urine is too concentrated. Excessive alkalinity may result from a diet too rich in starch and sugar; therefore, if this cause of enuresis be discovered, it is well to limit the carbohydrate intake, and to give benzoic acid, 2 grains three times a day, to a child three years old. If the quantity of urine passed is excessive, the amount of fluids taken by the patient must be restricted.

Not infrequently, microscopic examination of the stained sediment of a specimen will reveal the presence of the colon bacillus, showing an infection of the genito-urinary tract by this organism. Such an infection is accompanied by acidity of the urine and a highly irritable bladder, and requires the administration of potassium citrate and urotropin, of each 3 grains three times a day, to render the urine less irritating and inert.

The bladder, itself, should be examined carefully, and if atony of the sphincter be found, galvanic or mild faradic currents may be applied or the neck of the bladder massaged through the rectum. Excessive irritability of the bladder, whether caused by urinary infection or of nervous origin, demands the administration of vesical sedatives, injections of normal saline or boric acid solution, and rest in bed. Sometimes a stone is discovered in the bladder, and its removal is always attended by cure.

When remote conditions, such as enlarged tonsils and adenoids, are treated, the results are not nearly as good, with the possible exception of hypothyroidism, when the administration of thyroid extract usually effects a cure. Having remedied the cause of enuresis, the habit must usually be overcome before improvement sets in. One must consider that such a child is usually neurotic, and that, instead of harsh measures or punishments, it needs soothing but firm treatment. It should have the advantage of a change of climate to seashore or country, and be put on a good nutritious diet to build up the physical condition which in most cases is greatly impaired. Quiet is especially necessary, the nervous tension of present-day school life being a hindrance to treatment.

Certain routine instructions should be given in each case. The child should be made to urinate before going to bed, and be awakened late in the evening and put on the toilet. The foot of the bed should be elevated, and the child kept from lying on its back by means of a towel tied around the waist and knotted in the back. Sometimes a change from one bed to another will induce temporary relief. The diet should be bland and non-irritating; coffee, tea, and spices must be prohibited. The evening meal should be a light one, and no fluids be taken after 4 P.M. The bowels should be kept regular throughout the treatment, and no food or medication given that will irritate the urinary tract or produce constipation.

If the child is anemic or chlorotic tonics, such as arsenic and cod-liver oil, are indicated and may be given with a few grains of sodium bromide added to each dose. The two most valuable drugs in the treatment of enuresis are belladonna and strychnine. Belladonna owes its effectiveness to its sedative action on the muscular wall of the bladder; it should be administered in increasing doses until the physiological effect is obtained. A child of three may take 1 drop of the tincture, three times a day, increasing it 1 drop daily until there is dryness of the throat and a flushed skin. It may then be reduced 1 drop a day until the flushing ceases, and continued at this dose for several weeks. If there is no appreciable improvement after such administration of belladonna, it is useless to continue it.

Tincture of nux vomica is indicated where the sphincter of the bladder is weak; this accounts for the particularly good results obtained by the use of this drug when diurnal and nocturnal enuresis are combined. The dose is 2 drops three times a day, to a child of three years. This dose may be cautiously increased. Ergot has been given for its tonic effect on the bladder wall, but it is the consensus of opinion that it is useless when belladonna and strychnine fail.

Cathelin's treatment is worthy of trial and is justifiable when milder measures fail, for 80 per cent. of cures are recorded where it has been carried out. The patient is placed in Sim's lateral posture, and the coccygeal spine located. A lumbar puncture needle is introduced in the middle of a line joining the coccygeal cornua, and is passed directly upward, care being taken not to perforate the meninges. From 5 to 25 c.c. of normal saline solution, at body temperature, are then slowly injected. The process is almost painless, and the child may be sent home as soon as it is done. In some cases a second injection is necessary ten days later.

NEPHRITIS.

Nephritis is not uncommon in children at any period from birth to puberty, and may occur in any of the types seen in adults. Our classification of the various forms of nephritis is still unsatisfactory, the term nephritis, itself, being restricted to non-suppurative inflammation of the kidney, thus making it necessary to use a qualifying

adjective when a suppurative process is spoken of. Just as in adults, both acute and chronic nephritis occur in childhood, although the latter is rare; but, in addition to this classification, nephritis, as seen by the pediatrician, may well be classified etiologically. During infancy one may differentiate a distinct form of nephritis due to congenital syphilis, another arising from gastro-intestinal disturbances, a variety of other types produced by various infections and intoxications, as well as a type which resembles the contracted kidney of adult life. In older children one meets with nephritis which is usually secondary, and in which acute infectious diseases play an important part as primary factors. Thus, we may divide these nephritides into the scarlatinal and diphtheritic types, and those due to other infections and intoxications of unknown or doubtful nature.

Acute Congestion of the Kidneys.—Acute renal congestion, or renal hyperemia, is much more common in children than in adults because in the child it is so easily induced. The usual cause is an acute infectious disease; but it may be the result of severe digestive disturbance, high fever, irritating drugs used indiscriminately or taken accidentally, or of exposure to cold.

Pathology.—The kidney of active congestion is slightly enlarged, swollen, and, after the capsule has been removed, appears brown or mottled. On section the cortex is wider and darker than in health, the bloodvessels are engorged, and the cells are the seat of cloudy swelling.

Symptoms.—There are rarely any symptoms except a change in the urine, which may show albumin, with a few hyaline and granular casts. It is usually high colored, of high specific gravity, and scanty in amount—sometimes so scanty as to constitute partial suppression; yet rapid improvement is possible with no trace of kidney lesion remaining. The duration of the attack varies, recovery taking place promptly after removal of the cause.

Treatment.—The child whose urine shows evidence of acute congestion of the kidneys should be kept in bed on a milk diet until recovery has taken place. It should drink plenty of water in order to dilute the toxins circulating in the blood and acting upon the kidneys, and increased excretion of toxins through the skin and bowels may be promoted by means of vapor or hot baths and saline cathartics. Hot applications over the kidney region act as mild counter-irritants, and relieve the feeling of discomfort usually present in the loins in this condition.

Nephritis in Infancy.—No accurate estimate can be made as to the frequency with which nephritis occurs during infancy because of the difficulty in detecting its presence; this, however, makes it appear reasonable that it is more common than observations lead us to suppose. Several factors increase the difficulty of diagnosing nephritis in infants, not the least of which is the problem of collecting the urine in a suspected case; but far more puzzling is the frequent occurrence of nephritis without edema, also the possibility of both edema and albuminuria appearing in infancy with no demonstrable nephritis.

Etiology.—Primary nephritis in infants is rare, although it is probable that many mild cases escape detection. Syphilis and gastro-intestinal disorders account for most of the cases, but one cannot ignore its association with bronchopneumonia, erysipelas, and extensive skin affections, when it may be considered as a complication. It is also a complication of the acute infectious diseases when, by chance, they occur in infants. Occasionally a case is reported of nephritis associated with scurvy.

Pathology.—The syphilitic kidney exhibits microscopically an interstitial nephritis, but is normal to the naked eye, since there is no actual fibrosis, although the glomeruli and tubules may be imperfectly developed owing to the syphilitic process. It is only exceptionally that the preponderance of changes in the parenchyma justifies the diagnosis of parenchymatous nephritis, rather than the interstitial variety.

The kidneys of an infant with nephritis of gastro-intestinal origin are also normal macroscopically; but, in contradistinction to the changes in syphilitic nephritis, there is usually fatty degeneration of the epithelium of the convoluted tubules, but no change in the glomeruli.

Symptoms.—Acute nephritis, whether of syphilitic or gastro-intestinal origin is, as a rule, latent. The infant with hereditary syphilis whose kidneys are affected rarely shows any symptoms. The urine contains albumin and casts, but is not diminished in quantity, and, when tested for blood, is negative. Uremia practically never occurs, and edema rarely is seen. It is not fatal, and its severity bears no relation to the degree of the other syphilitic manifestations, although it readily clears up under treatment which corrects the disease elsewhere in the body.

The course of the disease is acute, and the symptoms latent. The nervous symptoms are very infrequent in nephritis of gastro-intestinal origin and in syphilitic nephritis; but it is believed by some authorities that the symptoms referable to the nervous system which appear in the course of fatal gastro-enteritis associated with nephritis may be uremic in character. The urine also shows more decided changes, and, in addition to albumin and casts, it sometimes contains blood.

It is, apparently, only in the severe primary acute nephritis of infancy that symptoms and signs are the predominating features of the disease. Here the disease is usually a diffuse nephritis, but the involvement of the parenchyma is secondary to the interstitial lesions which are more pronounced. The symptoms are high but irregular fever, vomiting, diarrhea, dyspnea in severe cases, anemia, and nervous manifestations. The urine may or may not contain albumin at the onset; but, together with casts, it appears at some stage of the disease in every case. Although there is no edema, this form of nephritis is very grave, and a mortality of over 70 per cent. is recorded. As a rule, the nephritis associated with scurvy is latent, and albumin and casts in the urine may persist for months after the total disappearance of the scorbutic condition.

Prognosis.—From the preceding description of the various forms of nephritis which occur in infancy it is evident that, with the exception of the severe, acute, primary type, the prognosis as to life is good, the syphilitic form offering, perhaps, the best ultimate chance for recovery. Some clinicians believe that the acute interstitial nephritis of infancy is of chronic nature, and results in a contracted kidney, such as is observed in adult life.

Acute Diffuse Nephritis.—Under this title will be included acute parenchymatous nephritis, acute exudative nephritis, acute desquamative nephritis, glomerular nephritis, acute tubular nephritis, and acute interstitial nephritis.

Etiology.—From an etiological stand-point, the term acute nephritis may well embrace all the various forms of this disease enumerated above, since they all result from inflammatory changes produced by the action of toxins and bacteria. It is true, however, that the action of toxins alone is limited to degeneration of the epithelial cells of the kidney, while bacterial invasion of the kidney results, not only in degenerative processes in the epithelium, but also in infiltration of the kidney with newly formed cells which are probably the precursors of the fibrosis which follows. Infection of the kidney is quite frequently predisposed to by a degenerative condition of the organ induced by the action of toxins; but, in a few cases, usually severe in type, the bacterial infection occurs with no antecedent changes in the kidney structure whatsoever, as typified in the nephritis of the first week of scarlet fever or diphtheria.

There can be no doubt that scarlet fever is the most common cause of nephritis in childhood. Why the kidneys should be so peculiarly vulnerable in this disease is not known; but the frequency with which scarlet fever is accompanied by acute inflammation of the kidneys is known the world over, even to the laity. The attack may have been mild, and the symptoms may have entirely disappeared; yet nephritis occurs which is suggestive of the operation of a variety of causes; and, although the toxin must be regarded as the primary factor, one cannot overlook the possible added effect of exposure to cold, errors in diet, constipation, and too short a period of rest in bed.

The toxins produced in scarlet fever have a particularly selective action for the glomeruli. Acute nephritis is an earlier complication in diphtheria than in scarlet fever, but by no means as common; and the toxins of diphtheria injure both glomeruli and tubules. The proper use of diphtheria antitoxin is largely responsible for the comparative rarity of nephritis as a complication of that disease. As a complication of the other common infections of childhood, nephritis is also comparatively rare; but it occasionally accompanies chickenpox, epidemic cerebrospinal meningitis, measles, German measles, mumps, and whooping-cough.

Other diseases of childhood which are sometimes accompanied or followed by nephritis are pneumonia, influenza, rheumatic fever, tonsillitis, typhoid fever, smallpox, tuberculosis, and, in the tropics,

malarial fever. With septicemia and pyemia due to the streptococcus, staphylococcus, pneumococcus, or gonococcus, febrile albuminuria is the rule, although nephritis is not uncommon.

Certain extraneous substances, if ingested, are capable of producing nephritis and in their action resemble the toxins of scarlet fever and diphtheria; for instance, arsenic, cantharides, and snake venom affect chiefly the glomeruli; while bichloride of mercury, uranium nitrate, and potassium and ammonium chromate affect chiefly the tubular epithelium, attacking the glomeruli but slightly, if at all. Other exogenous toxins which cause nephritis, but whose selective action is as yet not clearly defined, are turpentine, carbolic acid, potassium chlorate, salicylic acid, oxalic acid, the mineral acids, alcohol, chloroform, phosphorus, and lead.

In addition to these toxic substances which are introduced from without the body, we cannot fail to recognize the importance of various toxins produced within the body; for instance, from the gastro-intestinal tract in digestive disorders, these being most marked during infancy, also in jaundice, in diabetes, and in children of gouty ancestry. The importance of cold as an etiological factor in the causation of nephritis has been greatly overestimated, as in all probability it does nothing more than favor the action of pathogenic bacteria, thus differing in no respect from its influence on inflammation of other organs of the body.

Of the predisposing causes of nephritis, age and heredity are the only ones which influence this disease in childhood. There can be no question that the tendency to nephritis is sometimes transmitted; but this is infrequent because, in these cases, nephritis is chronic, whereas most of the cases of nephritis seen in children are of acute type.

Pathology.—In acute nephritis both kidneys are enlarged, are softer than usual, and have a more rounded appearance. The color varies; but, after removing the capsule which strips readily, the convex surface presents a pale, grayish, mottled appearance, with light red and quite dark red spots caused by hemorrhage and the congestion of the stellate veins. Upon incising the capsule the kidney substance may bulge through, and, upon sectioning, the cortex is found to be much swollen and grayish-yellow or light red in color. The pyramids seem unusually red in contrast with the pale cortex, its yellow areas or streaks marking the degenerated tubular epithelium.

Microscopic examination shows involvement of the glomeruli, tubular epithelium, and interstitial tissue, the extent to which each is involved depending somewhat upon the cause of the nephritis. The glomeruli are swollen and hyperemic, and the capillaries are tense with blood that may be in thrombus form. Bowman's capsular space is filled with exudate and débris which cause pressure upon the vessels of the tuft and upon the tubules, markedly disturbing the function of the glomeruli, and accounting in some measure for the albuminuria, oliguria, and, perhaps, the increased blood-pressure of nephritis.

The tubular epithelium undergoes cloudy swelling and fatty degeneration, and may desquamate; and the tubules themselves contain red blood cells, leukocytes, desquamated endothelium, and casts. In cases of short duration, inflammatory edema, round-cell infiltration, and hemorrhagic areas are found in the interstitial tissue; and, if the attack has been prolonged, there may be an increase in connective tissue. There is an exudative type of acute diffuse nephritis which is observed in infants and young children and is characterized by large accumulations of leukocytes, serum, and red blood cells in the glomeruli and tubules, with either marked or but little change in the parenchyma and interstitial tissue.

Symptoms.—Acute nephritis may manifest itself in two ways: either by an abrupt, frank onset with edema, pallor, headache, gastric disturbance, and conspicuous urinary changes, typified by scarlatinal nephritis; or by another type of the disease in which—though the onset may be sudden—there are no frank symptoms, and only by most careful study of the urine can any changes in its quantity and microscopic or chemical properties be detected. This latter form is characteristic of the acute nephritis which occurs during the course of typhoid fever or pneumonia.

There are three symptoms of acute nephritis in children which are fairly constant—edema, uremic manifestations, and urinary changes. The edema may be extreme and develop rapidly, although it sometimes varies so greatly that the child may be entirely free from it for weeks and even months at a time. It is rather firm at the onset, and is most noticeable in the face in the morning, causing puffiness, swollen eyelids, watery eyes, and a pasty expressionless look which is characteristic. A similar puffiness of the fingers, ankles, the back of the hands, and scrotum occurs; later on there is infiltration of the subcutaneous tissues over the back and abdomen. The increase in weight is sometimes 40 per cent. of the previous body weight.

As a rule, the urine is markedly diminished in quantity, and suppression is not uncommon. Albumin and casts are constantly present, and a trace of blood may be found occasionally. The color becomes very dark; the specific gravity is normal or slightly raised. One of the earliest signs of recovery is an increased output of urine.

The usual temperature range is from 100° to 103° F., but in very severe attacks it may reach 105° F. As a rule, the high temperature does not long persist, and a continuous elevation of temperature is to be regarded rather unfavorably. Headache, backache, extreme restlessness, and stupor are due to toxemia. In severe cases there may be uremic convulsions preceded by nausea, vomiting, deficient excretion of urea, and a urinous odor to the breath and perspiration.

A fulminating type of nephritis may occur in which the onset is very abrupt, accompanied by high temperature, scanty urine, rich in albumin, casts, and blood. The pulse is full, of high tension, and there is severe pain in the lumbar region. Nausea, vomiting, and diarrhea, with extreme restlessness passing into stupor, signify the

early approach of uremia; and, unless diuretic measures are prompt in relieving it, death rapidly ensues. Children, however, bear extreme oliguria and suppression of urine relatively better than do adults, and cases have been reported where suppression for three, five, or ten days was followed by recovery.

Duration.—The duration of an ordinary attack of acute nephritis is usually from one to three weeks. It depends largely upon the severity of the particular case, and may be prolonged greatly beyond its usual course; but, in view of the fact that recovery may take place after six months or a year, one month seems but a short time.

Complications.—The most frequent complications of acute nephritis are endocarditis, pericarditis, pleurisy, bronchitis, bronchopneumonia, and lobar pneumonia. Erysipelas, meningitis, and edema of the glottis are but rarely observed.

Diagnosis.—The only possible way by which a physician can accurately diagnose the cases of acute nephritis in children which come under his care is to carefully examine the urine in all cases, and make daily urinalyses of his scarlet fever patients. He must carefully rule out cyclic albuminuria, febrile albuminuria, congestion of the kidney, infarcts, amyloid disease, tumors, and calculi before attributing urinary changes to acute nephritis. While it is desirable, if possible, to recognize acute nephritis by urinalysis before clinical signs and symptoms appear, the disease is occasionally not suspected until the characteristic appearance of swelling about the eyes and ankles, with, perhaps, fever, vomiting, and headache, leaves no doubt as to the diagnosis.

Prognosis.—Acute nephritis is always a serious disease, yet the tendency in scarlatinal nephritis is to recovery, and the prognosis in severe acute nephritis is good if the case is properly managed from the onset until at least six months after convalescence begins. If not properly treated, a mild acute nephritis may pass into a chronic nephritis with doubtful prospects as to final cure. Acute nephritis in infants and young children often terminates fatally, and in a variety of ways; uremia is the most common cause of death in older children, but in the younger ones complications, especially of the respiratory tract, may lead to a fatal termination.

Chronic nephritis was formerly thought to be an unusual sequel of acute nephritis in childhood, but we have been misled, perhaps, by the relatively long period of apparent health which may elapse between the initial attack and subsequent ones; therefore the least we can say is that the kidneys are left in a condition which renders them susceptible to future attacks. Of the various aids to prognosis in a given case of acute nephritis, the presence or absence of uremic manifestations and the amount of urine voided daily are, perhaps, the most reliable, although each case must be studied and judged by itself.

Treatment.—Judicious treatment of any acute infection is the best prophylaxis of acute nephritis in childhood; therefore, in those dis-

eases which are especially liable to cause nephritis precautions as to diet, clothing, catharsis, the use of drugs, and proper exercise, should extend well into convalescence. Since it is the irritation of the kidneys by toxins eliminated during the course of infectious diseases which causes nephritis, an attempt must be made by free catharsis and stimulation of the excretory function of the skin to eliminate these excrementitious substances when present in the urine. The urine may be rendered less irritating if we increase the daily output by restricting the patient to milk or buttermilk, whey, koumiss, or junket, with, possibly, cereals and gruels at meal time, and fruit between meals.

The value of diphtheria antitoxin as a prophylactic against nephritis has already been emphasized; but the abuse of other therapeutic agents, chief of which is the too free administration of certain drugs, may induce nephritis. Alcohol and urotropin are, perhaps, foremost in the list of harmful drugs; extreme caution must be observed also in the use of salicylic acid, potassium chlorate, phenol, mercury, and other recognized renal irritants. The effect of exposure to cold in the development of nephritis during the acute infections has probably been overestimated; but prolonged exposure to cold, and especially to dampness and cold, should, of course, be avoided.

The active treatment of acute nephritis should be undertaken with three objects in view; viz., the removal of the cause; the reëstablishment of kidney secretion and the securing of rest for these organs by increasing elimination through the skin and bowels; and the treatment of symptoms and complications. Removal of the cause of acute nephritis may be brought about by the prompt and efficacious treatment of an existing infectious disease, or by discontinuing the use of such drugs as are known to irritate the kidneys. In order to lessen the work of the now crippled kidneys, free saline catharsis should be induced by the use of magnesium sulphate in 2-dram doses daily to a child of five years, and the skin kept acting freely by means of frequent sponges and warm baths, and not allowing the temperature of the sick room to fall below 70° F.

The diet should consist of food which will not aggravate the inflammation of the kidneys. For a child of five years, 21 ounces of milk daily, given 7 ounces at a time with a little cereal, and supplemented by some apple sauce or jelly, with stale bread or zweibach between meals, meets this requirement and is amply sustaining. This diet should be insisted upon for a month after the first trace of albumin makes its appearance in the urine, and the only indication for increasing it is the advent of anemia or steady loss of weight. Cognizant of the fact that in individuals with apparently healthy kidneys the ingestion of sodium chloride is followed by chloride retention which equals that in mild nephritis, the value of a salt-free diet is apt to be overestimated; but in all forms of nephritis associated with renal edema the restriction of sodium chloride to 1 or 2 grams (15 to 30 grains) daily for a child of five years is desirable, and only when edema

35

is marked should a salt-free diet be advised; even in this case it should not be maintained for a long period. The proteins ingested should never exceed an ounce a day, and a diet of fats and carbohydrates leaves less residue in the shape of solids for elimination through the kidneys. Fruits, such as oranges, grape fruit, apples, either baked or made into sauce, and lemonade form an acceptable addition to the diet, and are not injurious, in some instances acting as diuretics. Rest in bed is imperative, and should be insisted upon until albumin and casts have permanently disappeared from the urine.

Change of climate is only to be considered in subacute and chronic cases, for the danger of overexertion or exposure to cold makes traveling undesirable; the child is much better off at home under proper treatment.

Severe cases of nephritis marked by high fever, partial suppression of urine, and intense edema, require more active and radical procedures. The hot pack and vapor bath should now be resorted to for increased stimulation of the skin, and an effort be made to increase diuresis by the application of hot flaxseed or mustard poultices to the loins, or by dry cupping. Colonic flushings, using a pint of normal saline solution at 110° F. for a child of five years, will often increase the kidney action, and should be repeated every six hours until effective. Tincture of aconite, in 1-drop doses for a child of five years, may be repeated every two hours until there is slight diaphoresis; and nitroglycerin, $\frac{1}{200}$ of a grain may be given hourly until the high-tension pulse is relieved.

If uremia supervene, the convulsions can sometimes be controlled or prevented by the hypodermic administration of morphine, $\frac{1}{20}$ of a grain at a dose for a child of five years. Venesection is advisable in severe attacks, and though extremely difficult because of the collapsibility of the veins in children, at least a half-pint of blood should be withdrawn and a pint of normal salt solution injected. The withdrawal of a test-tubeful of spinal fluid by lumbar puncture gives relief in some cases, and may be tried in extremity. If the heart grows weak, tincture of strophanthus should be administered in 3-drop doses, every three hours, to a child of five years. Other symptoms may necessitate special treatment. If the edema be severe, paracentesis of the abdomen or pleura and puncture of the legs is sometimes required. Edema of the larynx may necessitate tracheotomy.

Convalescence is tedious; the anemia so often present is improved by the administration of Basham's mixture (liquor ferri et ammonii acetatis), in 1-dram doses to a child of five years. Sudden exposure to cold should be carefully guarded against, and exercise taken very moderately. The diet should be cautiously increased, avoiding much nitrogenous food. With convalescence fairly well established, or if the disease shows a tendency to become subacute, it is perhaps advisable to send the child to a warm equable climate; the winters, especially, should, if possible, be spent in Florida or Southern California.

Chronic Nephritis.—Chronic nephritis is one of the rare diseases of childhood. It is almost unknown in infancy, and practically never observed before the third year, most cases occurring between the fifth year and puberty. There are three forms of chronic nephritis in children which conform more or less to the adult types; *i. e.*, chronic parenchymatous nephritis (chronic diffuse non-indurative nephritis); chronic interstitial nephritis (chronic diffuse indurative nephritis); and the waxy or lardaceous kidney. The chronic parenchymatous type may assume the characteristics of the chronic interstitial variety as the disease progresses. Amyloid disease, which causes the waxy kidney, is a degenerative process which may be engrafted on a chronic nephritis of childhood.

Etiology.—The direct relation of chronic nephritis to acute nephritis in childhood is definite, which is in sharp contrast with the obscure connection between these two diseases in adult life. Most cases follow acute scarlatinal nephritis and, less frequently, the other acute infections. Amyloid disease of the kidney is most apt to be the sequel of syphilis, tuberculosis, chronic suppuration, rachitis, or chronic malaria. Hereditary syphilis may account for that very rare form of chronic nephritis in childhood—the interstitial variety—in which a gouty ancestry, tuberculosis, alcoholism, and chronic valvular heart disease are supposed to be predisposing factors. The occurrence of chronic nephritis in more than one child in a family also suggests the possible influence of heredity. All the other etiological factors of acute nephritis may be considered capable of indirectly producing a chronic nephritis.

Pathology.—The lesions produced in the kidneys by the three types of chronic nephritis which occur in childhood do not differ essentially from those observed in adult life. The kidney in chronic parenchymatous nephritis is enlarged, pale, of decreased consistence, and has a smooth surface; the capsule is not adherent. On section the cortex is found to be wider than normal and yellowish-white in color, on account of which it is called the "large white kidney."

The microscope shows the convoluted tubules to be thickened and dilated, the epithelium undergoing granular and fatty degeneration and exfoliation, but only exceptionally are the tubules atrophied. The glomeruli may be either compressed and atrophied or may show hyaline changes, swelling, cellular proliferation, and desquamation. The large red kidney—the result of multiple hemorrhages in the cortex —and the small white kidney, which is the same size or even smaller than the normal organ, are rarer pathological forms which the kidney may assume in chronic parenchymatous nephritis.

In chronic interstitial nephritis the kidneys are usually much atrophied, and appear drawn or shrunken, hence the synonym "contracted kidney." They are red or reddish-gray in color, and the cortex is much thinner than usual. The capsule is firmly adherent and strips off with difficulty, exposing a coarse, granular kidney surface. The

most characteristic microscopic change noted is the marked increase
in fibrous tissue distributed irregularly throughout the kidney
structure.

Other changes may be practically the same as those observed in
the chronic parenchymatous type. The tubules in one part of the
kidney may be completely atrophied, and in another they are dilated,
forming cysts. If no chronic congestion of the kidney has preceded
the inflammation, the glomeruli are atrophied; but if chronic conges-
tion has taken place they may be large, the capillaries dilated, and
the walls showing hyaline degeneration. Cardiovascular lesions are
commonly present, and include atheroma of the arteries and cardiac
hypertrophy. Cirrhosis of the liver is occasionally seen.

In amyloid disease the changes are not confined to the kidneys, but
these organs are considerably enlarged, grayish in color, and of putty-
like consistency. The lardaceous material is most abundant along
the renal vessels and in the vascular tufts of the glomeruli, and the
kidney substance atrophies as the amyloid deposits increase. Other
organs, such as the suprarenal glands, spleen, liver, and intestinal
villi are also involved.

Symptoms.—*Chronic Parenchymatous Nephritis.*—The onset of this
form of chronic nephritis is usually an exacerbation of an acute attack
of nephritis which in some cases immediately precedes it, while in
others it may be separated from it by months or even years. Rarely
do we find a case which is chronic from the beginning, with insidious
onset, and resembling the adult type; yet the undoubted existence
of such in later childhood demands recognition.

The physician is usually consulted on account of indefinite symp-
toms, such as malaise, anorexia, pallor, gastro-intestinal disturbances,
or slight puffiness of the ankles or about the eyes. There may be
dropsical accumulations in other parts of the body, even effusions
into the pleura, pericardium, or peritoneum; but the amount of
dropsical fluid varies greatly, being markedly increased during exacer-
bations, and diminished or entirely absent at other periods. Anemia
is always present, vomiting is common, and various nervous phe-
nomena, such as drowsiness, insomnia, fatigue, headache, and neuralgia
are frequently observed. Unless there are complications there is no
fever in chronic nephritis.

The amount of urine is diminished, but the daily output varies
greatly during the course of the disease. The reaction is acid, the
specific gravity normal or a little above normal, and on standing there
is an abundant sediment of urates, casts, epithelial cells, and blood
corpuscles. Albumin is constantly present, and greatly increased
during exacerbations, the total amount lost during the day reaching
as high as 20 grams (5 drams). The number of casts is usually in
direct proportion to the amount of albumin in the urine. They are
hyalin, granular, epithelial, and fatty, with broad outlines; oil
globules may be perceived upon many of them. Free fat droplets
are often found; but red and white corpuscles are not abundant in

the urine of chronic parenchymatous nephritis except during acute exacerbations.

Lardaceous or waxy degeneration of the kidneys is associated with greater ascites, also with amyloid deposits in the liver, spleen, and intestinal canal which cause enlargement of these organs, and often obstinate diarrhea which renders the prognosis grave. The urine is pale yellow in color, of low specific gravity, and contains hyalin and waxy casts. Albuminuria and polyuria are usually present, but there is rarely blood in the urine, and but little sediment on standing. Mild cases are not recognized because, instead of the typically weak, pale, cachetic, emaciated child with muddy complexion, the patient may have a good ruddy complexion, be fairly strong and fat, and show no evidence of dropsy.

The duration of these forms of renal disease depends largely upon the surroundings of the patient, the amount of renal tissue involved, and the treatment. Pulmonary edema not infrequently brings chronic parenchymatous nephritis to a fatal termination, and in both of these diseases death is usually due to pneumonia, pericarditis, pleurisy, or endocarditis. Uremia is uncommon in children; when it does occur, it is usually associated with chronic interstitial nephritis.

The symptoms of chronic interstitial nephritis seem in many cases to date from birth, but they are indefinite, vague, and few in number. The child is pale and delicate from infancy, anemic, and usually stunted in growth; but, when the case comes under observation, anemic pallor may be replaced by a peculiar dusky flush, due to capillary congestion. Dropsy is rare. The blood-pressure, high with hypertrophy of the left ventricle, and atheromatous changes in the arteries are not infrequent. Nervous disturbances are common, and include headache, neuralgia, albuminuric retinitis, retinal hemorrhages, and attacks of spasmodic dyspnea; toward the end of the illness convulsions and cerebral hemorrhage may also occur. The urine is pale, of low specific gravity, increased in quantity, and voided frequently in large amounts. The sediment is quite scanty; under the microscope it is found to be composed of a few epithelial cells, a few hyaline or granular casts, and an occasional red or white blood corpuscle.

Diagnosis.—In the early stages of the disease when few symptoms appear, chronic nephritis may be easily overlooked and not diagnosed until after careful urinalyses have been made, since albumin and casts may be present in the urine for quite a while without the appearance of any other symptoms of nephritis than anemia. Children presenting symptoms such as convulsions, persistent or frequent headaches, or cardiac hypertrophy with high arterial tension, and all cases of general malnutrition, should have their urine carefully examined, for it is failure to examine the urine of children routinely that accounts for the frequency with which chronic nephritis is undiagnosed in childhood unless it be accompanied by frank symptoms, such as dropsy with scanty urine. If the urine be carefully examined, functional albuminuria is the only condition which can

possibly need to be differentiated from chronic nephritis, and organic renal disease can be excluded only after repeated and thorough urinary analyses.

Prognosis.—One who expects permanent recovery from chronic nephritis will, in the majority of cases, be disappointed. Although these patients may live for a number of years with comparatively few symptoms, the outlook as to ultimate or complete recovery is unfavorable. The prognosis, also, largely depends on the circumstances of the parents; for, in the parenchymatous type particularly, life can be prolonged and made tolerable for years if the child can be kept midst ideal surroundings in an equable climate.

Chronic interstitial nephritis offers the least favorable prognosis; and in chronic parenchymatous nephritis, or cases of waxy kidney, the outlook is serious when there is a persistently increasing amount of urine of low specific gravity, this indicating fibrotic changes in the interstitial tissue and Malpighian tufts. Other unfavorable signs in chronic nephritis are a considerable amount of dropsy, valvular disease of the heart associated with nephritis, and a greatly diminished output of urea.

Treatment.—The principles of treatment in chronic nephritis are the same that apply in the acute form. The child should be carefully protected from any influence which might lead to an exacerbation. To this end, excessive muscular exertion, exposure to the acute infections, chilling, drugs that irritate the kidney, and dietetic errors should be avoided. Since outdoor life is very beneficial, it is always advisable to reside in a climate where the winters are mild.

Rest in bed is indicated only when there are uremic symptoms or large dropsical accumulations. While the child is in bed, the diet should be the same as that prescribed in acute nephritis. If it be up and about, the list may include fats, carbohydrates, well-cooked green vegetables, cereals, fruits, simple desserts, and meat three times a week. Of course, food or drinks known to be renal irritants should be avoided, and no salt should be added to any food except what is used in making bread. Water or milk may be taken as beverages in such quantities as the child may desire. The clothing should be warm, and woolens may be worn next the skin all the year.

Good elimination through the skin and bowels is important. A daily warm bath at 95° to 100° F. is of great benefit, and saline cathartics are advisable to keep the bowels active, especially if there is much edema. Diuretics also are indicated when edema is marked, and, for a child of five years, 1 dram of liquor ferri et ammonii acetatis, given three times a day, has the additional advantage of supplying iron to the impoverished blood. In severe cases cardiovascular stimulation is necessary.

If uremia develops, nitroglycerin in doses of $\frac{1}{200}$ of a grain may be given every hour to lower the pulse tension; even venesection may be resorted to. Uremic convulsions may require the hypodermic injection of morphine or the rectal injection of chloral hydrate. Active

diuresis, catharsis, and diaphoresis should be induced by the measures outlined in the discussion of the treatment of acute nephritis. In cases of chronic nephritis which fail to improve under medical treatment, and in which the specific gravity and urea output are constantly falling and uremia impending, Edebohls' operation of splitting the capsule of the kidney, while not curative, should be resorted to, for it sometimes prolongs life and adds greatly to the comfort of the patient.

PERINEPHRITIS.

Perinephritis is an inflammation of the tissues surrounding the kidney. As discussed in this article, the term perinephritis includes inflammation of the fibrous capsule, of the fatty capsule (epinephritis), and of the retroperitoneal fat (paranephritis). It is a rare condition in children, and usually terminates in abscess formation, although resolution may occur.

Etiology.—Perinephritis is usually a secondary affection, arising either from metastasis, the infection being carried by the blood or lymph stream, or by direct extension from a neighboring focus of inflammation or suppuration, such as a psoas abscess, rectal abscess, a pyonephrosis, or an appendicular abscess. The primary form is very uncommon; it usually develops from penetrating wounds, contusions, and blows in the region of the kidney, while some cases may be traced to a sudden strain, the lifting of heavy weights, or even exposure to cold.

Perinephritis, as met with in the acute contagions, is believed to be due to secondary pyogenic infection rather than to the primary disease. Bacteria are always the immediate cause, and the various organisms which may be responsible include staphylococci, streptococci, and pneumococci; influenza, colon, typhoid, and tubercle bacilli. In many cases no assignable cause can be discovered. Right and left kidney regions are affected with equal frequency, and both sexes are equally liable to the disease.

Pathology.—Abscess formation in this region of the body does not differ essentially from that occurring elsewhere. Pus may burrow down along the ureter into the pelvis, or form a fluctuating mass beneath the liver or spleen, or come to the surface posteriorly near the middle of the intercostal space, or point just above Poupart's ligament, or discharge its contents into the peritoneal cavity, vagina, or bladder.

Symptoms.—In primary cases the onset is acute with fever, and the symptoms are referred definitely to the perinephric region, while secondary perinephritis is apt to be masked by symptoms of the primary disease, and may not be recognized until a soft, fluctuating tumor is detected on bimanual examination. Pain in the lumbar region with tenderness on pressure is usually the first symptom, and may be extremely severe. The leg is held semiflexed, and extension is very painful; but it may be flexed on the abdomen without discomfort. There is no fixation of the hip-joint. Fever is variable and usually

intermittent, but may be remittent, or continuous; high, low, or absent; and in acute cases is preceded by a chill.

In the early stages there is no local swelling; but when the disease has existed for some time, a distinct tumor appears in the back, and by palpation a smooth, elastic mass, usually fluctuating and generally fixed, but not affected by respiratory movements, can be made out. Symptoms referable to the kidneys are present only when the inflammation involves these organs; hence the urine is, as a rule, normal, but if pyelitis exists it contains pus. As the disease progresses, lameness and deformity become prominent symptoms. There is deviation of the spine, its concavity being toward the affected side; pain is so increased by movements of the limb that standing or walking is rendered impossible.

Course.—The duration of perinephritis, primary in nature, is usually from one to two months, as recovery generally proceeds rapidly after evacuation of the pus; but secondary perinephritis is slow and insidious, and may last six months.

Diagnosis.—Hip-joint disease is so closely simulated by perinephritis that careful investigation and study are necessary to exclude it, especially if there be no localized abscess. Acute cases of perinephritis are, of course, differentiated by the rapidity of onset and the general symptoms of an acute inflammatory process; even those cases which are subacute or chronic in character run a different course and are never so insidious or chronic as hip-joint disease. There is also a marked difference in the limitation of motion which these two diseases cause; for, while in perinephritis there is only interference with extension of the thigh on the affected side, in hip-joint disease all movements of the joint are restricted, and there is tenderness in the hip-joint with pain which is frequently referred to the inner side of the knee. Psoas abscess from Pott's disease may cause deformity and lameness and thus simulate perinephritis; but on examination we find rigidity of the spine, angular prominence, and other evidences of spinal caries. Early diagnosis of perinephritis is always difficult because of the absence of tumor and the masking of the symptoms by the primary disease. In doubtful cases, puncture is justifiable, and the *x*-ray may sometimes be helpful.

Prognosis.—In primary perinephritis the prognosis is good, and the majority of cases in children terminate in recovery. If the abscess points externally this is invariably the case, especially if there be early surgical interference. The only condition likely to prove fatal is rupture of the abscess into the peritoneal cavity. Occasionally a persistent fistula results, but, as a rule, recovery is complete.

Treatment.—The patient should be put to bed and treated symptomatically until the diagnosis is made with certainty. Pain may be relieved by hot applications and poultices to the affected side, but is occasionally so severe as to require the use of morphine hypodermatically. When the diagnosis is established, and abscess formation becomes apparent, but one procedure is indicated; *i. e.*, the abscess

must be freely opened and drained, and all pockets of pus broken up to prevent burrowing and subsequent rupture into the peritoneal cavity. If any kidney involvement be suspected, this organ should be palpated or even incised, and nephrotomy or nephrectomy be performed, as the condition demands.

TUBERCULOSIS OF THE KIDNEY.

It is questionable whether, in the true sense of the word, there are any cases of primary renal tuberculosis; and in children, tuberculosis of the kidney, without clinical evidence of the disease in other parts of the body, is extremely rare. In general tuberculosis in children the kidneys are usually involved, but the knowledge that the kidneys are studded with miliary tubercles helps little, if any, in the subsequent treatment of miliary tuberculosis, and this phase of renal tuberculosis is, therefore, of little importance. When, however, tuberculosis of the kidney occurs with no demonstrable active lesions elsewhere, the so-called "primary renal tuberculosis," it tends to remain localized in the kidney, and prompt and proper treatment is followed by brilliant results in a large proportion of cases.

Etiology.—Miliary tuberculosis is by far the most frequent cause of renal tuberculosis in children; the infection is hematogenous, and both kidneys are involved. Hematogenous or descending infection may arise also from some focus of tuberculosis elsewhere in the body, independent of general tuberculosis. Ascending infection from other parts of the genito-urinary tract is extremely rare; but infection by continuity from some adjoining focus, such as spinal caries, from tuberculosis of the adrenals, or from tuberculous empyema is occasionally seen.

Pathology.—In general tuberculosis the kidneys, when involved, show many small tubercles which cover the surface and are disseminated throughout the substance of both organs. So-called "primary tuberculosis" is usually unilateral, and involves first the cortex, then the mucous membrane of the pelvis and calices to the pyramids, so that, in advances cases, nearly the whole organ may be destroyed and replaced by caseous material. Perinephric inflammation is quite common in renal tuberculosis as the result of direct extension; the other kidney may become involved by metastasis or, in rare instances, by direct extension *via* the bladder. The second kidney may also show signs of hypertrophy, of chronic fibrosis, or of amyloid degeneration.

Symptoms.—In acute miliary tuberculosis, involvement of the kidneys is rarely accompanied by any symptoms, and in the primary type of renal tuberculosis the symptoms, particularly at the onset, are vague and indefinite. There may be pain and tenderness in the region of the kidney after the disease has become established, but the first symptom is usually frequency of urination, with burning and cramp-like pains which increase toward the end of micturition and

cease when the bladder is empty. Incontinence is occasionally present. In uncomplicated cases the urine is acid in reaction, and contains albumin, pus cells, blood, and tubercle bacilli. Constitutional disturbances develop late, and include fever and digestive disturbances, while toward the close of the disease emaciation, anemia, and cachexia become marked.

Diagnosis.—The diagnosis is made by the symptoms and signs of tuberculosis elsewhere in the body, and by localized manifestations of kidney disease, such as pain and swelling on the affected side, and is confirmed by finding the tubercle bacilli in uncontaminated urine from the kidney.

Prognosis.—The prognosis in unilateral renal tuberculosis which is recognized early and treated by nephrectomy is good; but, of course, there is the danger of the operation. In bilateral tuberculosis of the kidney and in miliary tuberculosis the outlook is unfavorable.

Treatment.—Nephrectomy offers the only hope of real cure. Lesser operations, such as nephrotomy or nephrostomy, are justifiable only when the child's condition renders nephrectomy impossible. Operation should be followed by the general curative measures employed in treating tuberculosis, such as fresh air, sunshine, good nutritious food, and suitable climate.

RENAL CALCULI.

Large calculi are very rarely observed in children under four years of age, but in infancy there is a marked tendency to the deposition of fine granules of uric acid in the pelvis and calices of the kidney. These deposits have been found in more than one-half of the infants who live for only a few weeks, and are for the most part composed of uric acid. Under ordinary circumstances these crystals are dislodged during early infancy, and pass out in the urine. The affection is not accompanied by any severe symptoms, unless the granules be large, and should pass away at the end of the first or second week. No renal lesions follow, so that there is no danger to life, and the free administration of water will soon dissolve the deposits. The chemistry of larger renal calculi and the mechanism of their formation, as well as the symptoms, are the same as in the adult.

Etiology.—True calculi are composed of uric acid, calcium oxalate, and phosphatic concretions of which bacteria and cellular detritus usually form the nucleus.

Symptoms.—The passage of small uric acid granules is usually attended with no symptoms aside from their appearance on the infant's napkin, which is sometimes a matter of great concern to the parents. When, however, the stone is of such size that it passes down the ureter with difficulty, it produces paroxysms of excruciating pain, which is apt to be referred to the umbilicus, and also symptoms not unlike those of intestinal colic. In severe cases there may be nausea, vomiting, convulsions, and often collapse, until the stone reaches

the bladder, when these symptoms usually cease. If, however, the
stone becomes impacted and completely blocks the ureter, no urine
is passed, and hydronephrosis followed by pyonephrosis or pyeloneph-
rosis quickly develops. The older the child the more closely do the
symptoms of renal calculi adhere to the adult type; pain is referred
along the ureter to the loins and thigh of the affected side, and may
be reflexly felt in the penis. There is usually hematuria. The testicle
on the affected side may be drawn up to a higher level than its fellow.
The passage of a stone through the urethra will cause the child to
run about in pain, grasping the penis, and if the stone is not passed
it is not uncommon after an attack like this to find it lodged in the
urethra. In well-marked cases there may be slight fever, indicating
pyelitis, with pain over the affected kidney, marked tenderness in
this region, and accompanying pyuria. Occasionally renal calculi
are found at autopsy which have given rise to no symptoms during
life.

Diagnosis.—The diagnosis of renal calculus is sometimes very
difficult, but the presence of any of the foregoing symptoms, especially
when urinalysis reveals pus or blood, is strongly suggestive. The
x-ray is of actual use only when a positive result is obtained; but
should be resorted to in every case, as by its aid we frequently dif-
ferentiate renal calculus from appendicitis. In infancy, immediate
diagnosis from intestinal colic is impossible.

Treatment.—Surgical measures are warranted only when the presence
of a large stone in the kidney or ureter can be demonstrated, and is
giving rise to severe symptoms. In milder cases symptomatic treat-
ment is usually sufficient. It consists in the administration of plenty
of water, and the correction of a highly acid or alkaline urine by appro-
priate medication. Citric or acetic acid may be given for excessive
phosphatic concretions, hexamethylenamin, in 2-grain doses every
four hours to a child of five years, if the deposits be of uric acid; while
oxalic acid crystals call for the internal use of sodium phosphate. In
some cases the pain may be relieved by the application of heat either
as a poultice or hot bath; but not infrequently the hypodermic
administration of morphine with atropine is necessary to relieve pain.
General measures, such as regulation of the diet, with avoidance of
red meat, plenty of outdoor exercise, and careful attention to the
bowels, hasten recovery, and are prophylactic as regards future attacks.

TUMORS OF THE KIDNEYS.

Benign tumors of the kidney rarely occur during childhood, but
malignant growths are comparatively frequent, usually of develop-
mental origin, and sarcomatous in type. When benign tumors do
appear in children they generally prove to be adenomas, fibromas,
lipomas, or cysts, and, because of their slow growth, they cause few
if any symptoms, remain undiscovered, and, practically, are seen only
at autopsy. The most common malignant tumor in this situation is

the adenosarcoma, which is almost always primary. In some instances it contains several varieties of epithelial, adenomatous and connective-tissue elements.

Malignant tumors may form in the cortex or pelvis, and either invade the interior of the kidney or project from its external surface. As they adhere to surrounding tissues, and enlarge rapidly, they may fill the whole abdomen in a relatively short time. Malignant growths are, as a rule, very soft, and rarely cystic; they frequently show a tendency to hemorrhage within the substance of the tumor.

Ascites, hydronephrosis, and thrombosis of the vena cava may result from pressure, but general peritonitis rarely develops, although the growth may attain great size, not uncommonly weighing from five to ten pounds, and, in rare instances, even fifteen pounds. Metastasis is common, resulting in involvement of the liver, the lungs, the other kidney, the mesenteric nodes, the colon, the small intestine, and the adrenals; but, curiously enough, secondary growths are rarely found in the bladder or ureters.

Etiology.—Most kidney tumors have their origin in embryonal tissue, and little or nothing is known of the exciting, causative factors. The typical growth found in the kidney during childhood is peculiar to children, the majority of cases being seen before the fifth year; instances of kidney tumor occurring as early as the seventh month of intra-uterine life have been reported, while renal growths after the ninth year are exceedingly rare. The left kidney is more frequently involved than the right. Both sexes are equally liable to the disease.

Symptoms.—The three cardinal symptoms of renal tumor are hematuria, pain, and tumor. Constitutional symptoms appear late, and not infrequently the first suggestion of a growth is the appearance in the lumbar region of a mass which is very soft with smooth or irregular surface. It gives rise to no pain, grows rapidly toward the median line of the abdomen, and is easily palpable under the large bowel. At this time it is so symmetrical that its relation to either kidney may be impossible to ascertain. In some instances, careful physical examination is necessary to exclude enlargement of the liver or spleen as a cause of the distended abdomen, these organs being greatly displaced by the pressure of the invading mass on all of the abdominal contents.

Hematuria is a common, and often an early symptom of the disease, indicating an infiltration of the kidney structure by the growth, for in purely extrarenal and intracapsular tumors bloody micturition is very rare. In the majority of cases, the blood is so scant as to be seen only microscopically, although at times the hemorrhage is severe, and occasionally so profuse that pain is produced by mechanical obstruction of the ureter by clots.

In addition there may be paroxysmal attacks of severe colic, although the pain is usually merely a dull ache producing discomfort and irritability early in thedis ease. A quickly developing cachexia is always noticeable; it is unlike that of anemia or tuberculosis, and

is so characteristic of renal tumor as to be regarded as one of the cardinal symptoms of this affection.

Other constitutional signs appear late, and include emaciation, loss of strength, digestive disturbances from pressure on the stomach, dyspnea from pressure on the lungs, and edema from pressure upon the great veins of the abdomen.

Diagnosis.—This can usually be made with comparative certainty when there is a rapidly enlarging tumor mass in the kidney area with early emaciation and cachexia, and it is substantiated by the appearance of blood in the urine. It is clinically impossible to differentiate one form of kidney tumor from another; but in view of the fact that early operative procedure offers the patient the only chance for recovery, the diagnosis of any growth of the kidney should be made at the onset of the disease. Kidney tumor should always be considered as a possible cause of abdominal enlargement before the sixth year; but it must be differentiated from tuberculosis of the kidney, hydronephrosis, pyonephrosis, and other tumors of the abdomen, such as enlarged liver or spleen, ovarian cyst, and retroperitoneal sarcoma. The location of the colon, which runs along and above tumors of the kidney, and the position of the swelling near the costal margin and projecting into the loin, are of significance in differentiating kidney tumor from various abdominal tumors, especially enlarged spleen and liver.

Prognosis.—The prognosis is unfavorable, as this disease invariably ends fatally except in those cases where an early diagnosis is cleverly made, and an operation performed immediately. Even when this is done, we have the danger of death from shock during the operation, or from metastasis later.

Treatment.—Medical treatment is of no avail, and is resorted to only symptomatically in cases where hope of cure by operation has been abandoned because of the extent of the growth or the appearance of metastases. Early nephrectomy is justifiable if, by any chance, it is thought the child can withstand the operation. A few recoveries have resulted where this has been done; and, even if the chance of cure be remote, the severity of the symptoms is sometimes greatly diminished by surgical intervention.

CONGENITAL CYSTIC KIDNEY.

Congenital cystic disease of the kidney is a rare affection. It usually affects both kidneys.

Etiology.—The etiology of these congenital cysts has not yet been clearly demonstrated, but Shattuck, after systematic histological study, claims that the condition is due to a defect in development, and that the mesonephros, or Wolffian body, becomes attached to the kidney or melanephros, and the cysts are formed in remnants of the mesonephros embedded in the true kidney.

The fact that other congenital malformations frequently coexist

supports this theory of developmental malformation. The earlier view that these are retention cysts due to prenatal nephritis has been largely abandoned. Goodhart and Bateman have suggested the term "renal adenoma," which implies that these cysts are new growths.

Pathology.—The cysts vary in size from the diameter of an ordinary uriniferous tubule to that of a pigeon's egg, and are most numerous in the cortex. In some cases the kidney may become merely a collection of tiny cysts, none larger than a cherry, supported by intervening atrophied or sclerotic renal tissue, the whole mass weighing a pound or more. In other cases the cysts are quite large, some attaining the size of a child's head, and they may coalesce.

Kidney tissue may be wholly absent or, at least, not demonstrable in the fatal cases of congenital cystic kidney in children; but in the adult some renal tissue is always found. If only one kidney is involved, the other kidney compensates for the deficiency of renal tissue in the diseased organ.
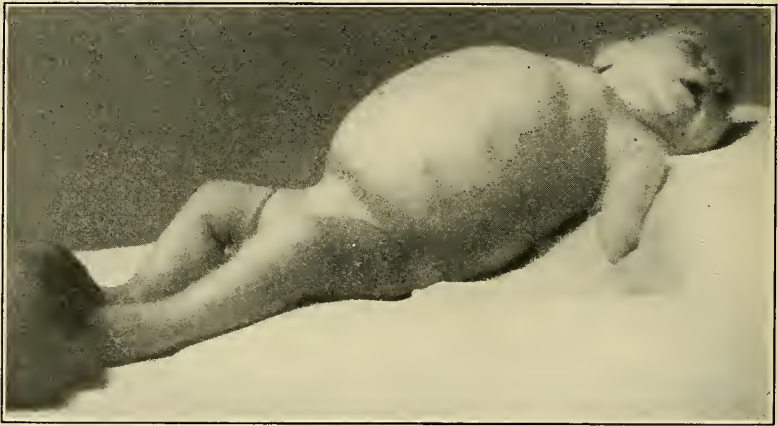
Fig. 54.—Congenital cystic kidney in an infant twenty-five days old.

The cysts contain a clear fluid, which is composed of cholesterin, albumin, blood pigment, fat, triple phosphates, degenerating cells, and, in rare cases, urea or uric acid. The cyst walls are usually fibrous and are lined with flattened or columnar epithelium. There is a distinct connective-tissue stroma, and here and there between the cysts recognizable glomeruli and urinary tubules with saccular dilatations. As a rule, there is no obstruction of the ureters.

Symptoms.—If there is no other malformation incompatible with life, and the kidneys are not so large as to impede labor, the child may be born alive; but death usually occurs during delivery or soon after birth. In many cases there is a palpable abdominal tumor which may cause pressure symptoms. Evidences of renal insufficiency are also present.

Uremic symptoms may occur; but several cases have been reported
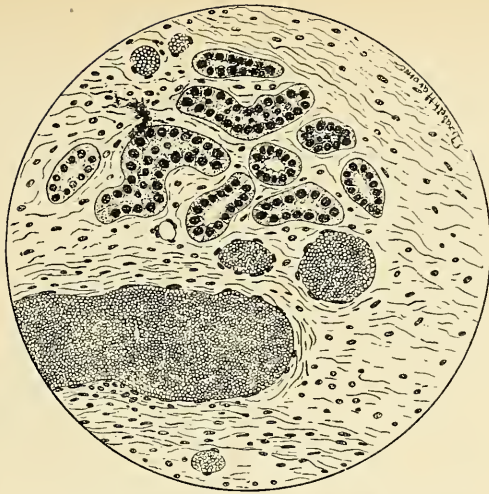
Fig. 55.—Tubular glands resembling sweat glands.

Fig. 56.—Cyst wall lined by columnar epithelium.
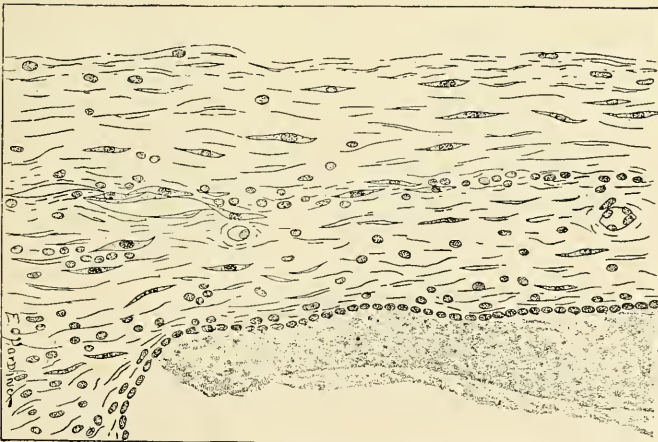
Fig. 57.—Cyst wall lined by flattened epithelium.

of children who lived several months with no renal symptoms. The
duration of life and the prominence of renal symptoms depend
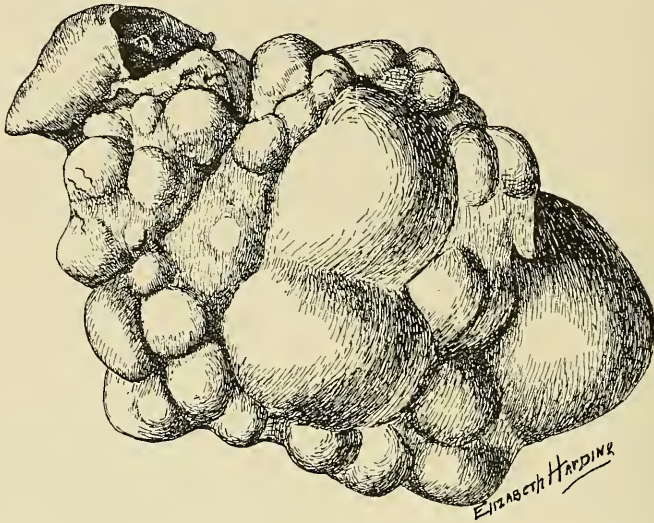
Fig. 58.—Congenital cystic kidney.

entirely on the amount of normal kidney tissue present. If, as in
Fig. 54, from which Figs. 55–59 were made, there is little or no
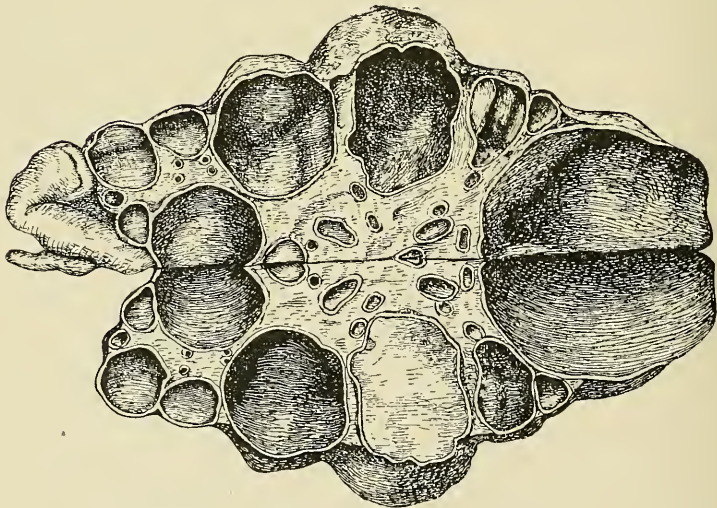
Fig. 59.—Congenital cystic kidney laid open.

normal tissue remaining in either kidney, the baby dies within a few
hours after birth. If only a small amount of kidney substance is

involved, the child may live well into adult life. Where life is prolonged for years, hematuria may appear, also changes in the heart and arterial system, similar to those in interstitial nephritis.

Diagnosis.—The diagnosis of congenital cystic disease is very difficult, because there has been little opportunity to study the symptomatology. The fact that it is bilateral is an aid in the differentiation of this disease from hydronephrosis and malignancy.

Prognosis.—The prognosis is in many cases unfavorable; but the consensus of opinion seems to be that a certain number of cases will live well into middle or advanced life, in spite of the disease. It is also claimed that the affection may persist for years without producing any serious symptoms, or even being suspected.

Treatment.—Treatment by drugs offers no hope of cure, and surgical intervention is rarely justifiable, the condition being usually bilateral.

HYDRONEPHROSIS.

Hydronephrosis may be either congenital or acquired, and is not a rare condition in children. The congenital form is the more common. It makes its appearance during infancy, while the majority of cases of acquired hydronephrosis develop late in childhood. One or both kidneys may be affected, according to the site of the obstruction, but double or bilateral hydronephrosis is so quickly fatal that in many instances the tumor is not detected, and the diagnosis is made only at autopsy. Acquired hydronephrosis is usually unilateral; therefore, more often diagnosed than the congenital form, which is frequently bilateral.

Etiology.—Hydronephrosis is caused by an obstruction to the outflow of urine somewhere along the urinary tract. The congenital form results from stenosis of the ureter or urethra, imperforate urethra, membranous septa, valves or cysts which impede the flow of urine, and to developmental defects of the ureter. The obstruction is generally incomplete, for total blocking of the urinary flow tends to produce atrophy of the kidney rather than an extreme degree of hydronephrosis, although in all types of hydronephrosis the kidney usually shows a certain amount of cirrhosis and dilatation.

Acquired hydronephrosis is most frequently seen as a result of stoppage of the urinary flow by renal calculi; but it may also be caused by inflammatory stricture of the ureter, pressure on the ureter by neighboring tumors, kinking of the ureter, which occurs when the kidney is movable, and by a tight phimosis.

In addition to these two forms of hydronephrosis, Holt describes a traumatic hydronephrosis, which develops after the early symptoms of trauma have subsided, and results in tumor formation two weeks to two months after the injury. The pathology of this condition is obscure, but it presents all the characteristics of hydronephrosis arising from other causes. The tumor may disappear spontaneously, or require surgical intervention for its cure.

36

Pathology.—The kidney is enlarged, often enormously, and loses its reniform shape. As the urine accumulates, it first impinges on the pelvis of the kidney and then, by pressure, causes dilatation of the kidney with atrophy of its substance, until in the later stages the organ is represented by a palpable fluctuating tumor. This enlargement is much slower than the growth of the kidney tumor, and at times may considerably decrease in size by the occasional escape of its contents past the constriction (intermittent hydronephrosis).

Some authorities claim that in unilateral hydronephrosis there is always a lesion of the opposite kidney, usually a chronic nephritis, induced by the nephrotoxic substances which are formed in the blood in these cases. Infection of the accumulation of urine in hydronephrosis results in pyonephrosis, and consequent general toxemia or septicemia. At postmortem, bilateral hydronephrosis has been found to have caused dilatation of the bladder in addition to distention of the ureters and kidneys.

Symptoms.—The chief symptom of hydronephrosis is the formation of a tumor in the kidney region. When the accumulation becomes very large its pressure may produce pain and local tenderness, which are relieved by the discharge of a large quantity of urine of low specific gravity. As a rule, the urine is scanty in amount, and may not reveal any abnormal condition unless infection has taken place in the hydronephrotic tumor. Bilateral hydronephrosis usually terminates fatally in such a short time that no characteristic symptoms appear.

Prognosis.—Unilateral hydronephrosis offers fair chances of recovery, provided that the fluid does not become infected, and the other kidney is normal. When both kidneys are involved, the case is invariably fatal.

Treatment.—The treatment of hydronephrosis, whether congenital or acquired, is surgical. Exploratory puncture, in addition to giving temporary relief, aids in the differentiation of hydronephrosis from cysts, new growths, and tuberculosis of the kidney. The ever-present danger of infection, and the remote possibility of rupture of the sac, seem to justify operation even in the absence of urgent symptoms.

If a stone is found, or some other obstruction is discovered which may be removed, and the kidney is still functionating, nephrectomy is by no means indicated; but if the kidney is infected or practically destroyed, and the obstruction cannot be removed, nephrectomy, to say the least, prevents secondary nephritis in the other kidney, and in many cases saves the life of the child. In view of the fact that infection of the accumulated fluid is a common occurrence, the administration of hexamethylenamin should be kept up throughout the whole course of this disease.

MOVABLE KIDNEY.

Movable or floating kidney is a very rare condition during infancy and childhood. At this early age it is usually a result of some develop-

mental defect, such as an elongated pedicle or a mesonephron, but has occasionally been caused by trauma or the pressure of a neighboring abdominal tumor. Of the few cases described in literature, the right kidney was in children the one most frequently affected. Floating kidney was usually associated with dyspepsia and dilatation of the stomach, which is probably best explained by the close relation between the solar and renal plexuses.

Other symptoms are few in number. They may consist wholly in a slight dragging-down sensation in the kidney region, with some tenderness and, perhaps, frequent micturition. In many patients there may be no symptoms at all, so that the condition is either discovered by accident or is brought to the physician's attention when the pedicle becomes twisted and causes paroxysms of pain which may simulate intestinal or renal colic, or vomiting and intense prostration which might cause one to mistake it for appendicitis. Hydronephrosis or pyonephrosis may result from obstruction of the ureter, and form a tumor in the loins.

In order to palpate the kidney satisfactorily, the physician should stand on the side of the patient corresponding to the kidney he wishes to examine, and place his right hand under the left loin, or his left hand under the right loin, and allow the child to rest upon it. The opposite hand should make gentle pressure upon the front of the same part, thus attempting to press the kidney between the two hands. If movable, it can be felt to slip up toward the liver, or down toward the crest of the ilium. Having ascertained that the kidney is movable, and if the symptoms are very mild, no treatment is necessary. A belt may be worn if discomfort is felt; but operation is only justifiable in those cases in which twisting of the ureter causes severe symptoms.

DISEASES OF THE BLADDER.

PYELITIS.

Pyelitis is in reality an inflammation of the mucous membrane lining the pelvis of the kidney; but so often is the bladder involved as well as the kidney that the terms pyelocystitis, pyelonephritis, and pyelonephrosis are, perhaps, more descriptive and accurate as applied to the various phases of this affection.

As a primary disease, pyelitis is probably not as rare as is supposed, and were urinalyses made routinely in the case of infants, its existence would, perhaps, explain some of the cases of obscure fever at this early age. Pyelitis is found secondarily in older children, but it is rare, and its manifestations are less striking and typical than in infants.

Etiology.—The Bacillus coli is by far the most common infecting organism in this disease; and while, in a fair proportion of cases,

staphylococci and streptococci are isolated, other organisms, such as the tubercle bacillus, the gonococcus, the Bacillus typhosus, and the Bacillus pyocyaneus, are much less frequently met with.

The bladder is probably always involved primarily in infants, and the invading organisms pass up the ureters to the pelves of the kidneys giving rise, in the majority of cases, to a bilateral affection of these organs, but rarely causing severe inflammation of the bladder. This route of infection probably explains satisfactorily the great number of little girls affected by this disease, and its relative infrequency in little boys, although vulvovaginitis accompanied by pyelitis is rarely seen.

The numerous cases in which pyelitis in infancy is preceded by diarrhea, constipation, or indigestion, strongly suggest intestinal disturbance as a preceding factor, and it is quite within reason to assume that, in male infants especially, the colon bacillus may gain entrance to the bladder directly from the intestines. The majority of cases of pyelitis occur in children under two years of age; after the fifth year it is rare, is usually caused by renal calculi, and is associated with tuberculosis or tumors of the kidney, or is secondary to scarlet fever, diphtheria, influenza, or typhoid fever. In exceptional instances there is a history of trauma, of suppurative processes in the urethra or vagina, or of the ingestion of such drugs as turpentine, cantharides, or carbolic acid. Pyelitis is said to be more common in summer than in winter, probably owing to the lowered resistance of infants at this season. An enumeration of the etiological factors in pyelitis would scarcely be complete without mentioning the hematogenous and lymphogenous routes by which the infection may have been carried from some distant part of the body.

Symptoms.—The most characteristic feature of pyelitis in infants is the presence of marked constitutional symptoms with little or no localized evidence of the disease; while, in older children, the local symptoms are severe and the constitutional disturbances mild. In infants, at the onset, there is a sudden and high rise in temperature, not uncommonly preceded by chills and rigors, and accompanied by other evidences of acute infection, such as vomiting, diarrhea, delirium, or a restless drowsiness, and also perhaps a stiffness of the neck and twitching of the limbs which suggest meningitis. There is usually little pain; but, if it be present, it so closely simulates intestinal colic, as to be impossible of differentiation. In some cases the abdominal muscles on one or both sides may be rigid. These symptoms are followed in a few days by the appearance of mucus or mucopus in the urine, which is strongly acid when passed, and, examined microscopically, is seen to contain numerous bacilli and pus cells with an occasional cast. Fever is usually of intermittent type, ranging from 101° to 105° or 106° F. for a week or several days. The chills, however, are seldom repeated, although the urine may contain pus for several weeks.

Older children frequently complain of distinct pain in the abdomen with tenderness over the loin; but, as a rule, the disease runs a subacute course, with anorexia, nervous irritability, and a variable temperature

range. The urine contains pus and desquamated epithelium from the various parts of the urinary tract involved. If cystitis also exists to a marked degree, dysuria and other bladder symptoms are present; and, if pyelonephritis or pyonephrosis develop, there may be evidence of tumor formation in the kidney region. Cautley described three types of pyelonephritis in children: (1) Acute, with rigors, fever, lumbar and abdominal pain, and sweating. The urine is unaffected for two or three days, then shows pus and organisms. Sometimes there is retention, occasionally blood. (2) Subacute, with unexplained fever, anorexia, malaise, anemia, and wasting. (3) Chronic relapsing, recurrent attacks with fever, headache, vomiting, malaise, painful micturition, and, perhaps, lumbar pain suggestive of stone.

In the interim, in chronic pyelitis, which is usually due to stone or malformations, pyuria may be the only symptom of pyelitis until suppurative pyelonephritis or pyonephrosis develops, and causes the localized symptoms of pain, tenderness, and swelling. Occasionally, perinephritis develops late in the course of the disease; as a rule, it rapidly causes an abscess from which the pus is sometimes discharged into the pelvis of the kidney, and subsequently voided in the urine. Tuberculous pyelitis, when observed, is usually associated with general tuberculosis and is evidenced by chronic pyuria and the presence of tubercle bacilli in the urine.

Diagnosis.—Although the diagnosis of pyelitis in infancy hinges more on the urinary findings than on any other signs or symptoms, the significance of a sudden chill, followed by high fever as being suggestive of pyelitis, cannot be too greatly emphasized. Typhoid fever, acute intestinal indigestion, influenza, and especially malaria, are simulated by the mode of onset of pyelitis; but careful study of the temperature range, which is intermittent with distinct afebrile periods following the discharge of pus, will serve to exclude all of these diseases. Other characteristic symptoms of pyelitis are oliguria, pyuria, and a variable degree of pain and tenderness over the kidneys. The relatively large amount of pus contained therein and the reaction of the urine, which is acid, will exclude inflammation of the bladder as the main source of the infection, while the severe constitutional symptoms, with few or no tube casts or renal epithelium in the urine, are sufficient to exclude acute nephritis. Pneumonia, meningitis, and otitis, which should always be considered when we have obscure fever in infants, may be easily excluded by an accurate history and the absence of signs of these diseases upon thorough examination. Very frequently cystitis coexists, and there are symptoms and signs of vesical irritability; also, with the development of pyelonephritis and pyonephrosis, distinct lumbar pain with, perhaps, tumor formation which indicates that one is dealing with more than simple pyelitis.

Prognosis.—When pyelitis remains uncomplicated, the prognosis is good, and the duration of the disease depends more upon the time when it is recognized and treated than upon the nature of the infection. In pyelonephritis and pyonephrosis, which usually develop in cases

of pyelitis secondary to acute infections, malformations, or tumors of the kidneys, the outlook is very serious, so that it is only in cases of secondary pyelitis where the primary cause is early recognized, and removed before renal involvement takes place, that recovery is to be hoped for. Relapses are not infrequent, and may occur even a year after the initial attack. The mortality in uncomplicated pyelitis is about 10 per cent.

Treatment.—The treatment of pyelitis varies with the acuteness and severity of the attack, and there can be no doubt that many infantile cases pass unnoticed and recover spontaneously. The patient should be kept at rest in bed, and the diet, which should be bland and non-irritating, restricted almost wholly to milk. Water should be taken in large quantities, and this may be encouraged in children by flavoring it with orange or lemon juice. The reaction of the urine should be neutralized. For this purpose, hexamethylenamin is largely used when hyperacidity is present, one grain every three hours being the average dose for an infant one year of age, and correspondingly larger doses for older children.

Citrate of potassium in massive doses is exceedingly beneficial, and preferred by many authorities to any other drug for the treatment of pyelitis. As much as one-half or even one dram (four grams) may be given an infant one year old and upward during the course of a day with no decidedly untoward effects aside from slight depression and gastrointestinal disturbance. If nausea, depression, and low temperature should ensue, sodium bicarbonate, in 10-grain doses every three hours, may be given to an infant under one year of age with splendid results and no harmful effects.

Pain is rarely as intense as in renal colic; but, if severe, may be relieved by the local application of heat or cold, or by the administration of paregoric, 10 to 15 drops, every two hours until relieved, or until four doses are taken, if the infant be a year old or less.

In chronic cases, the bowels should be watched and regular movements promoted by adding to the diet enough coarse food to insure a large residue, also by the administration of mild cathartics, such as compound licorice powder or saline waters. When the diet is greatly restricted, as in acute cases, free bowel movements should be obtained by the use of magnesium sulphate and enemata.

In protracted cases of pyelitis, which resist all other treatment, vaccine therapy should be instituted, using the autogenous vaccine whenever possible—for response to this line of treatment may avert the development of pyelonephritis or pyonephrosis. When pyelitis is secondary, the cause should be removed as quickly as possible, and if pyonephrosis develops, surgical intervention is indicated.

Perinephritic abscess may require merely incision and drainage; but if the kidneys be involved, as in suppurative pyelonephritis and pyonephrosis, the surgeon must decide upon either nephrectomy, nephrotomy, or pyelotomy, according to the extent of renal involvement and the functional activity of the other kidney.

CYSTITIS.

Primary cystitis is rare during infancy and childhood, but inflammation of the bladder secondary to disease elsewhere in the genitourinary tract is not uncommon.

Etiology.—The organisms most frequently found are the gonococcus and the Bacillus coli, which indicates the two sources of infection—*i. e.*, the external genitalia and the gastro-intestinal tract. Mechanical irritants, such as calculi and gravel, as well as various chemicals and medicaments, are also exciting factors, but in only a small proportion of the cases. Among the predisposing causes of cystitis are excessive cold or heat, strongly ammoniacal urine, trauma, or any condition which lowers the tone of the bladder or prevents its complete emptying.

Cystitis is much more common in girls than in boys owing to the short urethra in females, which renders extension of inflammation by this path to the bladder quite natural. Phimosis and a narrow urethral meatus are two of the causes of cystitis in boys, and, as a rule, the affection when due to either of these conditions is very severe. In rare instances, tuberculosis or tumors of the bladder may be the cause; in some cases, inflammation of the bladder may develop during the course of typhoid fever, although bacteriuria may exist indefinitely without giving rise to appreciable bladder lesion.

Symptoms.—The characteristic symptom of cystitis is pollakiuria accompanied by vesical spasm which causes great pain; but in mild cases in which there is no pain, the unduly frequent passage of urine is often attributed to nervousness, and the cystitis is overlooked. In severe cases there are pain and tenderness over the pubes and in the perineum, high fever, constitutional disturbance, and even convulsions. The urine is voided a little at a time; it contains epithelium, pus, mucus, many bacteria, and a trace of albumin. It is of a reddish color, alkaline or slightly acid in reaction, and may show the presence of blood, especially if the cystitis is caused by calculi. In chronic forms of cystitis the symptoms are less acute and usually exist unrecognized for an indefinite period until, for some reason, the urine is examined. If, however, a vesical calculus be the cause of chronic cystitis the symptoms are more severe, and although the pain may not be as great as in the acute form, pollakiuria frequently becomes so aggravated that the urine appears to be voided continuously. The urine presents the same characteristics as in the acute form, and the constant dribbling often gives rise to irritation and inflammation of the genitalia and adjacent skin.

Prognosis.—The prognosis in simple acute cystitis is favorable, and prompt recovery may be expected after removal of the cause and appropriate treatment. Chronic cystitis may be secondary to some primary condition, such as disease of the kidney, tumor, or tuberculosis of the bladder, and in these cases is very resistant to treatment.

Treatment.—The child with cystitis should be put to bed and the diet almost wholly restricted to liquids, milk being, perhaps, the best

form of nourishment. The child should be encouraged to drink a large amount of water, but other beverages, such as tea and coffee, should be prohibited. Potassium citrate in massive doses has proven, in my experience, to be the one drug *par excellence* in cystitis, and as much as 1 dram (4 grams) has been administered to an infant a year old. The child should take enough of the drug to keep the urine slightly alkaline or, at least, neutral in reaction until the pyuria has ceased.

Urinary antiseptics are also indicated in cystitis, the best results being obtained by the use of salol or hexamethylenamin in from 2- to 5-grain doses, three times a day, according to the age of the child. To relieve the severe pain caused by vesical spasm, atropine sulphate may be given in from $\frac{1}{1000}$ to $\frac{1}{500}$ of a grain doses, three times a day, according to the tolerance of the child. If this is not efficacious, a suppository of opium and belladonna should be inserted in the rectum, and hot compresses applied over the bladder. Unless the cystitis is very severe or shows a tendency to become chronic, irrigation of the bladder is not advisable.

Comparatively few cases of cystitis become chronic during childhood, but when this happens they are very stubborn and require radical measures and close supervision to control them Daily irrigation of the bladder with a 1 per cent. boric acid solution, or a 1 to 5000 solution of either silver nitrate or potassium permanganate, should be continued for a week, after which the number of irrigations should be diminished each week until recovery ensues, although it is sometimes a good plan to use a little stronger solution at each irrigation.

As yet, the exact value of autogenous vaccines in cystitis has not been definitely established, and although satisfactory results have been claimed by some observers, my own experience has not led me to use them except as a last resort in protracted cases, which have not responded to other lines of treatment.

VESICAL SPASM.

Spasm of the bladder occurs most frequently in the early years of childhood. When observed immediately after birth it is due to uric acid infarctions, and later is often caused by an excess of uric acid in the urine. It is a characteristic symptom of cystitis and vesical calculus, and may be the result of blood-clots, excessive phosphatic deposits, or renal gravel which obstructs the urinary flow. Occasionally, sudden chilling of the lower abdomen or trauma in the region of the bladder is followed by vesical spasm, and in rare instances, it is seen in association with hysteria, priapism, and masturbation. The symptoms are chiefly local, and indicative of great pain and difficulty in urinating. In very young children the pain may simulate intestinal colic; but the absence of any signs or symptoms of gastro-intestinal disturbance, and the distended bladder which abdominal palpation reveals, readily establish the source of pain. Urination is frequent, but the amount of urine passed each time is small.

Treatment.—Removal of the cause is essential to permanent relief of the conditon. Distention of the bladder frequently necessitates catheterization; but hot baths or hot compresses applied to the suprapubic region should be tried first in an effort to secure complete evacuation of the bladder. If pain is extremely severe, tincture of hyoscyamus may be given in doses of one to three minims, three times a day; or a suppository of powdered opium and extract of belladonna, each grain $\frac{1}{4}$, may be cautiously used for its relief.

The child should be encouraged to drink plenty of water, and by careful regulation of the diet an effort should be made to prevent hyperacidity or hyperalkalinity of the urine in the future. Potassium citrate is, perhaps, the best diuretic in this affection, and may be given to advantage through the attack.

VESICAL CALCULI (URETHRAL CALCULI).

Calculi in the bladder or urethra of children are only occasionally met with. Although, during infancy, the passage of renal sand or gravel is quite common, a concretion is rarely of sufficient size to be retained within the bladder. The majority of calculi contain uric acid and urates, the remainder being mainly oxalates and phosphates. The tendency to formation of concretions is greatly increased by any inflammatory process along the urinary tract. It has been estimated that vesical calculi are twenty times as common in boys as in girls, a fact readily explained by the ease with which a stone may escape from the bladder through the short and distensible female urethra.

Symptoms.—The symptoms of vesical calculus are, for the most part, associated with the act of micturition. There is great frequency of urination, accompanied by pain more or less intense, and referred to the end of the penis, to the perineum, or in some cases to the rectum. The stream is variable and uncertain, and may be interrupted only to be resumed after a change in posture. In some cases there is incontinence during the day, but rarely during sleep.

If the calculus be impacted in the urethra there may be a constant dribbling of bloody urine and efforts to dislodge it. Straining during the act of urination often causes a prolapse of the rectum. When violent exercise is taken, pain is usually felt in the bladder region, but this quickly subsides upon lying down. If allowed to remain in the bladder a stone quickly causes cystitis with characteristic changes in the urine; but, in the absence of this condition, the urine usually shows an excess of crystals, mucus, a little pus, and, occasionally, blood.

Diagnosis.—All other causes of vesical irritation must be excluded before one is justified in considering it due to a stone in the bladder. Once this has been done, the diagnosis may usually be readily confirmed by the x-ray, the use of the sound, and bimanual examination through the rectum.

Treatment.—Surgical removal of the stone is practically always necessary; in my opinion, suprapubic lithotomy is preferable to lithoplaxy.

If the stone is removed before complications have arisen, recovery may be expected; but, as a rule, these children are very weak and anemic, and should have all the benefits of a carefully regulated, nourishing diet, and change of location to seashore or country.

URETHRITIS.

Urethritis occurs in both sexes during childhood; but, owing to the prevalence of vulvovaginitis with which it is usually associated in females, it is far more common in girls than in boys. It may be either simple or specific, the simple form being but a slight inflammation of the anterior portion of the penile urethra caused, generally, by lack of cleanliness of the genitalia, and occasionally by injury or the passage of uric acid crystals.

The symptoms are very mild, and consist of a little pain on urination, frequent micturition, and a slight discharge of pus which contains leukocytes, various microörganisms, and a few epithelial cells. There is usually a prompt disappearance of symptoms if the parts are simply kept clean. Irrigation is rarely necessary unless the discharge persists, when the anterior urethra should be gently syringed out daily, using a 1 to 2000 potassium permanganate solution, or a 5 per cent. solution of argyrol.

Specific Urethritis.—Gonorrheal infection of the urethra in children is much more frequently seen than the simple form, but rarely occurs before the sixth year as it is usually contracted through direct contagion. While not as serious a disease as in adults, gonorrheal urethritis, in comparison to the simple or non-specific form, is a very severe infection, and is not confined to the anterior urethra, but may involve the posterior urethra as well, and is not infrequently complicated by balanitis, stricture, epididymitis, and inguinal adenitis.

Orchitis is a rare complication, but though there are few constitutional symptoms of gonorrhea in the child, arthritis and conjunctivitis occur not infrequently as complications. The symptoms are frequent micturition, accompanied by severe burning pain on urination and a profuse, thick, creamy discharge of pus. It is upon the microscopic examination of this discharge that the diagnosis of specific urethritis is based, and there is usually very little difficulty in making the diagnosis, for gonococci are, as a rule, present in great numbers.

Treatment.—Much attention should be paid to prophylactic measures, in order that the child's brothers, sisters, and playmates may not be contaminated, and also that the child may not infect other parts of its own body. The genitalia should be kept securely covered so that the child cannot touch the parts, for if the hands become contaminated the infection is easily spread.

The child should be allowed to drink plenty of water, and free diuresis should also be promoted by an alkaline diuretic, such as hexamethylenamin, which may be given in from 2- to 5-grain doses, three times a day, according to the age of the patient. If the

discharge is pro use, and tends to persist, it is best to irrigate the anterior urethra with a 1 to 500 bichloride solution, or a 5 per cent. solution of argyrol.

The diet should be bland and non-irritating, and all spices, seasoned foods, beverages, aside from milk and water, should be prohibited. The child should eat sparingly and confine the diet to cereals, bread, butter, milk, and puddings, with a few vegetables, and little meat.

Physical exertion, if carried to excess, is extremely harmful, but rest in bed is not necessary. Of great importance is the condition of the bowels, and special emphasis should be placed on the fact that the patient must have one or two soft stools daily. A properly fitting suspensory is often a source of great comfort and a help in retaining the dressing while the discharge persists.

DISEASES OF THE REPRODUCTIVE ORGANS.

PHIMOSIS.

Phimosis is a narrowing of the orifice of the prepuce which prevents the withdrawal of the foreskin over the glans penis, and frequently results in adhesions between the inner surface of the prepuce and the mucous membrane of the glans. Narrowing of the orifice of the prepuce is normal in very young infants, and should be corrected early by the mother or nurse, as a tight foreskin retains the smegma, and is a constant source of irritation. In time it may interfere with the flow of urine, and cause retention, or permit the passage of only a few drops at a time, which frequently gives rise to stubborn eczema of the genitalia.

Nocturnal incontinence is often a direct result of phimosis, and in rare cases, also of hydronephrosis. The nervous equilibrium of the child, as yet undeveloped, is severely deranged by the constant itching and irritation. As a result, there is restlessness and peevishness during the day, with night terrors and insomnia. In some instances it leads to the habit of masturbation, or an attack of hysteria or chorea comes on. Adherent prepuce in little girls is not fraught with as much danger as the same condition in boys; but it has been claimed that, if allowed to persist, the result will be hypererethism or sexual apathy in later life.

Treatment.—In many cases of phimosis, mere stretching of the foreskin and separation of the adhesions will relieve the condition, after which the prepuce should be drawn back, and the glans cleansed thoroughly every day. Circumcision, while more radical, is, perhaps, the best and safest measure with which to secure permanent results.

PARAPHIMOSIS.

Paraphimosis is a condition just the opposite to phimosis, although due to the same cause. The narrow foreskin is drawn over the glans penis, and forms a tight constricting band behind the corona. The glans soon swells, making it impossible to slip the foreskin back again over the head of the penis, and if the constriction is not relieved the intense edema and failure in circulation will result in gangrene.

Treatment.—The penis should be bathed in hot or cold water, and an attempt then made to relieve the constriction by holding the foreskin between the first two fingers of each hand and exerting pressure on the glans with the thumbs while gentle traction is being made on the foreskin with the fingers. When the edema is marked, multiple punctures of the glans are sometimes necessary to reduce its size. If these measures fail, and the circulation is not established, the constricting band must be promptly incised to prevent permanent injury to the glans.

BALANITIS.

Balanitis is an acute inflammation of the mucous membrane of the glans penis, and is frequently observed in children, its most common cause being phimosis. Lack of cleanliness results in the retention of smegma and urine between the glans and the prepuce, in consequence of which the mucous membrane of the glans becomes tender, swollen, and covered with pus, and the foreskin edematous and swollen. The discharge of pus may be so free as to suggest urethritis.

Treatment.—Daily cleansing of the parts, followed by an antiseptic wash with a saturated solution of boric acid, will usually effect a cure within a few days. Circumcision is indicated if the prepuce be tight or elongated, but should not be performed until all inflammation has subsided.

TORSION OF THE SPERMATIC CORD.

Torsion of the spermatic cord is a very rare occurrence, and practically never happens except in those children where the testicle fails to descend or only descends partly. When the testicle becomes twisted upon the cord, circulation is cut off because of its constricted condition, and gangrene may result if the blood supply is not resumed quickly. The symptoms resemble closely those of strangulated hernia, and it is also difficult to differentiate this condition from acute orchitis.

Treatment.—The treatment is surgical and depends upon the length of time the circulation has been shut off and the resultant condition of the tissues. The cord should be untwisted if possible, and the testicle slipped back into place, but if gangrene is present, cord and testicle must be excised.

ACUTE ORCHITIS.

Acute or simple orchitis is rare during childhood, and is usually secondary to some systemic infection. The inflammation begins in the epididymis. Hydrocele very frequently accompanies acute inflammation of the testicles, but has no bearing on the severity of the process.

Etiology.—In children, acute orchitis is seen occasionally following urethritis, but a fair proportion of cases occur during the acute infections, especially mumps, typhoid fever, and variola. Authorities differ as to the prevalence of syphilitic orchitis in infancy and early childhood, but from my own observations I should judge it to be relatively frequent. Injury to the testicles is often followed by a simple, mild orchitis; if the trauma be severe, and much damage has been done the the the tissues, gangrene may set in.

Symptoms and Pathology.—In simple orchitis the inflammation in the epididymis is essentially catarrhal, but when the testicle becomes involved the interstitial tissue is invaded, and here the inflammation tends to become almost entirely interstitial. The cardinal symptoms are pain and swelling, and the size of the scrotum may be increased by the presence of a hydrocele, which often accompanies acute orchitis. The pain may be felt in the loins, but is most severe in the scrotum, which may also be red and edematous. In most instances, in addition to the local symptoms, there are systemic symptoms, such as fever, anorexia, and malaise.

Treatment.—The child with acute orchitis should be put to bed and kept there until recovery ensues. The testicles must be supported by stretching a wide strip of adhesive plaster from the anterior portion of one thigh to the other, and allowing the scrotum to rest upon it. Ice-bags are useful if applied to the scrotum in the very early stages of the inflammation; but, after this time, heat is more serviceable, and should be applied by means of hot compresses renewed frequently.

A saline laxative preceded by a course of calomel, is of great benefit, and is practically the only internal medication that is of any value. The course of the inflammatory process should be watched closely, and if suppuration sets in the testicle should be freely incised and the pus allowed to escape. The child must wear a suspensory bandage for several weeks after the attack, for in some instances atrophy of the testicle follows acute orchitis. Free circulation of the blood should be encouraged in every way possible.

TUBERCULOUS ORCHITIS.

There are two forms of tuberculous orchitis occurring in children, and it is important that each form should be recognized as such. Involvement of the testicles in children with general tuberculosis is the most common type, and more rarely is it seen as a part of a

generalized tuberculosis of the genito-urinary tract alone.   Both testicles are involved, as a rule, the tuberculous process beginning in the epididymis.

Etiology.—Tuberculous orchitis is a secondary process in almost every instance, the infection having been carried to the epididymis through the lymphatics, blood stream, or vas deferens. Occasionally one may obtain a distinct history of trauma, which has probably been an important predisposing factor in determining the testicles as a site of the tuberculous lesion.

Symptoms and Pathology.—The testicle in which a tuberculous process is going on becomes a hard indurated mass as in adults, and if the child resists the effects of the systemic infection, which is usually present, this mass breaks down, suppuration takes place, the tunica vaginalis is involved, the skin ulcerates, and a sinus forms. The cord is always thickened. The local symptoms are pain and swelling, for in children tuberculous orchitis may be very acute, and a large tumor form in a very short period of time. Other symptoms of tuberculosis are usually present throughout the body and aid in the diagnosis, but even without these additional signs, if tubercle bacilli are found in the discharge from the sinus, the diagnosis is established without doubt.

Prognosis.—The prognosis of tuberculous orchitis is much better in children than in adults, as the tendency for the process to spread and involve the surrounding tissues is much less marked and the infection does not assume as serious an aspect.

Treatment.—The testicles should be treated locally, palliative measures at first, such as pressure and the application of iodide of lead ointment. Castration should be performed early where the danger of general infection of tuberculosis is to be feared, but should not be undertaken until the diagnosis is definitely established and suppuration is taking place. In every case, whether other lesions of tuberculosis are found elsewhere in the body or not, the child should receive the benefit of all the hygienic, dietetic, and medicinal measures with which tuberculous children are treated. Fresh air is indispensable both night and day, the food should be highly nutritious, and the child should obtain the proper amount of exercise and rest to materially build up the general physical condition.

TUMORS OF THE TESTICLE.

Tumors of the testicle are rarely seen during childhood, and, in not a few cases where the existence of a neoplasm is suspected, careful study and investigation will prove the growth to be a tuberculous process which has extended from the epididymis. Congenital growths are usually malignant, and either carcinomatous or sarcomatous, but occasionally one sees a benign growth, an enchondroma or myoma. Acquired growths of the testicle are, as a rule, extremely malignant, the round-cell sarcoma being the most common. Cystic disease of

the testicle occurs with relative frequency in childhood, and is always a serious problem, since it is impossible to differentiate simple cystic disease from sarcoma.

Symptoms.—Tumors of the testicle are most common from the first to the tenth year of life. There are very few symptoms of malignancy at the onset of the disease, but the tumor grows very rapidly in size, and with this increased growth there is a progressive feeling of discomfort and weight. The tumor sometimes becomes quite large before constitutional symptoms of malignancy and cachexia are apparent.

Treatment.—In view of the fact that most tumors of the testicle are malignant, and in those which are not so at the onset there is an ever-present possibility of malignancy, a surgical operation for the removal of the testicle should always be advised when it is the site of a new growth.

VULVOVAGINITIS.

Vulvovaginitis is not an uncommon condition in little girls between the ages of two and eight years, but rarely occurs in infancy. The mucous membrane of the vulva is inflamed and swollen, and the process may extend into the vagina, and, in rare instances, involve the urethra and cervix uteri. The majority of cases are not specific, but in the poorer classes, especially, gonorrheal vulvovaginitis occurs quite frequently. Diphtheritic and aphthous vulvovaginitis are extremely rare forms sometimes seen in large institutions.

Simple Vulvovaginitis.—Simple vulvovaginitis is usually a mild catarrhal inflammation, confined for the most part to the vulva, and accompanied by a serous discharge; but not infrequently one encounters a severe case with intense inflammation of the vulva and vagina, and a profuse purulent discharge. The urethral orifice and cervix uteri may be involved, and the whole aspect of the case resemble a specific infection. Microscopically, the discharge is found to contain numerous bacilli and cocci, but, while the cocci resemble gonococci in some respects, they do not exhibit all the characteristics of this organism.

Etiology.—Simple vulvovaginitis is caused by uncleanliness in the majority of cases, the mucous membrane of the genitalia becoming contaminated from collections of smegma, the irritation of decomposing secretions which have accumulated, or from eczema of the adjacent skin surfaces. Occasionally the inflammation is due to the habit of masturbation, local injury to the parts (attempted rape, etc.) or to scratching in the case of scabies, eczema, or threadworms.

Delicate and anemic children whose constitutions have been undermined by tuberculosis, malnutrition, poor hygienic surroundings, or a recent acute contagious disease, are especially liable to vulvovaginitis, and here we have the probable explanation of the rapidity with which an epidemic spreads through the wards of an institution when a child with vulvovaginitis is admitted. The exact means of transmission from one child to another has not been satisfactorily deter-

mined, but there is no doubt as to the highly contagious nature of the discharge.

Symptoms.—In mild cases, local symptoms of inflammation are so slight that the existence of the disease is first brought to notice by the appearance of the discharge, which is usually serous and scanty, and close inspection of the parts reveals a little redness and swelling which, however, are limited to the external genitalia. There may be a little local tenderness and a slight itching, intensified on urination.

Simple vulvovaginitis may be of such a severe type as to present all the characteristics of a gonorrheal infection, and in these cases it can only be differentiated by careful and thorough microscopic study of the discharge. The labia are red, swollen, and may be ulcerated; the vagina is inflamed, and the urethral orifice and hymen are also involved. The parts are bathed in a profuse, thin, greenish-yellow discharge, and urination is attended by much pain.

If the adjacent skin becomes excoriated walking is painful, and the child assumes a straddling gait to prevent friction of the parts. In some instances there is a moderate elevation of temperature at the onset, but this falls to normal after the acute symptoms have subsided.

Prognosis.—A mild vulvovaginitis may last two or three weeks, but the severe type usually continues for one or two months. Complications, such as cystitis and urethritis, are rare, and a complete and uneventful recovery will follow the institution of proper treatment.

Treatment.—Vulvovaginitis responds readily to gentle irrigation with either saturated boric acid solution, sulphocarbolate of zinc solution, or a weak solution of potassium permanganate. The solution should be fairly hot (108° F.) and its use should be preceded by thorough cleansing of the genitalia and adjacent skin surface with soap and water.

If the labia be excoriated, a soothing antiseptic ointment, containing 1 per cent. phenol and 1 per cent. hydrargyri ammoniata, will allay the burning sensation and protect the parts from the discharges, and a pledget of cotton interposed between the labia will, by keeping them separated, prevent friction. In my experience, I have found the use of an ointment preferable to dusting powders, which are apt to cake when they become moist, and thus cause additional irritation.

The general health of the majority of these children is poor, and improvement in the living conditions, together with the administration of a tonic, such as cod-liver oil or the syrup of the iodide of iron, will materially hasten recovery. In every case the etiological factor should be ascertained and removed, and prophylactic measures instituted to prevent the spread of the infection to other children.

Gonorrheal Vulvovaginitis.—Gonorrheal, or true, vulvovaginitis is by no means an uncommon affection in little girls of the poorer classes between the ages of two and eight years, since at this age it is not a venereal disease, but is contracted accidentally because of close seggregation. It is one of the most highly contagious diseases met with in institutions, and epidemics, once begun, are rarely checked until the

majority of the female children, if not all of the inmates, are attacked. The infection is far more virulent and serious than simple vulvovaginitis, is very resistant to treatment, and, as a rule, runs a protracted course.

Etiology.—In the majority of cases of vulvovaginitis seen in private practice, it has been contracted accidentally by children sleeping with adults, using their towels and toilets, or bathing in the same tub. Older children may be contaminated by their playmates manipulating the sexual organs, and contamination of the vagina is frequently traced to nurse girls, but rarely is vulvovaginitis the result of a criminal assault.

Infants have been known to become infected in their passage through the birth canal, but during infancy this disease is usually transmitted through the medium of the napkins.

The exact way in which the contagion spreads in institutions, despite strictest prophylaxis and antisepsis, is so obscure that the theory of atmospheric infection has been advanced, and although one hesitates to accept this, in view of the fact that nurses in charge do not become infected, it is only by isolation that the disease is finally eradicated. There can be no doubt that the prevalence of the disease is very much increased by the poor general condition of health so common in children of institutions and the slums.

Symptoms.—The severity of an attack of vulvovaginitis is extremely variable, and while the extent of the inflammation is modified by the virulence of the infecting organism and the constitutional condition of the patient, records of a large number of cases show that, as a general rule, the younger the child, the milder the attack. The inflammation is at first severe and the discharge profuse, but in many cases the disease is either discovered accidentally during routine examination of children, or the physician is consulted because of the discharge alone, other symptoms being so mild as to escape observation.

The discharge varies greatly and may be largely mucus, mucopus, or a thin, serous, greenish-yellow fluid, but the characteristic discharge in gonorrheal vulvovaginitis is a thick, creamy, yellow pus, which, when examined under the microscope, is found to contain the gonococcus in practically every case and but few other organisms. Infants and young children may show no other local sign of inflammation than a slight hyperemia of the mucous membrane of the vulva and orifice of the vagina, with a few crusts on the labia; but in older children there is usually redness and swelling of the vulva and vagina, and in some instances the skin and mucous membrane become excoriated and even ulcerated.

The urethra rarely escapes, although the bladder is not frequently involved. The cervix uteri becomes inflamed and an endocervicitis results, but extension of the process into the internal pelvic organs is extremely rare. The inguinal glands are found to be enlarged in some cases, and accompanying this there may be a slight elevation of temperature which, however, subsides after the acute symptoms have passed.

37

Irritation of the parts produced by walking causes the child to assume a straddling gait which is very much exaggerated if there are excoriations of the thighs and labia. Urethral involvement very often gives rise to painful micturition, and in every case there is increased frequency of urination, which causes nocturnal and diurnal enuresis.

Diagnosis.—A positive diagnosis of gonorrheal vulvovaginitis can only be made when gonococci are found in the vaginal smears; and, therefore, it is important to obtain a smear in every case of vulvovaginitis, even though the discharge be very scant. In some instances one may only be able to state that the cocci present the characteristics of the gonococci, and at times cultures must be made before it can be positively determined that the organism is the gonococcus; but the finding of gram negative intracellular diplococci is accepted as conclusive evidence of Nisserian infection.

Those smears in which we find the gonococcus usually contain few other organisms, and so true is this observation that, although the gonococcus may not be demonstrated, if very few other organisms are found, one should be highly suspicious of specific infection. In non-specific or catarrhal vulvovaginitis the discharge is usually composed of an abundance of several varieties of cocci and bacilli. If the facilities are not at hand thoroughly to examine smears and make cultures, any case of vulvovaginitis which is accompanied by a profuse, purulent discharge should be considered specific, and be treated as such.

Course and Complications.—Gonorrheal vulvovaginitis runs an essentially protracted course; from six to eight weeks is the shortest period in which recovery ever occurs, and in most cases three or four months or longer are required for its cure. The two complications which the practitioner must be warned against are ophthalmia and arthritis, for they are seen occasionally and should be recognized and treated immediately. Endocarditis, septicemia, and pyemia are rare.

Prognosis.—Despite its protracted course the ultimate outlook of a case of vulvovaginitis in the absence of complications is good. The fact that gonococci may remain latent in the genital tract for an almost indefinite period should not be overlooked, however, as this is no doubt responsible for the numerous relapses which occur.

Prophylaxis.—The extremely infectious nature of gonorrheal vulvovaginitis makes the consideration of prophylactic measures of utmost importance. In the home, if one child is affected, it should be isolated from the others in so far as this is possible, and at least all relations or associations with other children should be avoided. This refers particularly to the child's clothing, bed linen, towels and table silver, which should be kept apart at all times from those of the rest of the family.

The child should bathe in a tub which no one else is allowed to use, should use a separate toilet, and should wear pads which are changed frequently and the soiled ones burned. Too much attention cannot be directed to the necessity of absolute cleanliness, and this is espe-

cially important with regard to the hands, which, if soiled, may easily be the means of conveying the disease to other children.

Prophylactic measures must be carried out to even a greater degree if epidemics are to be avoided in institutions where children are congregated together. Every applicant for admission should be thoroughly examined for vaginal discharge, and even if the microscopic examination of a discharge be negative, the patient should be isolated for at least two weeks and repeated examinations made, for in a large number of cases where thorough study of the discharge is made one is able eventually to demonstrate the gonococcus.

The routine examination of all children in institutions twice or three times a week is recommended as the only means to eradicate the disease. Children isolated because of a discharge should be under the care of special nurses; for it is practically impossible for a nurse to attend a child with vulvovaginitis without conveying the disease to other children. Care must be taken to disinfect all linen and clothing before allowing it to go to the general laundry, and all pads and dressings should be burned.

A nurse treating more than one case of vulvovaginitis should carefully cleanse and disinfect her hands after attending each patient, and should see that the toilets and tubs are disinfected each time they are used in order to prevent reinfection of convalescent patients. It is unwise to allow any patient to mingle with other children until there is absolutely no vaginal discharge remaining, for in many instances fresh outbreaks of vulvovaginitis have followed where children have been taken out of quarantine, simply because there were no gonococci demonstrable in the vaginal secretions.

Treatment.—Cleanliness of the parts is the prime essential in the successful treatment of gonorrheal vulvovaginitis, and this is mainly secured by means of douching and flushing the vagina. In the initial stage, when the inflammation is severe, a vaginal douche should be given three or four times daily, using at least two quarts of either a 1 to 5000 potassium permanganate, or 1 to 5000 bichloride solution, the temperature of which should be about 106° to 108° F.

The use of various other irrigating fluids, such as normal saline, boric acid, and silver salts solutions will, perhaps, be found to give equally good results, since the real benefit of the douche is apparently due to its cleansing effect more than to anything else. The female catheter is undoubtedly the best irrigating nozzle for this purpose, but care should be taken not to insert it too far or injure the parts in any way, or force the solution into the uterine cavity by allowing the stream to flow too swiftly.

If the child be under the care of inexperienced persons it is sometimes wiser to omit douching, and simply allow the child to sit in a large basin partly filled with warm boric acid or saline solution, several times a day. In addition to this the vagina may be packed with cotton saturated with weak solutions of potassium permanganate, bichloride, or one of the various silver salts.

Strong applications, such as 2 to 5 per cent. silver nitrate or protargol solutions, or 10 to 20 per cent. solutions of argyrol, are often used advantageouly after irrigating, and may be either daubed on with a swab or instilled in small quantities into the vagina. A vulvar pad should be worn constantly, and changed as frequently as the amount of discharge necessitates, and whenever the underclothing is soiled, fresh linen should be put on lest the child be reinfected by the soiled garments.

Care should be taken that the child's hands do not become contaminated, and the danger of infecting the eyes should be explained to the nurses and attendants, who should be instructed to burn all pads and dressings and disinfect all soiled clothing before having it laundered. At the best, recovery is a slow and tedious process, and relapses are common; and if a case is unusually protracted vaccine treatment is sometimes resorted to with excellent results.

Stock vaccines are quite as effective as autogenous, and the dose should be gradually increased from an initial injection of fifty million up to one or two hundred million within a week. If the facilities are at hand it is always advisable to determine the opsonic index before and after a dose of the vaccine, in order to calculate the exact effect of the treatment; although no untoward effects have ever followed the administration of these vaccines in this manner.

Because of the poor general health of these children, they improve much more quickly if, in addition to local treatment, they are removed to the seashore or country, and given a good nutritious diet. The general constitutional condition should be built up by the administration of tonics such as the syrup of ferrous iodide, or cod-liver oil.

GANGRENE OF THE VULVA.

Gangrene of the vulva, or noma vulvæ, is a condition analogous to cancrum oris, and differs from that disease only as to the site of the lesion. It usually occurs in greatly debilitated children, following one of the acute infections, commonly measles or diphtheria, or may arise during the course of enteric fever or dysentery. It never occurs primarily except where great injury has been done to the parts, as in severe crushing or by the continued application of strong alkalies or acids.

A swelling of one of the labia is usually the first symptom of approaching gangrene, after which the part becomes indurated and finally breaks down, forming a foul-smelling, sloughing ulcer, which spreads rapidly, invading the surrounding tissues. The prognosis is nearly always hopeless, because of the already weakened condition of the patient, and the rapidity with which the gangrene spreads through the devitalized tissues, and affects the whole constitution by the absorption of toxins.

Treatment.—Wide excision offers the best chance of checking the disease, but is not always possible because of the close proximity

of important structures. Cauterization is sometimes followed by diminution in the severity of the gangrenous process, and may enable the physician to get it under control. The parts should be cleansed ever so often, and the child's strength should be supported by an abundance of nourishing food and stimulants at frequent intervals.

VICARIOUS MENSTRUATION.

Vicarious menstruation is exceedingly rare in children, but occurs with sufficient frequency to demand recognition. The cause is unknown. In many instances there is precocious development of the genitalia or of the whole body, and, although vicarious menstruation, in itself, is not particularly harmful unless the hemorrhage is severe, the resultant anemia from, for example, a periodical epistaxis every three or four weeks, lasting for from two to five days, would be quite serious in delicate, tubercular, or syphilitic children.

Treatment.—There is no known treatment which has any influence on the occurrence of the hemorrhages of vicarious menstruation, therefore attention should be directed to keeping up the child's nutrition and combating the anemia. The diet should be carefully regulated, so that the amount of nutrition received is the maximum, with a minimum effect on the gastro-intestinal tract.

Iron is, perhaps, the one best drug for these children, and may be given as the syrup of ferrous iodide in 10-drop doses, three times a day, to a child of five years, or one of the various easily assimilable preparations of iron may be included in a tonic mixture. Where the opportunity presents itself, these children should spend a few months each year at the seashore or in the mountains.

MENSTRUATION PRECOX.

Menstruation precox is very rare in early childhood, but it is not an uncommon occurrence in the United States for girls of eight and ten years to menstruate regularly. In these latter instances, the subjects are either very strong and healthy or very delicate, but very young children who menstruate are usually overdeveloped, mentally and bodily.

Many of the delicate children who menstruate too early have syphilis or tuberculosis, which may be responsible indirectly for the condition, because of its effect on the constitution.

Symptoms.—As in adults, the periods may last from one to five days, but they are much more irregular as to the time of their occurrence, and in some instances, after one or two periods, menstruation will cease till puberty. During the periods the symptoms resemble those in adult life, and the child is restless, nervous, and has colicky pains in the abdomen, slight fever, swelling of the mammæ, shows a change of disposition, and even a certain amount of sexual excitement.

When the period is missed there are also sensations of discomfort

and other signs like those in the adult. Before one may definitely state that a given case is one of menstruation precox, bleeding from the genitalia from other causes, such as masturbation and severe vaginitis, must be excluded, and the periodicity of the vaginal discharge of blood must be definitely established.

Treatment.—A child who menstruates several years before it should normally had better be kept in bed during each period. An ice-bag sometimes gives relief to pain if placed over the uterus, but cooling drinks are apt to cause pain, while warm liquids are often very soothing.

If bleeding be profuse, the fluidextract of ergot may be given in 10-drop doses three times a day to a child of five years; stypticin, grain $\frac{1}{6}$ to $\frac{1}{8}$ three times a day, may be used, the dose of this drug varying according to the age of the child and degree of hemorrhage; hydrastin may be given in the form of hydrastin hydrochlorate, in doses of from $\frac{1}{25}$ to $\frac{1}{10}$ of a grain.

MASTURBATION.

Masturbation is not an uncommon practice during childhood and is also seen quite frequently during infancy, although rarely before the first year. In older children this practice is much more common in boys, but from my observations of this condition in infancy I believe that it occurs in female infants much more frequently than in males. Thigh rubbing, and not manual manipulation of the genitalia, is invariably the method of exciting the orgasm in infantile masturbation, and for this reason it has been regarded as pseudomasturbation by some authors.

Etiology.—Thigh friction during infancy is usually begun because of some irritation of the genitalia or buttocks, which has induced rubbing of the parts to allay the itching. Lack of cleanliness of the thighs, buttocks, and genitalia is, perhaps, the most important factor which incites the habit of masturbation, for, as a result of accumulations of secretions and excretions, a balanitis or vulvitis of a mild degree exists, which is often the exciting cause. Seatworms, highly acid urine, eczema of the adjacent skin, and even too tight clothing are many times responsible for the habit.

There can be no doubt that the general physical condition of these children plays an important part in determining the establishment of the act of masturbation, and in many cases one may obtain a definite history of inherited neurotic tendencies, which have been fostered and aggravated by the child's environment and lack of care.

In rare instances it occurs in children where no local or general cause can be found, and under these circumstances one should always suspect and look for other signs of mental deficiency. In children of seven years and over who masturbate we must consider other factors which are in no way related to this habit during infancy, for at this age not uncommonly true sexual feeling is incorporated in

the act, especially when it has developed because of surroundings of immorality.

I am convinced that in but few cases of masturbation in older children has the habit been contracted accidentally, as in washing the genitalia, scratching to allay irritation, or in climbing; and that imitation of the act by younger children who see their more mature playmates perform it, and initiation of children to this habit by nurse girls and attendants, are its two most frequent causes at this age.

Symptoms.—Infants usually masturbate by holding the thighs close together and moving the whole body so as to produce friction; sometimes an infant will be seen to perform a series of backward and forward and side-to-side movements while lying on the back, and still others lie on the abdomen and rub the genitalia on a pillow or the bed.

The variety of methods adopted is endless, but in each case the effect produced is the same; the child's exertions become more marked with each movement and nervous excitation increases, the face becomes flushed and covered with perspiration; at the height of the act a few grunts may be emitted, after which the movements cease and the child falls back exhausted and pallid.

Rarely are any ill-effects noticeable in infants addicted to masturbation, although the already unstable condition of the nervous system is no doubt aggravated by the frequent excitation and stimulation.

The local symptoms are vague and insignificant. There may be noticed on close inspection a slight redness of the parts, and the prepuce or the nymphæ may be swollen.

The occurrence of frequent erections is a very suggestive sign of masturbation and I have found this symptom fairly constant in a number of the cases under my observation. After the fifth year the habit is usually practised manually, and these children often exhibit the so-called "tell-tale" signs of masturbation, such as sunken eyes, with dilated pupils and dark rings around them, palpitation of the heart, headache, and constant fatigue, but in most instances these are merely symptoms of neurasthenia.

When practised persistently, and to great excess after infancy, masturbation is highly suggestive of imbecility, or at least a certain amount of mental deficiency, and in these cases the child's physical condition may become greatly impaired; but too many dire effects have been attributed as results of this habit, for if carried on to a mild degree, unless one catches the child in the act, it may escape the notice of both parents and physician, so slight are the signs and symptoms.

These children are usually languid and shy to a greater or lesser extent, showing little or no disposition to play the usual games of childhood, and are often backward in school, peevish, and irritable, By far the greatest harm done by masturbation is the effect on the moral nature of the child who practises it and is conscious of wrong-doing, for from then on it is the mind which suffers and not the body.

Moral degeneration in one child just at the imitative age also results quickly in spreading the habit, and the harmful effects on his or her associates are far-reaching and unlimited. As soon as the child who masturbates comes to the realization that the act is wrong, it naturally practises it in secret, which produces that air of seclusiveness and the guilty expression so characteristic of the masturbator.

Prognosis.—The prognosis is favorable, but the duration of the habit depends greatly on the age of the child, and to a lesser extent upon the length of time it has been practised, and upon the child's mental and physical condition. In infancy spontaneous recovery usually takes place before the second year, but the duration of the habit may be appreciably shortened by appropriate treatment. Older children are not so amenable to treatment and some of these cases are most obstinate and persistent in spite of rigid discipline and thorough care, but unless great mental deficiency or imbecility is to be reckoned with, no case should be regarded as intractable.

Treatment.—Prophylaxis with regard to masturbation is very important, for prevention of the habit is far easier than the cure. Of prime importance is careful attention to cleanliness of the genitalia and avoidance of all sources of irritation to the parts. Male infants should have the glans exposed and cleansed each day at the time the bath is given, and if phimosis is present, or the prepuce is greatly elongated, circumcision is advisable.

In little girls the same amount of care and attention is required to prevent irritation around the clitoris. Older children should be trained never to touch the genitalia and should be carefully watched to see that these instructions are carried out. Masturbation is usually practised just after the child is put to bed, or before arising in the morning, and if the parents are told this they may be able to restrain the habit to a great extent themselves, by not allowing to child to lie awake alone before it goes to sleep or just after it awakens.

Children must never be allowed to sleep together when visiting each other, and even within the family circle the juvenile members should each have a separate bed wherever possible. Parents should investigate carefully the morals of their children's playmates, and when children attain the age of reason and understanding, explain to them the whole subject of sex hygiene rather than have them accept the perverted and misconstrued conception of these matters which they are sure to learn from their associates.

When the habit is once established in infancy there are two lines of procedure which must be followed out in order to break it up— removal of the cause, and forcible restraint to interrupt the practice immediately. In many instances removal of the cause is sufficient in itself to effect a cure, but the child should be watched closely for some time afterward and any suspicious movements should be discouraged. The method of restraining the infant from masturbation depends, of course, upon the particular way in which the act is performed.

If possible, a nurse should be detailed upon each case to watch the child continually and forcibly prevent the accomplishment of the act by taking the child up out of bed and holding it each time the movements begin. Since it is very inconvenient and often impossible to have a child under direct observation for twenty-four hours each day, mechanical appliances are employed, which many times are practical and of great service. These appliances may be improvised at home or purchased from any surgical instrument maker, and for the most part comprise triangular splints which are placed between the legs and adjusted so that the base of the triangle keeps the knees apart, thus preventing thigh friction, and knee braces have also been devised for the same purpose.

I have many times obtained excellent results by instructing the nurse to fasten one foot to one corner of the crib and the other foot to the opposite corner and keep the thighs separated in this manner. If manual masturbation is practised, the hands may be incapacitated by having the child wear a sleeveless gown or tying each hand to opposite sides of the crib.

In older children, forcible restraint is a much more difficult matter, and if punishment is given for each offense there quickly develops a tendency to perform the act in secret which renders these cases all the more difficult to treat. Moral suasion is often very effectual where secret indulgence is practised, if the patient's confidence is gained. Those in attendance upon such a case should employ the most tactful and diplomatic methods, thereby impressing the patient with their desire to help in breaking the habit rather than to accuse him of a shameful practice continually.

Moral cleanliness should be pointed out to these children as a virtue to be sought for and attained, and an attempt should be made to substitute healthy outdoor sports for the lewd, sexual excitement aroused in the mind of a child who is the victim of the habit.

I have rarely seen these children benefited by the administration of drugs and do not advise their use, since the amount of any drug sufficient to inhibit the act is often so great as to make the child stuporous. In infancy, however, the administration three times a day of bromide of sodium in 2-grain doses, or tincture of belladonna in 1-drop doses, will sometimes quiet the patient and aid materially in the general treatment of the case. The child who masturbates should be treated physically as well as trained mentally.

A change of climate, associations, playmates, nurses, and environment is of tremendous advantage when possible, since it supplies new material for the child's attention and engages his interest so completely that the habit may be unconsciously neglected. The diet should be carefully regulated so that an increased amount of nutrition is afforded without undue tax upon the digestive system. Medicinal tonics such as arsenic, cod-liver oil, or various preparations of iron are undoubtedly of value, but may be disregarded entirely in treatment if cold bathing and plenty of outdoor exercise are indulged in.

CHAPTER XX.

DISEASES OF THE SKIN.

In no other organ of the body is a healthful condition during childhood of greater importance than in the skin, which is intimately concerned in two vital processes—excretion and heat radiation. While the proper functionating of the skin is so highly necessary to the child, yet the skin is at the same time extremely tender and delicate, therefore much more susceptible to disease than in the adult. It is very sensitive in its response to systemic conditions, and normal, healthy skin in an infant is an indication of the efficiency of the other vital functions of the body.

Infection of an infant's skin may occur without any appreciable break in its continuity; mere stretching will traumatize it. Neglect of the skin, especially during infancy, is quickly followed by inflammation and infection. Clothing, if excessive, will cause sudamina, and even eczema. Flannel and wool are irritating, and should not be worn next to the skin. Excretions, such as sweat, urine, and feces, if allowed to remain in contact with the skin, quickly give rise to inflammation, therefore special attention should be given to the cleanliness of the infant. Bathing very young children, at least twice daily, is necessary to secure the proper function and healthy condition of the skin.

The common lesions of the skin during childhood are lichen, eczema, impetigo, ecthyma, furuncular eruptions, herpes, and erythema. Less frequently we see psoriasis, tinea, alopecia, and molluscum contagiosum. Occasionally, cases of pemphigus, keloid, and erysipelas are observed. In infancy, congenital disorders, nevi, ichthyosis, sclerema neonatorum, eczema, and, particularly, pustular infections appear. At puberty, acne, seborrheic dermatitis, and other disturbances of the sebaceous glands occur. Inflammations of the skin, because of its delicate structure, are, in younger children, usually acute in type.

ECZEMA.

The usual types of eczema in children are the vesicular and pustular forms, or a combination of the two. Although eczema is of an acute type, its course is frequently chronic, and it may last several years.

Etiology.—The most common causes of eczema in infants and young children are soap, hard water, rough clothing, and pathological secretions. The presence of toxins in the blood, due to deficient elimination, imperfect metabolism and assimilation of food, caused by improper

or irregular feeding, are important systemic causes. Local infection of the skin by pyogenic organisms is more common during childhood than at any other time of life, and may produce eczema.

In children eczema most frequently involves the face, head, ears, the creases of the neck, axilla, groin, serotal, and anal regions. It is often associated with scabies and pediculi, as rubbing and scratching because of itching from any cause may give rise to eczema. More than one-third of the cases of eczema seen in children occur during the first year, and most of these before the fifth month.

Symptoms.—Itching is the chief subjective symptom, and the one most difficult to relieve. The scratching which follows results in the rapid spread of the disease and the formation of thick crusts composed of blood and pus. The skin surrounding the encrusted area is reddened, thickened, and inflamed, with scaling at the margins.

In children pediculi are associated with eczema of the scalp more frequently than in infants, and they tend to aggravate the condition by increasing the severity of the itching.

Diagnosis.—The diagnosis of typical cases of eczema in children is easy, but it must be differentiated from erysipelas, scabies, psoriasis, impetigo, and syphilis. Erysipelas is distinguished by its rapidly spreading margin and by the high fever which accompanies it.

Scabies may occur with eczema, but should not be confused with it, the distribution being different. The itching is worse at night. The parasites may be demonstrated in scabies, and there is usually a history of scabies in other members of the family.

Psoriasis is essentially dry, while the eczema of children is usually moist. The elbows and knees and extensor surfaces are affected in psoriasis, while the flexor surfaces are the usual sites of eczema. The silvery scales of psoriasis are characteristic, and can be demonstrated.

Syphilides show no tendency to itch, and the lesions are not as acutely inflammatory as in eczema. There are usually other symptoms and signs of syphilis in the infant or child.

Treatment.—Success in the treatment of eczema is only attained after a thorough search for, and removal of the cause, and the persistent application of measures to relieve the condition. Regulation of the diet is, perhaps, the most important factor in treatment. The bowels should be thoroughly cleaned out, and thereafter kept regular. Alkaline diuretics are also indicated to relieve the hyperacidity of the urine.

Aside from these general measures, there is no internal medication of specific value. Local treatment is directed to the relief of itching and the prevention of scratching. If the eczema be of a mild degree, a dusting powder of zinc stearate, zinc oxide, or boric acid may be used after thorough cleansing of the skin, which, however, should never be attained by the use of soap. When water is used, it should preferably be boiled first, and should contain boric acid or a solution of bran.

In more severe cases, boric acid ointment (1 dram to the ounce of petrolatum) may be daubed on the eczematous area with a piece of cotton instead of cleansing the part with water. For the relief of

itching, phenol may be added, using 5 grains to the ounce of ointment. Lotions containing zinc oxide and lime-water, or calamine lotions, are very soothing and beneficial. In cases with much moisture, a solution of silver nitrate, one-half of 1 per cent., may be used to advantage.

Subacute eczemas in children are apt to appear after infancy, and call for mild stimulation with an ointment containing resorcin, zinc oxide, and bismuth. In chronic cases, stimulation should be somewhat stronger, and for this sulphur, resorcin, creosote ointment, Lassar paste, or tar ointment is advisable.

All dressings must be firmly secured, and in infants and young children, especially, this is very difficult. In eczema of the face and scalp, a mask which fits well down over the neck is excellent, as it not only insures the continuous application of the ointment, but prevents scratching. A good plan for preventing scratching of any affected part is to put a straight splint on each arm, thus making flexion of the elbow impossible.

When eczema of the scalp is complicated by pediculosis, as is frequently the case, the crusts resulting from persistent scratching are very hard and thick, and must be removed before the application of ointments, but must be removed carefully, or the condition will be aggravated. The best method is to apply olive or cod-liver oil, containing 1 dram of phenol and 2 drams of balsam of Peru to the ounce. After several hours of soaking, the crust may be easily taken off with the aid of warm water, and this should be followed by the application of the ointment or lotion desired. The scalp should be treated with petroleum, bichloride solution, or alcohol for the removal of nits and pediculi.

The following prescriptions have been employed with excellent results:

ACUTE ECZEMA.

R—Phenolis . . . . gr. v
Calaminæ . . . . ℥j
Ung. zinci oxidi . . . . ℥vij—M.

SUBACUTE ECZEMA.

R—Acidi salicylici . . . . gr. xx
Pulv. amyli,
Pulv. zinci oxidi . . . . āā ℥ij
Petrolati . . . . ℥iv—M.

CHRONIC ECZEMA.

R—Ung. picis liquidæ . . . . ℥j
Pulv. zinci oxidi . . . . ℥ij
Ung. aquæ rosæ . . . . ℥iv—M.

URTICARIA (NETTLE RASH: HIVES).

Urticaria in children differs slightly from the form seen in adults. It is characterized by a multiform eruption of whitish, pinkish, or reddish color, which suddenly appears, and as suddenly disappears, showing a marked tendency to recurrence. This eruption is accom-

panied by painful, pricking, and tingling sensations. Papules are most abundant in those forms in which the attacks last for weeks, while wheals appear in those of short duration. In severe cases, there may be vesicles, vesicopustules, and pustules. The eruption is most intense on the trunk; but the lesions shift about, and when they appear on the face may be quite disfiguring.

Etiology.—So frequently is gastro-intestinal disorder a cause of urticaria that the latter is now considered to be a cutaneous manifestation of toxemia caused by faulty digestion and metabolism. The mere presence of indigestible food in the stomach of an infant may cause urticaria, and certain cases may be traced to intestinal parasites. Sea food, canned meats and vegetables, pastry, confectionery, and certain fruits, particularly berries, are liable to cause urticaria.

A few cases may be traced to the bites of insects, giving rise to a localized area of urticaria which quickly spreads. Sudden emotion or excitement may bring on an attack in a susceptible child. Certain drugs, among which are quinin, arsenic, the salicylates, and opium, also the serums, will produce urticaria in some individuals.

Symptoms.—The local symptoms are the severe burning and intense itching which accompany the eruption. In children, an urticarial attack may often be preceded by gastric and nervous disturbances and a rise in temperature.

Diagnosis.—The diagnosis is easy, and is sometimes made by the parents. The rapidity of the onset and appearance of the eruption, and the severe itching which accompanies it, leave little doubt as to the affection. Usually there is a history of preceding attacks.

Treatment.—The chief considerations in treatment are restriction of the diet and regulation of the feeding. An initial purge is followed by light diet, free from all irritating foods. Alkaline waters internally and alkaline baths are very beneficial.

The administration of codein is sometimes necessary to quiet the nervousness produced by the itching. The skin may be sponged with a lotion containing from 0.5 to 1 per cent. of either phenol, dilute hydrocyanic acid, or menthol. Instant relief may often be obtained by the use of the prescription below:

R—Phenolis . . . . ℥ij
    Glycerinæ . . . . f ℥j
    Aquæ . . . . q. s. f ℥xvj—M.
Sig.—Use as a spray.

ICHTHYOSIS.

Ichthyosis is a congenital disease of the skin characterized by a hardness and dryness, also by a scaliness from which it gets its name of fish-skin disease. In some cases the skin is much thickened and furrowed, and resembles a suit of armor. This severe degree of ichthyosis is rarely seen; the children who are born with the disease so well developed are generally monsters, and do not live. In the common type, ichthyosis is not incompatible with life.

Etiology.—The cause of ichthyosis is unknown.

Symptoms.—The ordinary type of ichthyosis usually appears during the second year. The skin becomes dry, wrinkled, papery, and scaly. The whole body may be involved, but the disease is more severe on the outer surface of the arms and legs, the general health remaining unaffected.

Diagnosis.—Ichthyosis has such typical characteristics that it is rarely mistaken for any other affection of the skin, the absence of inflammation being, perhaps, its most distinctive feature.

Prognosis.—There is no cure for the disease, and in authentic cases no recoveries have ever been reported. The condition of the skin may greatly improve under careful, proper, and persistent treatment.

Treatment.—Children with ichthyosis are greatly benefited by residence in a moist, warm climate, and this should always be strongly advised. Frequent bathing in alkaline waters, followed by the application of an oily substance, will prevent the skin from becoming extremely dry and rough. Almond oil, cod-liver oil, cottonseed or olive oil may be used, and to any one of these may be added salicylic acid or resorcin. No internal medication seems to be of much value, although thyroid extract is recommended by various authorities. An ointment to keep the skin moist and smooth may be prescribed as follows:

℞—Acidi salicylici	gr. xv
Glycerinæ	♏xxx
Lanolin	ℨij
Resorcin	gr. xl
Adipis benzoate	℥vj
Ung. paraffin	℥ij—M.

INTERTRIGO.

Intertrigo is a very common disease during childhood, especially in infants. It is a chafing and rubbing off of the superficial skin, which has been macerated by constant moisture of the parts. It occurs in the natural folds of the skin, and is most frequently seen on the buttocks and scrotum, in the groin, and axilla.

Etiology.—Maceration of the skin which precedes the chafing is caused by the action of highly acid urine, feces, or even perspiration. Intertrigo may be found in any parts of the body where two skin surfaces rub against each other during movement. The superficial epidermis comes away as a result of this chafing and leaves a moist, red surface, which soon becomes infected.

Symptoms.—The lesions vary in degree from mere erythema to an encrusted area of infected skin, which is actively inflamed, painful, and tender. There are usually no constitutional symptoms, the cause being local.

Diagnosis.—The diagnosis is easy, the only other skin lesion simulating it closely being cutaneous syphilides in the natural folds of the body. In a given case, however, this syphilitic eruption will usually

be found in other locations and concomitant symptoms, such as snuffles or hoarseness, may be present. A Wassermann reaction will aid materially in a doubtful case.

Treatment.—Prophylaxis is very important and also practical in intertrigo. Strict cleanliness must be maintained without the too frequent use of soap. The buttocks should be sponged off with warm water after each evacuation of the bowels, and the napkins should always be fresh and clean. The urine may be highly acid, and if allowed to remain on the skin, or, if napkins are merely dried and reapplied, the intertrigo will be aggravated.

After washing off the parts they should be dried carefully, and boric acid powder, talc, or zinc stearate should be dusted on. If there is a tendency to chafing of opposing skin surfaces, they may be kept apart by wads of absorbent cotton. The digestive system should be investigated with a view of correcting any acidity in the discharges, and the urine should be tested for hyperacidity. In the early stages intertrigo usually yields readily to the application of the powder which follows:

  ℞—Zinci oxidi pulv. . . . . ℥ss
   Camphoræ pulv. . . . . ℈iss
   Amyli pulv. . . . . ℥j—M.
  Sig.—Dusting powder.

IMPETIGO CONTAGIOSA.

Impetigo contagiosa is a highly contagious and auto-inoculable skin disease, occurring most frequently among the children of the poor. Epidemics are often observed in institutions. The disease is caused by infection of the skin with the staphylococcus. An attack lasts from ten to fourteen days, but the child may suffer with impetigo for weeks, kept up by auto-inoculation.

These lesions are most commonly seen on the face, about the nostrils and corners of the mouth, also on the hands, and may extend to any part of the body. They first appear as vesicles, which vary in size from that of a pin head to the diameter of a five cent piece, and are flattened on top. The vesicles soon become pustules, which coalesce and rupture, and result in the formation of grayish-yellow crusts. The crusts appear as though they were merely stuck on the skin, but are usually attached to the hairs and, if removed, leave a raw, bleeding surface. There is no itching, and the lesions leave no scar.

The characteristic features of impetigo contagiosa are quite distinct, and the diagnosis is made on the superficial appearance of the lesions, their distribution, the absence of itching, with evidence of auto-inoculation or a history of contagion. By these definite features it can readily be distinguished from pustular eczema, varicella, and scabies.

Treatment.—It it were not for the danger of auto-inoculation in children, the lesions could safely be allowed to heal spontaneously. They should be kept clean and healed as rapidly as possible, however,

to prevent their spreading. The crusts may first be softened by a bland oil, and then washed off with green soap and warm water. After the crusts are removed an ointment containing 15 grains of sulphur to one ounce of Lassar's paste should be applied. The general health of these children is usually poor, and should be improved by regulation of the diet, good hygiene, and the administration of tonics, such as cod-liver oil or iron. The preparation appended below has been found invaluable in the treatment of impetigo.

R—Hydrargyri ammoniata . . . . gr. xx
Petrolati . . . . ℨj—M.

FURUNCULOSIS.

Furunculosis, in which multiple boils appear all over the body, or successive crops in certain regions of the body, is quite common during childhood. The boils are the result of a deep-seated infection of the skin, most frequently by the Staphylococcus pyogenes aureus, which attacks children whose vitality is low from malnutrition or debilitating disease. It rarely occurs in the very young, but may follow prickly heat in infancy. It is quite common to see a child's scalp, as well as the rest of the body, covered with boils.

These boils vary in size, burrow deeply, are acutely inflamed, and very tender and painful. They terminate in suppuration with rupture, and the pus which is discharged may then infect other areas of skin, if there be any abrasion. Constitutional symptoms sometimes appear, and the child is restless, loses weight and strength, and may have slight fever.

Treatment.—The local treatment of the individual boil depends upon the stage of inflammation it has reached. Suppuration may often be aborted by the inunction of 10 to 20 per cent. ichthyol ointment over the inflamed area. If suppuration has taken place the boil should be lanced, and the pus gently squeezed out. The cavity may then be wiped out with carbolic acid on the end of an applicator. In using pure carbolic acid care should be taken not to injure the surrounding skin, and, unless perfect control of the patient is assured, it is wiser to use hydrogen peroxide.

Vaccine therapy is very effective both in curing and preventing attacks. Both autogenous and stock vaccines are used with equal success. In addition to these measures the child's general health should be built up by change of environment, with country or seashore life, which will do much to prevent future attacks. The diet frequently needs regulation, and tonics are also indicated.

One or two applications of the following preparation will hasten the cure of a furuncle by rapidly bringing the pus to a focus.

R—Acidi salicyli . . . . ℨij
Emp. saponis . . . . ℨij
Emp. diachyli . . . . ℨj—M.
Sig.—Spread on cotton cloth and apply over the boil.

MILIARIA.

Miliaria is a disease of infancy and childhood, which results from the clogging of the sweat ducts, and is most common during hot weather. It may, or may not, be inflammatory. The non-inflammatory form is called sudamina. The lesions consist of papules of pin-head size, surmounted by vesicles which contain pure sweat. They are most abundant on the neck, chest, abdomen, and back.

There may be a slight erythema between the papules, and there is usually itching, also a burning sensation. An attack subsides in the course of a week; but, unless precautions are taken, there may be several outbreaks during the summer. Very slight desquamation follows the rupture of the vesicles.

Treatment.—Frequent bathing, the wearing of light outer garments, and of silk and linen-mesh underwear are important prophylactic measures. Locally, boric acid solution, bran baths, or alkaline lotions are soothing. Dusting powders of boric acid, zinc stearate, or starch will dry up the vesicles. A sojourn at the seashore with sea-bathing will hasten the cure.

ERYTHEMA MULTIFORME.

Erythema multiforme is characterized by the appearance of lesions of many types, from simple erythema to papules, or even tubercles. The papular form of eruption is, perhaps, the most common in children. The process is acutely inflammatory, but, as a rule, there are no subjective symptoms. The eruption may be ushered in by a feeling of malaise or vague rheumatic pains. It disappears as suddenly as it comes, lasting usually a week or ten days.

The eruption may appear on any part of the body, but is seen most frequently on the hands, arms, feet, and legs. There is usually some intestinal derangement, auto-intoxication, or ptomain poisoning, to which it may be attributed.

Treatment.—The child should receive at the onset of the attack a course of calomel, followed by castor oil. Quinine and the salicylates should be given in small doses. A lotion containing 1 per cent. of phenol, or a dusting powder, should be used locally. The diet should be investigated in every case and carefully regulated.

SEBORRHEA.

Seborrhea is marked by the appearance of crusts composed of the secretions from overactive sweat glands, and is commonly known as the "milk crust." It appears most frequently on the scalp, but may also occur on the forehead in infants. The crust is grayish yellow in color, shows a tendency to scaling, and is firmly adherent to the skin which, however, is not inflamed. Seborrhea is a skin disease which attacks poorly nourished children, and is most common in infancy and at puberty.

Treatment.—The crusts should be softened by applications of warm olive oil or an ointment of petrolatum containing salicylic acid, 15 grains to 1 ounce. They may then be washed off with warm water and Castile soap, and an ointment containing 10 to 20 per cent. of sulphur should be rubbed into the scalp. The health is usually poor, and any existing constitutional disorders should be remedied in order to improve the child's general condition.

PSORIASIS.

Psoriasis is quite a common skin disease during childhood, and occurs in apparently healthy children. Lesions may be found on any part of the body, but are most common on the knees, elbows, trunk, scalp, and extensor surfaces of the extremities. The eruption appears in patches, which are usually symmetrical, sharply defined, and composed of papules with silvery scales at the summit. The disease is essentially dry, but if the scales be removed a few bleeding points may be found underneath them.

The course of psoriasis is generally chronic, but it sometimes undergoes spontaneous cure during the summer only to return in the fall. Occasionally there is a history of constipation, autointoxication, or of a rheumatic tendency. Psoriasis resembles seborrhea, but is more widely distributed in children, not being confined to the scalp. It is inflammatory, and the scales are pearly white, while seborrhea is non-inflammatory and the scales are yellow and greasy-looking. Squamous syphilides may look like psoriasis, but the distribution is altogether different. Eczema is usually moist during infancy, and is not sharply defined, as is psoriasis.

Treatment.—Psoriasis may clear up under treatment, but shows a persistent tendency to recur. Applications of 2 per cent. chrysarobin ointment are very beneficial after the scales have been removed by washing with warm water and Castile soap. Ointments containing ammoniated mercury, 20 grains to the ounce, or tar, 2 drams to the ounce may be used. Arsenic should be given internally after the inflammatory symptoms have subsided. X-ray treatment gives splendid results in some cases. Children with psoriasis are usually much benefited by seashore life and salt water bathing. Regulation of the diet is of prime importance. The following ointment is often of great benefit, if applied to lesions after removal of the scales:

R—Liquoris picis carbonis . . . . ℥ij
  Chrysarobin . . . . gr. x
  Hydrargyri ammoniata . . . . gr. xxx
  Adipis benzoate . . . . . : . . . q. s. ad. ℥ij—M.

TINEA.

The two most common vegetable parasites which infest children are tinea tonsurans, or ringworm of the scalp, and tinea circinata, or

ringworm of the body. Favus is so very rare that it barely deserves mention here. Infants are rarely affected by tinea, but it is common in older children.

TINEA TONSURANS.

Ringworm of the scalp occurs especially in children. It is highly contagious, and epidemics are not infrequent in schools and institutions where children are closely congregated. The spores of this parasite are found on the hair shafts, and are much like the roe of fish in appearance. They are smaller than the spores of the parasite which causes ringworm on the body.
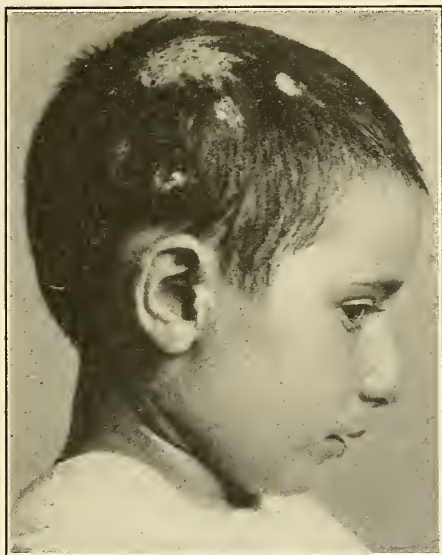
Fig. 60.—Ringworm of the scalp. Tinea tonsurans.

Symptoms.—The eruption may be preceded by itching and swelling of the scalp. The lesions are reddish, greenish, or grayish-yellow elevated patches, which appear on the scalp and cause the hair on the head to fall out. The underlying scalp may become inflamed, and exude a yellowish gelatinous material. The hair follicles become erect, and give the scalp a goose-flesh appearance. A few stumps of broken hairs are to be found on the bald areas, and the long hairs are loosened.

Diagnosis.—The diagnosis of tinea tonsurans is made by microscopic examination of the hair stumps to which a drop of liquor potassæ has been applied. The presence of the spores is pathognomonic (Fig. 60).

Treatment.—The hair of the scalp should be cut as short as possible, and the scalp then well scrubbed with tincture of green soap. After thorough cleansing, a 1 per cent. solution of bichloride of mercury, or

a 5 per cent. carbolic acid solution should be applied to the scalp for several successive nights. Tincture of iodine, painted on the scalp, is also an excellent remedy.

If there are only a few patches, these may be blistered with glacial acetic acid, after which a parasiticide should be used, and, in my experience, an ointment containing nitrate of mercury, sulphur, and phenol is most useful. Chronic cases must be treated patiently and vigorously, since they are very stubborn and resist treatment for months before they are overcome. Acute or recent cases usually respond well to treatment.

The following has been found to be a most effective ointment:

R—B. naphthol . . . . ℥j
    Ol. cadini . . . . ℥ij
    Ung. sulphur . . . . q. s. ad. ℥ij—M.

TINEA CIRCINATA.

Tinea circinata, or ringworm of the body, is a much milder disease of the skin than tinea tonsurans. It may occur on any part of the body, but is most common on the face, hands, and arms. It appears in the form of small circular spots, which spread rapidly, and several rings may coalesce. The spores in tinea circinata are larger than those found in ringworm of the scalp. There is usually an itching sensation around the site of the lesion. Successive applications of tincture of iodine or glacial acetic acid bring about rapid recovery in a few days. The preparations given below may be also used with equally good results:

R—Hydrarg. ammoniata . . . . gr. v
    Adipis . . . . ℥j—M.
    Sig.—For very young children.

Or

R—Resorcini . . . . gr. xx
    Sulphur præcip.,
    Zinci oxide . . . . āā ℥j
    Petrolati . . . . q. s. ad. ℥ij—M.
    Sig.—For ringworm of the body in older children.

HERPES.

Herpes, or fever blisters, are frequently seen during childhood, usually on the face. The eruption consists of vesicles upon a reddened base and containing a clear fluid. There are four varieties of herpes: that on the face, herpes facialis; on the lips, herpes lingualis; about the genitals, herpes genitalis; and on the body, herpes zoster.

Etiology.—Quite frequently, herpes breaks out spontaneously, and some children seem predisposed to it. In the course of fevers, especially cerebrospinal meningitis, malaria, influenza, pneumonia, and tonsillitis, herpetic eruptions are quite common.

Symptoms.—Before the appearance of the vesicles there is usually a sense of itching and burning, followed by a reddened area which is

later the site of the eruption. A crop of tiny vesicles soon appears, which may later coalesce and form one large crust. The clear fluid contained in the "blister" never becomes purulent unless infected by scratching, and, if let alone, soon dries up. The attack usually lasts a week or ten days, but may be prolonged by the appearance of successive crops of vesicles.

Diagnosis.—The diagnosis of herpes is easy, but some cases may suggest eczema or impetigo. Eczema itches, while herpes burns. The formation of pus and crusts is more common in eczema than in herpes. Impetigo is essentially pustular, does not occur in single patches, as a rule, and is characterized by its contagiousness and auto-inoculability.

Treatment.—The treatment of herpes should begin with an initial purge and correction of the diet. Locally, applications of camphor, alum, stearate of zinc, or calomel are beneficial. Fowler's solution, gtt. i–ij, three times a day, is administered to children who show a disposition to recurrent outbreaks of the disease.

HERPES ZOSTER (SHINGLES).

Herpes zoster is quite different from other forms of herpes, and must be discussed separately. The eruption is on the trunk, usually the upper half, and follows the course of a nerve. As a rule, it is unilateral.

Etiology.—Exposure to cold, inflammation of the nerve trunks and ganglia, trauma, and the season of the year, particularly winter and spring, are all predisposing factors.

Symptoms.—Herpes zoster is preceded and accompanied by neuralgic pain, which may or may not be so severe as to require morphine for relief. The vesicles vary in size from that of a pin-head to a split pea, and, as a rule, do not coalesce, but dry up in a few days. Successive crops appear during the attack, which usually lasts from ten to twenty days.

Diagnosis.—Before the eruption is visible the neuralgic pains of herpes zoster may suggest pleurodynia, or pleurisy. With the appearance of a well-defined unilateral eruption, following the course of a nerve trunk, the diagnosis is easy.

Treatment.—The treatment of herpes zoster should be directed first to the eruption, which must be protected from injury by a dressing of absorbent cotton, after the application of tincture of benzoin, 2 per cent. powdered camphor, or a 10 per cent. ichthyol and collodion dressing. Pain is occasionally so severe as to call for the administration of phenacetin, heroin, or codeine. Following the attacks there may be a troublesome neuritis, which will improve with the use of the galvanic current locally, and salicylates internally.

WARTS (VERRUCÆ).

Most warts are congenital or develop soon after birth. They are composed of epithelium with a central axis of bloodvessels and

connective tissue, and vary in size from that of a split pea to the dimensions of a small tumor. Warts usually appear on the hands and faces of children at the age when they begin to crawl around and handle everything within reach. For this reason they are believed to be contagious, as well as auto-inoculable.

The two varieties most commonly met with during childhood are the ordinary wart, verruca vulgaris, and the plane wart, or verruca planus juvenilis. The common wart needs no description; it is painless, and, except for a disfigurement, causes no trouble except that it may be torn and become infected.

Plane warts are usually very numerous; they consist of soft, elevated, disk-like planes, varying in size from a pin-head to a split pea. They may be pale brown in color, or the same tint as the skin. The duration of a wart on the skin cannot be estimated; they may come and go suddenly or gradually, and may last for weeks or months.

Treatment.—A favorite method for the removal of warts is to paint them on several successive nights with salicylic collodion, 10 per cent. strength. Glacial acetic acid, containing 1 per cent. perchloride of mercury, applied repeatedly on the end of a match-stick, will eradicate a wart. If the wart has a pedicle, or stem, it may be snipped off with the scissors and cauterized, silver nitrate or tincture of iodine being then applied to the base. Plane warts may be treated with unguentum acidi salicylici, grains xv to the ounce. Good results may follow the use of small doses of magnesium sulphate three times a day. The children affected are usually undernourished, and will be benefited by a well-regulated diet and improvement in their personal hygiene.

ALOPECIA.

Congenital alopecia, or absence of hair, is very rare. Diffuse alopecia follows febrile or debilitating diseases, and a patchy alopecia may result from inflammation or disease of the scalp. These forms of alopecia are all rather uncommon, but alopecia areata is seen more frequently in childhood than in adult life.

Alopecia Areata.—In alopecia areata an irregular surface of the scalp is entirely free from hair, up to the clean-cut margins where the long hairs are found. The appearance of the skin over this spot is quite normal.

Etiology.—The exact cause of alopecia areata is unknown. It occurs with greatest frequency in dark-haired children, both sexes being affected equally. In some cases there is an element of heredity. In others there is evidently a neuropathic taint shown by a history of shock, grief, anxiety, or fright, as, for instance, from a lightning stroke.

Reflex irritability from injury to other parts of the body, defective teeth, and errors in refraction followed by severe headache, may also be responsible for this condition. Syphilis is regarded by some authorities as the most important etiological factor. Epidemics of

alopecia areata have suggested a parasitic element, but this has not been supported by the isolation of any particular organism.

Symptoms.—There are usually no symptoms preceding the appearance of an area of alopecia on the scalp. The spot appears suddenly, and the loss of hair is complete. The long hairs at the margin of the bald spot become loose, and the spot tends to grow larger, while other spots appear. The whole scalp may become involved and complete baldness result (Fig. 61).
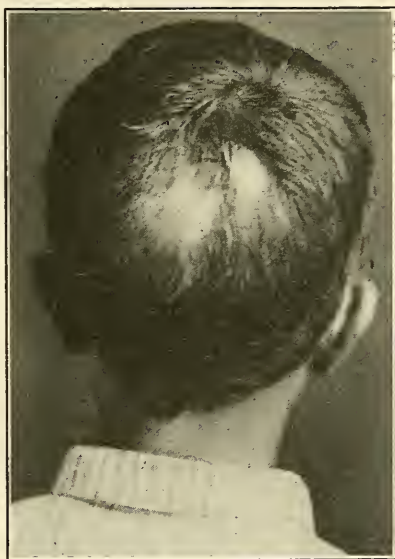
Fig. 61.—Alopecia areata.

Diagnosis.—Alopecia areata must be differentiated from ringworm; but ringworm of the scalp rarely presents the perfectly bald patches of areata, for fungi are present, and broken hairs are found over the affected areas.

Treatment.—Alopecia areata is very difficult to cure, and responds but slowly to external and internal medication. The general health of the child should be built up, the nervous system quieted, and any associated disease treated. Tonics containing cod-liver oil, strychnin, and iron should be administered. Salt-water bathing is very beneficial.

Stimulating antiparasitic remedies should be applied locally, and of these alcohol, resorcin, turpentine, cantharides, and sulphur or the mercurials are, perhaps, the best. Hot compresses, used on the affected areas daily, promote the circulation and favor the growth of new hair. No hope should be held out of cure in less than a year; but, unless the condition becomes general, recovery is to be expected.

A very serviceable prescription for alopecia areata is made up as follows:

R—Ammonii carbonatis . . . . gr. xx
  Tinct. cantharides . . . . ʒvj
  Aquæ . . . . q. s. ad. ℥iij—M.

NÆVI.

Nævi are congenital localized overgrowths of any element of the skin, and are usually associated with other developmental defects. Two forms of nævi occur in children, the pigmented nævus, or mole,

FIG. 62.—Hairy mole.

FIG. 63.—Hairy mole under treatment with CO_2 snow.

and the vascular nævus, or angioma. Nævi are found in all parts of the body, but occur most frequently on the face, neck, and back. Their etiology is obscure. Aside from disfigurement they are of little importance except that vascular nævi may be injured and bleed pro-

fusely, and moles sometimes begin to grow quickly, and become malignant.

Treatment.—Small cutaneous and subcutaneous angiomata may disappear spontaneously. Larger ones are most successfully treated with radium, or may be removed by applications of CO_2 snow, but the port-wine colored variety is very hard to efface. Electricity is sometimes employed with great success in the removal of angiomata.

Moles require no treatment unless they begin to grow rapidly, when they should be excised at once. Hairy moles are best treated with CO_2 snow (Figs. 62 and 63).

GANGRENOUS DERMATITIS.

This disease is a multiple gangrene of the skin seen during infancy, usually before the third year. It follows variola, varicella, vaccinia, and rubeola. In some cases a co-existing tuberculosis, syphilis, or rickets has been observed.

Symptoms.—After one of the exanthemata, an ulcer follows the eruption, and a slough is thrown off. If several ulcers have coalesced the tissue loss may be great. Occasionally the gangrenous points appear on normal areas of skin. The head, shoulder, and trunk are the most common sites. If the ulcers are numerous, they may cause marked constitutional symptoms. After sloughing a process of repair sets in, and a vacciniform scar is left at the site of each ulcer.

Treatment.—The general condition of the child should be supported. Local treatment consists in dressing the ulcers antiseptically, and applying deodorants to the sloughing tissue. The outlook in the majority of cases is very grave, in spite of most careful treatment.

PEMPHIGUS NEONATORUM.

Pemphigus neonatorum is the title given to impetigo contagiosa bullosa occurring in the newborn. It appears in infants both sporadically and epidemically, epidemics being more commonly seen in the obstetric wards of large hospitals. The disease is of infectious origin, and probably due to the staphylococcus aureus.

Symptoms.—The eruption commonly appears between the fifth and twentieth days of a child's life, and shows a predilection for the abdomen, inguinal region, face, and hands, but the lesions may appear on any part of the body. The eruption consists of vesicles and bullæ situated on an erythematous base. They vary in size, and contain either serous or purulent fluid which spreads the infection when the lesions burst. In mild cases there are few constitutional symptoms; when severe, the lesions are very numerous and are accompanied by diarrhea, anorexia, fever, and exhaustion.

Diagnosis.—If there are no other signs of syphilis present, the absence of lesions on the soles and palms will differentiate pemphigus neonatorum from syphilitic pemphigus.

Treatment.—Absolute cleanliness is essential, and when the involved area is large, warm baths should be given twice daily. Dusting powders of starch, boric acid, bismuth subnitrate, or zinc stearate may be used. The vesicles may be opened, and an ointment of 2 per cent. ammoniated mercury applied. Supportive treatment is indicated in severe cases, as it is the bottle-fed, cachetic baby that usually succumbs to this disease. Healthy babies generally recover in from two to three weeks, and in these infants the disease never approaches a severe degree.

DERMATITIS EXFOLIATIVA NEONATORUM.

This is also known as Ritter's disease. It is very rare, and is believed to be closely related to pemphigus. It occurs in infants between the first and fifth weeks, most commonly in foundling asylums. The cause of Ritter's disease is unknown. Its relation to pemphigus is upheld by some authorities, while others consider it to be merely an exaggeration of the normal exfoliation of the newborn. The majority of cases have been observed in boys.

Symptoms.—In some cases the disease begins with a dryness of the skin, while in others there is inflammation of the oral mucous membrane. This is followed by a diffuse redness all over the body. As the hyperemia spreads, the area which first became reddened begins to desquamate. The desquamation may be perfectly dry, or accompanied by the formation of bullæ filled with fluid. After desquamation, the skin is irritable for a few days, and then returns to normal. There is usually no fever, and uncomplicated cases show no systemic disturbances.

Delicate children who have Ritter's disease are apt to develop complications, such as furunculosis, gangrene, gastro-intestinal disturbance, or pneumonia, which usually brings about a fatal termination, but healthy children quite frequently recover. The disease shows a mortality-rate of about 50 per cent.

Diagnosis.—The differentiation between Ritter's disease and pemphigus is very difficult, since the two diseases are related. Ritter's disease is differentiated from the general exfoliation of the skin caused by syphilis by the absence of other evidences of lues.

Treatment.—Prophylaxis consists in cleanliness, and the avoidance of all local irritation. The child's state of nutrition usually needs improvement, and the warmth of the body should be maintained. Locally, an ointment containing 1 per cent. salicylic acid or phenol should be employed, or the following:

$$\text{℞—Hydrarg. ammoniata} \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad \text{ʒij}$$
$$\text{Petrolati} \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad \text{ʒij—M.}$$

MOLLUSCUM CONTAGIOSUM.

Molluscum contagiosum is a form of contagious wart within which there occurs a peculiar degeneration. The affection is much more

common in children than in adults, and epidemics are not uncommonly seen in institutions. The etiology of molluscum contagiosum is still obscure, but the theory of the parasitic nature of the disease has been universally accepted, although no definite organism has been isolated.

Symptoms.—The eruption appears as small, firm nodules, which are tense and shiny, of whitish color, and waxy appearance. There is a central umbilication from which sebaceous material may be expressed. The eruption occurs principally upon the face, neck, and arms.

Diagnosis.—Molluscum contagiosum does not resemble any other skin disease. Warts of the ordinary type may possibly be mistaken for it, but they do not commonly appear on the face, and have no central umbilication.

Treatment.—Multiple punctures of the nodules will usually cause their disappearance in a few days. If there be but few lesions, the sebaceous material may be squeezed out of each individual nodule, and a cure thus effected. If the disease is widespread over the body, applications of tincture of green soap or 5 per cent. sulphur ointment are advisable.

PARASITIC SKIN DISEASES.

Parasitic skin diseases are more common among the children of the poor, but may be seen in all classes, on account of the close contact of children at school and at play. Both animal and vegetable parasites infest the skin. Of the animal parasites, pediculi and the itch mite are most frequently encountered, while fungi which produce ringworm of the scalp and favus are the most common vegetable parasites.

Pediculosis.—Pediculosis is that condition in which the child is the host of pediculi or lice. In young children the pediculosis capitis, or head louse, is most frequently found; occasionally the crab louse, or pediculosis pubis, will be observed on the eyelids of young children. Later in childhood one finds the pediculosis corporis, or body louse, and the pediculosis pubis.

The pediculi found in the scalp, pubic hair, and body hair form respectively three distinct varieties of lice. They are very prolific, each female when three weeks old laying about fifty eggs. The eggs of the body louse are deposited in the underclothing, but the eggs of the other types are found attached to the hairs, and are known as nits.

Symptoms.—After attaching itself to the skin the parasite sucks the blood, and causes intense irritation and itching, which result in vigorous scratching on the part of the child. Many cases of eczema, especially of the scalp, have their beginning as pediculosis. In every case there is severe inflammation of the skin from the irritation and scratching, and the lymphatic glands draining the affected area may become enlarged, as is often the case with the enlargement of the postcervical glands seen in pediculosis capitis.

Diagnosis.—The diagnosis is made by identifying the pediculi, which may readily be seen under a low power glass. The nits are

visible to the naked eye. Pediculosis corporis is sometimes confused with scabies; but the distribution of the lesions differs, and no burrows are to be found in pediculosis corporis.

Treatment.—Removal of the hair from the affected regions is a great help in every case. If this be objected to, the hair must be gone over carefully with a fine-tooth comb. Kerosene is a good application, but is disagreeable, and a solution of bichloride of mercury, 1 grain to the ounce of water, is just as effective. To this may be added dilute acetic acid, which will loosen the nits, and facilitate their removal from the hairs.

If the irritation is severe, a 5 per cent. phenol solution may be applied to the scalp, or a 10 per cent. boric acid ointment rubbed in. The head should be thoroughly washed each morning, and the parasiticide reapplied until all pediculi and nits have been removed.

Pediculosis corporis may be eradicated by daily bathing, and a change of underclothing after each bath. The discarded undergarments should be soaked in a 5 per cent. carbolic solution before they are used again. An ointment containing 5 per cent. phenol or the following prescription is often beneficial:

> ℞—Olei olivæ .   .   .   .   f ℥iij
>   Olei petrolei .   .   .   .   f ℥vj
>   Balsam Peruv. .   .   .   .   f ℥j—M.
> Sig.—Apply to scalp on retiring.

SCABIES.

Scabies is a contagious skin disease due to the presence of the itch mite, or acarus scabiei, in the skin. The female buries herself in the skin, making a burrow in which she deposits eggs and feces. This produces on the skin an eruption of vesicles, pustules, and nodules. The itching is severe, and is worse at night, so that scratch marks are added to the eruption. The burrows are elevated, and grayish in color; with the aid of a magnifying glass the parasite may be seen at one end.

The distribution of the eruption is characteristic, for the mites seek moist, warm places for burrowing, therefore the lesions of scabies are most abundant in the interdigital spaces, the wrists, the flexor surfaces of the forearms, between the toes, in the under surfaces of the thighs, and on the scrotum and penis.

Diagnosis.—The diagnosis of scabies is certain when the burrows have once been demonstrated in the skin of the child. The distribution of the eruption, the history of contagion, and the itching, worse at night, are points which differentiate scabies from pediculosis or eczema. An eczema frequently appears as a result of scabies, and may tend to mask the original condition.

Treatment.—The treatment of scabies consists, for the main part, in the destruction of the itch mite, but this is difficult to accomplish without causing a dermatitis or eczema by the parasiticide used. The

first step should be to remove the clothing and bedclothes, and to sterilize them. The child should be given a warm bath, using plenty of soap, followed by a vigorous rubbing with a rough towel. The skin should then be anointed with an ointment containing balsam of Peru and sulphur, 1 dram each to the ounce of petrolatum. This ointment should be reapplied for three successive evenings following the first application, but no baths should be given during this time, and the same underclothing should be worn. After the fourth application of the parasiticide, another warm bath is given, and fresh underclothing put on.

If dermatitis or eczema results from the treatment, this should now receive attention. If not completely eradicated, a second course of treatment similar to the first will usually result in the disappearance of all evidences of scabies.

When treating scabies in very young infants, a solution of styrax, one-half ounce to the ounce of lanolin, is just as effective as the sulphur preparation, and is less liable to cause dermatitis.

R—Sulphuris . . . . ℥ij
Hydrarg. ammoniata . . . . gr. xx
Creosote . . . . gtt. xx
Lard . . . . q. s. ad. ℥iv—M.
Sig.—Apply locally.

Fig. 64.—Bromide rash.

DERMATITIS MEDICAMENTOSA.

This includes all eruptions caused by the administration of drugs. As a rule, the skin lesions appear after some days or weeks of continuous dosage; occasionally, however, they may develop after a very small amount of the medicine has been taken.

Certain drugs, especially the bromides, belladonna, the iodides, quinin, salicylic acid, mercury, arsenic, and many coal-tar derivatives, are apt to produce cutaneous eruptions. Some of these, as the bro-

mides, iodides, and belladonna, usually produce a rash, others less regularly do so.   Some children, too, are very susceptible.

The rashes caused by the administration of bromides are commonly pustular, less often furuncular and carbuncular.   There may occasionally be some superficial ulceration; very rarely papillomatous or vegetative lesions occur.   Bromide lesions do not leave any permanent disfigurement, although the rash may persist for four or five weeks after the bromides have been discontinued (Fig. 64).

Fig. 65.—Tuberculosis cutis.

TUBERCULOSIS CUTIS.

The tubercle bacillus may also produce lesions of the skin, usually suppurative or ulcerative in character.   These lesions are generally due to the extension to the skin of some underlying tuberculous process. The superficial skin is more or less destroyed, the edges are red and undermined, and the granulations thinly covered with pus.   The lesions spread slowly and cause very little pain.   Occasionally the disease assumes a papulopustular form, which is most commonly seen on the face and upper extremities.   An ulcerative papillomatous tuberculosis occasionally occurs, and is, as a rule, found on the lower leg or hand (Fig. 65).

CHAPTER XXI.

DISEASES OF THE EAR.

DISEASES of the ear are quite common in children at all ages. During infancy, otitis is usually primary, but in later child life it is commonly secondary to one of the exanthemata.

FOREIGN BODIES IN THE EAR.

The presence of foreign bodies in the ear is not usually attended with severe symptoms or grave consequences, but efforts at removal, if not skilfully directed, may do much harm. Children are in the habit of putting things in the ear, and for this reason the variety of articles which has been removed from the ears of children is large. They are divided into three groups: animate objects, such as bugs and insects—inanimate objects influenced by moisture, such as beans, onions, tea and wheat—and inanimate objects uninfluenced by moisture, such as beads, pebbles, and buttons. Insects should be killed by filling the auditory canal with warm water or whisky before an attempt is made to syringe them out.

Objects in the ear influenced by moisture may be reduced in size by instilling pure alcohol before attempting extraction. Even if the object be uninfluenced by moisture, instillation of alcohol will tend to reduce any swelling of the auditory canal and facilitate its removal. Syringing an object out of the ear is preferable to using instruments, and should be successful if the stream can be directed back of the foreign body. In some cases turning the head sidewise with the ear lowermost, and striking the head from below, may dislodge the object.

ACUTE OTITIS MEDIA.

Acute otitis media is an inflammation of the middle ear, and may be catarrhal or suppurative.

Etiology.—In severe cases of scarlet fever, diphtheria, and to a less degree in measles, especially when the throat symptoms are severe, there is a distinct tendency to inflammation of the middle ear, and there is certainly a probability of more or less inflammation of the ear in all infectious diseases of childhood. In bathing infants, water may gain entrance to the tympanic cavity through the upper segment of the drumhead, which is not always closed at birth, and this gives rise to otitis media.

The mucous membrane lining the tympanic cavity is embryonic in type during infancy, and this also predisposes to inflammation.

Cachexia and bronchitis are two important factors in the etiology of otitis media in infancy. Coughing, vomiting and sneezing may force matter through the Eustachian tubes into the middle ear and thus give rise to suppuration. Adenoids and diseased tonsils, syphilis and tuberculosis, all predispose to middle-ear disease during childhood.

It has been stated that pathogenic bacteria may be found normally in the tympanic cavity, and, therefore, any local change in the condition of the mucous membrane, such as congestion from any cause, may result in an otitis media.

Otitis media is sometimes present in the newborn, and is supposed to be caused by the forcible entrance of amniotic fluid into the middle ear during delivery.

Pathology.—The pathological changes vary from the simple catarrhal inflammation which usually accompanies catarrh of the rhinopharynx or measles to the phlegmonous form with infiltration and purulent secretion seen after scarlet fever, influenza or diphtheria. The simple catarrhal form of otitis media is characterized by swelling and clouding of the mucosa, and the production of a slight quantity of sero-pus. There is usually very slight pain and the inflammation subsides in the course of a few days with no serious results following. This form is seen most frequently during infancy.

In older children the suppurative form is most common. It is caused by the action of a great number of virulent bacteria on a devitalized mucous membrane in the tympanic cavity. The membrane is first hyperemic, then it becomes infiltrated, and there is a purulent exudate. This inflammatory process may extend to the cellular tissues above the tympanum. The Eustachian tube becomes blocked up and the tympanic membrane is finally ruptured by the force of the pent-up secretion in the tympanic cavity. Necrosis of the ossicles may result, and, by extension, the inflammation may give rise to periostitis, pachymeningitis, thrombosis of the lateral sinus, and cerebral abscess.

Bacteriology.—The organisms found most frequently in otitis media are the streptococcus, staphylococcus, and pneumococcus. The tubercle bacillus may be present in chronic cases, and the Klebs-Loeffler bacillus has been isolated in cases of otitis secondary to diphtheria.

Symptoms.—The symptoms of acute otitis media are very often variable and obscure. In infants with cachexia as an underlying cause, there may be no subjective symptoms. On inspection, the drumhead is slightly reddened and a small amount of slimy secretion may be found in the auditory canal. The nutrition is often disturbed in otitis of infancy, and the child nurses poorly. It may refuse to nurse except on the side which allows it to place the affected ear to the mother's breast. It is peevish, twists its head or drops it to one side, and cries out continually. The pain is worse at night and the fever is higher than during the day, so that the child is restless and may make sudden outcries in its sleep.

Pain and temperature are the two constant features of otitis in children. The temperature range in these cases is from 100° F. to

104° F. In some cases there is anorexia, nausea, vomiting, and marked apathy. These symptoms usually persist for a week or ten days when a purulent secretion is found in the canal. Examination of the ear drum will usually reveal the site of rupture of the tympanic membrane. The discharge of pus is followed by a drop in temperature and rapid subsidence of all symptoms. As a rule there are no complications in this form of otitis during infancy.

In older children the symptoms are more severe. The pain is intense and becomes excruciating just before rupture of the drumhead. Evidences of meningeal and labyrinthine irritation are present and include nystagmus, vomiting, convulsions, and unconsciousness. Headache and delirium are also not uncommon. A child with otitis will often complain of noises heard in the ear. It is very restless, cries incessantly and rubs or strikes the ear with its hand.

Otoscopic examination reveals the ear drum markedly congested and irregular in contour. The upper and posterior walls of the meatus are swollen. The mastoid is very tender and there is a swelling of the tissues covering the bone and extending downward along the whole side of the neck and forward to the retromaxillary fossa. The inflammation may not go on to suppuration if the symptoms are mild, but as a rule there is pus formation which causes first a bulging of the tympanic membrane, and then, as the tympanic cavity fills up with pus, the ear drum is ruptured.

The temperature in otitis media purulenta is the most constant sign and may be the only indication of a complication in one of the exanthemata. It usually reaches 102° F., but the course is irregular and many cases range from 103° F. to 105° F. until rupture of the membrane, when the temperature falls and other symptoms subside. If the drumhead be resistant so that rupture is delayed, the suppurative process may extend down the neck into the throat and cause a retropharyngeal abscess, or into the middle fossa of the skull, or to the lateral sinus.

During the course of diphtheria, measles, scarlet fever or typhoid fever, many of the symptoms of an otitis media may be easily overlooked or attributed to the primary disease, so that a routine examination of the ears should be made during the course of all exanthemata.

Diagnosis.—An early diagnosis of acute purulent otitis media, while it is apt to be very difficult if the otitis is secondary, is important, as the child's future hearing may depend upon the time that it is made. A diagnosis after rupture of the drumhead and perhaps threatened mastoiditis or necrosis of the ossicles, is, of course, a simple procedure. An otoscopic examination is always necessary, since many of the symptoms of a secondary otitis may be attributed to meningeal irritation from the primary disease.

Some cases of otitis media in infants, when the symptoms are mild, may even fail to show any positive signs of otitis on otoscopic examination. Fever may be the only manifestation, and in the absence of any signs in the ear, the diagnosis must be made by exclusion of any

39

cause for fever in the throat, lungs or gastro-intestinal tract. Older children will complain of pain or tenderness about the ear, or of deafness and noises heard in the ear.

Furunculosis or foreign bodies in the auditory canal may produce symptoms which simulate otitis media, but their presence is quickly revealed by otoscopic examination. A case of primary acute suppurative otitis media presents no difficulty in diagnosis in older children, and, if its frequency in infants during cold weather be borne in mind, it should not be overlooked in infancy.

Prognosis.—The prognosis in a catarrhal otitis media is uniformly good. The outlook in an acute purulent otitis media depends upon the early diagnosis, and presence or absence of complications. The most unfavorable cases are those following scarlet fever. The mortality rate of middle-ear disease in children is below 1 per cent.

Complications and Sequelæ.—Mastoiditis is the most frequent complication of purulent otitis media.

Meningitis.—Meningitis is more often a complication of chronic otitis media but may follow an acute attack. There are two varieties: the serous and purulent. It results from infection through the roof of the tympanum in most cases, but may be secondary to thrombosis of the lateral sinus, or rupture of a cerebral abscess. The symptoms come on suddenly and there is headache, contraction of the pupil and nystagmus toward the affected ear. The pulse is rapid, the respirations increased and projectile vomiting may occur. The patient is ataxic, the muscles of the face and extremities are drawn or contracted, and the head is retracted by the contraction of the head muscles.

As the disease progresses, delirium is followed by somnolence or apathy and loss of memory. The distinction between serous and purulent meningitis is made by examination of the spinal fluid, which shows no pus or bacteria in the former, and an abundance in the latter. The prognosis of the serous meningitis is good, while the purulent form is almost invariably fatal.

Brain Abscess.—Middle-ear disease is the most frequent cause of abscess of the brain, but it usually follows chronic otitis media. The infection gains access through the bone, veins or dura mater, and in most cases the abscess is in the temporosphenoidal lobe. The symptoms are pain, vomiting, subnormal temperature, slow pulse, constipation and retention of urine. The child is usually apathetic. Localizing symptoms depend upon the site of the abscess. The prognosis of brain abscess is, as a rule, bad, but early operation may often result in recovery.

Septic Sinus Thrombosis.—This complication is caused by the occlusion of the lateral sinus by an infected clot, due to a loss of integrity of the intima of the sinus. It usually follows mastoiditis or labyrinthine inflammation. The symptoms are severe. There is a widely fluctuating temperature with chills, violent headache and a marked tenderness over the posterior triangle of the neck and jugular vein. Vomiting is a frequent occurrence and convulsions are not unusual.

The child grows apathetic and stuporous and coma precedes death which usually occurs in twenty-four to forty-eight hours.

Facial Paralysis.—Facial paralysis is more frequently a complication of chronic otitis media, and is due to an extension of the inflammatory process from the bone to the seventh nerve where it passes through the canal.

Labyrinthitis.—Labyrinthitis may be either suppurative or non-suppurative. It is rarely seen as a complication of otitis media where treatment has been prompt and thorough.

Treatment of Otitis Media.—Because of the dire results which sometimes follow an acute attack of otitis media in children, prophylaxis is an extremely important part of the treatment. Tonsils and adenoids, as common predisposing factors, should be removed. During the course of the exanthemata and other acute febrile diseases, the nasopharynx should receive special attention and careful cleansing. Warm salt water or liquid albolene should be instilled into the nose twice daily with a dropper; the use of nasal douches or syringes should be avoided. The ears should be syringed daily with warm boric acid solution.

A mild attack of acute catarrhal otitis media will often subside if the ear be carefully irrigated with saline solution at a temperature of 110° F. The child should be put to bed, the diet restricted, and a thorough purge administered. Local blood-letting may abort suppuration in a child, but should never be practised in an infant. The artificial leech is usually employed.

Hot applications are soothing for the pain, and dry heat is preferable to wet heat. The best results are obtained from the use of rays of a high-candle-power electric light (200 to 300 c. p.), but a hot-water bag or a bag of salt heated may serve the purpose. It may be necessary to instil a 4 per cent. cocaine or 5 per cent. phenol solution into the ear for the pain, but the mother should be warned against dropping oil of any kind into the auditory canal.

Frequent otoscopic examinations should be made if a child has acute otitis, and as soon as suppuration is indicated the drumhead should be incised. Myringotomy is such an important measure in treatment that it should be done if there is the slightest suspicion of pus, for in many cases where the drumhead is incised and no pus obtained there has been instant amelioration of symptoms without pus formation. The incision should be long and curved to allow the discharge to flow through it. The after-treatment may be either wet or dry.

The dry method consists in wiping out the exudate from the external auditory canal and the use of silver nitrate, 3 to 5 grains to the ounce, as an application in the tympanic cavity. The ear may then be dusted with equal parts of boric acid and zinc oxide. The wet method is carried out by repeated syringing of the ear with hot salt solution or copper or zinc sulphate, gr. viii to the ounce of water.

If the discharge lessens and the temperature persists, the drainage

is imperfect and a secondary incision may be necessary. If the discharge persists, a few drops of 2 per cent. silver nitrate should be dropped into the tympanic cavity, or the tympanic membrane may be cauterized with trichloracetic acid. A fetid discharge may be alleviated by the use of hydrogen peroxide or compound tincture of benzoin in a syringe. An aurist should be consulted if the discharge does not cease within a month. The presence of adenoids and other conditions in the nasopharynx also has a tendency to prolong an attack of otitis, and these conditions should be corrected as soon as possible.

The use of vaccines has been the subject of much discussion recently, and my experience with them has been very satisfactory. Autogenous vaccines are made by suspending dead bacteria in normal salt solution after a culture has been obtained from the pus. In this way the specific organism is isolated and used after subjecting it to a temperature of 60° C. for forty-five minutes. Because of the reaction it produces, vaccine therapy is contra-indicated in cases where there is a debilitated condition of the patient, or if the child be in a state of profound sepsis.

Subacute and chronic cases are benefited most by the administration of vaccines, for the child is, as a rule, in better physical condition than during acute attacks. Cases which respond to vaccine treatment undergo rapid recovery and complications of otitis are rarely seen. A marked improvement is often observed when vaccines are given following operation for any of the various complications of otitis. It is usually administered a week or so after operation.

If vaccine therapy is decided upon in a case of otitis, injections should be given in increasing doses every three or four days, but as soon as reaction symptoms, such as a feeling of exhaustion, etc., are noticed, the injections should be discontinued for a week or so until the vital forces are reëstablished.

Stock vaccines, which may be had from the biological departments of the large drug firms, do not give as uniformly good results as the autogenous vaccines, because they do not contain the same strains of organism which the patient must react against. The antibodies which are formed following their injection are therefore not exactly suited to counteract the specific bacteria and endotoxin present.

In many cases they have the objectionable feature of devitalizing the patient with no beneficial results in return. The leukocyte extract of Hiss may be given with safety in those cases of debility and profound sepsis where the use of vaccines is contraindicated. It contains the elements of the leukocyte necessary to combat the bacteria and endotoxin, and its administration in no way exerts any influence on the vital forces.

CHRONIC SUPPURATIVE OTITIS MEDIA.

Inflammation of the middle ear which has become chronic is seen quite frequently during childhood. It is usually characterized by a

fetid discharge and an absence of subjective symptoms except during acute exacerbations, which are sometimes quite frequent.

Etiology.—Chronic suppurative otitis media usually follows scarlet fever, measles or influenza, and is also frequently seen after diphtheria and typhoid fever. Occasionally a case will develop in certain cachectic conditions without previous acute phenomena, but, as a rule, there is a history of an acute purulent otitis media with spontaneous rupture of the drumhead.

On otoscopic examination, there is generally found a state of imperfect drainage caused by the location of the perforation of the drumhead or the size of the aperture. Catarrhal conditions in the nasopharynx and the presence of adenoids are also predisposing factors. It usually develops in weakly, anemic or tubercular children, and tuberculous otitis is chronic from the onset. Chronic otitis involves the tympanic membrane, external meatus, bony walls of the middle ear, and sometimes the labyrinth.

Symptoms.—Persistent discharge of pus is perhaps the most constant symptom. In some cases it is very slight, but can usually be found on otoscopic examination. In neglected cases it becomes fetid and may also be blood streaked due to ulceration, in other cases the discharge is more like mucus than pus.

There may be headache or fulness in the head, and often the child may complain of noises in the ear. Pain is usually due to ulceration or pus retention. Acute exacerbations, in which there is pronounced pain, mastoid tenderness and elevation of temperature, are of frequent occurrence.

Prognosis.—The future hearing of the child, with a chronic purulent otitis media, depends upon the site of the perforation and the extent of involvement. If there is a central perforation, the hearing may be but slightly affected, but if there is marginal perforation with bone necrosis, there may be serious impairment of hearing.

Treatment.—The treatment of chronic otitis media in children should be conservative. If drainage is poor because of a small perforation in the tympanic membrane, an incision should be made and the opening enlarged. The ear should be syringed with bichloride solution, 1 to 10,000, or hydrogen peroxide, saturated solution of boric acid, or normal salt solution, if the wet method is to be followed.

The dry method consists in wiping out the secretion, the instillation of an astringent, and the application of a powder which is blown into the ear. Silver nitrate 3 to 10 per cent., alcohol 50 per cent., and argyrol are largely used in the middle ear, and in some cases the cautery may be necessary for polypi and granulations. Boric acid, aristol and iodoform are used as dusting powders, but they must be finely pulverized, and care should be taken that one does not put enough into the canal to block drainage.

If there is bone necrosis or a large number of granulations, operation is necessary to effect a cure and prevent intracranial complications or mastoiditis. If the child has adenoids, they should be removed and other nasopharyngeal defects remedied.

Autogenous vaccines are especially to be recommended in chronic suppurative otitis media, as very many excellent results have followed their administration. In addition to these measures for treatment of chronic middle-ear disease, attention should always be directed to the general health of the child. Frequently the patient is found to be in poor physical condition—anemic, rheumatic or syphilitic, and proper treatment of these conditions will aid materially in the subsidence of the ear trouble.

ACUTE MASTOIDITIS.

Acute infection of the mastoid process in children is practically always secondary to a suppurative otitis media. The occurrence of primary mastoiditis as a result of cold, or trauma, or furunculosis of the external auditory canal posteriorly, is so rare as to be disregarded in the discussion of mastoiditis during childhood.

In children, probably all suppurative middle-ear infections also involve the mastoid cells, and it is difficult to separate suppuration of the middle ear from that of the mastoid. If, however, a case of otitis media presents no symptoms of mastoiditis, it is regarded as a simple case of otitis media.

Etiology.—The ease with which pus may enter the mastoid antrum causes mastoiditis to be a frequent complication of acute purulent otitis media in children. The external bony wall is thinner than in adult life, the petrosquamous suture, which is persistent in some cases, passes through a foramen on the inside of the skull and appears externally behind the glenoid fossa and tympanic ring, and pus may also find its way externally more readily through the open fissura mastoidea squamosa.

In addition to this, the Eustachian tube is short and has a wide calibre, which invites infection through this source. Involvement of the mastoid is much more apt to occur where there is not sufficient drainage or when incision of the drumhead has been too long delayed in an attack of purulent otitis media. As a general rule, streptococcal infection is most liable to give rise to mastoiditis. Mastoiditis has been observed as early as the second month, but most cases are seen after the second year.

Symptoms.—The symptoms of mastoiditis are both local and general. Those referable to the mastoid are due to pressure from retention of secretion within the cells. Pain is severe only when the cortex is thin, with more or less necrosis due to otitis and periostitis. Redness, tenderness on pressure and swelling over the mastoid process are present in almost every case. Otorrhea is usually increased and, in the absence of other signs of mastoiditis, a profuse discharge is indicative of mastoid involvement.

Otoscopic examination reveals a bulging or drooping of the postero-superior wall of the osseous canal. There is usually a small central perforation in the drumhead and, in some cases, granulations may

be observed protruding from this aperature and obstructing the drainage. Of the general symptoms of mastoiditis, elevation of temperature is the most constant. It ranges from 99.5° to 105° F. The pulse and respiratory rates are increased correspondingly with the elevation of temperature. The child is restless at night and may cry out while asleep. During the day it is cross and irritable. Occasionally there is vertigo, vomiting and a convulsion at the onset of the disease.

Diagnosis.—An early diagnosis of acute mastoiditis in children not only prevents the occurrence of complications, but may save the life of a child, for delay in operation always subjects the patient to certain dangers. The diagnosis is made chiefly on the clinical symptoms, physical signs, and the findings on otoscopic examination.

Symptoms, while important, are not alike in all cases, and for this reason careful examination is sometimes necessary to detect mastoid inflammation. A continuous discharge from the middle ear for over four weeks, with fever and impaired hearing, strongly suggests an abscess of the mastoid. One of the most important diagnostic signs is the drooping of the posterosuperior wall of the osseous canal. The x-ray and transillumination are sometimes used as an aid to diagnosis.

Acute mastoiditis must be differentiated from furunculosis of the external auditory canal and scalp, from erysipelas of the auricular region and from lymphangitis of the scalp. Furunculosis of the external auditory canal may give rise to many of the local signs of mastoiditis, including pain, swelling, tenderness and edema over the mastoid. An otoscopic examination quickly reveals the site of the furuncle, and shows a normal tympanic membrane and osseous canal. Hearing is affected in furunculosis only when there is mechanical obstruction by the furuncle. The temperature is rarely elevated.

Erysipelas, involving the region of the auricle, may be accompanied by symptoms suggesting mastoiditis, but careful inspection will usually reveal the nature of this disease, and otoscopic examination shows a normal tympanum and osseous canal. In children with pediculosis capitis, a lymphangitis may occur which will involve the mastoid lymph glands, and in some cases simulate mastoiditis.

The differentiation between this affection and mastoiditis is based upon the absence of any ear condition on otoscopic examination, and the finding of the pediculi and the bilateral involvement. An important differential feature in all of these conditions is the absence of any previous history of ear disease (except rarely as a coincidence).

Complications.—The complications of acute mastoiditis are Bezold's mastoiditis, caries and necrosis of bone extending in any direction, facial paralysis and brain abscess.

Treatment.—The most important prophylactic measure against mastoiditis is early incision of the drumhead in acute purulent otitis media. In otitis following influenza, typhoid fever or pneumonia, the drumhead should be incised immediately, not waiting for bulging. When a case of acute mastoiditis is seen before spontaneous perfora-

tion of the tympanic membrane has taken place, the drumhead should be freely incised at the point of greatest bulging. This relieves pressure and thus retards necrosis.

The child with mastoiditis should be put to bed and be given a course of calomel and a dose of aconite. If the case is seen at the onset, an ice-bag may be placed over the mastoid for twelve to twenty-four hours, but once removed, should not be reapplied. Dry heat obtained by the use of the hot-water bag is often preferable for the relief of pain, and in some cases an opiate must be given. Leeching is of great value, but should not be used in infants and is difficult to perform in children.

In every case it is most desirable to obtain a reaction to inflammation, and this may be promoted sometimes by hot irrigations, using a 1 to 5000 bichloride solution. Bier's hyperemia has been resorted to with this object in view, but authorities disagree as to its efficiency, and some writers claim that the disease is found more widespread after its use. The leukodescent light may be mentioned as another means of securing a reaction to inflammation, but its use is by no means universal.

The treatment outlined so far should always be carried out in an endeavor to abort a threatened mastoiditis, but once it becomes apparent that the acute symptoms are not modified or the infection eradicated, operation should be advised without further delay.

If performed early, a mastoid operation is not formidable and the results are good, but a delay of a few days or a week may make a radical operation necessary because of the extent of involvement. In advanced tuberculosis, general debility or bilateral involvement in bottle-fed babies, however, operation should be attempted only if life is endangered by the virulence of the infection.

THE SPECIFIC INFECTIOUS DISEASES.

TYPHOID FEVER.

Typhoid fever is an acute general infection caused by Eberth's bacillus, the bacillus typhosus. It occurs during intra-uterine life, may exist in the newborn, is comparatively rare in infancy, and more common in childhood. It is generally contracted by drinking water which contains typhoid bacilli, by the use of infected ice, by drinking milk which has been diluted with contaminated water, by using dishes, cans, or other utensils which have been washed with such water, and by eating uncooked vegetables, such as lettuce, celery, and water cress, also oysters. In an infected locality the only safety lies in boiling for five minutes all water used for drinking and other household purposes. With infants and children, the water used in the daily bath may be a source of infection owing to their habit of splashing it about and putting their wet fingers in their mouths.

Etiology.—Typhoid bacilli are present in the feces and urine of all typhoid fever patients, and the prevalence of typhoid fever in any locality points unmistakably to some imperfection in the disposal of sewage in that district. It may be carried by flies or other insects, or by dust containing dried human excreta.

While the disease is not contagious, and cannot be spread by personal contact, still it is true that a child ill with typhoid fever should be regarded as a source of infection to all those with whom he comes in contact, and all bed linen, articles of clothing, dishes, etc., may be soiled by the fecal or urinary discharges or by the sputum. These should be soaked in a 1 to 20 carbolic solution and boiled. The hands of all attendants should be dipped in a creolin solution and washed often, especially before eating.

The disease is more common in temperate climates, and in the autumn months. During infancy both sexes are equally affected; in childhood boys are more exposed, consequently more cases occur among them than in girls.

The typhoid bacilli are found especially in Peyer's patches, the mesenteric glands, and the solitary follicles of the intestines, also in the liver, spleen, bile, bone-marrow, and blood, as well as in the rose-colored rash. In the first few days of the disease they can be demonstrated in the stools by cultural methods. They may occasionally be found in the exudate of pleurisy or meningitis occurring during typhoid. They often remain virulent in water and milk for a period varying from a few days to three months—an important fact, con-

sidering that milk and water form almost the exclusive diet of the
infant. They may also remain virulent in the ground for long periods
of time, and may not be killed by freezing temperatures. The bacilli
are not always destroyed by the dilute acid condition of the gastric
secretions, and may pass in a virulent state into the intestinal canal
where they infect the system at large, and multiply in the lymphatic
tissues.

Pathological Anatomy.—In intra-uterine typhoid the infection is
mainly one of the blood, and the typical intestinal and mesenteric
lesions are absent, while in infants and children the intestinal lesions
are usually not as marked as in adults. Peyer's patches and the
solitary follicles are enlarged, but ulceration is uncommon, and, if
present, is usually not deep, hence perforation seldom occurs. In
typhoid fever the length of the ulcer corresponds with the long axis
of the intestine, whereas in tuberculous ulceration of the bowel the
greatest length of the ulcer is transverse in direction. Ulceration,
if present, is more often found in the lower portion of the ileum; next
in frequency in the upper colon, which is also the most common seat
of perforation. The older the child the more closely do the intestinal
lesions resemble those in the adult. The mesenteric glands are espe-
cially apt to be involved in the vicinity of the ileocecal valve.

Peyer's patches and the solitary and mesenteric glands may show
only moderate inflammation and swelling; but, as these conditions
are often found in intestinal diseases in infants and children, one can
not assume that the case is, therefore, one of typhoid. The disease
is often seen in children after five years of age, and but 8 per cent.
of the cases under five years, while 42 per cent. are between five and
eleven years, and 50 per cent. between ten and fifteen years. The
period of incubation is from nine to twenty-one days.

Typhoid Fever in the Fetus.—**Symptoms.**—If the mother has
typhoid fever the fetus is often infected with the disease, although
the mother may have typhoid, and the fetus escape. Miscarriage
occurs in more than one-half the cases of pregnant women who have
typhoid, and the fetus is usually born dead, the death of the fetus
being commonly the cause of the miscarriage, although the high
temperature or the accumulation of toxins in the maternal blood may
also cause the premature expulsion of the fetus. If the fetus is born
alive it usually lives only a few days, quickly succumbing either as
the result of the intra-uterine typhoid infection or because of its
undeveloped, frail, and delicate physical condition. Occasionally a
child, if born near the end of the pregnancy, may outlive the attack
of typhoid.

The typhoid bacilli may pass from the mother through the placenta
into the fetus, although it is questionable whether they ever pass
through a healthy placenta.

The infection occurs through the blood, the bacilli entering the
fetus through the umbilical vein, and the system at large being sub-
sequently infected. The disease is practically from the beginning

a septicemia, which explains the great mortality in this form of the disease. The typical intestinal lesions are not found; indeed, in the majority of cases, no intestinal lesions are present, and when found they are but slight. This is undoubtedly due to the absence of function of the bowel before birth, and to the fact that no bacilli enter the intestinal tract from the mouth. The spleen may be enlarged, the kidneys may show slight changes, and the liver may present the usual changes of typhoid; occasionally, slight lesions are found in the intestinal mucosa. The condition is, however, a blood infection, the typhoid bacilli being found in the spleen, liver, kidneys, and heart's blood. The Widal reaction may be obtained from the blood of the fetus. In these cases the agglutinating factor may have passed from the mother to the fetus.

Typhoid Fever in the Infant.—The disease is less common in the first two and a half years of life than in older children, largely because the infant is fed either on breast milk or on certified or pasteurized milk. It is undoubtedly true that many cases of the disease are overlooked in infants, the possibility of typhoid not being considered. In the older child or adult a continued fever invariably suggests the possibility of typhoid, and a Widal test is made. Owing to the belief that infantile typhoid is rare, this test has not commonly been applied to infants; and, while the disease is less common than in later child life, still those who apply this modern means of diagnosis will not infrequently meet with cases in infancy.

The disease runs a shorter course in infants. Prodromata are not apt to be marked. Drowsiness, disturbed sleep, loss of appetite and indigestion are among the most common symptoms. The onset may be sudden, with vomiting and fever. The duration of each stage is usually shorter in the infant than in the child.

The mortality in infantile typhoid is not easy to determine. In many of the reported cases, proven to be typhoid by a Widal, the disease was severe and the mortality high. If, however, many cases of typhoid in infants have been overlooked, and, personally, I believe this to be the case, then many of these cases were mild in type, which tends to make the prognosis more favorable. The infection in fatal cases in infants is general in character—not, as in older children, largely intestinal—and the temperature curve is not so regular and characteristic as in the older child and adult.

In infants the average duration of the fever is not more than two weeks. Diarrhea is more common in the early part of the disease, and vomiting with loss of appetite is not an unusual early symptom. Weakness and prostration are common, but epistaxis seldom appears. While the infant may be drowsy and dull, convulsions are rare. The tongue is coated, and not apt to be dry. Sore throat occurs in a fair proportion of the cases. Abdominal distention is the rule. Bronchitis is usually present, but not severe. The pulse is often rapid and weak, but, as a rule, the circulation is well maintained. The infant is restless and fretful, but rarely has any other marked nervous symptom.

The fever rises rapidly, usually reaches the maximum in four or five days, remaining high for seven or eight days, and returns to normal by a rapid decline or a more gradual fall in from four to seven days. The rash is seen in about 70 per cent. of the cases. It may appear earlier than in later childhood, its development on the fifth, sixth or seventh day being not uncommon. The spleen is usually enlarged, often after the fifth day. A positive Widal reaction is obtained in 90 per cent. of the cases. The leukocyte count is low, ranging from 4000 to 12,000. The urine may show albumin, and occasionally hyalin and granular casts, especially in the severe cases.
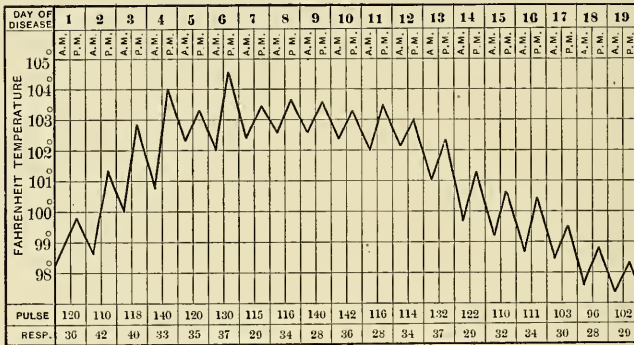
FIG. 66.—Typhoid fever in a child two years of age.

Typhoid Fever in the Older Child.—*Fever.*—The first stage shows a fever higher each morning than the preceding morning, and an evening fever higher each evening than the preceding evening. In the second stage the fever remains more or less continuously high, with a morning fall of 1.5° to 2.5°, and an evening rise of about the same extent. In the third stage the temperature gradually returns to normal, the morning temperature being lower than the evening, and reaching normal several days before the evening temperature. The average duration is eighteen to twenty-one days.

In the so-called abortive cases the duration may be much less; yet, again, the fever may continue for four or five weeks. The usual height of the fever is 102° to 103° F., but high temperatures of 104° to 106° F. are not uncommon. Increased fever often apparently produces no special increase in the symptoms. The temperature may remain subnormal during the first two or three days of convalescence. A sudden and marked fall in temperature usually means intestinal hemorrhage or perforation. With intestinal hemorrhage the pulse is weak, the face pale, and the extremities cold. With perforation there may be localized abdominal pain which increases, also increasing tenderness, rigidity of the abdominal muscles, vomiting, and collapse.

Pulse.—The pulse, as a rule, is only moderately increased, and is slower than in other diseases with the same temperature. It may,

however, be rapid and dicrotic. It is not as slow in proportion to the fever as in adults, a pulse rate of 160 to 170 not being uncommon, and not necessarily a sign of danger. The older the child the slower, as a rule, the pulse. Dicrotism is often observed. As the pulse is not as slow in proportion to the fever as it is in the adult it is of less assistance in forming a diagnosis.

A murmur, systolic in time and heard best at the apex, is not uncommon toward the end of the third week. It is temporary in character and disappears after the patient has entirely recovered from the illness. Myocarditis may be the cause of the murmur, or it may be hemic. By acting on the pneumogastric centre the toxins may possibly affect the pulse, and by their action on the heart muscle produce the murmur.
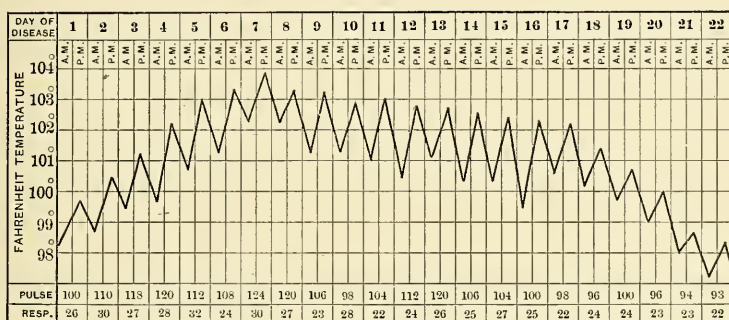
Fig. 67.—Typhoid fever in a child seven years of age.

Bowels.—Constipation may be present, especially in younger children, but there is slight diarrhea in about 50 per cent. of all the patients. The stools are not characteristic. Severe diarrhea is uncommon except in the worst cases, and intestinal hemorrhage occurs in only about 2 or 3 per cent. of the cases, and is most often seen at the end of the second week. The mortality after hemorrhage is about 50 per cent. Hemorrhage is uncommon in children under ten years of age.

Perforation rarely occurs, but, when this dangerous symptom develops, the life of the patient usually depends upon its immediate recognition. With perforation we have a sudden fall in temperature, sudden abdominal pain, and changes in the respiration and pulse. The pain continues, there is tenderness with some rigidity of the abdominal muscles, and there may be vomiting and sweating. The movements of the abdomen on respiration are restricted, there is decrease in the area of liver dulness, with movable dulness in the flanks, and leukocytosis.

Abdominal distention is present in 70 per cent. of the cases, more often in older children. Abdominal pain and tenderness seldom appear,

although vomiting is a common early symptom in the younger child. Diarrhea is often seen in the severe cases.

Tongue.—The tongue is not so dry as in adults, is usually coated early in the disease, the edges and tip being clean.

Rash.—Rose-colored spots appear in over 60 per cent. of the cases, being scattered over the abdomen, chest, back, and thighs. The number of spots is less than in adults. They usually develop at about the seventh to the ninth day, coming in crops, which last about ten days. They are of great diagnostic importance, and usually reappear in a relapse.

Spleen.—The spleen is enlarged in almost all cases, this enlargement being demonstrated by palpation or percussion in about 80 per cent. of typhoid children. A decrease in the size of the spleen is a favorable sign, and usually means that the child is progressing favorably. An enlarged spleen that does not decrease in size indicates the continuance of the infection and persistence of the disease. During a relapse the spleen, as a rule, enlarges.

Lungs.—Bronchitis is so often present that it may justly be considered a normal part of the disease. Bronchopneumonia is an unfavorable complication, especially in delicate children and in hospital cases. Lobar pneumonia and pleurisy are seen occasionally; lung abscess is rare.

Headache.—Headache is present in 80 per cent. of the cases, and is usually complained of by children old enough to describe their symptoms. Vertigo is less common, appearing in about 20 per cent. of cases.

The Nervous System.—Delirium in mild form is common; in the severe cases it may be marked. Dulness is especially apt to be seen in the younger children. True meningitis is rare, although a meningitis due to the typhoid bacillus may occur. Post-typhoidal temporary insanity occasionally develops, in the mild form is not uncommon, and is seen especially in those children whose nutrition is poor, and who take a subnormal amount of nourishment; it may also be toxic in origin. It usually ends in complete recovery.

Delusions of fear are common, or the child may become greatly excited on the slightest cause, or may be greatly depressed mentally. The nervous symptoms, as a rule, improve in proportion to the possibility of increasing the child's food. Chorea and neuritis occasionally appear, and paralysis may develop in those muscles supplied by the affected nerves.

Convulsions are dangerous and often a cause of death. Hemiplegia is a rare complication, but may be caused by embolism or thrombosis. In severe and protracted cases the mental powers may be more or less impaired, and melancholia, various degrees of mental excitation or depression, or temporary dementia may develop. The tendency of these conditions is to end in complete recovery, except where there are permanent brain lesions, as in some cases of hemiplegia. Epistaxis is less common than in adults, occurring in about 20 per cent.

Kidneys.—Degenerative changes of mild degree probably occur in most cases. At the height of the disease a trace of albumin is detected in the urine, and occasionally a few casts. Typhoid bacilli are found in the urine in from 20 to 50 per cent. of the cases. Ehrlich's diazo-reaction in the urine occasionally appears as early as the fifth day, although the usual time is from the seventh to the tenth day; or it may not appear until the end of the fifth or sixth week, consequently it may appear before or after the Widal. It is absent in about 15 per cent. of all cases.

Blood.—The leukocytes are reduced in typhoid fever, leukopenia being the rule. In 10 per cent. of the cases the white cells are 5000 or less; in 70 per cent., 5000 to 10,000; in 30 per cent., 10,000 to 16,000. If leukocytosis exists, it is usually due to some complication. As the disease progresses secondary anemia develops, both the red cells and the hemoglobin being diminished, and during convalescence the red cells return to the normal more rapidly than does the hemoglobin.

Blood cultures are of great assistance in a case of doubtful diagnosis. The younger the child the more difficult it is to locate and puncture the vein, but those who have mastered the technic have comparatively little trouble in obtaining from $\frac{1}{2}$ to 2 c.c. of blood by puncturing a vein in the arm. Busquer recovered the typhoid bacillus in 43 children he examined, and Rotch and Conrodi have also proven the efficacy of this means of diagnosis. The Widal reaction is of great diagnostic importance, and is present in 95 per cent. of children with typhoid fever. It is present in 13 per cent. by the seventh day; in 63 per cent. by the fifteenth day; and in 89 per cent. by the twenty-fifth day. Occasionally it is not present until the patient is entirely free from all typhoid symptoms. In children with jaundice there is a positive Widal, hence in a case of jaundice the Widal loses its significance.

Relapses.—The number of relapses varies in different epidemics. In some years it may be only 4 per cent., in others, 15 per cent. In my own cases it has been 9 per cent. The duration of the relapse is usually about fifteen days; ordinarily it is shorter than the original attack. In a relapse the spleen is enlarged, the roseola is present in 75 per cent. of the cases, leukopenia in 60 per cent., and mild intestinal symptoms in about one-half the cases.

Diagnosis.—In the diagnosis of a doubtful case of typhoid fever, the temperature, rose spots, enlarged spleen, Widal reaction, blood cultures, Ehrlich's diazo-reaction, typhoid bacilli in stools and urine, and the intestinal symptoms, are all worthy of careful study. Usually the diagnosis is easy, occasionally it is difficult, but it is almost always possible to decide positively whether typhoid is present or not.

Differential Diagnosis.—*Appendicitis.*—A child may be taken suddenly ill with severe pain in the right iliac fossa, high fever, vomiting, and often little or no resistance of the abdominal walls. The early subsidence of the symptoms in the region of the appendix, the appear-

ance of rose spots, enlarged spleen, and a positive Widal clear the diagnosis. Yet in a case of typhoid fever, a typhoid ulcer in the appendix may cause a perforation.

Paratyphoid Fever.—The symptoms are very like those of typhoid fever, but usually less severe and of shorter duration. The paratyphoid bacillus, which may be found in the stools, urine, and blood, differs only slightly from the typhoid bacillus, and an agglutination of paratyphoid cultures occurs with the blood of the child with paratyphoid. There is no Widal reaction.

Acute Miliary Tuberculosis.—In this disease the onset is slower than in typhoid. There may be a family history of tuberculosis, or a history of the child's exposure to tubercular infection. A preceding pneumonia, pertussis, or measles, with incomplete recovery is suspicious. Cough, irregular fever, and nervous symptoms may be present in both, and in miliary tuberculosis the physical signs in the lungs are not usually significant.

The presence of tuberculous lymph glands, a persistent cough, gradually increasing physical signs in the lungs, the appearance of tubercles in the choroid, a positive tuberculin test, and the finding of tubercle bacilli in the mucus removed from the pharynx, will confirm the diagnosis of tuberculosis. A positive Widal, rose spots, and typhoid bacilli in the stools, urine, or blood make certain the diagnosis of typhoid.

Prognosis.—The mortality varies in different epidemics according to the severity of the infection, the general health and vitality of the child, and whether it comes under observation early or late in the disease. In hospitals the mortality-rate is about 8 per cent.; in private practice 4 per cent. The prognosis is, of course, influenced by all complications, in direct proportion to their mildness or severity. In my experience, most of the deaths were associated with complications, such as bronchopneumonia, hemorrhage, or perforation. The prognosis is really better than is apparent from statistics, as mild cases of typhoid are easily overlooked. A continued fever in a child should always suggest the possibility of typhoid fever, and a systematic examination for typhoid in all such cases is the only method by which one can avoid error.

NUMBER OF DEATHS IN PHILADELPHIA—TYPHOID FEVER.

Age period.				1911.	1912.	1913.	1914.	1915.
Under 1 year	.	.	.	0	0	0	0	0
1 to 2 years	.	.	.	4	3	2	1	0
2 to 5 "	.	.	.	10	7	10	5	2
5 to 10 "	.	.	.	14	7	13	8	5
10 to 15 "	.	.	.	15	18	17	12	13
15 to 20 " .	:	.	.	24	29	26	17	19
All ages	.	.	.	.	223	200	255	124	109

Complications.—Among the more common complications may be mentioned otitis, pneumonia, diphtheria, hemorrhages, furunculosis, and nephritis. Among the rarer complications are measles, scarlet

fever, gangrene of the skin, chorea, and neuritis. Aphasia is more common in children than in adults, and usually appears in the third week in severe cases associated with nervous symptoms. It ordinarily ends in complete recovery in three or four weeks. Meningitis may be due to the typhoid bacillus, and may end in recovery.

Treatment.—Prophylaxis is of the greatest importance; and, as the drinking of infected water is the most common cause of the disease, it is wise to order that all drinking water, and water used for washing such vegetables as celery or cress, be boiled. If a case of typhoid fever exists on a dairy farm, milk from such a farm should not be allowed to be sold. An infant should be weaned if its mother has typhoid, and all children who are apt to be exposed to typhoid fever infection may be protected by typhoid vaccine. The feces and urine of a typhoid child should be disinfected by covering them with three times their bulk of 1 to 20 carbolic acid, or 1 to 1000 bichloride solution.

All dishes, spoons, etc., should be similarly treated with 1 to 20 carbolic acid, and all bed linen should be soaked in 1 to 20 carbolic solution, and then well boiled. All typhoid cases should, if possible, be kept apart from both well and sick children, as it is not uncommon for those who handle, nurse, or come in intimate contact with typhoid cases to contract the disease. The hands of the nurse, especially, are in danger of being contaminated by the fecal or urinary discharges, and infection thus take place; therefore, the hands of all who come in contact with typhoid cases should frequently be washed in a solution of lysol, 1 to 200, particularly before eating.

The child should be kept in bed, in a large, well-ventilated room, until the temperature has been normal for a week.

Diet.—Nourishment should be given every three hours during the day and every three or four hours during the night. The nourishment should be liquid, milk being the best single article of diet, and this should be administered in such quantities and such dilutions as the child can digest. It may be diluted with water, or barley-water, and, if not well digested, should be peptonized. If the symptoms of gastro-intestinal infection are present, milk is contra-indicated until the acute symptoms have subsided.

Mutton, beef, and chicken broths may be given in addition to the milk, and albumen-water is often well borne and answers a useful purpose. A raw egg, either alone or beaten up with milk, is very nourishing, especially in the early stages of convalescence. Cereal gruels, thoroughly cooked and well thinned with milk, may be given throughout the illness.

If the disease runs the regular course of about three weeks, milk, animal broths, and cereal decoctions with milk are all that is required. If, however, the fever continues for four, five, or six weeks, yet no relapse has occurred, and especially if there is extreme prostration with emaciation, but no marked symptom of gastro-intestinal infection, raw eggs, or soft eggs, junket, and custards may be added to the

40

diet. After the temperature has been normal for one week, milk toast, scraped meat, zweibach, and soft foods may be allowed. Water should be given freely throughout the disease.

Hydrotherapy.—If the child's temperature remains below 103° F., a morning and evening sponge with water at 90° F., followed by an alcohol rub, is all that is required. The best method of treating pyrexia and the associated nervous and respiratory disturbances is by the bath. The temperature of the water should be 90° to 85° F., and the child should remain in the tub, its entire body, except the head, being covered by the water, from five to twelve minutes. Friction of the body and sponging of the head should be kept up throughout the time of the bath.

The length of the bath depends upon the effect produced on the temperature, and the nervous and respiratory symptoms. After the bath the child should be well rubbed and returned to its bed. The rectal temperature should be taken ten minutes after the bath, and the reduction in the fever will serve as a guide in deciding upon the duration of future baths, and the temperature of water to be employed. If there is prostration, whisky, in doses appropriate to the age of the child, may be given before and after bathing.

The bath should be repeated every three to six hours if the temperature remains at 103° F. or higher, and an ice-cap applied to the head is often beneficial in those cases where the temperature remains high and the nervous symptoms marked. Bromide of soda, 5 grains, repeated in three hours if necessary, is of assistance in quieting the nervous symptoms and inducing sleep. Cold-water baths are not well borne by children with typhoid, and, in my experience, the water should never be below 80° F.

Bowels.—During the first few days of the disease constipation may be overcome by fractional doses of calomel, and while the fever continues it may, if marked, be partially relieved by an occasional dose of castor oil. The safest and best means of overcoming this condition is by the daily use of an enema of salt solution, soap and water, or small amounts of sweet oil. If the number of stools in twenty-four hours exceeds four, especially if they are large and watery, it is well to control the diarrhea by subnitrate of bismuth, 5 to 10 grains, every three or four hours, and in older children, 1 to 2 grains of salol may be added to each bismuth dose. The bismuth and salol are only to be given as long as the diarrhea continues. Paregoric, 10 to 20 drops, may be given in addition to the bismuth if the diarrhea is not easily checked, the dose being in proportion to the age of the patient and the severity of the diarrhea.

The stools should always be carefully examined, as the presence of undigested food materials may be of service in the selection of the diet to be given. Tympanites is best controlled by a hot turpentine stupe applied to the abdomen and covered by oiled silk and the giving of a warm enema of salt solution. If this does not afford relief, a rectal tube may be cautiously used.

Alcohol.—Whisky is not required in the mild cases. In the asthenic cases whisky, 20 drops to 1 dram, may be given every three hours; it is especially valuable when there is low muttering delirium and a weak pulse. Strychnine and camphor are of benefit when the heart-sounds are weak, or if a murmur develops due either to changes in the myocardium or to general toxemia.

Hemorrhage from the bowel demands absolute rest for the patient, an ice-coil to the abdomen, sufficient opium by the mouth to check intestinal peristalsis and bowel movements, the stopping of all food, and the giving of only small pieces of ice or very small amounts of water by mouth. Parotitis is best treated by the application of an ice-bag. Perforation demands the opening of the abdomen, and the closing of the perforation in the intestine. An early diagnosis of perforation is essential if the child's life is to be saved.

PARATYPHOID FEVER.

Paratyphoid fever in many cases bears a close resemblance clinically to typhoid fever, but it is due to an entirely different organism. In paratyphoid there are no intestinal lesions.

Etiology.—The organisms which cause the disease are called paratyphoid bacilli, and are divided into two groups, "A" and "B," according to their cultural characteristics. Many cases of paratyphoid fever are the result of food poisoning, and there is considerable evidence also to show that it is a water-borne disease.

Symptoms.—The symptom-complex is practically the same as in typhoid fever; the disease may occur in epidemics, and is not uncommon. It may be differentiated from typhoid fever by a negative Widal reaction which, however, is only obtained when the tested blood is diluted more than 1 part in 50. The paratyphoid organisms cause an agglutinin reaction, but belong, according to their cultural and other characteristics, between the typhoid bacillus and the Bacillus coli communis. The symptoms are usually less severe than in typhoid, but one may observe nosebleed, headache, and anorexia; the temperature may be about 101° F. at first, and so continue for two weeks, rising a degree, to 102° F., every day, and falling the next morning. In some cases the symptoms closely resemble Asiatic cholera, and there is little, if any, evidence to suggest typhoid. The spleen is usually enlarged, but Peyer's patches, the mesenteric glands, and solitary follicles are not involved. In the worst cases the symptoms are largely those of septicemia.

Diagnosis.—The diagnosis is made by examination of the blood, which gives a positive agglutination reaction with the paratyphoid bacillus.

Prognosis.—The prognosis is usually favorable, and death occurs only in the most severe cases.

Treatment.—The treatment is, for the most part, the same as in typhoid fever.

SCARLET FEVER (SCARLATINA).

Scarlet fever is an acute contagious disease, occurring sporadically or in epidemics. It is characterized by sudden onset, sore throat, a punctate eruption, scarlet in color, which covers the entire body, and is followed by desquamation of large flakes of superficial skin. There is a tendency to otitis, cervical adenitis, and nephritis.

Etiology.—The disease is essentially one of children. It is rarely met with in infants under six months of age, and is uncommon even in children from six to twelve months. Numerous instances have been reported where the mother having scarlet fever continued to nurse her infant under six months of age, and the child did not contract the disease.

A very few cases of infants being born with scarlet fever are on record, the mother having the disease at the time of the infant's birth. After the age of one year the susceptibility to the disease increases with each year until the extreme is reached at five or six years; after this age the number of cases gradually decreases and it is uncommon after the sixteenth year. It is occasionally seen in adult life, but after the period of childhood is passed the disease is usually in a mild form.

In Philadelphia during the years 1911 to 1915 inclusive only 18 deaths occurred from scarlet fever in children under one year of age. More than 60 per cent. of the cases occur in children under five years of age, and 90 per cent. in the first ten years of life. Unlike measles, immunity in adult life does not depend upon the fact that most children contract the disease, as it is estimated that not more, and probably less, than 50 per cent. of children have scarlet fever.

Second attacks are rare. Family predisposition to contract scarlet fever is occasionally observed, and not only may all children in the family contract the disease, but it may appear in them in a most severe form, while the other cases seen at the time may be of quite a mild type. It is more common in the winter months, possibly because children are kept indoors more, and are brought more often and more closely in intimate contact.

Epidemics differ greatly in severity and in the liability to dangerous complications. It is more common in city than in country districts, and the white race is more susceptible and shows a greater mortality than the colored race.

The specific virus of scarlet fever is as yet unknown. The immunity that one sees so often in those exposed to scarlet fever is not always permanent, as a child who has been brought in close contact with a scarlet fever patient may, if afterward exposed, contract the disease. The virus is present in the discharge from the nose, mouth, and ears, and while this dried discharge containing the poison may be carried for short distances by the air, it cannot be carried far, as those children living in the neighborhood of scarlet fever hospitals do not show any unusual tendency to contract the disease.

Scarlet fever has been produced in children by inoculation with the mucus from the mouth and throat, also by the blood and contents of the vesicles, so that undoubtedly the virus producing the disease must be present in these fluids.

It is still a matter of doubt as to whether it is possible to produce scarlet fever by inoculating a child with the scales; the evidence is against the possibility and probability of the disease being transferred by such inoculations. The poison lives for long periods of time outside the human body, and may remain virulent and cling to the bed linen, carpets, wall paper, and any portion of the room for months, in spite of the most thorough scrubbing, disinfecting, and flushing with fresh air.

The disease is usually spread by direct personal contact. The mild cases are especially to be feared as sources of infection, as they may not be ill enough to go to bed, and may even continue to attend school; they are consequently brought in close contact with a large number of well children, and may easily transmit the disease.

Epidemics have been traced to milk, and boiling the milk destroys the scarlet fever poison. While the lower animals do not contract the disease, a pet cat or dog may carry the poison from the sick-room to children in the same or near-by house.

The disease may be carried by a third person, and all persons coming in contact with a scarlet fever patient, especially the nurse and physician, should take definite precautions to avoid carrying the infection. Numerous instances have been reported where the disease has been known to be conveyed considerable distances by letters or packages sent by mail.

The scarlatinal poison enters the body through the nose and throat; less often through wounds and burns, and in puerperal women through the genital tract, although it is certain that many of the so-called cases of scarlet fever in puerperal women have been puerperal sepsis and not scarlet fever.

The streptococcus is, by some, considered the cause of the disease, and streptococci have been found in the blood, skin, and in the internal organs at autopsy. The general belief is that while the streptococcus may and often does exist as a mixed infection, and does produce many of the dangerous symptoms and complications of scarlet fever, as pseudomembrane in the pharynx, otitis, cervical adenitis, endocarditis, synovitis, and nephritis, that it is present only as a complication, and is not the true cause of the disease.

A protozoön has been found in the skin by Mallory, and the belief that the disease is caused by a protozoön is steadily growing in favor. The contagion is slight in the beginning of scarlet fever, whereas in measles the contagion is marked from the very beginning of the disease. Scarlet fever is very contagious during the eruption and for quite a long time after the eruption has disappeared, but it is not at all certain that the contagion is due to the skin that is peeling off; in fact many believe that this desquamating epidermis is incapable of producing the disease.

A scarlet fever patient can transmit the affection before desquamation begins and after it has ceased, and a discharge from the nose, ear or throat persisting after desquamation has ceased is a not uncommon source of infection. Nothing is more common than for a scarlet fever patient to be released or escape from quarantine before desquamation is complete, and as the general belief at present is that the contagion is contained in the scales, a child who is desquamating would, if the scales carry the contagion, expose to the disease all those with whom he comes in contact.

The disease is especially contagious when the fever is high, and the throat symptoms severe, and is more or less contagious as long as any discharge is present from the nose, ears, or throat, and until desquamation is complete, and quarantine in an ordinary case should continue for six weeks. Infected articles and clothes that have been kept in closed drawers and closets may transmit the disease months or even years later.

Measles, whooping-cough, chicken-pox, diphtheria, typhoid fever, or erysipelas, may coexist with scarlet fever, although a pseudomembrane in the pharynx or larynx may be diphtheritic or streptococcic. It is estimated that from 2 to 4 per cent. of the scarlet fever cases discharged from the hospital transmit the disease to other children. This estimate I believe is too small.

Pathological Anatomy.—In an uncomplicated case of scarlet fever pathological changes are found in the skin, tongue, throat, and the lymphatic glands. The rash disappears after death, except in those locations where it has been especially marked during life. If the rash is of the hemorrhagic form it is visible postmortem.

The skin shows a marked congestion of the bloodvessels, and a dilatation of the lymphatics with moderate cellular proliferation around the bloodvessels, hair follicles, and sweat glands. The epidermis is destroyed and is later thrown off in scales or flakes. The tongue shows similar changes in its epithelium to that occurring in the skin, but the changes occur earlier in the disease and are more severe. The pharynx shows an inflammation which may be of a mild catarrhal form, or of a severe type with pseudomembrane.

The anterior and posterior nares show a more or less severe involvement of the mucosa, and the ears are not infrequently involved. The infection may extend from the nose to the antrum of Highmore or from the ear to the mastoid with possible involvement of the meninges, and later of the brain.

The lymphatic system throughout the entire body is involved to a marked extent in scarlet fever. Not only are the superficial lymphatics, as the cervical, axillary and inguinal, enlarged, but the deeper glands, as the tracheal, bronchial, mesenteric, and retroperitoneal, are also affected, and the tonsils, liver, and spleen show a distinct hyperplasia. The cervical lymphatics may undergo suppuration, and the infection may spread to the surrounding tissues, resulting possibly in abscess, and rarely gangrene.

In severe cases the mucous membrane of the stomach shows distinct inflammatory changes and similar changes are found in the mucous membranes of the intestine with marked swelling of the lymph follicles.

The heart may show either cloudy swelling or fatty degeneration; less commonly, an endocarditis or pericarditis develops. Hypertrophy and dilatation may ensue from a nephritis. Bronchopneumonia is not uncommon in the fatal cases and a seropurulent pleurisy, pulmonary gangrene, and abscess may occur. Acute interstitial nephritis is the most common form of nephritis associated with scarlet fever, and in all fatal cases more or less marked pathological changes in the kidneys are found.

Symptoms.—The period of incubation is short, usually from one to seven days; commonly the disease develops within two to four days after exposure. The shorter the incubation, the more severe is, as a rule, the attack.

Stage of Invasion—The child is taken suddenly ill without any prodromes; vomiting is often the first symptom. The vomiting may occur a number of times and be very severe. In young children, a convulsion is not uncommon, and very occasionally the disease may begin with a chill. Fever is present from the first, and usually reaches its maximum in twenty-four to forty-eight hours and may even in a few hours rise to 104° or 105° F. The higher the initial fever, the more severe, as a rule, is the illness, although in the mild cases the temperature may not exceed 101° F.

Sore throat is an early symptom, but as many children will not complain of soreness in the throat, even when a considerable amount of inflammation is present, it is always necessary and advisable to make an examination of the pharynx. The severity of the inflammation, which commonly involves the soft palate, tonsils, and pharynx, with small red points on the hard palate, is in direct proportion to the severity of the infection.

Headache is also an early symptom, and the severity of these initial symptoms, as convulsions, vomiting, fever, sore throat, and headache, will usually be sufficient to indicate the probable severity or mildness of the future course of the disease.

The tongue is coated, the tip and edges being red, the child refuses nourishment, complains of thirst, is restless, drowsy, or delirious, and passes only small amounts of high-colored urine. In younger children, especially in hot weather, diarrhea may occur.

Stage of Eruption.—The rash appears first on the neck and chest, and rapidly spreads over the body and face; ordinarily the entire surface of the body is covered in twenty-four to thirty-six hours after its first appearance. The rash may appear on the face only as a flush, or the face may be almost or quite free from rash. The eruption usually appears in the first twenty-four to forty-eight hours, although, rarely, its appearance is delayed until the third, fourth, or fifth day.

It appears first as very small red points, set very closely together;

the points are larger and not so close together on the legs. The rash areas are scattered in patches over the body, and, spreading quickly, cover the entire surface. The general color of the skin is red, and the appearance of the red skin and innumerable fine, darker red points is very characteristic. The color of the skin is usually a dull red, and becomes dusky red as it fades.

The body has the appearance of being covered with a rash of uniform reddish color, but close inspection reveals the skin to be a lighter red, and the fine points to be distinctly darker. Irregular patches of skin on the buttocks, arms or legs, may be entirely free from rash, giving the skin a blotchy appearance not unlike measles.

The face may be entirely free from rash, but usually the forehead and cheeks are flushed. The lips, also alæ and tip of the nose, are, as a rule, free from rash, giving the so-called white rim around the mouth which is quite characteristic. The rash disappears on pressure, is usually accompanied by itching, and, if severe, edema and swelling are present.

In the mild cases, the rash may be very slight, may only appear over very small areas, may not even show itself on the face, is most often under such conditions seen in the axillæ, groins, the back of the thighs or buttocks, and in these locations may last for only twenty-four hours. The eruption continues at its height for twelve to forty-eight hours, and lasts usually from three to seven days.

A careful examination of the skin will show small vesicles to be present in a fair proportion of cases, and especially when the rash has been very severe. In the worst cases the rash often varies, not only in its appearance, but also in the time of its development. It may be of a dark, almost purple color, or again it may closely resemble the rash of measles. If the eruption is very faint, a hot bath may cause it to better develop.

Stage of Desquamation.—Usually within forty-eight hours after the rash has disappeared, desquamation begins. It appears, as a rule, first on the neck and chest, and the entire body with the exception of the hands and feet continues to shed fine bran-like scales for about two or three weeks. The peeling appears later on the hands and feet, the thicker skin of the palms, fingers, soles and toes being slower to separate, and the desquamation continuing for a longer period of time, usually about four or five weeks, and often peeling off in large flakes. Occasionally the thickened epidermis of the fingers or palms may separate in one large piece like the ragged finger or palm of a glove.

The character of the desquamation is often, but not always, in direct proportion to the intensity of the rash, being quite extensive and flaky in the severe, and slight and bran-like in the mild eruption. In the very light cases, desquamation should be looked for at the tips of the fingers, under the nails, and in the groins and axillæ. Desquamation may occur twice in one or more portions of the body, prolonging the period of quarantine. Falling of the hair and shedding of the nails are occasionally seen.

Desquamation is the most typical sign of scarlet fever, and it is not uncommon in hospital dispensaries or among the poor to see a child in the peeling stage of scarlet fever, the history being that a mild and transient rash occurred several weeks previously; with this history and desquamation, scarlet fever can be positively diagnosed. In very few diseases is such a great variation seen in the severity of the symptoms as appears in scarlet fever. Sudden onset, fever, sore throat, and rash are present to a greater or less degree in every case, but in the light cases these symptoms may be very mild and even pass unnoticed.

Diagnosis.—Frank typical scarlet fever offers no great difficulty in diagnosis, since it can easily be made from the presence of the more prominent symptoms, such as a sudden onset accompanied by vomiting, a rapid rise in temperature, the appearance of the throat, enlarged papillæ on the tongue, and the typical eruption appearing within forty-eight hours. These symptoms constitute an unmistakable syndrome, but no one symptom is pathognomonic.

A diagnosis can rarely be made before the eruption appears; on the other hand, the diagnosis cannot be made upon the appearance of the rash alone, since many other eruptions resemble that of scarlet fever, such as the rash in rötheln, diphtheria, septic erythema, and influenza, also the drug rashes and antitoxin rash.

Scarlatiniform erythemas and rashes are also produced by severe burns, intestinal auto-intoxication, measles, varicella, variola, and vaccinia, also in rheumatism, pyemia, malaria, and typhoid fever.

In attempting to decide whether a rash is that of scarlet fever or not, the time of its onset and its persistence are important points, and the amount of scaling which follows a rash of given intensity is greater in scarlet fever than after any other rash which might simulate it. Many eruptions cause desquamation, some more freely than scarlet fever; but well-marked desquamation after an illness which simulates scarlet fever is a point of great diagnostic value.

In making a diagnosis after the eruption has faded or changed, a white line at the junction of the finger with the nail showing beginning desquamation is of great value. Another important clue to the diagnosis is an accurate history of the diseases the child has previously had and the diseases to which it has been exposed.

Differential Diagnosis.—While the throat involvement in scarlet fever may suggest diphtheria at first, yet the eruption which appears usually indicates the true nature of the disease, and the prevalence of one or the other of these diseases is also confirmatory. The diagnosis of diphtheria can, as a rule, be made with absolute certainty by a culture from the throat, and this test should always be made when there is any doubt.

Measles.—The typical eruption of scarlet fever does not resemble the rash of measles, and when the differentiation between scarlet fever and measles is rendered obscure by an atypical rash, we must bear in mind what a wide difference there is in the other symptoms

of these two affections, and this will usually permit us to make a definite differential diagnosis. The onset of scarlet fever is more severe, the fever higher, the attacks of vomiting more frequent, and the rash appears earlier than in measles; while photophobia, coryza, cough, and hoarseness are peculiar to rubeola. Sore throat, strawberry tongue, and cervical glandular enlargement are characteristic of scarlatina, and the Koplik's spots of measles are absent. .

Rubella.—The eruption of German measles resembles that of scarlet fever more closely than does that of rubeola; but, as a rule, it is not followed by desquamation, and there are no severe throat symptoms. The constitutional disturbances are much milder in rubella than in scarlatina, and the course of the disease is shorter. In German measles the posterior cervical glands are enlarged.

Drug Rashes.—The various drug rashes may be diagnosed from the history and other untoward symptoms which appear with the rash. In acute exfoliative dermatitis there are no throat symptoms and the tongue lacks the appearance so characteristic of scarlet fever.

Complications.—Acute nephritis is the most common complication of scarlet fever, and usually occurs in the second, third, or fourth week, after desquamation is more or less complete. A slight albuminuria often appears at the height of the fever, but does not signify acute nephritis, or predispose to it, although the cells lining the tubules are at this time in a state of cloudy swelling.

The nephritis of scarlet fever is in all probability caused by the specific toxin or bacillus, and the frequency with which this inflammation of the kidneys occurs in scarlet fever is to be attributed to the increased work thrown upon the kidneys because of the impairment of the function of the skin. Acute parenchymatous nephritis is the most common form of the kidney affection, but intersitial nephritis also occurs. The majority of cases progress favorably, although the disease may appear in all degreees of severity.

Throat.—Scarlatinal angina is a very common complication, and is the result of streptococcic infection of the throat. It differs from diphtheria in that it rarely spreads to the larynx or causes paralysis. True diphtheria, however, is occasionally associated with scarlet fever.

If the non-diphtheritic membrane seen in scarlet fever shows a tendency to spread, it may prove a source of danger by extending into the nasopharynx and along the Eustachian tube to the middle ear. Swallowing may become both difficult and painful; irritating discharges may exude from the nose; the breath is foul. Ludwig's angina may appear. Gangrene of the tonsils and ulceration may occur, and sometimes involve the uvula, fauces, and pharynx.

In severe cases the cellular tissues of the neck become infiltrated and slough or suppurate, occasionally causing hemorrhages from the tonsils or vessels of the neck, and thrombosis of the jugular vein. Death may ensue from hemorrhage or from septicemia. In these cases the constitutional symptoms are all aggravated, while asthenia, cachexia, and, eventually, collapse precede the fatal termination.

In the rare cases in which the larynx is involved edema of the glottis occurs, and, if there be suppuration in the throat, the pus may pass into the mediastinum and neighboring structures, setting up purulent pleurisy and pericarditis, with symptoms of embolism, thrombosis, or septicemia, and usually terminates fatally.

Ears.—The ears become involved in scarlet fever by extension from the throat along the Eustachian tubes. This occurs more frequently in younger children, and causes the usual symptoms of otitis media, such as earache, restlessness, and a rise in temperature, with congestion and bulging of the drum membrane, which, in the majority of cases, eventuates in perforation of the tympanum. When the drum membrane is resistant, and rupture is delayed, mastoiditis and meningitis may supervene. It is estimated that 10 per cent. of chronic deafness is due to scarlatinal otitis, which in very young children may produce deaf-mutism.

Lymph Nodes.—The submaxillary glands are always enlarged in scarlet fever, and when the throat symptoms are severe there is considerable swelling of these and of the cervical lymph nodes. Suppuration may take place, or the inflammation may not proceed beyond the acute inflammatory stage.

Joints.—Scarlatinal arthritis may appear early in the disease, and in these cases the inflammation is migratory, affecting the same joints which are commonly involved in adults who have acute inflammatory rheumatism. These cases simulate acute inflammatory rheumatism, and respond to treatment with the salicylates, but are regarded as scarlatinal. Occasionally a less transient arthritis, which is septic in origin, appears late in the course of scarlet fever, and involves one or several joints.

Nervous System.—Among the occasional complications of scarlet fever are convulsions and meningitis. Chorea and hemiplegia are quite rare, and but few cases of paralysis have been reported as following scarlatina.

Heart.—Transient murmurs are not infrequently heard during the course of scarlet fever, and of these the mitral systolic is the most common. There is often extreme irregularity of the heart's action, and a *"bruit de galop"* is occasionally heard. Transient murmurs are thought to be due to a loss of cardiac muscular tone, and consequent imperfect closure of the heart valve.

Acute dilatation of the heart may take place during the height of the fever, and endocarditis and pericarditis occasionally appear. Cases of malignant endocarditis are less numerous than would be expected from the virulence and wide-spread action of the infecting organism, and it is evident that the heart is affected more by scarlatinal toxins than by the germ which produces the disease.

Lungs.—Pneumonia is one of the early complications of scarlet fever, and may be either lobular or lobar in type; but pulmonary complications are not as frequent or as severe as in measles. A septic or aspiration pneumonia (bronchopneumonia) is liable to occur in the severe septic anginal cases.

Miscellaneous.—Among the various other complications of scarlet fever may be mentioned the following: Gastro-intestinal disturbances in which vomiting is a marked feature, hyperpyrexia, osteomyelitis, ophthalmia, and other infectious diseases of which diphtheria, measles, and varicella are the most common.

Prognosis.—The prognosis in scarlet fever depends upon the character of the epidemic, the prevalent type of the disease, the age of the patient, and the presence or absence of complications. When cases are isolated and skilfully treated from the onset of the disease, the prognosis is usually favorable and the mortality is not high except in very young children. In severe cases the prognosis should always be guarded, and persistent albuminuria, profuse diarrhea, and marked angina or otitis are danger signals.

The hemorrhagic form is always serious, affections of the endocardium or pleura may prove fatal, and nearly all of the malignant septic cases die. A child with scarlet fever should not be pronounced out of danger until after the fourth week has passed with no complications.

NUMBER OF DEATHS IN PHILADELPHIA—SCARLET FEVER.

Age period.	1911.	1912.	1913.	1914.	1915.	Total mortality for five years.	Per cent. of mortality compared with mortality at all ages.
Under 1 year .	7	3	3	4	1	18	3.0
1 to 2 years .	19	8	13	no data	no data	. .	8.8
2 to 5 " .	85	55	71	60	8	279	45.0
5 to 10 " .	39	34	54	34	4	185	·27.1
10 to 15 " .	7	6	9	5	3	30	5.0
15 to 20 " .	7	1	2	4	2	16	2.6
All ages . .	179	113	162	129	25	608	

Treatment.—Prophylaxis is an exceedingly important part of the treatment in scarlet fever, the organism and infectious principle of the disease being so difficult to exterminate. The patient should be isolated as soon as the diagnosis is made, and strict quarantine maintained.

If several children in the same house are affected, each child should be assigned to a separate room, or reinfection may occur. The sickroom should be one that can be isolated completely, and every portable object should be removed from the room except those absolutely needed. All the children in the family should be kept from school, and contacts who have angina should not be allowed to mingle with well persons.

Sheets soaked in a 1 to 2000 bichloride solution should be hung in front of the door of the sick-room, and all bed linen and clothing used by the patient should be soaked in a 1 to 5000 bichloride solution before being sent to the laundry, then boiled and sun-dried. Clothing of the nurse or attendant should be washable, and she should wear a close-fitting cap to prevent the hair from becoming contaminated. She

should also spray her throat twice daily with 1 to 5 glycothymoline solution, liquor alkalinus antisepticus, or with hydrogen peroxide.

The attending physician should never enter the sick-room without removing his coat and vest and donning a linen gown and close-fitting cap. At the termination of his visit he should remove the robe and cap just outside the sick-room, disinfect his hands and face, and go home to change and air his clothes before visiting other patients.

The sick-room should be thoroughly disinfected after the patient is convalescent before it can again be used, and the mattress had better be burned. During occupancy as much sunlight as possible should be allowed to enter the room, and it should be kept at an even temperature of 70° F., and be well ventilated; an open fireplace is of great advantage.

While the fever is high milk is the best food, and after the temperature declines a bland soft diet may be allowed. The child should drink plenty of water, and when the fever runs high (103° F. and above) tepid or cool spongings will afford relief.

The skin should be anointed with a boric acid ointment or with cocoa butter to lessen the tension; if the itching is severe, with a 1 per cent. phenol ointment. Cold cream, sweet oil, 5 per cent. ichthyol ointment, and oil of eucalyptus have all been used to relieve the skin irritation. The scales should be removed by daily baths of tepid water and green soap.

The throat symptoms require treatment according to their severity; when mild no local applications are necessary, although saline or boric acid washes and sprays may be used as prophylactic measures. If an exudate is present in the throat, 25 or 50 per cent. hydrogen peroxide solution, or 1 to 5000 bichloride, or 1 to 60 carbolic acid solution may be used locally as a spray. If the nose is involved, it should be gently irrigated with a normal saline solution.

Cold applications externally, such as ice-bags to the throat, afford great relief and are very soothing. Careful attention to the throat tends to prevent otitis; but, if it occurs, the pain can often be relieved by gently syringing the ear with warm normal saline solution. The ears should be inspected daily, and if bulging of the tympanic membrane is observed myringotomy should be performed immediately.

Nephritis is best guarded against by prolonged rest in bed and daily urinary examinations. The actual treatment of a complicating nephritis consists in restriction of the diet to milk and enforced rest in bed. In addition, the bowels should be kept opened freely, 1 or 2 grains of calomel in divided doses being given at the onset, and followed by magnesium citrate, 2 to 6 drams, or magnesium sulphate, 1 to 2 drams. Alkaline diuretics are also indicated and 5 to 20 grains of potassium citrate may be given every three hours.

Hot packs tend to make the skin more active, thus relieving the overburdened kidneys, and may prevent uremia and convulsions. If convulsions or uremic symptoms develop, the child should be put

in a hot bath. If this has no effect, a hypodermic injection of $\frac{1}{30}$ to $\frac{1}{20}$ of a grain of morphine, and $\frac{1}{400}$ of atropine may be given. Lumbar puncture often gives excellent results in these cases.

If a diphtheritic-looking exudate appears during the first week, it is usually a safe plan to administer 2000 to 5000 units of antitoxin, which can be repeated if it proves to be true diphtheria. In adynamic cases 20 to 40 drops of brandy may be given every three or four hours, and if the heart is weak $\frac{1}{3000}$ to $\frac{1}{100}$ of a grain of strychnine with 1 to 3 drops of digitalis may be administered at like intervals.

Malignant cases require continuous and powerful stimulation with whisky, 15 to 40 drops, caffeine, gr. $\frac{1}{4}$ to $\frac{1}{2}$, and camphorated oil, 10 to 15 drops hypodermically every two to four hours, according to indications.

Cerebral irritation may call for the administration of either sodium bromide, 5 to 10 grains, Dover's powder, $\frac{1}{2}$ to 1 grain, or codein sulphate, $\frac{1}{30}$ to $\frac{1}{15}$ of a grain, three times a day. Vomiting and diarrhea, if troublesome, may be allayed by giving bismuth subnitrate, 10 to 20 grains, with paregoric, 5 to 15 drops, or Dover's powder, $\frac{1}{4}$ to 1 grain, three times a day. For digestive disturbances in young children, small doses of calomel and bismuth are very efficacious.

When the joints are involved they should be immobilized, methyl salicylate applied, and the joint then warmly wrapped in cotton-wool. Sodium salicylate, 5 to 10 grains, combined with twice this amount of sodium bicarbonate, should be given three or four times daily. If suppuration takes place in the joint, surgical intervention is necessary.

The constitutional treatment of scarlet fever is largely symptomatic, since there are no known specific drugs. If the rash does not appear within the usual time, warm baths, a mustard bath, foot-baths, or a hot pack should be given, also hot drinks containing 10 grains of citrate of potassium, or 5 to 10 drops of sweet spirits of nitre. Constipation must be prevented. Antipyretics are contra-indicated; but ice-bags to the head are often a great comfort when the fever is high.

Serum Therapy.—Good results have recently been reported from the use of antistreptococcic serum and from serum obtained from convalescents. In those cases where the effect is favorable, one may note after an injection a drop in the temperature, a decrease in the pulse rate, and improvement in the force and rhythm of the heart. From 20 to 80 c.c. of antistreptococcic serum a day should be given in all malignant and complicated cases, as long as the dangerous symptoms continue.

Convalescence.—During convalescence the child should be on a full, nourishing, and easily digested diet. A hot soapsuds bath should be given every second day, and this followed by an inunction of olive or sweet oil. After desquamation, if there is no nephritis, the child may play about out of doors; but should not be allowed to come in

contact with other children for six weeks after the onset of the disease. If there is anemia, Basham's mixture, 10 to 20 drops, or tincture of ferric chloride, 2 to 5 drops, may be given three times a day after meals. The syrup of ferric iodide, 10 to 20 drops, and cod-liver oil, ½ to 1 dram, are also valuable tonics at this time. If possible, these children should be sent away to the shore or mountains to recuperate.

VARICELLA (CHICKEN-POX).

Varicella is an acute contagious disease, characterized by an eruption of vesicles on the skin together with mild constitutional symptoms. In the vast majority of cases, one attack of chicken-pox protects the individual against the disease for life, and because nearly everyone contracts it during childhood it is rarely seen in adults. Varicella is quite separate and distinct from vaccinia or variola, and an attack of chicken-pox affords no protection against these diseases.

Furthermore, inoculation with the virus of varicella, when successful, will produce varicella, and no other disease; likewise, variola inoculation will always produce smallpox. Vaccinia, varicella, and variola may occur successively in the same person within a relatively short space of time.

Etiology.—Chicken-pox occurs both in endemic and epidemic form, though sporadic cases are frequently seen. No specific microorganism has as yet been isolated. It is usually contracted by direct contact or conveyed by a third person, and may also be air-borne for a short distance. It is chiefly a disease of childhood, occurring in the vast majority of instances between the second and sixth years; but no age is absolutely exempt, as it is occasionally observed in very young infants as well as in adults. It is highly contagious, in this respect being somewhat like measles. Most cases are observed during the autumn months. The period of incubation is usually between two and three weeks, but it may be a little longer or a little shorter.

Symptoms.—In the majority of cases there are no prodromes, but there may sometimes be a feeling of malaise during the later stages of the incubation period, while a day or so before the eruption appears there may be slight fever, headache, restlessness, and muscular pain. In exceptional cases a prodromal scarlatiniform rash has been seen upon the trunk.

The onset occurs with fever, often with chills, and there may be angina, conjunctivitis, and even convulsions in severe cases. Occasionally there is vomiting, with pains in the back and legs. Often the first symptom noted is the eruption, which usually appears first either on the face or the trunk, but may occur without any characteristic grouping upon any part of the body. In some instances exanthemata may be seen upon the mucous membranes of the mouth, palate, tongue, and throat, and exceptionally on the nasal and vulvar mucous membranes.

As a rule the eruption is scant upon the extremities, the hands and feet being rarely affected. It appears in the form of small, red, elevated papules which, within a few hours, are transformed into vesicles containing a clear fluid. The papules generally appear in crops, soon followed by succeeding crops of new ones on the same or other parts of the body. The first crop of papules generally dries up before the appearance of the next one. In consequence, all stages of the eruption may be seen at one time upon the same part of the body, this being a diagnostic sign of great value.

There is usually no umbilication of the vesicles. In the majority of cases they are flat, and vary in diameter from one-eighth to one-fourth of an inch. Seldom is the skin about the vesicle reddened or infiltrated. After the first or second day the fluid in the vesicles becomes purulent and, within twenty-four to forty-eight hours later, the vesicles show signs of drying up, this beginning in the centre, and producing a slight depression which somewhat resembles umbilication.

Crusts form which, according to the degree of skin involvement, fall off any time between the seventh and twenty-first days. Only after the most severe cases, and when secondary infection has taken place from scratching, are any marks left, pitting depending upon whether or not there has been involvement of the true skin. Even in such an event, the pits are few, and most apt to be on the face. A diagnostic point of value is the fact that pocks are almost invariably seen on the scalp, which is rarely the case in smallpox.

Frequently the rash becomes so modified that it assumes a peculiar appearance. Necrosis may occur about the pock, thus producing a condition known as *varicella gangrenosa*. This type of eruption is most commonly seen in institutions, where the children are apt to be puny and poorly nourished, and in these cases the disease may be fatal. It is believed to be due to a mixed infection. Or, the vesicles may become exceedingly large, and resemble bullæ, having all the appearance of pemphigus, this condition being known as *varicella bullosa*.

The constitutional symptoms in varicella are usually mild. The fever rises to 101° or 102° F. on the appearance of the eruption, and does not usually decline until the second or third day. It is slight except in severe cases, when it may rise to 104° or 105° F., and last four or five days. Usually it returns to normal as the rash gradually disappears, but may again rise with the appearance of a fresh crop of vesicles. Only when the skin lesions of varicella become secondarily infected does the temperature remain elevated for more than a few days.

Hemorrhagic varicella is exceedingly rare; it is usually accompanied by bleeding from the mucous membranes.

Diagnosis.—This is obvious if the case has been seen from the onset. The marked predominance of the rash on the trunk, the appearance of the eruption in crops of papules which change into vesicles and crusts, and the slight umbilication resulting from the drying of the

vesicles, ought to render the diagnosis easy. In the absence of an epidemic of smallpox, the differentiation between smallpox and chicken-pox is not difficult; but when there is an outbreak of variola mild cases of smallpox may closely simulate varicella, and a severe attack of chicken-pox closely resemble variola.

It should always be borne in mind that in varicella the temperature is lower and the duration of the fever shorter than in even very mild smallpox. The history of a recent successful vaccination renders the possibility of smallpox in that particular individual most unlikely. The superficial character of the pocks and their appearance in successive crops, the absence of infiltration and true umbilication, together with the mildness of the constitutional symptoms, are important factors in eliminating smallpox.

Impetigo may be simulated by the dried crusts of chicken-pox; but in impetigo there are no constitutional symptoms, no lesions upon the mucous membranes, and the eruption persists for a longer period than do the skin manifestations of varicella. If the suspected case be chicken-pox, it can probably be traced to other cases, and it will run a shorter course than impetigo, the lesions disappearing without treatment.

Complications.—Complications and sequelæ are seldom observed, but occasionally pneumonia, pleurisy, bronchitis, laryngeal stenosis, otitis media, synovitis, or arthritis may be encountered. Polioencephalitis is a very rare complication which causes stupor or extreme restlessness and paresis of the extremities. A fatal termination· is rare, even in these cases.

Where there is suppuration about the pocks erysipelas may develop, and often proves fatal. Nephritis, though not a frequent complication, may occur either at the height of the disease or during convalescence. The urine passed by children who have chicken-pox often shows a trace of albumin.

Other contagious diseases, particularly scarlet fever, may complicate varicella, especially in institutions; occasionally we may see simple or suppurative adenitis.

Prognosis.—Simple uncomplicated cases almost invariably recover, for varicella is the mildest of all the acute contagious diseases with the possible exception of rubeola. Even in complicated cases death is uncommon, and a fatal outcome is never to be feared except when erysipelas, sepsis, or varicella gangrenosa occurs in weak, debilitated children.

Treatment.—Children suffering from the disease should be isolated from other children for a period of at least two weeks, or until the crusts have completely disappeared, as it is while the vesicles are present that the contagion is at its height. If there is elevation of temperature, rest in bed should be enforced, and the diet restricted to liquids while the symptoms are acute. An initial purge of castor oil, 1 to 4 drams according to age, or 2 to 6 drams of citrate of magnesia, is also advisable.

41

In the majority of cases the constitutional symptoms are so mild as to require little or no treatment. The itching may be allayed by the application of a 2 to 3 per cent. solution of carbolic acid, or a saturated solution of sodium bicarbonate, or by the use of tincture of iodine or carbolized vaseline (2 to 3 per cent.). Warm baths will also relieve the itching, as will the application of the following ointment:

R—Acidi salicylici,
    Thymolis . . . . āā gr. xx
    Ung. zinci oxidi . . . . ℥ij—M.
Sig.—Apply locally night and morning.

Cases in which the rash is abundant, particularly upon the face, should be prevented from scratching the pustules by keeping the finger-nails closely trimmed and by applying splints to the elbows, if necessary, so that the affected parts cannot be reached; also by enclosing the hands in mittens tied securely at the wrists.

If the vesicles are punctured as they become pustular, and a saturated solution of boric acid is applied, secondary infection and resultant pocking may be averted. A mild ointment, such as vaseline, may subsequently be applied to the crusts.

MEASLES (MORBILLI—RUBEOLA).

Measles is an acute contagious disease, occurring in epidemics, and characterized by an early eruption on the buccal mucous membrane and catarrhal symptoms, later by a rash which covers the entire body, and by the development of bronchitis.

Etiology.—The specific microörganism that causes measles is as yet unknown, but its viability outside the human body is not great. The disease can be reproduced in man by subcutaneous inoculation with the blood taken from a measles patient during the twenty-four hours before the eruption, and thirty hours after the first appearance of the eruption—in all fifty-four hours.

According to Anderson and Goldberger, the virus can pass through a Berkefeld filter, and may resist desiccation for twenty-five and one-half hours. Its infectivity is destroyed by heating at 55°C. for fifteen minutes. It resists freezing for twenty-five hours, and possibly retains some infectivity after keeping twenty-four hours at 15° C. A monkey which has acquired the disease by contact or inoculation remains immune.

The virus exists in the blood and in the secretions of the nose, eyes, mouth, and bronchi of infected individuals, especially during the height of the disease, rapidly lessens as the rash fades, and disappears entirely with the disappearance of the rash. The disease is only slightly, if at all, contagious after the fading of the rash. It has been produced by inoculation with the blood, and the discharge from the eyes, nose, mouth, pharynx, bronchi, and vesicles of a patient

who has measles, but the contagion does not exist in the scales. No growth in any way connected etiologically with the disease can be obtained from the blood of a measles patient.

Measles is easily spread from one child to another. A very short exposure is sufficient for contracting it, close contact not being necessary. Infants under six months are less susceptible than older children, which is also true of rubella, and, in a less degree, of scarlet fever, but all other children, if exposed, usually contract it. The non-susceptibility of the newborn to measles is shown by the following case which came under my notice:

Mrs. S. was seen on the morning of February 28, with coryza, a cough, a temperature of 102.4° F. The rash of measles covered her face and upper neck, and there were also a few isolated spots on her body. The following morning, after a normal labor at the end of the full period of uterogestation, a male infant, presenting absolutely nothing abnormal, was born. By this time the rash had spread over the entire body of the mother. The baby was free from rash, had a normal temperature, and presented nothing unusual. It was at no time isolated from its mother, was nursed regularly every two hours, and treated exactly as if it were not exposed to the contagion of measles. With the exception of a severe laryngitis, the mother made an uninterrupted recovery. When the infant was nine days old, another child of the family, a boy aged eighteen months, developed a typical attack of measles. Up to the age of six weeks, the baby had shown no symptoms of the affection.

The disease may be acquired *in utero*, the mother having the disease, and the child show the typical signs of measles at birth or within a few days after birth. A. Bartsch reports a case where the infant was born on the day when the rash first appeared on the mother. When the baby was three days old, it presented the rash, temperature, and, indeed, a perfect clinical picture of measles. The child recovered without any complications.

Another illustration of measles in early infancy is shown by the following case which occurred in my service in the Philadelphia Hospital:

A baby was born December 4, 1912. The mother showed the rash of measles January 6, and it appeared on the baby on January 17. On January 20, the baby's temperature was normal, and the rash had faded. The rash in this case appeared forty-four days after birth.

Epidemics are more severe in the winter and spring. The period of incubation is from nine to seventeen days, usually about fourteen days. The catarrhal symptoms generally appear eleven days, and the rash fourteen days after exposure to the disease. Measles occurs in all parts of the world. Boys and girls are equally susceptible.

About 60 per cent. of all cases occur under five years of age, and almost 30 per cent. of the remaining cases in the next five years of life. If, however, measles is brought to a community which has never previously been infected, people of all ages are attacked by the disease,

the old as well as the young. Many cases happen in young men and women who come to cities from isolated rural districts where they have never been exposed to the infection. Taking up their residence in a community where measles is always present they contract it. In the adult it is rare simply because almost everyone has the disease in childhood. It is, however, more common in young adult life than is scarlet fever.

A third person may convey it, but only for a short time or distance, or it may be carried on the clothes, or on fomites which have been used by the patient. The disease is often contracted by large numbers of children from a single case occurring in a school or at some entertainment given for children. It is contagious from the earliest catarrhal symptoms, and almost all who are exposed contract the disease. A second attack is rare, but may occur, and three attacks in one individual have been reported.

Measles is endemic in all large cities, but most of the cases occur in epidemics which recur at irregular intervals of two, three, or five years. As almost all children of school age are exposed and contract the disease, the epidemic disappears only when all of the susceptible children who have been exposed have contracted measles, and it reappears when there is a new enrolment of pupils. As school epidemics commonly spread the disease, a single case calls for prompt disinfection. It is especially contagious from the first symptom of the catarrhal stage, and, to a less degree, during the later or desquamating period. The infection does not cling or last after convalescence and at the end of three weeks the patient and the sick-room are free from it.

Pathological Anatomy.—The main lesions are in the skin and mucous membranes. The skin shows a superficial inflammation, with congestion and swelling which involve the glandular structure and the papillæ. There is invariably a catarrhal inflammation of the eyes, nose, mouth, pharynx, larynx, trachea, and larger bronchi. The cervical and bronchial glands are swollen, there is swelling of Peyer's patches, and, occasionally, degenerative change in the kidneys. The inflammation in the eyes, nose, larynx, trachea, and bronchi is a constant and integral part of the disease, and is not to be considered as a complication.

The severity of this inflammation corresponds with the severity of the attack of measles. In severe cases in weak and delicate children, especially in hospitals, the inflammation may extend to the air vesicles, and produce bronchopneumonia, or the inflammation in the pharynx or larynx may assume a membranous form. The severe cases with marked involvement of the pharynx, larynx, or lungs are usually complicated with staphylococcic, streptococcic, or pneumococcic infection.

An epidemic of measles in a hospital where a large number of foundlings are housed is often a most serious disease, owing to the frail and delicate condition of the young children, and their liability to

infection with the streptococcus and pneumococcus. From a personal experience with a number of such epidemics in the Children's Department of the Philadelphia Hospital I am convinced that measles is a disease to be dreaded under such circumstances. General septicemia, due either to the pneumococcus or streptococcus, may also occur.

Symptoms.—Prodromes, such as drowsiness, loss of appetite, restlessness, and disturbed sleep are common.

Stage of Invasion.—The onset of the disease is gradual, with watery eyes, coryza, cough which is often croupy, and slowly rising fever, which usually reaches 104° F. by the fourth day, the highest temperature ordinarily coinciding with the full development of the rash. The catarrhal inflammation of the mucous membranes also gradually increases, the eyes become more watery, photophobia appears, severe coryza develops with a croupy cough, hoarseness, pain on swallowing, and soreness of the throat due to congestion of the tonsils and pharynx.

Koplik's spots—minute rose-colored spots with a central white speck—are seen on the mucous membranes of the cheeks or lips. The tongue is coated, headache and pain in the back are common, and nausea and vomiting may occur. The duration of this catarrhal stage is usually three or four days, although it may last only one or two days or may continue for seven days. Occasionally the onset is sudden with high fever, vomiting, even convulsions, and severe respiratory symptoms that suggest acute pneumonia. The symptoms during these first three or four days are quite constant, varying less than is the case in scarlet fever, although occasionally they differ greatly from those described, especially in children under three years of age. Occasionally the disease sets in with a chill followed by high fever.

The temperature in measles conforms usually to a definite type. The fever increases each day until the eruption is at its height, and falls quite rapidly after the eruption is fully established.
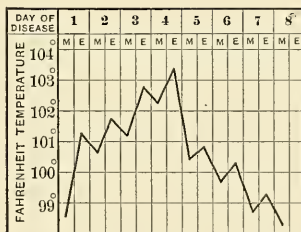
FIG. 68.—Typical Fahrenheit temperature chart of measles in a child of six years; rash over entire body on evening of fourth day.

Occasionally the fever is high at the onset—103° F.—falls to 100° F. on the second day, rises to 102° F. on the third day, and again reaches 104° F. with the appearance of the rash on the fourth day. A decline of the temperature on the second or third day may lead to

an error in diagnosis unless one appreciates that the gradual increase in the severity of the catarrhal symptoms is of great significance. The symptoms and fever are most marked at the time the eruption appears. During this stage of invasion, the diagnosis can usually be verified by Koplik's spots. If the mucous membrane of the mouth is examined in a strong sunlight, a few or many stellate or round rose spots are seen, and in the centre of each is a bluish-white speck. They appear from one to three days before the rash on the skin, usually on the inner surface of the cheeks and lips, and are of great assistance in forming an early diagnosis; occasionally, they do not appear until the rash is present, and they cannot be detected by artificial light.

Stage of Eruption.—The rash is first seen on the upper portion of the forehead, behind the ears, or on the neck, spreads over the face and scalp, and gradually extends over the trunk, arms, and legs. From the time of its first appearance until the entire body is covered is usually about thirty-six hours. When first seen it consists of small scattered red spots or macules, which rapidly increase in number, coalesce, and form slightly elevated papules. The rash is irregular in shape, often crescentic or oval; some blotches are large, others small, with more or less normal or slightly reddish skin intervening between the areas of rash. On the face the eruption is usually confluent, and the swollen face with discharging nose and eyes is very characteristic. The rash disappears on pressure, except in those cases where its color is a rather dark red, owing either to severe congestion of the vessels or to hemorrhage. This does not necessarily mean that the child is suffering from more than a moderately severe case. I have not infrequently seen this hemorrhagic rash in cases of measles that were of ordinary type and ran the usual course; sometimes, however, these cases were more than ordinarily severe. Cases with a rash that is more or less hemorrhagic must be carefully differentiated from the true hemorrhagic and the malignant forms of the disease.

The rash remains at its height for twenty-four to thirty-six hours, and then begins to fade, disappearing first from those portions where it appeared first, as the face and neck, and remaining latest on the lower extremities. It is often seen on the palms of the hands and soles of the feet. After the disappearance of the rash, a brownish staining of the skin persists for about a week. During this week, desquamation of the entire body occurs in the form of fine, branny scales, the scales being so minute that desquamation may, and often does, pass unnoticed. The more severe the rash, the more noticeable is the desquamation. During the spreading of the rash, the catarrhal symptoms, restlessness, nervous symptoms, fever, and cough are most severe. The catarrhal symptoms gradually disappear as the rash fades, although, during this period as well as earlier in the disease, the cough is often persistent and annoying, disturbing the child's sleep and adding greatly to its discomfort.

After the temperature declines to normal, the cough more or less rapidly lessens, loses its metallic or croupy character, and commonly

disappears during the second week, although it may persist much longer. The appetite is poor, the tongue coated, and symptoms of gastro-intestinal indigestion are common. The child complains of itching and heat in the skin, all the symptoms being most severe during the height of the rash. The rash does not always follow the typical course. It may be either distinctly papular or vesicular. The enanthem, or spots on the mucous membrane, may not appear; in rare cases the rash on the skin may be absent, although all the other symptoms appear. In mild cases the rash may be slight, of short duration, and followed by little or no desquamation. It is not uncommon for the rash to be darker than normal, of almost a purple color, and not to disappear on pressure.

Leukocytosis is present in the latter part of the period of incubation, but during or after the appearance of the rash is present only if some complication develops. Conjunctivitis persists during the time of eruption, and the eyes are sensitive to light. Convalescence in uncomplicated cases is rapid, and one week after the rash is fully developed the child is free from symptoms.
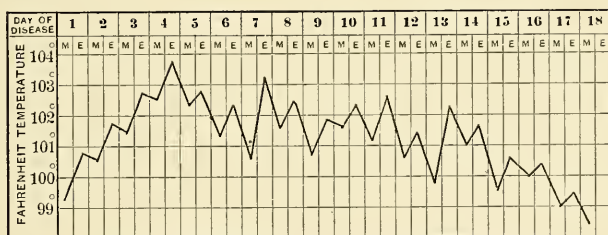
Fig. 69.—Typical Fahrenheit temperature chart in measles complicated by bronchopneumonia. Temperature normal on morning of eighteenth day; normal convalescence.

Hemorrhagic measles is marked by deep stupor, delirium, and convulsions. Hemorrhages in the skin and mucous membranes occur, the systemic symptoms are very severe, and a fatal issue is not uncommon. The so-called malignant cases are characterized by hyperpyrexia, marked prostration, severe nervous symptoms, decided toxemia, unusually severe inflammation of the affected mucous membranes, and either no rash or merely a few spots on the face. It is fatal on the second or third day. Hemorrhages may occur from the mucous membranes of the nose, mouth, stomach, or bowel, and blood may appear in the urine. A petechial rash may appear. Malignant measles may occur in epidemic form, or, occasionally, in an isolated case.

Lack of hygiene, general weakness, and poor nutrition increase the liability to malignant measles, and the infection in these cases is of unusual virulence. The lung symptoms may be severe, the tongue is dry, sordes appear on the teeth, and diarrhea is not

uncommon. The eruption is dark blue, and does not disappear on pressure.

Diagnosis.—Koplik's spots, which in the large majority of cases appear one, two, and, less often, four or five days, before the rash on the skin, are of great aid in forming an early as well as a differential diagnosis. They can best be seen under strong sunlight, but often must be carefully searched for. These spots are slightly raised and firmly adherent, but can be removed by firm rubbing or by forceps. They disappear soon after the appearance of the rash. Leukopenia, coryza, and fever are present at this early period, and a diazo-urinary reaction when the rash appears on the skin.

Rubella may closely resemble a very mild attack of measles, and a mild case of measles may be mistaken for rubella, but in rubella Koplik's spots do not appear, there are few prodromes, and none, or very mild catarrhal symptoms. The fever is usually 101° F. or lower, and is much shorter in duration. The rash is paler and smaller, spreads quickly, and does not persist as long as in measles.

Cases of scarlet fever in which the skin eruption is atypical and resembles measles can also be differentiated, as Koplik's spots are not present. The rash of sepsis may closely resemble measles; but, as a rule, it is not as regularly distributed as in measles, being more apt to be on the extensor surface of the extremities, and the catarrhal symptoms of measles are absent. Serum rashes can be similarly differentiated. The rashes occasionally produced by quinine, antipyrine, the salicylates, and turpentine may more or less closely resemble the rash of measles, but their distribution is usually irregular, and neither fever nor catarrhal symptoms appear.

Complications and Sequelæ.—In private practice, in robust children, where the child is seen early, and receives skilful nursing and systematic treatment, few complications occur. If, however, the child is delicate and under three years of age, complications are more common and more dangerous. I have had the opportunity of observing a number of epidemics of measles in the Children's Department of the Philadelphia Hospital, and in hospital practice, especially in delicate and poorly nourished children under three years of age, complications are common and often serious, and the mortality is high. Older children in hospitals do much better; complications are less common, less severe, and less dangerous.

Respiratory System.—Catarrhal inflammation of the larynx is present in the majority of cases, severe laryngitis is not uncommon, and membranous laryngitis occasionally occurs, being seen more often in hospital than in private practice. It may be streptococcic or diphtheritic. The membrane may appear in the pharynx, may be limited to the larynx, or may be present in both pharynx and larynx. The tendency to membranous laryngitis is much greater in measles than in scarlet fever. Severe streptococcic infection of the throat may produce marked local and systemic symptoms, and may cause death by a widespread streptococcic infection; moreover, as broncho-

pneumonia is often associated with a streptococcic infection, this dangerous complication may be added. Streptococcic laryngitis is usually associated with bronchopneumonia.

If membranous inflammation of the larynx is suspected, cultures should be taken immediately, and diphtheria antitoxin at once given. The pseudomembrane that develops in the larynx previous to and during the stage of eruption is usually due to the streptococcus; the membrane that develops later than this is usually true diphtheria. Both are much more common in hospital than in private practice, and most frequent in children under four years of age. Measles following diphtheria is not especially dangerous; Hellström had 8 deaths in 117 cases. Diphtheria following measles is much more serious, often being laryngeal in these cases, and early treatment with diphtheria antitoxin is necessary if life is to be saved.

Bronchopneumonia is the most common and the most important complication of measles, being seen much more often and being more dangerous in hospital than in private practice. The younger the child the greater the danger, especially in children under four years of age. Most of the deaths from measles are due to bronchopneumonia; even when pneumonia is not the direct cause of death, more or less pneumonia is revealed at autopsy. This is, however, only what might be expected, since bronchitis is present in all cases of measles, and the tendency in children, especially if frail and delicate, to develop secondary bronchopneumonia is very common.

Lobar pneumonia is occasionally a complication in older children, and usually ends in recovery. Pleurisy, either serous or purulent, may be associated with pneumonia, and increases the gravity of the prognosis. The association of measles and tuberculosis is not uncommon. Tuberculous meningitis and miliary tuberculosis frequently follow measles, but tuberculous bronchopneumonia is the most common tuberculous sequel. While it is, of course, possible that the tuberculous infection may occur subsequent to the development of the measles, it is nevertheless true that, in the large majority of cases, latent tuberculosis had previously existed, and the infection of measles, with the general catarrhal inflammation of the upper respiratory tract, had lighted up and made active this previously latent tuberculous process. Tuberculosis of the bones and of the cervical lymphatics are unusual sequelæ

Otitis is less apt to occur and is less severe than in scarlet fever; but is more common in children under four years of age. Usually both ears are involved, but without serious damage.

Stomatitis.—Catarrhal stomatitis is generaly present, and herpetic and ulcerative stomatitis occasionally occur. Gangrenous stomatitis, or noma, is fortunately very rare, although it occurs more often with measles than with any other disease. It is a very dangerous complication, but, if recognized early and energetically treated, is not necessarily fatal. It is rare outside of institutions and hospitals. Slight gangrene of the face, ears, genitals, and toes is not rare.

Eyes.—An ordinary catarrhal conjunctivitis is present in almost every case, and in institutions, or in weak and badly nourished children, this inflammation shows a tendency to continue and become chronic. Blepharitis and keratitis, acute or chronic, may occur. Choroiditis is unusual.

Digestive System.—Loss of appetite is common. Diarrhea is of frequent occurrence, especially after the fading of the rash, and may be mild or severe, and in young children is especially common in hot weather. Its prompt treatment is important, as, in the young and poorly nourished, it may prove a serious complication.

Nephritis as a complication of measles is rare, although in severe cases a trace of albumin is usually found.

Fig. 70.—Noma, or gangrenous stomatitis, following measles.

Nervous symptoms are not common. Convulsions occasionally occur in very young children, and may be associated with affections of the ear, brain, or lung.

Cervical adenitis is common, and may be subacute or chronic; suppuration rarely occurs.

Tuberculous adenitis is common, and *myocarditis, endocarditis* or *pericarditis* may develop. The skin is, as a rule, not affected other than by the rash; but furunculosis, pemphigoid eruptions, impetigo, and erysipelas are, in rare cases, complications. Measles may be associated with diphtheria, pertussis, or scarlet fever, and the association of pertussis and measles is quite common, the one often following the other; if the two diseases coexist bronchopneumonia is a common complication. Hemorrhages from the mucous membranes are rare. Second attacks of measles are extremely rare. A few cases have been reported of children who have undoubtedly had measles twice. Relapses are more common, the relapse being separated from the primary attack by an intervening period of a few days or weeks.

Prognosis.—This depends upon the severity of the prevailing epidemic and the previous health of the child. Epidemics are usually

more severe in cold than in warm weather. Age is an important factor; the younger the child the worse is the prognosis. Measles among the well-to-do and educated class is usually a mild infection, and in robust children is attended by but little danger. Among the poor and ignorant, especially if the child is frail and badly nourished, it is often a serious disease. If there is latent tuberculosis, it may be stirred into activity by an attack of measles, and become rapidly progressive.

NUMBER OF DEATHS IN PHILADELPHIA—MEASLES.

Age period.				1911.	1912.	1913.	1914.	1915.
Under 1 year	.	.	.	72	7	49	19	45
1 to 2 years	.	.	.	104	19	86	32	81
2 to 5 "	.	.	.	96	21	53	20	38
5 to 10 "	.	.	.	20	2	7	4	4
10 to 15 "	.	.	.	4	0	1	1	0
15 to 20 "	.	.	.	4	1	0	0	1
All ages	.	.	.	305	50	199	77	169

If the temperature has a tendency to remain high after the rash begins to fade, some complication should be suspected, and this often proves to be bronchopneumonia. Severe diarrhea following measles, especially in the young and wasted infant, is dangerous, particularly in the hot summer months. Convulsions usually occur only in severe cases, and then add greatly to the gravity of the prognosis.

Pseudomembrane in the pharynx or larynx demands the immediate use of diphtheria antitoxin, and I heartily approve of the practice, now so common in hospitals, and which I follow in my wards in the Jefferson Hospital, of giving each child on admission, an immunizing dose of diphtheria antitoxin. In institutions where a large number of young children are housed, and especially in foundling asylums, an outbreak of measles which attacks most of the children over six months of age is often associated with an epidemic of bronchopneumonia, and the mortality may be as high as 20 to 30 per cent. Eighty per cent. of all deaths in measles occur in children under five years of age.

Prophylaxis.—The patient should be isolated in an upper room or, preferably, in two rooms with a bath-room. Free ventilation is of the utmost importance. Measles patients require as much fresh air as any others, and in my wards for measles at the Philadelphia Hospital, as well as in my private practice, an abundance of fresh, moving air is allowed. A sheet is hung at the door and kept wet with 5 per cent. carbolic solution; all articles, such as bed linen, dishes, etc., removed from the room should be immersed in 5 per cent. carbolic solution.

It is important that all infants and young children be protected from the disease, also all delicate or badly nourished children, and those who have any tendency to respiratory diseases or to tuberculosis.

In hospitals and institutions all measles patients should be immediately removed from the ward. The quarantine for measles should

be twenty-one days; that is, the patient should be quarantined for seventeen days from the day the rash is fully developed. At the end of this period of quarantine, the room occupied by the patient should be thoroughly house-cleaned and disinfected, and the windows left open for three days to admit sunshine and moving fresh air. The room may then safely be occupied by other children.

Any children in the family where there is a case of measles who have been exposed should not be allowed to return to school until after the period of quarantine has expired. In a hospital ward, the daily examination of all exposed children for Koplik's spots aids in the early recognition of such patients, and their temporary isolation.

Treatment.—The eyes are cleansed frequently with a boric acid wash, and 1 per cent. unguentum hydrargyrum oxidum flavum applied to the lids. The nose, mouth, and throat should be kept clean by spraying with a mild alkaline solution. Liquor alkalinus antisepticus, diluted with four parts of water, is an agreeable and efficient preparation. This cleansing has a tendency to prevent the spreading to the ears and larynx of the catarrhal inflammation always present in the nose.

The patient should be kept in bed until the rash has entirely disappeared and the temperature has been normal for two or three days. He should have only enough bedclothes to keep him comfortable, and compresses, kept wet with cold water, should be applied to the eyes. The child's eyes should be shielded from direct sunlight, and the room should be kept rather dark. Dark glasses may be worn by older children.

An abundance of fresh, cool, moving air is essential unless the child has severe laryngitis, when warm, moist air is to be preferred. The child is kept on liquid diet during the period of fever, and the bowels kept moved each day. A tepid bath once or twice a day, or sponging with a bicarbonate of soda solution, a teaspoonful in two quarts of warm water, will partially allay the burning and itching of the skin.

The cough is usually the most annoying and distressing symptom, as, if persistent, it disturbs the sleep and aggravates the nervous symptoms. It is best controlled by heroin, gr. $\frac{1}{24}$ to $\frac{1}{36}$, or codein, gr. $\frac{1}{10}$, or Dover's powder, gr. j to ij, every two or three hours.

Nervousness, restlessness, and headache are best relieved by gr. j to ij of antipyrin or phenacetin, repeated in three hours if necessary. For sleeplessness, trional, gr. v, once in 24 hours, is of service. The child should be given each day a warm cleansing bath followed by an alcohol rub. If the fever has been 103° to 104° F. for only 24 to 48 hours at the time of the development of the rash, it requires no special treatment, but if of longer duration, and associated with restlessness and nervousness, it is best treated by an ice-cap, and a tepid bath, 95° to 85° or 80° F., with friction, the duration of the bath, five to twelve minutes, depending upon the resulting reduction of temperature.

In severe cases, if the hands and feet are cold, they should be kept warm with gloves, stockings, and hot-water bags. Cardiac depression should be combated with whisky, f℥ss to f℥j, every two to three hours; strychnine, gr. $\frac{1}{200}$ every three hours; camphorated oil, gtt. xv, hypodermically every four hours; tincture of digitalis, gtt. j to iij, every six hours, or caffein citrate, gr. j to ij every three hours.

Children with pneumonia as a complication should be separated from other measles cases. In hospitals and institutions all measles cases should receive an immunizing dose of antitoxin, and in private practice any suspicious evidence of diphtheria should be similarly treated. Bronchopneumonia should be treated as directed under that disease.

If, after the disappearance of the rash, cough persists, especially if associated with fever, bronchopneumonia is to be suspected, and the well-known tendency of such cases to become tuberculous should be borne in mind. Fresh air, rest, abundance of nourishing and easily digested food, the keeping of the digestion in good condition, and a suitable climate, with tonics—iron, arsenic and cod-liver oil—are all indicated.

Laryngeal symptoms should be treated with a croup tent, creosote being added to the water in the croup kettle. If the symptoms indicate the development of laryngeal membrane, diphtheria antitoxin should be given, and a culture taken. If fever persists without apparent cause, suspect the ears, and if any local evidence of inflammation can be detected, puncture of the ear-drums should be performed. It does no harm, and the escape of the pus or sero-pus may at once relieve the symptoms. The child should lie on the affected side after the puncture.

A child with bronchopneumonia following measles, especially in a hospital ward where there are other children between one and four years of age, should, if possible, be immediately isolated from all other measles cases, as the danger of an epidemic of bronchopneumonia is well recognized; it has twice occurred in my wards in the Philadelphia Hospital.

RUBELLA (RÖTHELN—GERMAN MEASLES).

Rubella is a contagious disease occurring in epidemic form, characterized by mild catarrhal symptoms, a diffuse eruption of rose-colored macules which may resemble either measles or scarlet fever, a slight fever lasting three or four days, and swelling of the superficial posterior cervical lymph glands.

Etiology.—It is less contagious than measles, and is most common in the winter and spring. The virus is longer-lived outside the human body than is the virus of measles. It usually occurs in epidemics which recur at intervals of two to four years, and is almost, if not quite, as common as measles. It is generally transmitted by direct contact but can be carried for a short time or distance by a third

person or by fomites. It is contagious from the earliest period of the disease until the disappearance of the eruption.

Infants less than six months old rarely contract rötheln, although it may occur before the age of six months, and may be present at birth or a few days after birth. Children between the ages of six and twelve months are more susceptible and after the age of one year all those exposed to rubella are likely to contract it. Like measles, all ages are susceptible. The specific virus of rubella is as yet unknown, but an attack of rubella does not protect from either measles or scarlet fever.

Symptoms.—The period of incubation varies from 5 to 21 days, although it is usually 10 to 20 days. Second attacks and relapses are rare.

Stage of Invasion.—The symptoms are generally very trivial and transient, and may be easily overlooked. There is slight coryza, pharyngitis, and laryngitis, associated with general malaise. The fever is moderate, 100° F. (37.8° C.) to 102° F. (38.9° C.). In the rather rare cases where the children are quite ill, the symptoms may be severe; headache, chills, a sensation of weakness, pain in the back, catarrhal symptoms about as severe as in measles, and a temperature of 100° to 103° F., with mild delirium, convulsions, and epistaxis; but these severe cases are not common. The prodromal stage lasts, ordinarily, only about 12 hours, although it may occasionally last three or four days. As a rule the longer the stage of invasion, the more severe is the attack.

Stage of Eruption.—The eruption generally appears in 12 hours after the initial symptoms; very often the rash is the first sign of the disease, the prodromes having been so slight that they passed unnoticed. The rash usually appears first on the face, and spreads over the entire body in 24 hours. The eruption remains at its height on any one portion of the body usually for about 6 to 12 hours, and passes like a wave over the face, thorax, abdomen, and extremities. It may be well-developed on the face, thorax, and abdomen before it has spread to the legs. It consists of small, round, or oval macules of a pale rose-color, slightly elevated and usually discrete, with areas of skin paler in color intervening.

As the rash develops it often becomes confluent, and may simulate closely the eruption of measles. The lesions on the skin are usually smaller and more regular than in measles, and are not apt to be crescentic. The rash is more apt to be confluent on the face than on any other portion; it is quite profuse over the scalp and is usually least upon the legs. It fades first where it appears first, lasts from two to three days, is followed by a fine, bran-like desquamation, and later by an indistinct pigmentation which lasts for only a few days. In other cases the rash is very fine, pin-head in size, and spreads over portions of the body like a blush, resembling closely the rash of scarlet fever.

In rubella and measles the rash appears on the lips; in scarlet fever,

the margin of skin around the mouth is usually free from rash. There is slight itching and the posterior cervical lymph glands are enlarged; usually, however, the lymphatic swelling persists for only a few days. The spleen is usually moderately enlarged; slight cough, a mild pharyngitis, slight laryngitis, coated tongue, and occasionally nausea and vomiting are present.

The severity of the symptoms varies greatly in different epidemics and, while the fever and constitutional symptoms are usually mild, epidemics have been reported where temperatures of 102° to 104° F. were common and associated with well-marked eruption and severe catarrhal and nervous symptoms.

Forcheimer has described an enanthem which lasts for 24 hours or less. The enanthem is seen upon the uvula, soft palate, and, less often, on the hard palate. It consists of macules of pink rose-red color, the spots are the size of large pin-heads, and are only slightly elevated above the mucous membrane. The prodromal period of rubella is short, usually about 12 hours, and this enanthem which appears and fades away in the first 24 hours is present at or before the time the rash is appearing on the skin, so that at the most only a few hours intervene between the appearance of the enanthem and the exanthem. It is important to remember that the enanthem only persists for 12 to 24 hours.

Diagnosis.—It is the variations in the rash that usually cause one to hesitate in making a diagnosis in the exanthemata, and in those cases where the rash is irregular and atypical, the diagnosis must often be made from studying the period of incubation, the prodromes, the initial and usual symptoms, the duration of the rash, the character of the desquamation, and the complications and sequelæ. In a doubtful case it is best to quarantine and withhold the diagnosis for a few days.

Rubella is to be diagnosed from measles and scarlet fever, and while this is usually an easy matter, in some cases it is difficult. If an epidemic of rubella exists, the diagnosis is simplified. Rubella shows a slight fever, an enlarged spleen, enlarged posterior cervical lymphatics, an eruption that appears in the first 12 hours, and is of short duration, and a rose-pink enanthem, the size of large pin-heads, on the uvula and soft palate, that appears either slightly before or at the time of the rash, and lasts for six to 12 or 24 hours.

Measles has well-marked catarrhal symptoms, a higher fever, rash on the third and fourth day, an eruption that persists longer and is often crescentic, Koplik's spots, and more frequently complications, especially bronchopneumonia. The difficulty arises in differentiating a mild case of measles, with very slight prodromes and moderate fever, mild catarrhal symptoms and slight rash, from a severe case of rubella.

In such cases, if there is an epidemic of measles, if the period of incubation is definitely known to have been 11 days before the catarrhal symptoms, and 14 days before the rash appeared, these

facts, and the knowledge that the catarrhal symptoms even in a mild attack of measles are usually much more severe than in a severe attack of rubella, point strongly to the diagnosis of measles and not rubella.

If the eruption of rubella is of the scarlet-fever type, it may be impossible to make a diagnosis from the appearance of the rash. If, however, other features of the case are studied, a correct diagnosis is usually possible. Scarlet fever has a shorter period of incubation (from three to seven days), the initial symptoms usually develop suddenly and are severe, with high fever, 104° F., and severe throat symptoms, and the enlarged cervical glands are at the angle of the jaw. There is pallor around the mouth, and a white line appears at the union of the finger-nail and pulp of the finger. The eruption persists for five or six days, and the desquamation is in flakes. The typical strawberry tongue is present, and ear and kidney complications are common.

Complications and Sequelæ.—The disease is so mild that complications are rare. Albuminuria, stomatitis, bronchitis, bronchopneumonia, erysipelas, and severe sore throat are occasionally seen. Less often, pleurisy, enteritis, rheumatism, endocarditis, otitis and eye complications develop.

Treatment.—The child should be isolated and quarantine should be maintained for two weeks. In hospitals or institutions, infants over six months of age, and young children should be especially protected from the disease. The child should be kept in bed until the rash has entirely disappeared, should be given liquid nourishment, the bowels should be moved each day, and any symptoms which develop should receive appropriate treatment.

DIPHTHERIA.

Diphtheria is an acute infectious disease characterized by the production of a false membrane on the mucous surfaces of the throat or respiratory passages. The tonsils, soft palate, pillars of the fauces, the uvula, the pharynx, and the nares are the parts most frequently affected. The larynx is often involved, either primarily or by extension, and the disease may be limited to this one part.

In mild cases constitutional symptoms may be totally absent, but in severe forms there is marked prostration and cardiac depression. Frequently pneumonia and nephritis are complications, and paralysis, local or general, may follow. Diphtheria occurs endemically, epidemically, and sporadically, and is, perhaps, less contagious than most of the other acute infections of childhood.

Etiology.—The disease is the result of infection by a specific microorganism, and is at first local; later, as the result of absorption of toxins produced by the bacilli, it becomes systemic.

The Bacillus diphtheriæ, also known as the Klebs-Loeffler bacillus, was first described by Klebs in 1883, and later isolated by Loeffler.

The organism is rod-shaped with rounded ends, and is either straight or slightly curved, varying in diameter from 0.5 to 0.8 micron, and in length from 2 to 3 microns. These bacilli vary greatly in form, and in the same specimen may be found either singly or in pairs. Sometimes they form chains or parallel lines, and often an obtuse angle.

The culture medium used influences greatly the form and size, the bacilli appearing smallest and most regular on glycerin agar, and as segmented regular staining forms when grown upon Loeffler's blood serum. The bacillus stains well with ordinary aniline dyes and with the Gram stain. The stain most frequently used is Loeffler's alkaline solution of methylene blue, which makes the granules easily seen.

In 1897, Neisser advocated the following method of staining in order to differentiate the bacillus from other organisms which may closely simulate it. As described by Abbott it is as follows: The culture tested should be grown upon Loeffler's blood-serum mixture, solidified at 100° C. It should develop at a temperature not lower than 34° C. or higher than 36° C., and should not be younger than nine, or older than twenty-four hours. A cover-glass preparation is made from such a culture, and stained from one to three seconds in the following solution:

Methylene blue (Grubler's)	1 gram
Alcohol (96 per cent.)	20 c.c.

When dissolved, mix with:

Acetic acid	50 c.c.
Distilled water	950 c.c.

The preparation is thoroughly rinsed in water, and then stained from three to five seconds in vesuvin (Bismarck brown), 2 grams dissolved in a liter of distilled water, filtered, and allowed to cool. It is again rinsed in water, and is examined as a water mount, or dried and mounted in balsam. The bacilli, when stained in this manner, appear as faintly stained brown rods in which can be seen one to three brown granules. The granules are usually oval, occupy one or both poles of the cell, and bulge slightly beyond the contour of the bacillus in which they are found. In most instances the bacilli that do not stain in this manner are considered distinct from diphtheria organisms (Abbott). The bacillus is non-motile, aërobic, liquefying, and does not form spores. It is also a facultative, anaërobic organism (Sternberg). In the dry state it maintains its vitality for a long period of time. Abel found bacilli on children's toys, which had been kept in the dark, five months after exposure.

The diphtheria bacillus may be found in the heart's blood, lungs, liver, spleen, kidneys, and lymph nodes, and is more easily demonstrated in these organs when the cases are uncomplicated. When diphtheria complicates scarlet fever, measles, and other diseases, there is usually a mixed infection, and staphylococci and streptococci are found, in addition to the Klebs-Loeffler bacillus.

42

Modes of Communication.—All cases of the disease have their origin in a previous case, either recognized or unrecognized. An individual may become infected by organisms in the air, or they may be taken into the mouth by kissing, or by the handling of toys, infected clothing, or other articles upon which they may have lodged.

Occasionally diphtheria organisms are found in the throats of apparently healthy individuals without their having contracted the disease. Such persons are known as diphtheria carriers, and may be a source of infection to others. But there seems to be much evidence in favor of the theory that there are non-pathogenic as well as pathogenic diphtheria bacilli; unquestionably there is a great difference in the virulence of the various types.

Pharyngeal diphtheria is more contagious than other types because of the excessive amount of discharge laden with the organisms, which may readily be a means of infecting others. It is sometimes complicated by retropharyngeal abscess, the result of streptococcic infection of the lymph nodes in that region.

A patient suffering with diphtheria may convey the disease to others for some time after the disappearance of the membrane. In some instances organisms have been found in the throat six to eight weeks after recovery. In large cities the presence or absence of bacilli in the throat after the termination of the disease is determined by means of a culture. Usually two negative cultures, taken on successive days, and at least twenty-four hours apart, are required before the quarantine is removed, provided fourteen days have elapsed from the date of onset. It is essentially a disease of children, and occurs only occasionally in adults. It is less common in the first year of infancy than in older children.

Individuals are frequently infected directly through furniture, hangings, dishes, spoons, and other articles which have been in contact with a patient suffering from the disease. Milk may be a mode of transmitting diphtheria, and in rare instances water. The chief predisposing factors in children are enlarged tonsils and adenoid growths in the pharynx. The bacilli may lodge in the crypts of the tonsils and in the cavities of decayed teeth, and remain there for some time. Certain of the acute infectious diseases, particularly measles and scarlet fever, so affect the mucous membranes as to make them extremely susceptible to diphtheria. It is most prevalent during the winter and autumn months. Predisposing factors are unhygienic surroundings, exposure to cold and dampness, faulty sanitation, overcrowding, neglect of the teeth and mouth, and chronic catarrhal conditions of the nose and throat. One attack in no way confers immunity against subsequent attacks of the disease.

Children between the ages of two and six years are more susceptible than any others, susceptibility becoming less after ten years of age.

Period of Incubation.—This period varies according to the virulence of the organism and the resistance of the individual. From two to five days usually elapse before the disease manifests itself. Con-

stitutional symptoms do not appear, as a rule, until after the character-
istic exudate is seen. Nevertheless, in the majority of instances, the
period of incubation is short.

Symptoms.—Frequently there are slight prodromes, such as head-
ache, anorexia, nausea, vomiting, and general malaise, prior to the
appearance of characteristic symptoms. The lymphatic glands near
the angle of the jaw may become tender, and there may be slight
difficulty in swallowing. Sometimes convulsions may be the only
symptom to usher in the disease. There is slight elevation of tempera-
ture, frequently accompanied by a sense of chilliness. Gastro-intestinal
disturbances are usually of no moment. In the laryngeal form the
earliest symptom is huskiness of the voice, which is noticeable long
before the appearance of the membrane, and is usually accompanied
by a brassy cough together with dyspnea due to slight laryngeal
spasms.

The Throat.—In the throat, as a rule, is seen the first evidence of diph-
theria, the tonsils being the favorite seat for lodgement and multiplica-
tion of the organisms. Usually the tonsils are swollen, the fauces
congested, and on the surface of the tonsils and in their crypts may be
seen spots of exudate which rapidly spread and coalesce, often covering
the entire tonsil. In the severe forms, especially when both tonsils are
involved, the exudate has a tendency to spread to the fauces, covering
both the anterior and posterior pillars, also the posterior wall of the
pharynx, uvula, and soft palate. The nares and larynx often become
involved.

The exudate itself is either yellowish-white or dark gray, may be
either thick or thin, and is usually extremely adherent, so much so
that when removed it frequently leaves a bleeding surface. A distinct
odor is observable after a few days, and often this alone makes one
suspect the presence of the disease.

Microscopically the membrane is found to consist of a network of
fibrin, enclosing within its meshes epithelial and round cells which
show evidences of degeneration; in the superficial layer are usually
found a variety of other organisms as well as diphtheria bacilli. The
epithelial cells of the affected mucous membranes show degeneration
with fragmentation of the nuclei, and there is leukocytic infiltration of
the mucosa which sometimes extends into the submucous and muscular
layers.

In some cases there is a resemblance to follicular tonsillitis, with
practically no membrane formation, the fauces exhibiting merely an
angina. The larynx is rarely involved in the mild and localized cases,
and there may be few, if any, symptoms referable to the throat, so
that, unless discovered on systematic examination of the throat, the
diphtheritic aspects of these cases may pass unrecognized.

In certain other types the diphtheritic lesions appear as strips and
specks of exudate scattered over the tonsils, uvula, and posterior
pharyngeal wall. These cases usually present mild constitutional
symptoms, and are most common in older children. The onset is

gradual, and is accompanied by a slight feeling of malaise and sore throat. The temperature rises to 101° or 102° F., and the lymph nodes behind the jaw become slightly enlarged.

In some cases the children do not go to bed, so mild are the symptoms. When these mild localized forms occur during infancy, gastro-intestinal manifestations often predominate, and there may be few symptoms referable to the throat, except slight swelling of the lymph nodes at the angle of the jaw. The fever ranges from 101° to 102° F., and the pulse and respiration are accelerated. In many infantile diphtherias, anorexia, and diarrhea with green, foul-smelling stools are the most marked symptoms. Owing to the prompt recognition of diphtheria and the liberal use of antitoxin, the majority of cases do not progress beyond this mild stage.

Severe types do occur, however, and in these instances the membrane not only forms extensively in the throat, but may also be in the nose, and occasionally in the larynx. The membrane changes from a grayish-white to a dirty greenish hue, and the child becomes extremely toxic. There is notable depression of the pulse, also extreme prostration, difficulty in breathing, great swelling of the cervical glands, and of the lymph nodes at the angle of the jaws. The temperature is at no time very high, and varies between 101° and 102° F. throughout the greater part of the attack.

The child is apathetic, with spells of extreme restlessness, has no appetite, suffers pain on swallowing, vomits whenever it attempts to eat, and frequently has diarrhea. Pain in the back and headache are usually quite severe.

Albuminuria is always present in these cases, and not infrequently hyaline and granular casts may be found in the urine. In the severe types of diphtheria, a well-marked diffuse nephritis occasionally causes death.

Nasal Diphtheria.—The *nose* is the common seat of involvement, after the fauces. Although the lesions here are frequently primary, the nose more often becomes involved by extension of the exudate from the throat. The mouth is kept open, the child snores, has a foul breath, and is almost unable to swallow. As the membrane increases in thickness, the nasal cavities become occluded, and prevent the discharge from escaping; it now changes from a serous type, and becomes ichorous and blood-stained. This in turn prevents nasal breathing, and gives to the voice a nasal tone. On examination of the nose a whitish or greenish membrane is seen, and the anterior nares are eroded. After the detachment and expulsion of the membrane, which may sometimes be in the form of a distinct cast, there is a return of the nasal discharge, which may be copious.

In considering the subject of nasal diphtheria it is important to remember that the Klebs-Loeffler bacillus may produce only the slightest irritation and discharge, no membrane being present; or it may produce the most intense inflammation with swelling and edema and membrane so extensive in amount as to more or less com-

pletely block the nares. The amount of contagion is usually in direct proportion to the amount of discharge from the nose and nasopharynx. However, diphtheria may be, and not uncommonly is, spread from a case where little or no discharge is present, and yet Klebs-Loeffler bacilli in small or large numbers, and virulent, may be demonstrated as present in the nose.

When one considers that these cases are apt to be overlooked; are not ill enough to wish to be confined to bed or within a certain room or rooms; have perhaps very slight local manifestations and no constitutional symptoms, it is not to be wondered at that parents fail to see the necessity for strict isolation, and that a more or less casual examination by the physician does not disclose the real nature of the affection; and as membrane is more commonly limited to the posterior than to the anterior nares, its presence may be overlooked.

Such cases are often the focus from which epidemics in a school or hospital ward may have their origin, and, while perhaps not dangerous to the infected individual, are a great menace to the community. It is impossible to say in any given case how long the contagion may exist, whether a severe inflammation with membrane and profuse discharge is present, or a mild case with slight angina and no membrane; but it is absolutely necessary, for the safety of others, that all such cases should be isolated until no local manifestations remain, and all the diphtheria bacilli have disappeared.

If, however, as will occasionally happen, the bacilli are found for more than a reasonable time, say two weeks after all local evidences of the disease, such as membrane, inflammation and discharge, have disappeared, then it is quite possible that these bacilli are not virulent and a guinea-pig should be inoculated, to test their virulence. It is also necessary to draw particular attention to the fact that it is often extremely difficult to distinguish between the true and pseudodiphtheritic bacillus, many bacteriologists claiming that it may be impossible to do so. If inoculations with guinea-pigs were more commonly carried on, and thus the virulent Klebs-Loeffler bacilli separated from those non-virulent, and the true bacilli from the pseudobacilli, there would be a more earnest desire upon the part of physicians at large to coöperate with health boards in the reporting of cases, and many cases would be released from an unnecessary and uselessly prolonged quarantine. In considering the length of quarantine it is well to remember that diphtheria bacilli may often retain their virulence for at least four or five months outside the human body.

The nose may be infected through the anterior nares. These are the cases where the infection is most likely to be limited to the nose. Both sides are more often involved than one side alone. The involvement of the sinuses and the antrum of Highmore may explain the persistence and the difficulty of removal of the bacilli in certain nasal cases.

Only those cases are called nasal diphtheria that show an involve-

ment of the nose alone. It does not include cases where pharyngeal, tonsillar or laryngeal diphtheria is present.

Mild cases may be described as those with few or no constitutional symptoms, little or no membrane, very little nasal discharge.

In moderate cases there are slight constitutional symptoms and persistent and often copious nasal discharge, which may be purulent and blood-streaked, excoriating the upper lip. It may persist for several weeks and yet the child be in apparently very good health. Membrane is usually present.

In severe cases the nares are obstructed and the child breathes with difficulty through the nose, the mouth is kept open, respiration is labored, the tongue dry. Membrane can be easily seen, and small or profuse nasal hemorrhages may occur. The discharge often has a decided odor. The submaxillary and glands at the angle of the jaw are swollen and a distinct toxemia may exist. The child is asthenic, the pulse is rapid, weak and perhaps irregular; there is stupor and decided anemia. The blood shows a reduction in red cells and of hemoglobin, the reduction being in proportion to the severity of the case. There is usually a leukocytosis.

The temperature is, as a rule, moderate—100° to 101.5° F. Albumin is usually present in the urine in severe cases. Hyaline and granular casts are present, but not in large numbers. Dropsy is uncommon. Nausea, vomiting and diarrhea are often present. A moderate degree of delirium is quite common. The membrane disintegrates slowly; it may be dislodged in a large cast or mass by a violent sneeze.

The constitutional disturbances which accompany this form of diphtheria may be more severe than those in the tonsillar type. They are due to the ease with which toxins are absorbed by the injured capillary bloodvessels at the seat of membrane formation. Nosebleed occurs even in the mildest types of the disease, and becomes in many instances extremely annoying. It is in this form of diphtheria that postdiphtheritic paralysis is most apt to follow. At times the disease assumes a chronic form, and has the appearance of chronic rhinitis. This is one of the modes in which infection is frequently conveyed to others, the individual affected being totally ignorant of the true nature of the discharge.

The local and constitutional symptoms lessen rapidly with the disappearance of the membrane. The anemia and evidences of a weak heart are usually, however, slowly recovered from.

The membrane which appears in the nose in most cases of scarlet fever and measles is not commonly diphtheritic, especially if it occurs early in the disease or during the height of the disease. Membrane occurring in the later stages of measles or scarlet fever is often diphtheria, and, if diphtheritic, the inflammation of the surrounding mucous membrane is, as a rule, not severe.

As a diagnostic aid it is well to remember that the membrane in diphtheria may be found only in the nose; membrane not diphtheritic is rarely so limited. Albumin in the urine is suggestive of diphtheria,

but if the case is a mild one, and no toxemia exists, albuminuria would, of course, not be expected to occur. Paralysis, especially regurgitation of liquids from the nose, would be in favor of diphtheria.

A cover-glass smear will often enable one to quickly make a diagnosis, but a culture is more accurate, and as the local and constitutional symptoms are usually very mild, a culture is much to be preferred. Klebs-Loeffler bacilli may disappear early in the disease, and may not be found when the membrane has largely disappeared. If membrane is present all bacilli, which culturally and morphologically are diphtheritic, should, in my opinion, be considered true diphtheria bacilli, unless proven pseudodiphtheritic by inoculation of a guinea-pig.

Any child who is shown to have diphtheria bacilli in the nose, even if it presents no clinical evidences of the disease, is a possible source of danger, and the child should be isolated until the bacilli are proven to be non-virulent. Children exposed to diphtheria are known occasionally to have the bacilli in the nose, and animal inoculations prove that these bacilli may be virulent, and yet these children may not clinically develop diphtheria. Children in bad hygienic surroundings and in institutions, particularly, show this tendency to harbor the bacillus, and to have the disease develop into clinical diphtheria, also to spread it to other children, to a much more marked degree than where the conditions from a hygienic standpoint are first-class.

In a doubtful case, especially if the child has been exposed to diphtheria, and a nasal discharge persists, a single negative culture is not positive proof that the case is not diphtheritic.

An interesting question, and one which requires much careful future study, is the report of return cases. I believe that future investigation will conclusively prove that return cases occur in a greater proportion of instances than is now believed, and I do not approve of the doctrine that a child discharged from quarantine with diphtheria bacilli in the nose, who is clinically free from diphtheria, is, as is claimed by some careful observers, not able to transmit the disease. In fact, one hears continually from physicians abuse of health boards because two successive negative cultures must be obtained before the child is allowed out of quarantine. What is the risk incurred by children who come in contact with this child who has been clinically free from diphtheria for some days, and who, nevertheless, has Klebs-Loeffler bacilli present in the nose? Is one warranted in sending these cases out of private homes and hospitals without inoculating a guinea-pig to test the virulence of the bacilli? Return cases to hospitals do not show a very large percentage of infections from such patients. C. B. Ker, of Edinburgh, states that "Dr. Cameron found that 1.2 per cent. of the total diphtheria cases were, after their discharge from hospital, supposed to have infected persons with diphtheria."

It is an interesting point, and one well worthy of discussion, as to how many of these return cases are real infections from the discharged

case, and how many are cases of coincidence; by that, I mean, happened to contract diphtheria at this special time from a source other than the discharged patient, but contracted it just at the time when it would appear that infection had occurred from the discharged case. It not uncommonly happens that a second child from a family is admitted to the hospital a few days *before* the first child is returned home. If this second child had entered the hospital a few days after, instead of a few days before the first child was returned home, it would have been classed as a return case. However, there is no doubt in my mind but that all such cases with diphtheria bacilli in the nose, all clinical evidences of the disease having disappeared, are possible sources of danger.

Every suspected case of nasal diphtheria should be quarantined and isolation continued until a bacteriological examination proves the case to be non-diphtheritic. Cultures should be taken from all exposed children; and those showing diphtheria bacilli should be isolated and the nose and throat appropriately treated.

In nasal diphtheria the bacteria may show an unusual tendency to persist, and it is believed this may be due to the nasal sinuses and antrum being involved. The danger of harboring virulent bacilli in these cases of long standing is not very great; the bacilli are usually few in number, and experience proves that the risk of contagion is comparatively slight. It is unwise to admit into a hospital ward or institution any child who shows a membrane in the nose or has a nasal discharge, unless a bacteriological examination has shown the absence of diphtheria bacilli; and all children in hospitals or institutions should be carefully watched for the development of such symptoms. It has been the experience of almost all physicians with hospital experience to see epidemics of diphtheria in hospitals originate from such cases. The number of visiting days and the number of visitors to children's wards should be limited as far as possible, and no children should ever be admitted as visitors. All visitors, and, in fact, all persons entering a children's ward should wear a sterilized cap and gown. Such a rule has been inaugurated at the Jefferson Hospital, and, I believe, is of decided aid in preventing outbreaks of the disease.

If the discharge is small in amount and there are no constitutional symptoms, it is unnecessary to irrigate the nose. In those cases where the discharge is abundant and the nasopharynx is more or less blocked with secretions, it is important to keep the nose and naso- pharynx cleansed with warm, mild antiseptic solutions. Normal salt solution, weak boric acid solution, 3 grains to the ounce, or borate and bicarbonate of soda, 5 grains of each to the ounce, may be used. All solutions should be used warm; no unnecessary force should be exerted, and the child should always be firmly wrapped from head to foot in a blanket. As the treatment is designed especially to wash out the nasopharynx, the fluid entering a nostril should escape from the opposite nostril and the mouth. A fountain syringe, in my opinion, is better than a piston syringe. Enough fluid should be used

to at least fairly well cleanse the nose and nasopharynx. It is rarely necessary to employ irrigation oftener than once in four hours. Nasal hemorrhage calls for great care in the use of the syringe, and unless the case urgently demands syringing, it is better to discontinue it, if hemorrhage has occurred.

Antitoxin should certainly be given in every case of nasal diphtheria, and, of course, the earlier it is given, the better the result will be. In an infant it is dangerous to wait until a diagnosis can be confirmed by a bacteriological examination. In older children with only slight nasal discharge and no toxemia one may postpone giving antitoxin until the bacteriological examination confirms the diagnosis. To postpone the early use of antitoxin in a case of profuse nasal discharge, with membrane present, is, in my opinion, utterly inexcusable.

Laryngeal Diphtheria or Membranous Croup.—This may either appear as true diphtheria or may be diphtheroid in character. In the true form there is a pseudomembranous exudation into the larynx and trachea which results in croup. The cause in this type is the same as in other types of true diphtheria, namely, the Klebs-Loeffler bacillus. In the diphtheroid type the pseudomembrane formed is the result of invasion by bacteria other than the diphtheria bacillus, chiefly streptococci, and occasionally staphylococci.

It has become the custom to treat all cases of membranous croup as diphtheria, even though it is conceded that other organisms than diphtheria bacilli may be the factors in pseudomembranous productions. In this type of the disease there is often little or no involvement of either the tonsils or the fauces, though the affection is believed in most instances to be the result of downward extension from these parts. Not infrequently cases are seen in which the disease is confined solely to the larynx. It is in this type that the exudate may extend into the trachea and bronchial tubes.

In both the true and false types the symptoms closely resemble each other, beginning with hoarseness, slight at first, and with a cough, rough in character, to which the name of croup is given, and which lasts for a period of one or two days. Suddenly these symptoms become intensified and the breathing paroxysmal as a result of spasm of the glottis. Dyspnea develops, inspiration is accompanied by retraction of the lower intercostal spaces and the epigastrium, and expiration is difficult.

The skin becomes livid, owing to imperfect oxygenation of the blood, restlessness is extreme, and an anxious expression due to fear of suffocation appears on the face, a condition which, once seen, is not soon forgotten. The fever is usually moderate throughout the course of the disease and attracts little or no attention. The pulse is rapid, and varies in frequency from 120 to 200. Usually in true membranous croup there is little, if any, amelioration of the symptoms during the daytime, such as often occurs in simple croup. Cough is paroxysmal in these cases, and membranous casts are frequently expelled.

In laryngeal diphtheria the early symptoms are vague, and are apt to be overlooked unless one is ever mindful of the possibility of diphtheritic involvement of the larynx. A little hoarseness, a croupy cough, and slight inspiratory dyspnea, as shown by recession of the suprasternal notch, supraclavicular spaces, and epigastrium are noted early in the case. These symptoms are progressive, and within six to twenty-four hours the disease is often fully developed.

The voice becomes hoarse, husky, and finally lost. The dyspnea becomes more and more pronounced, the inspiratory stridor gradually and continuously increases, the pulse becomes rapid, the face turns pale and is anxious, the alæ of the nose dilate with each inspiration, the child is restless, perhaps pulls at its throat, and puts its fingers into its mouth. All the accessory muscles of respiration are brought into action.

Gradually the child becomes weaker, drowsy, and heavy. Cyanosis of the lips and finger-nails develops; the skin is cold and covered with a clammy sweat. Since little air enters the lungs owing to the occlusion of the larynx with membrane, we hear very little vesicular murmur on auscultation.

Early in the disease the temperature is not higher than 100° or 101° F.; but as symptoms of suffocation increase, the fever rises to 103° or 104° F. The child becomes more and more quiet, it ceases to struggle, and in infants death may ensue in thirty-six to sixty hours after the development of the disease. Older children usually live three to five or six days.

Improvement in the symptoms usually occurs in those cases in which the false membrane is expelled in whole or in part from the larynx by a violent paroxysm of coughing or vomiting. Usually, however, the membrane quickly reforms, and the symptoms return.

In membranous croup, the result of invasion by bacteria other than the diphtheria bacillus, the onset is not so sudden, but the fever is usually high, and frequently there is involvement of the glands of the neck. Often a false membrane forms on the tonsils or fauces. In this type of the disease a culture taken from the membrane is the only method of determining the infecting organism.

The mild constitutional symptoms which accompany the laryngeal type may be accounted for by the fact that the lymphatic supply to the trachea and larynx is extremely small. The chief complication is bronchopneumonia, either the result of membrane extension into the bronchial tubes or inability to expel the mucopurulent material from the tubes.

Other parts which may become involved are:

1. *Middle Ear.*—By extension of the exudate through the Eustachian tube the middle ear may become involved, this resulting in acute otitis media. The tympanic membrane ruptures and suppuration takes place, the pus containing the bacillus of diphtheria together with other organisms. Although this is of rare occurrence, yet it is a possibility, and may produce temporary deafness. In some instances the deafness

may be permanent, this depending upon the extent to which the tympanic cavity is affected.

2. *The Eyes.*—These may be involved, either primarily or secondarily. Conjunctival diphtheria is rare, but when it occurs the membrane spreads rapidly from one eyelid to the other, and there is an associated chemosis of the bulbar conjunctiva. There is also a severe form of diphtheritic ophthalmia in which the cornea is perforated, and destruction of the eye often follows. In the milder form there is simply a slight seropurulent discharge.

3. *The Skin.*—The skin surrounding wounds and abrasions may become involved, and a membrane form over the wound or around it. This is most frequently seen about the mouth and lips as the result of extension in cases of pharyngeal diphtheria. When it occurs the diphtheria bacillus may be found in the exudate.

Diphtheritic processes have also been observed in the trachea and bronchi, in the stomach, and on the genitalia. Wounds made by tracheotomy and circumcision are occasionally infected by the Klebs-Loeffler bacillus, and in the newborn the umbilicus may be involved.

Septic Diphtheria.—During the course of diphtheria a septic condition frequently arises which is the result of infection by other associated organisms; namely, streptococci and staphylococci. This type of infection is most apt to be seen in the nasal form of the disease with associated diphtheria of the fauces, the exudate showing every evidence of decomposition, and resulting in a fetid discharge from the mouth and nose. This process is often followed by ulceration of the nasal mucous membrane with resultant hemorrhage. The lymphatic glands of the neck rapidly become involved, are swollen and tender, and may eventually suppurate. In this type of diphtheria, in addition to high fever and a rapid pulse, there is frequently a rash over the skin, at first erythematous, and later becoming petechial.

Other Symptoms of Diphtheria which Call for Special Attention.—1. *Fever.*—There is almost invariably fever at the beginning of the attack. It varies in intensity according to the virulence of the infection, and usually ranges from 100° to 102° F. during the first forty-eight hours of the disease, falling to normal two or three days after the exudate makes its appearance. Should the temperature remain high throughout the course of the disease, it should suggest the existence of such complications as adenitis and suppuration. In laryngeal diphtheria the temperature is high in the terminal stage.

2. *Disturbances of the Circulation.*—Very notable in diphtheria is the rapid pulse rate, which is out of proportion to the degree of temperature. The heart sounds become distant and weak.

3. *Nervous Disturbances.*—In children convulsions may usher in the disease. They are most often seen in children who are delicate and of a nervous temperament. Delirium, though usually mild, may be seen in any of the forms of the disease. Occasionally there is perversion of the senses of taste and smell.

Morbid Anatomy.—*Lymphatic System.*—The cervical lymphatics are most frequently affected. There is marked leukocytic infiltration together with minute hemorrhages, which ultimately result in resolution or suppuration.

Spleen.—This organ becomes congested and swollen. The splenic tissue is soft, and degeneration of the cells is almost always present, together with enlargement of the follicles. Infracapsular hemorrhage is of common occurrence.

Liver.—Degeneration and necrosis of the hepatic cells and leukocytic infiltration of these areas are frequently observed.

Kidneys.—As a result of the toxins circulating in the blood, degeneration of the epithelial lining of the uriniferous tubules occurs, and in the severe types of the disease a diffuse nephritis may be seen.

Heart.—Myocarditis, particularly of the left ventricle, is apt to appear and is of varying intensity, according to the duration and severity of the infection. Cardiac thrombi account for a certain percentage of sudden deaths, and also result in the production of emboli in the lungs, viscera, and arteries of the extremities.

Arteries.—Hyaline degeneration of the visceral arteries, infiltration of the adventitia, and degeneration of the endothelial layer, are the processes most likely to take place.

Blood.—Reduction in both hemoglobin and the number of red cells occurs in practically all severe cases of the disease. There is usually leukocytosis, though this may be wanting.

Nervous System.—The brain may be the seat of both hemorrhages and embolism, and the spinal cord reveals evidences of hemorrhage. The cord itself is apt to show degeneration of the ganglion cells of the anterior horns of the anterior and posterior nerve roots, also at times of the columns of Goll and the pyramidal tracts. In cases of mild diphtheritic paralysis the lesions found are usually peripheral. Certain nerves, such as the spinal accessory, hypoglossal, pneumogastric, oculomotor, and cardiac nerves may show changes of degenerative type as the result of the toxin, and when so affected produce lesions common to the disease, namely, multiple neuritis.

Lungs.—Bronchopneumonia is frequently found, and in a great number of instances is the responsible factor in causing death. It is usually the result of the aspiration of particles of membrane from the air passages, these containing streptococci and diphtheria organisms. Emphysema, when found, accompanies the laryngeal type, and, depending upon the duration of the illness, may be either vesicular or interstitial.

Complications and Sequelæ.—Hemorrhage may result from ulceration of either the nose or the throat, but is usually of minor importance.

Kidneys.—Nephritis is one of the most frequent complications, and is seen earlier in this disease than in scarlet fever, although its course is somewhat similar. In practically every instance of the disease albuminuria appears from the second to the fourth day, though the amount of urine may not be altered; in fact, the quantity is often

increased. When nephritis does develop it is the result of injury to the parenchymatous tissue by the toxins during their elimination. This will show its presence by the appearance of casts, both epithelial and blood, and occasionally hyaline and granular. The amount of urine becomes scanty, and at times there is suppression.

Lungs.—In laryngotracheal diphtheria the lungs frequently become the seat of bronchopneumonia, whereas in faucial diphtheria simple bronchitis is more apt to ensue. Lobar pneumonia may occur during convalescence, and the same may be said of pleurisy.

Heart.—It has been stated by Jacobi that, no matter how mild the case may be, the heart's function becomes affected to a certain extent. Endocarditis and myocarditis are both the result of the action of the toxins circulating in the blood. Heart paralysis occurs with greatest frequency in the severe forms of diphtheria in which the child is apathetic, and shows evidences of extreme prostration. These evidences of heart failure, when seen, usually manifest themselves at the height of the disease, which is usually about the seventh day. This failure may result from paralysis of the cardiac nerves without any change in the heart muscle. The symptoms of heart failure are frequently preceded by vomiting. There then occur cyanosis and rapid pulse, which later becomes slow and dicrotic, and the muscular sounds are indistinct. The extremities become chilled as a result of the failing circulation, though the mind is often clear until death.

All this may occur at any time during the stage of convalescence, often as late as the sixth or seventh week, and the child may have several attacks before one proves fatal. In other cases, sudden death may occur during convalescence from cardiac failure when there have been no preceding cardiac symptoms. Cardiac irregularity is common in children during convalescence, but does not necessarily prove fatal, and may disappear in the course of several weeks. Both measles and scarlet fever may complicate diphtheria, the reason being difficult to explain. When complicated by the former, the disease is apt to be more severe because of the changes in the mucous membranes produced by measles.

Diphtheria may also be a serious complication of pertussis, frequently causing bronchopneumonia. When it occurs during the course of typhoid fever or in tuberculous children it usually proves fatal.

Paralysis.—This is the most important of the sequelæ, and occurs after the acute stage of the disease, most often during convalescence. The exact cause is not definitely known, but it is regarded as a toxic neuritis which affects the peripheral nerves. Most frequently it involves the palate, this resulting in nasal speech and the passage of food through the nose instead of into the stomach. There may also be anesthesia of the mucous membrane of the pharynx. The muscles of deglutition are the next in point of frequency to be involved, and then the eye muscles, chiefly those concerned in accommodation. At times strabismus and ptosis may be seen, more rarely facial paralysis. There may be paralysis of the lower extremities, from which complete

recovery seldom occurs. Respiratory and cardiac paralysis may occur at any time during the stage of convalescence, and result in sudden stoppage of the heart's action and cessation of respiration.

There may be no preliminary symptoms, even in severe forms of cardiac paralysis. In other cases there may have been preceding cardiac irregularity, and often there is slight albuminuria. In many instances, the child is apparently in good health, when suddenly there is abdominal pain, dyspnea, cyanosis of the lips, slow, thready pulse with weak, scarcely audible, heart action, and coolness of the extremities. Not infrequently a child will survive one or two of these attacks only to succumb finally.

Gastro-enteritis.—Disturbances of the gastro-intestinal tract are dangerous, and not uncommon, complications of diphtheria in infants. The diarrhea may become very severe, and in rare instances death has resulted from extension of the membrane into the esophagus, stomach, and intestine.

Diagnosis.—The disease may be diagnosed both clinically and bacteriologically, though ofttimes the diphtheria organisms may be found in the throats of healthy individuals without any symptoms of the disease, consequently their mere presence does not constitute a diagnosis of diphtheria. Nevertheless, when they are present together with certain clinical evidences the diagnosis is complete. In a great number of instances the diagnosis is to be made from the clinical evidences alone, especially in cases where the exudate is rapidly spreading. In the milder atypical forms it frequently becomes necessary to determine by culture the presence or absence of diphtheria organisms in order to make a diagnosis and protect other members of the family.

Clinically, the following factors ought to be of some help in making a diagnosis. The presence of a flocculent nasal discharge which later becomes blood-tinged, enlargement of the cervical lymphatic glands, the early appearance of croup, which becomes progressively worse, the presence of a grayish patch on the tonsils which rapidly spreads, at times over the soft palate and the uvula, are very significant of diphtheria.

In instances where diphtheria is associated with scarlet fever or measles the variations may be so marked as to make diagnosis difficult. In cases of pseudodiphtheria the onset is, as a rule, rapid, the constitutional symptoms are marked, and there is a decided elevation in temperature. It rarely begins primarily in the larynx and only in extremely few instances is albuminuria an early symptom. The mucous membranes in pseudodiphtheria become exceedingly inflamed and the exudate present is easily removed by the use of a swab.

A diagnosis of the unusual types of the disease may be made by a bacteriological examination. The following is the method used by the Philadelphia Board of Health: Physicians can obtain from the various police stations envelopes containing two tubes. One tube contains two small wires; at one end of each wire a small amount of cotton is attached. The other tube contains the necessary culture medium

which is inoculated by rubbing the swab over any exudate present, and then gently passing it over the surface of the culture medium. The cotton stopper is replaced in the culture tube, the swabs are placed in their original tube, and both tubes are returned to the police station. They are then sent to the Laboratory of the Bureau of Health, where the tube containing the culture medium is incubated for a definite length of time, and then examined microscopically. The physician is notified by telephone whether or not any diphtheria organisms were found on the medium. Just prior to taking a culture no antiseptics should be used upon the throat, and great care should be exercised not to contaminate the culture medium with other organisms than those from the exudate. Sometimes we obtain a negative culture from a case of diphtheria which presents all the clinical evidences of the disease; again, we may obtain a positive culture from the throat of an individual who clinically has not the disease. In all cases, when clinical evidences are present, a positive culture constitutes a positive diagnosis.

The conditions with which this disease is most apt to be confused are the various forms of diphtheroid faucitis and the faucitis of scarlet fever.

Follicular Tonsillitis.—This disease is more often mistaken for diphtheria than any other throat affection. The exudate in this type does not show a tendency to spread and usually affects the tonsil crypts. It is easily detached, and when crushed gives off an extremely fetid odor. The constitutional symptoms are often intense. There are high fever, headache, chills, pains in the back and extremities. The throat is sore, swallowing becomes difficult, and there is tenderness about the neck at the angle of the jaw. Sometimes the disease may closely simulate mild diphtheria, and in such instances a bacteriological culture may be necessary to determine its true nature.

Scarlet Fever.—The absence of eruption is a great aid in making the diagnosis, although the eruption in scarlet fever may be wanting or extremely mild, and an erythematous blush may accompany diphtheria. In such instances a history of exposure or the prevalence of an epidemic should form a clue. Of course, when desquamation occurs the question is readily settled, but often, as in follicular tonsillitis, a bacteriological culture is necessary.

In scarlet fever, however, the redness in the throat is much more diffuse than in diphtheria, and when there is a membrane in the throat it appears to be embedded in the tonsil instead of upon it, as in diphtheria. Diphtheritic membrane, as a rule, forms late in scarlet fever, early membrane formation being due in most cases to streptococcus infection.

Catarrhal Croup.—During the early stages this is frequently confused with membranous croup. In true membranous croup there is no amelioration of symptoms during the daytime, but they become progressively worse, the hoarseness more intense, the breathing more difficult. Restlessness and cyanosis appear, and the typical tugging

of the diaphragm is seen. In catarrhal croup an emetic frequently relieves all of the symptoms, whereas in true membranous croup this is not the case and there is usually no exudate in the fauces.

Stomatitis.— In severe cases of this disease the minute ulcers may coalesce and form patches, which may cause confusion in diagnosis. The exudation in such instances is usually thin and never membranous, although here also it may be necessary to resort to bacteriological differentiation.

Prognosis.—The prognosis should always be guarded, for a positive one is impossible.

The mildness or severity of the attack does not necessarily have any influence on the prognosis because of the complications which may result and the rapidity with which the membrane frequently extends. At times what appears to be a very mild case suddenly becomes an exceedingly severe one; again, cases that are most severe in the beginning may suddenly change for the better, and make a rapid and uneventful recovery.

Much depends on an early diagnosis, and the giving of antitoxin immediately upon seeing the case, if the local or constitutional symptoms are severe. If the case is a mild one, we may wait for the result of the culture; but it is worse than foolish to do so in severe cases.

In uncomplicated cases in which antitoxin is given on the first day, the mortality is almost *nil*. If injected on the second day, the mortality is not more than 2 per cent.; if antitoxin is not given in these uncomplicated cases until the third day, it will be 6 per cent.; at the fourth day, 11 per cent.; and after this period much higher.

There are times when the nature of the epidemic has a bearing on the prognosis. In some epidemics the cases are all exceedingly mild, whereas in others they are all of severe type, and the death-rate is consequently increased. The general health of the child has but little influence on the severity of the attack or the ultimate result, and the social status of an individual has little or no bearing upon the prognosis. Frequently children in families whose surroundings are poor show better recuperative powers than those who have all the necessities and comforts of life. Of course, where the surroundings are good, and everything desirable, the care of the patient is easily obtainable, the probability of recovery is naturally greater, and greater still are the chances of recovery from the sequelæ of the disease.

NUMBER OF DEATHS IN PHILADELPHIA—DIPHTHERIA.

Age period.				1911.	1912.	1913.	1914.	1915.
Under 1 year	.	.	.	33	26	22	19	24
1 to 2 years	.	.	.	93	77	87	72	70
2 to 5 "	.	.	.	229	163	152	141	119
5 to 10 "	.	.	.	100	79	79	70	68
10 to 15 "	.	.	.	10	12	8	9	10
15 to 20 "	.	.	.	2	5	1	1	4
All ages	.	.	.	481	388	361	323	188

The mortality in children under five years of age is greater than at any other time of life. Infants and very young children frequently

succumb to laryngeal diphtheria, and at all ages diphtheria of the posterior nares and nasopharynx is the most rapidly fatal type, death being caused by toxemia. Diphtheria of the anterior nares is usually very mild; but mixed infection in diphtheria render the prognosis grave.

Other conditions being equal, the extent of the membrane is an indication of the severity of the attack. Malignant and septic cases are often fatal. The presence or absence of complications also influences the prognosis to a certain extent; thus, hemorrhage, acute nephritis, or the involvement of other organs is of serious import.

Postdiphtheritic paralysis develops as frequently in strong children as in weaklings, and the outlook in these cases depends upon the condition of the heart and respiratory muscles. Scrofulous children and those suffering from other constitutional dyscrasiæ usually succumb. More male children are affected than females, but this is merely due to the fact that male children are more likely to be exposed because of the general tendency to allow them to play about the streets, whereas the girls are kept at home. Of great importance in prognosis is the question whether or not antitoxin is used early in the disease. In some cases the diagnosis is made so late that the use of antitoxin is of little benefit. Yet there may be exceptions to this, and children who, to all appearances, were not likely to recover, have, especially after an intravenous injection of antitoxin, been brought back from what seemed certain death to complete recovery.

Prophylaxis.—Every case of sore throat which looks at all suspicious should be promptly isolated from other members of the family until an accurate diagnosis can be reached, either by awaiting clinical developments or by a bacteriological culture. Other children in the family should be prevented from attending school or coming in contact with children outside the family until the diagnosis is settled, because of the fact that children in a family in which there is a case of diphtheria often have diphtheria organisms in their throats and, if allowed to be at large, may possibly convey the infection to others. Should any children in the family show the presence of diphtheria organisms in their throats, although not suffering with the disease clinically, they should be isolated, but not with the child who is ill.

The sick-room should be, preferably, on the top floor. It should have the sunniest exposure, and all hangings, draperies, and unnecessary furniture should be removed from it. A sheet should be hung at the door and kept saturated with a weak solution of carbolic acid, formalin, or a solution of chloride of lime. The surfaces of the rooms should be wiped off daily with a bichloride of mercury solution 1 to 2000. Eating utensils and refuse from the room should be placed in a solution of carbolic acid, 1 to 30, and allowed to remain there for at least an hour before being taken out of the room. The nurse in attendance should remain in the room, and not come in contact with other members of the family. She should wear a long gown, that can be easily disinfected, to cover her ordinary dress, and a cap that will

43

cover her hair. These should be removed upon leaving the room, and left just outside where they can be readily put on before reëntering. All towels, as well as handkerchiefs or other pieces of cloth used for cleansing the mouth and nose of the patient, should be immersed in a solution of chloride of lime and subsequently burned.

In view of the fact that diphtheria organisms remain active in the throat of an individual who has suffered from the disease, often for weeks after apparent recovery, the child should be kept isolated until two negative bacteriological cultures are obtained on succeeding days, provided that a period of two weeks has elapsed from the onset of the illness. After two such cultures are obtained the patient should be removed to another room, and the sick-room thoroughly disinfected. This is best done by the use of formaldehyde gas. When the above principles of isolation and quarantine cannot be carried into effect it is wise to send the child, if possible, to a hospital for the treatment of contagious diseases. In this manner secondary cases are prevented and the health of the community at large is not jeopardized.

All exposed children should not only be cultured, as previously stated, but should also receive an immunizing dose of antitoxin, provided they are not asthmatic. When a case of diphtheria develops in an institution, all children who have been in contact ought immediately to be cultured, immunized, and isolated, and should show a negative culture prior to being allowed to mingle with other children, even though they show no clinical evidences whatsoever of the disease. The dose for immunizing is from 100 to 1200 units, depending upon the age of the child. Individuals who nurse cases of diphtheria should receive a dose of at least 1200 units.

In addition to immunization with antitoxin it is also advisable for all contacts to cleanse the nasopharynx and throat three times a day with a 1 to 5 solution of liquor alkalinus antisepticus or Dobell's solution. This precaution is particularly essential in those cases in which there are contra-indications to the administration of antitoxin.

The Schick reaction, which has lately become popular, particularly in institutional work, is of some advantage in determining the susceptibility of different children to the disease, thus indicating those to whom it is necessary to give antitoxin. The method of obtaining this reaction is as follows:

Following the observations of von Behring, Schick determined that if the serum of an individual contained as much as $\frac{1}{100}$ of a unit of antitoxin per cubic centimeter he possessed a sufficient degree of immunity. He further demonstrated that when an amount of diphtheria toxin equivalent to $\frac{1}{50}$ of a minimal lethal dose for a guinea-pig weighing from 250 to 300 grams was intracutaneously injected, and was not followed by a reaction, the serum of the individual contained $\frac{1}{30}$ or more of antitoxin per cubic centimeter.

Should the amount of antitoxin present be less, there will be a local inflammatory reaction at the site of injection, due to the fact that the toxin injected is not neutralized. A negative reaction indicates

that the individual possesses sufficient immunity against the disease, whereas a positive reaction indicates that he is still susceptible and therefore should receive a sufficient amount of antitoxin to produce immunity. Before the introduction of the Schick reaction it had been our custom in the children's ward of Jefferson Hospital to test for anaphylaxis when immunizing by first giving a small dose of antitoxin and then a larger one if no untoward symptoms developed, but recently we have used the Schick reaction instead of the small dose of antitoxin with, perhaps, more accurate results.

Treatment.—The treatment of diphtheria has been properly classified into hygienic, local, constitutional, and with antitoxin.

Hygienic Treatment.—The room occupied by the patient should be free from all carpets, hangings, and unnecessary furniture. It should be well ventilated, and the temperature kept at about 70° F. A room with an open fireplace and several windows is most desirable. The nurse should be the only one to occupy the room beside the patient. The physician in attendance should take every precaution to prevent the spread of the disease. While in contact he should wear a linen gown with a hood that completely covers his head. Upon leaving the room his face and hands should be thoroughly washed in a 1 to 3000 solution of corrosive sublimate. Absolute quarantine should be enforced, so as to prevent the possibility of infecting other members of the family.

Local Treatment.—In view of the fact that the organisms of the disease are present in the exudate in the throat, and knowing that they continuously multiply and produce toxic materials which are responsible for most of the constitutional symptoms, it is the belief of some clinicians that local measures should be resorted to that will tend to prevent the multiplication of the bacteria. Numerous drugs have been used locally for some time past, and each physician has his favorite method and drugs for swabbing the throat. Today active cauterants are not often used, but reliance is placed upon antiseptics that will cause no damage to the tissues with which they come in contact, yet have destructive effect on the organisms present. Those most frequently used are potassium permanganate, 1 to 5000, carbolic acid, 1 per cent., subsulphate of iron, potassium chlorate, and chloral hydrate or glycothymoline, 25 per cent. solution.

As a rule these drugs are applied to the throat by means of a swab, brush, or spray. Another drug which may be used either in the form of a dry powder or a liquid is the salicylate of sodium. In young children and infants it is often extremely difficult to employ local treatment. In very young infants solutions can be dropped into the nostrils, and in this way they gain access to the fauces.

The opinion regarding local measures has changed considerably within the past few years, and today the idea is not so much to prevent further multiplication of the organisms as to keep the nose, mouth, and pharynx clean. This is easily appreciated in view of the fact that by so doing the bacteria are to some extent prevented from gaining

access to the larynx and bronchial tubes. Absorption of toxins is also delayed, and further toxemia prevented. Ordinary salt solution, Dobell's solution, or a saturated solution of boric acid will suffice. These can be applied either with an atomizer or a fountain syringe. The solution should be warm and in sufficient quantity thoroughly to irrigate the parts affected.

For hemorrhage from the nose, if severe, solutions which are more or less astringent, such as alum, or a 1 to 3000 solution of adrenalin chloride, are required. The number of douchings necessary depends upon the amount of discharge. In mild cases three times a day is usually sufficient, whereas, in the severe types with profuse nasal discharge, it may be necessary to syringe the nose every third hour. For the relief of pain caused by the cervical adenitis, an ice-bag may be placed on the neck, or a 25 per cent. ichthyol ointment applied.

Constitutional Treatment.—The child should remain in bed throughout the attack. A purgative should be given at the beginning of the disease, preferably calomel, 1 to 3 grains, depending upon the age of the child. If the fever is high, nothing is more beneficial than tepid baths, that is, at 98° F. The use of such drugs as the coal-tar products should be discouraged, because of their depressing effect.

The diet should be liquid, consisting chiefly of milk, alternating with broths. Feeding the child with diphtheria is an all-important, but often a difficult task, since the appetite may be completely lost. The nursling must be taken from the breast, and given breast milk from a bottle, unless there is diarrhea, when milk-feeding should be temporarily suspended until improvement is noted. The older child should be given broths, beef juice, fruit juices, and ice-cream in small quantities at frequent intervals.

Everything possible should be done to build up the child's powers of resistance, and in some cases 1 to 2 drams of wine may be given four times daily with beneficial results. Water should be given freely.

For many years past *iron* has been used internally during the course of the disease and has gained in favor because of its good effects, both generally and locally. It should be administered in fairly large doses and at frequent intervals, the amount, of course, depending upon the age of the child. Children one to three years of age may be given 10 drops of the tincture every three hours, and older children 20 drops. An excellent method of administering it is in combination with glycerin. In instances where marked stimulation is necessary, the tendency today is to omit iron for a time, and rely upon stimulants.

Bichloride of mercury is another drug which exerts a beneficial effect, and has been used for many years. It is frequently combined with the tincture of ferric chloride, and in this way the action of both drugs is obtained, and is unquestionably beneficial. The amount of bichloride of mercury to be given needs some consideration, although there are few instances in which it has had any injurious effect. Jacobi states that a child one year old may be given $\frac{1}{2}$ grain every twenty-

four hours in divided doses for many days in succession without any intestinal disturbance.

Another drug used internally is calomel, in fractional doses, but because of its constant purgative effect it has lost favor, the weakness produced being greater than the benefit derived, save when given as a purgative at the onset of the disease. Chlorate of potash, because of its beneficial influence in stomatitis, has been extensively used in diphtheria; but because of the injury to the kidneys when given in doses large enough to have any good effect, its use has been more or less discontinued.

Caffein citrate, 1 grain every three hours; strychnine, $\frac{1}{200}$ to $\frac{1}{300}$ of a grain hypodermically; digitalis, 1 to 5 minims, and alcohol are all useful in combating cardiac depression, especially the last, in view of the fact that there are few cases which do not need stimulation, in one way or another. It is most frequently used in the form of whisky or brandy. In numerous cases it is necessary to give it in comparatively large doses, particularly in the more severe types of the disease, when as much as 1 dram hourly may be required, although in most instances half of this amount is sufficient.

Serum Treatment.—To Behring belongs the credit of having developed the use of antitoxin. In 1890 he announced that the serum of an animal which had been immunized against diphtheria was capable, when injected into other animals, of giving immunity to the disease, and also to effect a cure in those suffering from it. As to the exact manner in which antitoxin acts, no satisfactory explanation has as yet been given, but in all probability it exerts its good effect by neutralizing the toxins of the disease. It is probably a globulin, which combines with the globulin of the blood, and so prevents the growth of the organisms.

The serum itself should be clear, though its color varies. It frequently contains a preservative such as carbolic acid, but should be kept in a cool place in an hermetically sealed bottle. As a rule each cubic centimeter is equivalent in strength to from 100 to 500 antitoxin units. The animal commonly used is a horse which is positively free from glanders or tuberculosis. According to Park, the serum is produced by the Health Department of New York City in the following manner:

A virulent culture of a Klebs-Loeffler bacillus at the end of a week's growth is rendered sterile by the addition of 10 per cent. of a 5 per cent. solution of carbolic acid. This is filtered through sterile filter paper, and after standing twenty-four hours is placed in bottles and kept in a cool place. Several horses are then injected with sufficient toxin to kill 10,000 guinea-pigs, each weighing 250 grams. Ten thousand units of antitoxin are injected with each injection of toxin. This produces fever, but after it has subsided, which is usually from three to five days, another and larger dose is injected. The doses of toxin are increased, and given at intervals from five to ten days, and at the end of two months' time anywhere from ten to twenty times the

original amount is given. The horses are then bled, and the blood serum is tested for antitoxin. Those animals which yield less than 200 units to each cubic centimeter are considered unfit for use. The remainder are subsequently given toxin in ascending doses and at the end of three months the serum from such animals should contain from 300 to 800 units of antitoxin to each cubic centimeter. The serum is obtained by inserting a cannula into the jugular vein of the horse, and the blood is received in Ehrlenmeyer flasks, allowed to clot, and the serum removed. The antitoxin unit is the amount of antitoxin sufficient to prevent the death of a guinea-pig weighing 250 grams from the effect of 100 fatal doses of toxin.

Dosage.—Large doses of antitoxin are to be preferred under certain conditions to small ones, large amounts being required to neutralize the harmful "toxin" and the so-called "toxone." The latter is supposed to be responsible for the paralysis, and is only loosely taken up by antitoxin, and not in any great degree until all the "toxin" has been neutralized. This hypothesis seems to explain why antitoxin exerts a less favorable effect upon diphtheritic paralysis than one would, perhaps, expect.

The severity of the attack, and to a less extent its duration, should be the most important guides in regulating the dosage. For many years small doses, namely, 1000 to 2000 units, were given, and these repeated at frequent intervals, if necessary. Studies made in recent years have demonstrated that it is more advantageous to give a larger dose early in the disease, such as 4000 to 5000 units, and that it is unnecessary to give doses at such frequent intervals, it being sufficient to repeat the dose after the lapse of twelve to eighteen hours. The total amount of antitoxin to be given during the course of the disease depends entirely upon the reaction obtained; in the majority of cases it is not often necessary to administer more than 15,000 units during the course of the illness, and in many cases a single injection of 5000 units is all that is required.

The method of administration is as follows: A syringe is necessary which can easily be disinfected by boiling, and should be sufficiently large to hold 10 c.c. The site of injection is of little import, though the abdominal wall and the small of the back are favorable locations. The skin should be cleansed with soap and water, followed by alcohol, and all air should be expelled from the syringe. The serum is introduced slowly, and, after withdrawing the needle, a small wad of sterile cotton should be placed over the seat of puncture. Slight pain and general discomfort may follow its use.

Limitations of Antitoxin.—When given early in the course of the disease the membrane ceases to spread, becomes soft, and loosens, often in the most striking manner. The nasal discharge diminishes, and within a few hours the temperature often subsides, the heart action improves, and the nervous symptoms lessen materially. When given late, antitoxin cannot be expected to undo the harm which has already been done, nor is it likely to have any influence on the other organisms

so frequently associated with the Klebs-Loeffler bacillus. Again, in some instances, even though antitoxin be given early, the poisoning seems to be so virulent that the antitoxin has little or no effect. In these cases, owing to the more rapid result produced, the intravenous or intramuscular injection of antitoxin is of advantage.

Bronchopneumonia is, of course, commonly associated with the streptococcus and pneumococcus. But recent studies have proven that the diphtheria bacillus is more commonly than formerly supposed the cause of bronchopneumonia, and it may be the sole cause. Pneumonia has been produced in rabbits by the Klebs-Loeffler bacilli inoculated into the larynx. Large doses of antitoxin have proven of marked benefit in some pneumonia patients, and it is well to give the child the benefit of the doubt in appropriate cases.

The immunizing power which antitoxin has is shown by the fact that it will protect one from an attack of the disease, although the immunity so produced lasts but three or four weeks; this, however, can be prolonged by subsequent injections. The curative power of antitoxin is well attested by the lessened mortality since its use, the death-rate having dropped at least 50 per cent. The following table shows the average annual deaths from diphtheria and croup per 10,000 of the population:

	Before antitoxin, 1887 to 1893. Per cent.	Since antitoxin, 1896 to 1900. Per cent.
London	4.8	4.7
Berlin	10.2	3.7
Paris	6.5	1.3
New York	14.5	6.3
Chicago	13.1	5.0
Denver	12.9	1.7
Philadelphia (1890–1894)	11.9	9.6

In Berlin and Paris, where the serum was employed freely, the beneficial results can be seen by a glance at the above table; whereas in some American cities, where the serum was not adopted by the local Bureaus of Health, the reduction in mortality was not so great.

From the foregoing facts may be summarized the following:

First.—In view of the lessened mortality and the marked influence upon the course of the disease, Behring's antitoxin is practically a specific remedy, if given early in the disease.

Second.—In doubtful cases it should be given rather than await the findings of a bacteriological culture. This is especially recommended in younger children, in view of the fact that antitoxin is harmless when given to perfectly healthy persons.

Third.—The amount of antitoxin given depends upon the severity of the illness and its duration, larger doses at long intervals being of more benefit than frequently repeated small doses.

Fourth.—Diphtheria antitoxin cannot be expected to have any beneficial influence upon conditions which are the effect of other organisms associated with the Klebs-Loeffler bacillus.

Fifth.—All individuals, especially children, who have come in contact with a case of diphtheria, should be given an immunizing dose of serum.

Sixth.—As some hours must elapse, probably twenty-four, at least, before we get the full effect of antitoxin injected under the skin, therefore in toxic, laryngeal, and all other cases where a prompt reaction from the antitoxin is desired, it is advisable to inject it intravenously, since the benefits from intravenous injection are often noticeable within a few hours after its administration. If, owing to the small size of the vein, it is impossible to administer it intravenously, the antitoxin should then be injected into the muscle instead of under the skin.

In the present state of our knowledge, should anaphylaxis influence in any degree the giving of diphtheria antitoxin? Elmer E. Heg, in an article on "Review of Theories of Anaphylaxis" in *Northwest Medicine*, writes: "As we all know, quite a number of sudden deaths have followed the use of diphtheria antitoxins, a number of them in cases of asthma, though some without history of asthma. In most, no history is given as to whether or not a previous injection of serum had ever been given. Goodall reports 90 cases in a children's hospital, in which, during a period of five years, there was a record of a second injection after the incubation period. Sixty gave evidences of reaction, mostly a rash of more than usual severity; 7 had severe symptoms; 1 convulsions; 2 collapse, and 7 had chills and temperature as high as 105° F.; all recovered."

Much literature has accumulated on the theories, phenomena, lesions and mode of death in anaphylaxis in the lower animals, but one has some difficulty in finding much in literature regarding the serious results of anaphylaxis in man.

C. B. Ker, of Edinburgh, advises caution in the administration of antitoxin in the treatment of relapses. He says: "Some caution should be exercised in the administration of antitoxin in the treatment of relapses. It is, on the whole, wiser to withhold serum if it has been given during the original attack. Owing, doubtless, to what is termed 'anaphylaxis' the sequelæ of antitoxin are much more severe and much more frequent than we are accustomed to see in a primary case. Very profuse and irritating rashes, severe joint pains, and smart febrile reactions are all liable to occur early. A relapse is usually, though not always, comparatively mild, and, if there is not much evidence of toxemia and the larynx is not implicated, the serum can be dispensed with. I never hesitate, however, to give liberal doses if the symptoms are in the least urgent."

Dr. E. H. Funk has given me some interesting data from the Municipal Hospital in Philadelphia. During a period of some months he had an opportunity of seeing all the children in the scarlet fever hospital, and during a similar period all the children in the diphtheria hospital. All children in both of these hospitals are given one or more doses of antitoxin, and the curative doses are large ones. In the aggregate, a number of these cases have previously received either

immunizing or curative doses of antitoxin. Some children are known to have been given antitoxin on three different occasions, the intervals between the administrations being months or years. Rashes, joint pains and slight febrile disturbances were occasionally seen, and yet no symptoms of a serious nature were ever observed.

A very unusual opportunity has been offered to study the results of repeated injections of antitoxin at the Widener Memorial School for Crippled Children, in Philadelphia. Dr. Albert D. Ferguson and Dr. William J. Merrill have kindly furnished me with the following data: From 60 to 90 children are in the home, the buildings being all modern and thoroughly up-to-date. Many of these children remain for years as inmates. In spite of all precautions an occasional case of diphtheria occurs. An immunizing dose of 1000 units is always given to all children who have been exposed, and large curative doses are administered. As a result of this policy, and the fact that children often remain for years in the home, it has happened that many children have received a number of doses of antitoxin at intervals of a few months. It is understating the case to say that many children have received diphtheria antitoxin on at least six different occasions. Rashes, joint pains and slight fever are occasionally seen, but no fatal case, or even alarming symptoms, have ever occurred. It seems only just to conclude from this that with our present knowledge one should disregard completely anaphylaxis in treating diphtheria cases, and use antitoxin, according to modern ideas, at the earliest possible moment, both for immunizing and curative effects.

Future study is required to explain the symptoms in certain cases. Several cases have been reported where physicians, taking an immunizing dose of 1000 units, have had marked symptoms, such as unconsciousness and severe rash.

To those who are influenced by anaphylaxis it may be pointed out that the concentrated serum is safer, inasmuch as a smaller measure by quantity is required. And as the intravenous injection gives a more marked and rapid result, the subcutaneous method is to be preferred, since the dangerous symptoms develop soon after the injection is given, and it is reasonable to suppose that the slower absorption of the subcutaneous method is safer, therefore, than the intravenous.

A conservative and safe method has been advocated, which should appeal to physicians and be applicable to a large number of cases; in fact, could be used in all patients, except where the symptoms were so urgent that a delay of a few hours might be dangerous to the life of the individual. This consists in giving a small initial dose of antitoxin, about the usual immunizing dose, in all cases where a previous injection of antitoxin has been administered. If no immediate reaction is observed, it is safe in a few hours to administer the remedy in appropriate curative doses. If the case shows an immediate reaction it is wiser not to administer a large dose, as it might not be safe. If the immediate reaction has occurred, the patient will have milder symptoms from the smaller dose, on the theory that the larger

the dose, the more severe the immediate reaction. Recently, in the Jefferson Hospital, we have been using the Schick reaction with fairly satisfactory results.

By its use one is able to separate the children that should receive diphtheria antitoxin from those to whom it should not be administered. In my judgment the Schick test is practicable only in those institutions that have adequate observation wards. Children who are admitted for adenoid and tonsil operations often leave the hospital in twenty-four hours after admission. In these cases the time in the hospital is often insufficient for the study of the Schick test. In the hospitals where children are admitted directly to the wards, one or two days must elapse after the test is made before it is possible to know to whom antitoxin should be given. During this interval the child that requires antitoxin, either as a prophylactic measure to protect himself or as a protective measure for the other children in the ward, has received no treatment.

Antitoxin Eruptions.—In certain instances a cutaneous eruption follows the use of antitoxin. This eruption may make its appearance at any time from the date the antitoxin is injected up to a period of a month after its use, but usually between the fifth and the twelfth days. Such rashes are generally urticarial in type, sometimes erythematous, and the latter often closely resemble the rash of scarlet fever.

Other types of eruption which may occur are vesicular, bullous, and purpuric. Accompanying antitoxin rashes is an edema of the skin, most noticeable on the face. Though this rash may make its appearance on any surface of the body, it is generally first visible about the point of injection. Constitutional symptoms in the form of fever which lasts two or three days, headache, and at times vomiting, usually accompany such rashes. Joint involvement, chiefly pain, at times swelling, is a notable feature in many instances.

The diseases with which serum rashes are apt to be confounded are scarlet fever and measles, and sometimes it is almost impossible to make an early diagnosis. As a rule the appearance of a rash after the use of the serum, especially its appearance first at the site of the injection, the sudden rise in temperature, together with certain joint symptoms, should be a guide to the differentiation.

Operative Measures.—If, after the use of antitoxin and other measures, there is a marked increase in cyanosis and a gradual rise of temperature, intubation or tracheotomy should be performed. Which is indicated should be decided by the attending physician. In this country intubation seems to have the preference, though there are instances in which tracheotomy is necessary, either because of the location of the membrane, a lack of skill in performing intubation, or some other concurrent condition. Too frequently is tracheotomy postponed, consequently the results are often not as good as they might be.

Intubation.—We are indebted to Dr. Joseph O'Dwyer, of New York, for this operation. A set of instruments consists of seven tubes, an

introducer, an obturator, a mouth gag, a gauge for measuring the size of the tubes, and an extractor. The tubes vary both in size and calibre so that they may be inserted into the larynx of a child of any age. They are made of hard rubber lined with metal. The head of the tube is oval, and on its left side is a small hole through which a string can be passed. This is to enable the operator to withdraw the tube should it be introduced into the esophagus instead of the larynx. Each tube contains an obturator which is fastened to the introducer before the tube is inserted. The extractor is so constructed that when its tip is introduced into the tube the action of a lever makes its jaws separate, and the tube is grasped and extracted.

For the introduction of the tube the child should preferably be upon its back, with the hands fastened to the sides. A gag is then placed in its mouth, and the mouth opened widely. The tube is then attached to the introducer, and the index finger of the left hand is inserted into the pharynx until the epiglottis is located and pulled forward. Then, with the right hand, the operator passes the introducer with attached tube backward to the glottis, and on releasing the introducer by means of an attached thumb-piece, the tube immediately passes into the larynx.

The obturator is then removed, while the tube is held in position by the tip of the finger. That the tube is in the proper position is soon indicated by the sudden relief of the dyspnea. In the meantime the attached string should be firmly held for fear the tube may have been inserted into the esophagus. Should this occur, the tube must immediately be pulled out. The string may be removed by cutting it, and pulling it through the hole in the head of the tube.

Sometimes it is necessary to let the string remain attached because of the possibility of the tube becoming obstructed by membrane from below. In such cases the string may be securely fastened to the side of the face by a piece of adhesive plaster, or the loop may be carried over the ear. In addition to the sudden relief of dyspnea when the tube is properly inserted in the larynx, we usually note a severe metallic cough, and that the breath sounds assume a hissing character.

Dangers of Intubation.—In the hands of an unskilful operator much harm can be done, such as forcing the exudate downward into the trachea by the tube, thus causing instant death. False passages may be made by forcible introduction, or asphyxia may result from too frequent attempts.

After-treatment.—Sometimes, after intubation, the tube is expelled, usually owing to the fact that it is of too small size. In all cases of dyspnea after intubation, a careful examination should be made to ascertain whether or not the tube is really in the larynx, for it is possible for it to be coughed up unnoticed, or it may be coughed up and swallowed by the child.

The most perplexing question after intubation is in regard to the feeding. In some cases it is practically impossible for the patient to swallow liquids, whereas in others they are swallowed without any

difficulty whatsoever. Coughing is brought on probably by some of the fluid getting into the trachea through the tube, and in consequence most of the liquid is expelled. This condition happily disappears after two or three days, but during the time it lasts it is often necessary to resort to semisolid food, such as scrambled eggs, junket, etc.

Casselberry, of Chicago, advocates having the child lie with its head lower than its body while on its back, so that liquids which may gain access to the tube may have a better chance to run out. Should this fail, it may be necessary to resort either to gavage or feeding by the rectum. The former is accomplished by the introduction of a small soft-rubber catheter through the nose into the stomach. Infants who are breast-fed are usually able to swallow with little difficulty. A nipple shield should, however, always be used, or the child should be fed breast milk from a nursing bottle.

To prevent the possible expulsion of the tube, children should not be held with the face downward nor should they be allowed to lie upon the face; in either of these positions the tube may be readily expelled with the slightest cough.

The length of time necessary for the tube to remain in the larynx varies greatly in each case. Usually it should be allowed to remain for at least five days, though there are instances where it may be removed after two or three days. Sometimes the tube is coughed up with a mass of membrane, and in such instances it is well to withhold its reintroduction, as this may be unnecessary, but should signs of dyspnea return, it must be reintroduced immediately. Should the tube become obstructed, it must be removed, and if coughed up or swallowed no alarm need be felt, as it is invariably passed from the bowel.

Extubation or Removal of the Tube.—This procedure is considered more difficult than its introduction, and the technic is identically the same as that of intubation. The child is placed in the same position, the mouth is held open by the use of the gag, the index finger of the left hand is passed backward until the tip of the finger touches the head of the tube, the epiglottis is then tilted forward, the extractor is inserted with the right hand along the side of the finger, and the beak of the instrument is inserted into the opening of the tube. The extractor is then pressed down, which causes the two ends of the instrument to separate, and thus grasp the tube, which is then lifted out. Should there be any difficulty in removing the tube, it is wise not to continue the attempts too long, but to stop for awhile, and later try again. Should dyspnea occur, reintubation is imperative, consequently all cases of extubation should be carefully watched for the following hour.

Morphine is usually given hypodermically after extraction, and an ice-bag placed over the larynx. Immediately after extubation all irritative influences should be guarded against, and the child should be kept in bed for at least two days.

Prolonged Intubation.—There are cases in which it is necessary for the tube to remain in the larynx for a long period of time, the children

becoming markedly dyspneic soon after the tube is removed, which necessitates its immediate replacement. In such cases the tube may be expelled at frequent intervals, necessitating tracheotomy in order to prevent possible suffocation should they be where it is impossible immediately to replace the tube.

Various changes in the larynx, such as thickening, cicatricial contractions, paralysis of the muscles, persistence of false membrane, etc., may necessitate prolonged intubation, though frequently the true cause is undeterminable. Inasmuch as tubes may produce ulceration of the larynx, they should be removed early when possible, but not until the condition of the patient warrants it. After the removal of a tube its reinsertion is sometimes impossible. In such instances, should alarming symptoms occur, tracheotomy is necessary. For the ulceration which frequently follows the use of the tube, O'Dwyer has advised the coating of tubes with astringents.

For chronic inflammatory conditions of the mucous and submucous membranes of the larynx, Rogers has advised increasing the intra-laryngeal pressure by gradually inserting larger tubes until the largest sized tube has been worn for several weeks. This is then removed. In this way he has succeeded in dispensing with tubes which, in some cases, had been used for two or three years. In chronic stenosis of the larynx, O'Dwyer has advised opening the trachea, producing dilatation from below, and then inserting an intubation tube.

Tracheotomy.—In recent years this method of relief from laryngeal obstruction has fallen into comparative disuse, largely owing to the beneficial results of diphtheria antitoxin, and to the practice of intubation which has replaced it. However, there are still occasions which call for this radical procedure, *i. e.*, (1) When the membrane has become loosened in the larynx, and intubation may force it farther down. (2) When the membrane has been forced down into the larynx by unsuccessful attempts at intubation. (3) When membrane formation is too extensive to be relieved by intubation, and when cases have been intubated, and the membrane has formed below the tube. While the operation of intubation appears to be a simple one, it is often very difficult to perform, and a competent and experienced surgeon should always be secured to do it.

The occurrence of laryngeal diphtheria can to some extent be prevented by an early diagnosis and promptitude in treatment, thus lessening the possibility of extension of the membrane into the larynx. When the larynx becomes involved the membrane slowly disintegrates, becomes detached, and is expelled, consequently it is sometimes advisable to give an emetic to promote its expulsion. Of course, this ought not be done unless the physician feels positive that the membrane is detached, and this is certain only when a peculiar flapping sound is heard in the larynx.

Syrup of ipecac, in $\frac{1}{2}$- to 1-dram doses, is in all probability the most efficacious emetic to use in such circumstances, because it leaves no after-effects. Apomorphine, $\frac{1}{20}$ grain, may also be used.

Inhalations of steam were formerly quite generally used to detach the false membrane, but because of their depressing effect they are now seldom, if ever, resorted to. In addition to inhalations of medicated vapors, such as the compound tincture of benzoin, turpentine, etc., the internal use of mercury has gained much favor. It may be given in the form of calomel, in divided doses, extending over a period of from twelve to twenty-four hours.

Before the introduction of antitoxin, calomel by fumigation was frequently employed in the treatment of laryngeal diphtheria, and there is much clinical evidence in favor of its use. Ten grains of calomel are dropped on a strip of tin, which is placed over a chamber. A lighted alcohol lamp or candle is placed in the chamber under the tin. The crib or bed is surrounded by sheets to form a tent, and the patient and nurse remain in the tent for ten to fifteen minutes, the nurse frequently taking long breaths of outside air from the opening in the tent. The patient apparently runs no risk of salivation, and the above precaution will protect the nurse. The fumigation may be repeated three or four times a day.

PSEUDODIPHTHERIA.

Synonyms: False Diphtheria—Scarlatinal Diphtheria—Croupous Tonsillitis—Streptococcus Diphtheria.

Under this term may be grouped those inflammatory conditions of the mucous membrane of the throat and upper air passages in which is produced a false membrane which is not associated with the Klebs-Loeffler bacillus. Because of the fact that the Streptococcus pyogenes is frequently found in such membranes, it is often called Streptococcus diphtheriæ. A diplococcus, called the Roux coccus, has also been isolated, and the Staphylococcus pyogenes may frequently be found in cases of pseudodiphtheria. Among the organisms which may cause membranous formations in the mouth and throat are the pneumococcus, the Bacillus coli, and even the gonococcus. The affection is frequently confounded with diphtheria, and it has been estimated that from 25 to 35 per cent. of cases formerly thought to be diphtheria were nothing more than pseudodiphtheria.

The diseases in which pseudomembranous inflammation is most apt to appear are scarlet fever, measles, whooping-cough, and typhoid fever. It may also develop as a primary affection. The general hygienic surroundings seem to have some bearing upon the development of the disease, in that it is more frequently seen in children living in insanitary tenement houses, or among the inmates of institutions. The disease itself is but slightly contagious, which renders isolation and disinfection during its course unnecessary.

Lesions.—The tonsils are, as a rule, the chief seat of membrane formation, save in secondary cases where the entire pharynx may be involved with extension to the mouth, nose, middle ear, and, in exceptional instances, to the larynx, trachea, and bronchial tubes.

The membrane differs from that of diphtheria in that it is softer, and microscopically is found to contain a greater proportion of cells than the membrane of diphtheria. Nevertheless, it is practically impossible to distinguish the membranes of the two diseases microscopically, and in the majority of instances a bacteriological culture is necessary in order to make a diagnosis. The non-adherency of a pseudomembrane in primary cases is characteristic, whereas in secondary cases the pseudomembrane may extend deeper, and the adjacent tissues become markedly congested and edematous, this affecting chiefly the tonsils, soft palate, and uvula. In some cases membranous casts of the larynx and trachea have been expelled.

Symptoms.—The onset is sudden, accompanied by headache, vomiting, chills, loss of appetite, difficulty in swallowing, and sore throat. On inspection the tonsils are found to be reddened and swollen, and are soon after the seat of membranous patches. The membrane is usually yellow or gray, and loosely adherent, and, after three or four days' duration, disappears. The surrounding tissues are at the same time markedly inflamed, and the lymphatics behind the angle of the jaw are tender and swollen, and may suppurate. The constitutional symptoms usually subside by the third or fourth day.

Secondary cases of the disease in severe forms are usually seen in scarlet fever or measles. When accompanying scarlet fever, evidences of the disease may present themselves either at the beginning or from the third to the fifth day; whereas, when complicating measles, they are usually at their height when the eruption begins to disappear. Sometimes the larynx is involved, and then bronchopneumonia is apt to develop.

In the secondary type of the affection the constitutional symptoms are usually quite severe. There is high fever, the pulse becomes rapid and feeble, prostration is great, and delirium or stupor may set in. Following such an attack there may be either suppuration or necrosis of the adjacent tissues. Fortunately, such occurrences are rare.

Diagnosis.—The differentiation between pseudodiphtheria and true diphtheria can in most instances be made only by means of a bacteriological culture. When the disease is secondary to scarlet fever or measles, and appears during the height of the primary disease, the diagnosis is not so difficult, since true diphtheria more often develops after the primary fever has abated. If, in such cases, the first culture is negative, a second should always be made.

Prognosis.—Primary instances of the disease usually terminate in recovery, the mortality being extremely low (3.5 per cent.) whereas, when secondary to one of the diseases mentioned, the mortality is usually from 15 to 20 per cent.

Treatment.—All cases of membranous inflammation of the throat should be isolated and regarded with suspicion. In very young children antitoxin ought to be administered immediately, and a bacteriological culture then made to determine the organisms present. If the bacteriological findings prove the disease to be non-diphtheritic,

the administration of antitoxin should be discontinued; but in no case should antitoxin be withheld until a positive diagnosis of diphtheria can be made.

Locally the nose and throat should be thoroughly syringed with mild antiseptic solutions, such as liquor alkalinus antisepticus, 25 per cent. In instances where the swelling and edema are marked, a spray of 1 to 10,000 adrenalin chloride solution often gives great relief. The drugs most frequently used as a direct application to the membrane itself are nitrate of silver in the form of a 10 to 15 per cent. solution, and a solution of bichloride of mercury, 1 to 1000.

Should there be evidences of adenitis or cellulitis, the external application of an ice-bag affords great relief. Frequently a gargle containing chlorate of potash, 10 grains to 1 ounce, in combination with tincture of ferric chloride, $\frac{1}{2}$ dram to 1 ounce, is of decided benefit in preventing further membrane formation. When laryngeal obstruction occurs, intubation or tracheotomy may sometimes be necessary as in true diphtheria.

PERTUSSIS (WHOOPING-COUGH).

Whooping-cough is a contagious disease, characterized by catarrhal inflammation of the mucous membrane of the respiratory tract, together with irritability of the nervous system. It occurs in both epidemic and endemic forms.

It is essentially a disease of child life, being seen most commonly in the first five years, and about 65 per cent. of all cases occurring during the first two years. It often attacks infants only a few weeks or months old, and in this respect differs from measles, scarlet fever, and diphtheria, which are rarely met with at this early age.

It, however, occurs quite often in older children, is occasionally seen in adult life, and even in old age. It is very contagious, and almost all children, especially if they have a coryza or bronchitis, will, if exposed, contract the disease.

In the majority of instances one attack confers immunity, second attacks being rare. Winter and spring are the usual seasons for the prevalence of epidemics, and such epidemics frequently either precede or follow epidemics of other contagious diseases, particularly measles.

The disease is easily communicated from one person to another, most frequently by direct contact, though it is often contracted in the open air. The height of contagion appears to be in the early or catarrhal stage, although it is also contagious until the typical whoop disappears in the stage of decline.

Children who are delicate or anemic are more susceptible than others. Bordet and Gengou have apparently isolated the true organism. They describe it as a small, ovoid bacillus, sometimes elongated, but more often so short as to resemble a micrococcus. The bacillus stains a pale blue with Kuhne's blue stain, and is negative to Gram's stain; it is believed by some authorities to belong to the influenza group.

Pathology.—No constant pathological lesions are found, except a catarrhal inflammation of the respiratory tract, and sometimes a serous pleurisy. The paroxysms have been attributed to irritation of the upper air-passages in the region supplied by the superior laryngeal nerve, or to an irritation in the posterior part of the larynx between the arytenoid cartilages. Another view is that the paroxysms of coughing are due to a plug of mucus in the trachea. The most frequent complication is bronchopneumonia. In protracted cases the lungs are apt to show some degree of emphysema. In severe cases which prove fatal, hemorrhages may take place in the eye, ear, and brain.

Histological studies have shown that in fatal cases of whooping-cough the action of the infecting bacilli is largely mechanical, since they are found in great numbers in the epithelial lining of the respiratory tubes. There is also produced in pertussis a mild toxin which causes exudation of leukocytes into the lumen of the trachea and the bronchi from blood-vessels lying external to them.

The changes which occur in the splenic lymph structures, in the lymph nodes of the gastro-intestinal tract, and elsewhere are also produced by this mild toxin, while in the blood there is lymphocytosis and the formation of an antibody which acts specifically against the Bordet-Gengou bacillus.

Symptoms.—The period of incubation, though variable, is usually from seven to fourteen days. For convenience's sake, the symptoms are divided into three groups: the catarrhal, the spasmodic, and those in the stage of decline.

Catarrhal Stage.—The catarrhal stage gives all the appearances of a simple cold; namely, fever, nasal discharge, and cough. These symptoms may be preceded by a period of anorexia, languor, and restlessness, especially at night. The cough also is most troublesome at night. The fever may be slight and remittent, and is present only in the first few days unless complications, such as severe bronchitis or bronchpneumonia, develop. Up to this time no suspicion is aroused unless there is a history of direct contact. After five to ten days, however, it is noticed that the cough, instead of abating, is more frequent, and becomes spasmodic in character, occurring in paroxysms which gradually increase in severity and are especially troublesome at night, until the appearance of the typical whoop which marks the paroxysmal stage. Examination of the chest at this time shows a moderate degree of bronchitis.

Spasmodic Stage.—In this stage the symptoms and physical signs of bronchitis are present, and the paroxysms of coughing gradually increase in number and severity. During the coughing the child's face becomes congested, and the eyes suffused. A typical seizure consists of a number (5 to 10 or 15) of short expiratory coughs without a single inspiration intervening. At the end of these expiratory coughs there is a long drawn out inspiration, and it is this inspiration which produces the whoop. This is followed by another series of expiratory coughs, then by another whoop, and this is repeated in a single

44

paroxysm two, three, or even six times, until the child coughs up a portion of thick, tenacious mucus, and the paroxysm temporarily ceases.

Infants usually foretell the approach of a paroxysm by beginning to cry, and older children run to their parents, or support themselves against some object when the aura of vomiting or sneezing warns them of the coming attack.

As the attacks become more severe, there may be hemorrhages into the skin, nose, throat, or conjunctivæ. The face is puffy, especially about the eyelids, and there may be a great amount of venous stasis. Not infrequently well-marked bronchitis sets in at this stage of the disease.

Vomiting often follows, and may be so persistent as to produce emaciation. Epistaxis sometimes occurs during the more severe attacks, although the amount of blood lost is usually slight. Some degree of prostration often follows the paroxysms, and convulsions occur in severe cases.

The number of attacks during the day varies from five to fifty, according to the severity of the infection, and they may be provoked by laughing, crying, overloading the stomach, or any irritation of the nasopharynx and larynx. The paroxysms are always worse at night.

In very young children the whoop is frequently absent, though the cough may be severe. The duration of the paroxysmal stage is usually between four and five weeks; in the milder cases it may last but a week, and in the more severe ones may continue for eight or ten weeks, and then recur upon the slightest provocation, such as a cold. As a rule, it reaches its height at the end of the second week, remains constant for two weeks, and then gradually subsides, the cough assuming the appearance of simple bronchitis.

Stage of Decline.—This period is characterized by a decrease in the severity, and diminished frequency, of the attacks. The cough loses its paroxysmal form, gradually becomes catarrhal, and disappears in the course of about two weeks.

Complications.—Hemorrhages frequently occur as a result of intense venous congestion, epistaxis being the most common, though rarely of any severity. Conjunctival hemorrhages, bleeding from the ears, petechiæ, and, occasionally, hemoptysis may appear. Convulsions sometimes occur, probably as the result of engorgement of the cortex, although hemorrhagic or tuberculous meningitis may set in, and encephalitis has been reported.

Hemiplegia, monoplegia, facial paralysis, aphasia, and disturbances of sight, hearing, and sensation may result from meningeal hemorrhages. Various psychoses, such as melancholia and hallucinations, have been known to complicate pertussis, and more severe forms of mental derangement, such as imbecility and idiocy, may supervene.

In some instances emphysema may result from a severe paroxysm. Atelectasis, bronchiectasis, and edema of the glottis are among the rarer complications in the respiratory tract. Pulmonary affections

are by far the most serious. Of these bronchopneumonia is responsible for the majority of deaths; it occurs more frequently during the winter and spring months. Miliary tuberculosis or phthisis may also be excited by an attack of pertussis.

Other complications may occur, such as pleurisy, valvular heart disease, and nephritis, while gastro-enteritis is not an uncommon sequel in infants, and may terminate fatally.

Diagnosis.—The characteristic whoop of the disease renders the diagnosis easy, although it must be borne in mind that there are cases in which this is absent. If the disease is prevalent, however, if there is a history of exposure, and the cough is uninfluenced by treatment, the disease can be no other than whooping-cough. Particularly is this true if there is occasional vomiting after the paroxysms of coughing.

The chest examination in the catarrhal stage reveals, as a rule, only a slight bronchitis to account for the severity of the cough, and this is quite characteristic of pertussis, although it is scarcely possible to diagnose the disease before the whoop is heard. A severe cough, worse at night, which brings on vomiting, and is unaccompanied by fever, is almost certainly pertussis. Leukocytosis may be of some value in doubtful cases.

Recent experimental researches would seem to indicate that the diagnosis of pertussis may be made at any stage of the disease by means of the complement-fixation test. The procedure is the same as for other complement-fixation tests, pure cultures of the Bordet-Gengou bacillus being employed.

Prognosis.—In early infancy, especially during the first year, whooping-cough is an extremely fatal malady, owing to the serious complications so often encountered. The mortality has been estimated as at least 20 per cent. during the first year, and most of the deaths occur in bottle-fed babies who also suffer from gastro-enteritis, and finally succumb. After this age, the percentage of deaths decidedly decreases, and the prognosis depends upon the previous health of the child, and the presence or absence of complications or of constitutional diseases, such as tuberculosis or rachitis.

Children affected during the summer months are more apt to escape that much dreaded complication—bronchopneumonia—than those attacked during the winter months. Fifty to 70 per cent. of deaths occurring during whooping-cough are due to bronchopneumonia. Following this are the various intestinal disorders, chief and most common of which is diarrhea. Other causes of death are convulsions, cerebral hemorrhage, asphyxia, and emphysema.

According to Dr. Paul Luttinger, of the Bureau of Laboratories, New York City, the highest incidence and mortality—56 per cent.— have been observed among females, the incidence and mortality in males being 44 per cent. This has been attributed to anatomical differences in the construction of the larynx, and is, perhaps, also due to a nervous system which is more susceptible in girls than in boys.

The influence of sex is apparent at all ages, and it would appear from our present knowledge that it becomes more evident the older the girls are.

It is difficult to state positively what the true case incidence mortality is. All cases of pertussis are not reported, especially the mild and atypical ones. The law, in Pennsylvania, placards the house, but excludes from school only the children who actually have the disease, allowing the other children to go to school if they are free from symptoms. If this law were better understood by physicians and the laity, more cases would undoubtedly be reported.

The true case mortality, when one includes the mild and atypical cases, is certainly much less than statistics ordinarily indicate.

NUMBER OF DEATHS IN PHILADELPHIA—WHOOPING-COUGH.

Age period.	1911.	1912.	1913.	1914.	1915.
Under one year	41	53	51	135	18
1 to 2 years	37	18	25	74	17
2 to 5 "	32	22	22	45	10
5 to 10 "	5	6	8	10	2
10 to 15 "	0	0	0	1	0
15 to 20 "	0	1	0	0	0
All ages	116	100	106	266	67

Treatment.—Children suffering from this malady should be promptly isolated from other children for a period of not less than six weeks, owing to the fact that the disease is invariably contracted by direct contact. Rest in bed is imperative in cases of unusual severity, together with an abundance of fresh air day and night.

The treatment of pertussis is a subject which has always absorbed a large degree of medical attention, and the mere enumeration of all the drugs, inhalations, and cures which have from time to time been brought forward would consume far more space than is here possible. I will not, therefore, attempt to mention the drugs and plans of treatment that might properly be considered in a historical sketch of this much-to-be-dreaded disease, but will call attention to only a few of the drugs and methods of treatment that have in my judgment stood the test of long experience, such as fresh air, careful attention to feeding and digestion, local applications to the nasopharynx, inhalations, belladonna, antipyrin, bromides, codein, trional, heroin, and chloral. Among the newer methods of treatment are: quinine given intravenously and intramuscularly, adrenalin, suggestion, and vaccine treatment.

As pertussis is contagious from the very beginning of the catarrhal stage until the end of the spasmodic stage—a period of about ten weeks—it is important that children free from the disease be kept apart from those who have it, and in order that quarantine be effective all unexposed children should, if possible, be sent away from the house during the entire period of contagion. This applies particularly to infants, in whom pertussis is associated with considerable danger; and all children who are not robust or who have a tendency to tuber-

culosis should be especially protected. A child with pertussis should not be allowed to attend school, and quarantine should be continued until the end of the spasmodic stage.

All children with pertussis should be given an abundance of fresh air and should be kept in bed if their temperature is 100° F. or higher. If the patient is sufficiently ill to be in bed the windows should be open day and night; if not in bed the child should spend as much time as possible out of doors. However, as excitement and violent exercise increase the tendency to attacks, an effort should be made to keep the child interested and amused with the minimum amount of exertion on his part. An acute laryngitis or rhinitis is, however, not benefited by cold air; for such cases the air in the sick-room must be kept fresh but not cold. Cold, fresh air is of benefit in all other pertussis cases. A change of air, especially to the seashore, is often of benefit, and a change from a raw, damp climate to a warmer and milder one is often followed by improvement.

If the child has been confined to bed any considerable time it is of advantage to remove him to another room and thoroughly house-clean the room before returning him to it. Wards where pertussis patients are treated should, if possible, be cleared of all patients and house-cleaned and fumigated before the patients return to them. Many cases of whooping-cough in a hospital ward suffer with mixed infections, and at least a temporary benefit will follow their removal to a recently cleaned and fumigated ward.

All children with pertussis should be fed in small quantities and often, and the younger the child the more necessary it is to preserve its strength by proper attention to its food and digestion. If a child vomits soon after receiving nourishment, it should be given another feeding to replace the one vomited. Gastro-intestinal disorders in the young child suffering with pertussis often constitute a dangerous complication, and should receive early and careful dietetic and medicinal treatment.

Local applications to the nasopharynx, if made early in the disease— during the first two weeks—may be of decided value. They are of assistance only when the infection is localized in the upper air passages, as in rhinitis and pharyngitis. At this period an application of 2 per cent. nitrate of silver solution to the nasopharynx may, by producing death of the superficial mucous membrane, and, possibly, destroying some of the specific pertussis bacilli, tend to prevent the spread of the infection to the deeper respiratory passages.

Ochsenius, of Chemnitz[1] has recently reported 107 children treated by this method, with improvement in 84 of them. He makes the application every second day, and claims that eight days after the beginning of the treatment the number of paroxysms is slightly diminished and the severity of the attacks decidedly lessened, and by the third or fourth week improvement is marked. He lays special

---

[1] Therap. der Gegen., Berlin, 1913, liv, 502–509.

stress on the importance of using the nitrate of silver solution early in the disease, when the infection is limited to the upper respiratory tract. Phenol (1 per cent.) has also been used successfully as a local application. It should be employed early and may be repeated every second day.

Various drugs have been used by inhalation, particularly creosote and carbolic acid. They may be used in the ordinary inhaler covering the nose and mouth, or the vapor may be generated in the room of the patient. They act as a sedative to the inflamed mucous membranes and, at least in some degree, as a local antiseptic. As children are especially susceptible to carbolic acid poisoning, the urine must be closely watched. Chloroform may also be given cautiously by the physician when the paroxysms are especially severe and frequent. When the spasm of the glottis is unusually severe, intubation may be done and often gives relief.

The medicinal treatment of pertussis may, for convenience, be divided into two parts: (1) drugs or other methods that *per se* have a tendency to lessen the number and severity of the paroxysms. (2) drugs or other methods directed to the treatment of the complications of pertussis. To the first an unusual amount of attention has been given; to the second, comparatively little. Among the drugs that *per se* are useful in pertussis must first be mentioned belladonna. Personally, I prefer using the tincture, beginning with one drop, three times a day, and increasing the daily quantity by one or two drops until mild physiological effects of the drug appear, after which the dose must be increased very cautiously.

Antipyrin is a useful drug, but I have never used it in the frequent doses so often advised. A single dose at bedtime, or a morning and evening dose, has been, in my experience, as much as it was wise to employ. To a child of two years I would give 2 or 3 grains each night, or morning and night.

Bromide of soda, grains v, three or four times a day, to a child of three years, is often of benefit, and codein, trional, heroin, and chloral will often allay the cough and induce sleep. They may be given in a single dose at bedtime, or, if necessary, two or three times a day. A combination of quinine, two parts, and veronal, one part, has been used successfully by Professor Winternitz, of Vienna. He claims distinct improvement in 26 out of 30 children in whom he used the combined drugs, but believes that it acts only as long as given, and is not a cure. During its administration the paroxysms became milder and occurred less frequently; there was less vomiting, and the appetite improved. Fränkel and Hauptmann[1] also advise this combination of veronal and quinine. The dose ordinarily employed for a child of two years was veronal, $\frac{1}{2}$ grain; quinine, 1 grain, repeated three or four times a day, according to the effect produced. They do not advise its use in children under six months of age. Soolman and Hatcher[2]

[1] Med. Klinik, Berlin, 1912, viii, 1871.
[2] Jour. Am. Med. Assn., 1908, ii, 487.

report favorable results from a combination of quinine, grains i–ij, and bromide, grains i–ij, repeated three or four times a day, for a child two years of age.

Quinine has been used in large doses and when so given may reduce not only the number, but also the severity of the paroxysms. It is not an antispasmodic, and any effect it produces in these large doses must be due to some effect on the causal bacillus. Lenzmann[1] has reported some interesting results from the giving of quinine intravenously and hypodermically. He secured very prompt and positive results from doses of 5 grains injected intravenously every second day. He claims that the paroxysms rapidly disappear and that the treatment acts like a charm. Quinine lactate, 10 grams; saline solution, 100 grams, of which $2\frac{1}{2}$ c.c., warm, is injected intravenously, may be similarly used. If given hypodermically into the muscles the effect is favorable, but not as prompt nor as satisfactory as when given intravenously.

Hydroquinine has been employed both intravenously and intramuscularly, but its action is more favorable when injected into the vein. It has been put up in ampules, the dose being in proportion to the age of the child. A daily dose was given for five or six days, then a dose every second day; it is claimed that marked improvement was perceptible after the first week of treatment. No disagreeable local or constitutional symptoms followed either the intravenous or muscular injections, and, as so much is claimed for this treatment, it is worthy of more extended trial. Hydroquinine has been used both intravenously and intramuscularly as a prophylactic with satisfactory results.

Adrenalin is strongly advised in the treatment of pertussis by Fletcher,[2] and since the publication of his article others have reported favorable results following its use. Wm. J. Lord,[3] when all other means failed in a very delicate child, gave, by the mouth, 3ℳ of a 1 to 1000 adrenalin solution every four hours. The dose was soon reduced to three times a day, and the child rapidly improved; not only the number of the paroxysms became less, but their severity decidedly diminished. Carta Mulas,[4] who had read Fletcher's article, used adrenalin in a small epidemic of pertussis. He treated 15 cases, giving two to three drops of a 1 to 1000 adrenalin solution every two or three hours. The cough and vomiting rapidly diminished, no complications or bad effects followed the treatment, and in two or three weeks the patients were well. He claims that the rapid cure of these cases prevented the spread of pertussis, as only 5 per cent. of the infants contracted it. In his opinion, adrenalin exerts a specific action on the causal agent of pertussis.

Kilmer, of New York City, has devised a whooping-cough belt which supports the abdomen, and is evidently a great comfort to the

[1] Med. Klinik, Berlin, 1912, viii, 1789.
[2] British Med. Jour., London, 1912, ii, 1784.
[3] Ibid., 1913, ii, 122.
[4] Gaz. d. Osp., Milano, 1913, xxxiv, 1295–1297.

patient, for children who have once worn this belt insist on keeping it on during the paroxysmal stages of the disease, and like it to be buckled tight. In severe cases in which the paroxysms are violent, a plaster bandage may be applied around the ribs to give additional support. Some authorities encircle the chest with strips of belladonna plaster. If these supports are properly applied and fit snugly, they often modify the intensity of the paroxysms and, to a great extent, relieve the vomiting.

Every close student of pertussis must be impressed with the psychic element that so often enters into the disease, especially in neurotic children. If, as often happens, the mother and those who are brought in contact with the nervous child are also neurotic, conditions are favorable for the development of marked psychic phenomena in this nervous child suffering from pertussis. Given a neurotic child in whom the psychic element is present, and suggestion may be advantageously used as an aid to treatment. Oberholtzer,[1] in discussing the psychic element in pertussis, narrates the case of a boy, aged seven years, whose nurse, believing the paroxysms were largely induced by the desire of the boy to be carried in her arms, refused to carry him any more. His paroxysms ceased and he made a prompt recovery. His little sister of twenty months always began to cough as soon as the boy had a proxysm. The nurse reproved her and the paroxysms of cough ceased. One day while taking a trip on the lake, and after the nurse had told the boy that if a paroxysm threatened, they would have to return home, his cough disappeared for four hours.

Hamburger[2] also calls attention to this psychic element, and reports the case of a girl, aged three and one-half years, cured by suggestion. The child suffered with pertussis for five weeks and was cured in two days by the faradic current. Hamburger infers that in this case the paroxysms were pure neurosis after five weeks—what he calls a "half-voluntary reflex."

Space does not permit me to discuss the treatment of the complications of pertussis, but several cases that I have seen this winter deserve passing mention. A boy, aged thirteen months, with a tuberculous family history and himself tuberculous, passed successfully through a severe case of pertussis complicated with bronchopneumonia. In the stage of decline he became more and more drowsy, lumbar puncture was performed, and a clear fluid removed in which the tubercle bacillus could not be found, and a guinea-pig injected with 3 c.c. of this fluid failed to develop tuberculosis. There had never been any discharge from either ear, but Dr. MacCuen Smith obtained a drop or two of fluid by swabbing the ear with cotton. Pus was obtained from both ears by puncturing the drums; the day following, a double mastoid operation disclosed a well-advanced double mastoiditis with secondary otitic meningitis. The temperature had been normal for ten days before the operation. We all, of course, appreciate how

[1] Corr. Blatt f. Schweizer Aerzte, December 20, 1912.
[2] Wien. klin. Wchnschr., 1913, xxvi, 1869.

latent mastoiditis may be in a child, but with no local evidence of mastoid disease, no discharge from the ear, and no fever, the condition is easily overlooked.

Another case of interest was the seven-year-old son of Dr. C., of Philadelphia, who during an attack of pertussis had an unusually severe paroxysm on February 15. The next morning he had fever and vomiting. Vision continued to decline until March 13, when vision was found to be: left, 11/200; right, 20/100. The disks are pale; retinal arteries reduced in size; field of view is concentrically contracted. Previous high-grade double optic neuritis (choked disk). At present, total secondary atrophy. Disseminated choroiditis, both eyes. Divergence. Vision reduced to light perception. Pupils moderate in size and no reaction. Under ethyl chloride narcosis, pupils contract *ad maximum*. Diagnosis: hemorrhage into the sheath of nerve at the optic chiasm. These two cases illustrate the necessity of a correct diagnosis of complications in order properly to carry out appropriate treatment.

The Vaccine Treatment.—I was the first to try this method of treatment and have carefully read the report of all cases treated by this method since the publication of my paper in January, 1912. A large number of articles have appeared upon vaccine treatment, and from the close study of my own series, as well as many cases seen in consultation where the vaccine treatment has been used, since the publication of my paper, I am convinced that it is a distinct addition to our treatment, and is also of more or less use as a prophylactic. In a case of moderate severity in either an infant or an older child, when the number of the paroxysms is small and of a mild character, the vaccine treatment is not necessary but it is often of distinct benefit in the severe cases, in children of all ages, and its influence as a prophylactic, especially where infants have been exposed, or where a frail or possibly tuberculous child has been infected with the pertussis bacillus, should be carefully investigated, and its prophylactic power tested. There seems to be a growing tendency to treat pertussis with a mixed vaccine.

In connection with the mortality tables of typhoid fever, diphtheria, scarlet fever, measles, and whooping-cough, showing the number of deaths in Philadelphia during the five years, 1911 to 1915, inclusive, the following table (No. 1) shows the percentage of deaths to the total number of cases of these diseases. Table No. 2 may be of special interest; it shows the number of deaths in Pennsylvania from the above diseases during the years 1909–1912, inclusive, in children under one year of age, in children under five years of age, also at all ages, as well as the total mortality in Pennsylvania from all diseases during these years. Table No. 3 shows the mortality in Pennsylvania, during the years 1911 and 1912, from the diseases tabulated, embracing the number of deaths at all ages, also the mortality in children under five years of age.

TABLE 1.—TABLE SHOWING PERCENTAGE OF DEATHS TO TOTAL NUMBER OF CASES OF MEASLES, DIPHTHERIA, SCARLET FEVER, WHOOPING-COUGH, AND TYPHOID FEVER IN PHILADELPHIA FROM 1911 TO 1915, INCLUSIVE.

	Number of cases.					Total No. of cases in 5 years.	Total No. of deaths in 5 years.	Percentage of deaths to total No. of cases in 5 years.
	1911.	1912.	1913.	1914.	1915.			
Measles	11,640	2,279	15,611	7,096	14,089	50,715	798	1.5+
Diphtheria	3,792	3,080	2,623	2,610	2,615	14,720	1,741	11.8
Scarlet fever	1,928	2,872	3,400	1,944	1,072	11,216	608	5.4
Whooping-cough	1,410	1,369	1,438	4,152	1,092	9,461	655	7.0
Typhoid fever	1,382	1,514	1,698	793	787	6,174	911	14.7
					Total	92,286	4,713	

TABLE 2.

State of Pennsylvania.	At all ages.	Under one year.	Under five years.
Total mortality, 1909, from all diseases	111,062	25,638	36,216
" " " " typhoid fever	1,712	8	87
" " " " diphtheria	2,002	139	1,350
" " " " scarlet fever	1,216	72	754
" " " " measles	1,060	286	934
" " " " whooping-cough	910	500	870
" " 1910, from all diseases	119,815	28,377	40,495
" " " " typhoid fever	1,892	3	77
" " " " diphtheria	2,235	178	1,443
" " " " scarlet fever	1,094	64	657
" " " " measles	1,237	306	1,083
" " " " whooping-cough	1,114	581	1,068
" " 1911, from all diseases	111,292	24,195	33,788
" " " " typhoid fever	1,716	4	66
" " " " diphtheria	2,111	122	1,346
" " " " scarlet fever	749	46	460
" " " " measles	804	202	704
" " " " whooping-cough	998	567	960
" " 1912, from all diseases	111,842	24,110	33,468
" " " " typhoid fever	1,310	8	51
" " " " diphtheria	2,042	146	1,371
" " " " scarlet fever	552	34	337
" " " " measles	845	202	735
" " " " whooping-cough	809	476	777

TABLE 3.

Mortality in State of Pennsylvania.	1911.		1912.	
	All ages.	Under 5 years.	All ages.	Under 5 years.
Acute anterior poliomyelitis	93	61	116	78
Convulsions of infants	1062	..	960	
Diarrhea and enteritis (infantile)	8156	..	7469	
	(Under 2 yrs.)		(Under 2 yrs.)	
Rickets	69	63	90	87
Congenital malformations	1345	1319	1389	1368
" " of heart	881	862	877	867
Injuries at birth	552	552	593	593
Congenital debility, icterus, and sclerema	5942	5942	6420	6420
Diseases of spleen	14	2	10	1
Hernia, intestinal obstruction	855	174	930	191
Pneumonia	3573	1525	7130	2352

MUMPS (EPIDEMIC PAROTITIS).

Mumps is an infectious disease, characterized by swelling of the salivary glands, particularly the parotid gland, together with mild constitutional symptoms.

Etiology.—It has not been proven to be due to any one type of organism, but Catlin and Laveran have isolated from the blood and glandular lymph of the parotids and testes a diplococcus which produced parotitis in dogs and monkeys when injected into Steno's duct. An intracellular diplococcus has also been isolated from Steno's duct, and it is generally believed that the infection enters the parotid from the mouth by way of this duct.

The mode of infection is usually by direct contact, though it is possible to convey the infection by a third person or by clothing. It occurs endemically in large cities, particularly in spring and autumn. It is more common in boys than in girls. Children between the ages of three and ten years are more susceptible than young infants and adults. Owing to the fact that the disease is communicable for several days after the subsidence of the swelling, such children should be isolated for a period of at least three weeks from the date of onset. The period of incubation is from ten days to three weeks, and during this time there are rarely any symptoms of the affection.

Morbid Anatomy.—Owing to the trivial nature of this disease, there has been little opportunity to note the pathological changes which take place; but there is, as a rule, simply a serous infiltration which usually ends in resolution. Suppuration is very rare.

Symptoms.—There is usually a prodromal period during which the child is chilly and may vomit; following this the acute symptoms appear, varying in character and intensity according to the severity of the attack. The onset is usually marked by fever ranging from 100° to 101° F., headache, anorexia, vomiting, and pains in the back and legs. Pain on swallowing or moving the jaw may be the first objective symptom, and the child complains of pain beneath the ear. A slight swelling begins, usually on one side, which reaches its height on the second or third day, remains stationary for two or three days, and then gradually subsides. The swelling causes the lobe of the ear to be lifted and passes forward in front of the ear and backward to the sternomastoid muscle. The other salivary glands, the submaxillary and sublingual, are often not involved or they may at the same time show evidence of swelling, or may not become enlarged until after the parotid swelling has completely disappeared. More frequently both parotid glands become involved, but not simultaneously; the inflammation appears in one, and the maximum swelling is reached in forty-eight hours, after which the other side becomes involved, and swells with equal rapidity. When one alone is affected, it is more often the left than the right. During the height of the disease the mouth becomes dry, the salivary secretions are diminished, and there is excessive pain on swallowing. Mastication is both difficult

and painful, and in extreme cases it may be almost impossible to open the mouth because the parts are so tense and swollen. In young infants there is drooling. There may be earache, otitis media, and frequently also slight impairment of hearing. In the more severe cases there may be high fever, ranging from 103° to 104° F., delirium and marked prostration. Relapses are rare, but cases of recurrent mumps have been reported which persisted for six weeks.

Diagnosis.—The diagnosis is usually easy, though the disease may be confused with acute swelling of the cervical lymph nodes. The latter is usually behind the jaw, and does not extend to the face. In swelling of the parotid gland, the lobe of the ear on the affected side becomes elevated, and occupies the centre of the tumefaction, the swelling extending in front of and below the ear. The rapidity of the swelling and its short duration are characteristic of mumps. Inflammation of the parotid is usually, but not invariably, due to mumps; a history of other cases in the vicinity, is additional evidence in favor of true mumps.

Complications.—In childhood complications are rare, but in puberty orchitis may occur, usually making its appearance between the second and third weeks of the disease, and occurring most frequently in those who are allowed to be up and about. Either one or both testicles may become involved and the swelling is marked. Usually the testicle proper and not the epididymis is affected; but occasionally there may be acute epididymitis, acute hydrocele, edema of the scrotum, or inflammation of the spermatic cord and inguinal glands. Frequently it is accompanied by fever and chills, and the testes are painful and heavy.

The acute symptoms continue for from three to seven days, though frequently the testicle remains enlarged for some time afterward, and the swelling persists even longer when the parotitis is bilateral. Atrophy of the testes with resulting sterility has been reported as following mumps.

In some cases the urine contains a trace of albumin. Urination may be painful, and there may be a urethral discharge. In females there may be congestion of the breasts, ovaries, and labia majora, though such complications are not at all common.

The thyroid gland may show some enlargement during the attack. Delirium and high fever are occasionally observed, coma may appear, more rarely there is acute mania, and, in exceptional instances, insanity. Hemiplegia may occur, and in rare cases meningitis. Suppuration of the parotid gland is rare, and when it occurs is probably the result of a mixed infection.

There may be disturbances of the special senses, such as deafness and optic neuritis. Nephritis has in some cases followed an attack. Pneumonia, endocarditis, and pericarditis occur in rare instances. As a rule complications do not appear until after the parotitis has subsided. Parotitis itself may be a complication of pneumonia, influenza, measles, varicella, or typhoid fever.

Course and Prognosis.—In the vast majority of instances mumps is a mild disease, from which complete recovery takes place in a few days, and the disease generally runs its course in a week or ten days. Severe cases are rare; if uncomplicated, the prognosis is very favorable. When such complications as edema of the glottis, suppuration of the parotid, or meningitis occur, the outlook becomes serious; but involvement of the testicles or ovaries and of the mammary glands, while painful, is rarely dangerous.

Treatment.—Usually little or no treatment is necessary. The patient should stay in bed for at least one week. The bowels can be opened freely by an initial dose of 1 to 3 drams of castor oil, after which the aromatic syrup of rhubarb, 1 to 2 drams, or the aromatic fluidextract of cascara sagrada, 20 to 40 drops, may be administered whenever necessary. The patient should be kept upon liquid diet for a week. If the fever is high, 5 to 10 drops of sweet spirits of nitre may be given every three hours, or the temperature may be reduced by sponging the child in water at a temperature of 75° to 85° F. Hexamethylenamin should be given in 1- to 2-grain doses, three times a day.

If pain is severe, 1 to 2 grains of Dover's powder may be combined with 3 grains of salol or 1 grain of phenacetin, and administered every four hours until the child is relieved.

It is advisable to keep these children warm, and to prevent any chilling of the body throughout the whole course of the disease, since this will expedite recovery, and also because it is believed that complications may be induced by sudden chills.

Locally either cold or hot applications may be used on the gland; usually the latter are more agreeable; they should be covered with a pad of cotton wadding, and over this oiled silk. Relief will often be afforded by anointing the gland with some simple ointment, such as cold cream.

For severe pain warm oil of hyoscyamus may be applied twice daily, or 25 per cent. ichthyol, or such remedies as witch hazel, lead-water and laudanum, or menthol may be employed locally. The mouth should be frequently cleansed with liquor alkalinus antisepticus, 25 per cent. dilution, in order to prevent infection of the gland through the duct. Children suffering with mumps should be isolated and kept apart from other children for a period of not less than three weeks.

Should orchitis develop, rest in bed, together with support of the affected testicle with cotton-wool and adhesive straps, will usually suffice.

INFLUENZA (LA GRIPPE: CATARRHAL FEVER).

Influenza is an acute infectious disease characterized by a catarrhal inflammation of the mucous membrane, particularly of the respiratory tract, together with fever, muscular pain, and marked prostration.

Etiology.—The bacillus of Pfeiffer, discovered in 1892, is found in the sputum and nasal discharges. It is 0.8 to 1 micron in length, and

0.1 to 0.2 micron in breadth. In glycerin agar it forms colonies, which, under the microscope, appear as clear, separate drops. They are best stained by hot Loeffler methylene blue solution, or dilute solution of Ziehl-Neelson carbol-fuchsin. The bacillus is easily transmitted from one individual to another, because of its presence in the sputum and nasal discharges. Other organisms associated with the bacillus of Pfeiffer are the Streptococcus and Diplococcus pneumoniæ.

Influenza occurs epidemically, but after epidemics may remain endemically for some time, although in the majority of endemic cases of influenza it is impossible to isolate the Pfeiffer bacillus. The diplococcus of Fränkel, however, is quite commonly found in these cases, therefore it is often called pneumococcus grippe. Epidemics usually occur during the winter and spring.

The disease also appears in pandemics. This form is not as common in children as in adults, but the endemic form of influenza, which is encountered at all seasons of the year, and the development of which is favored by sudden and great changes in temperature, is very prone to attack children. The disease is rather uncommon in infants, but children between the ages of two and ten are very apt to contract it. One attack does not confer immunity against a second, and some children have it every winter. Among the predisposing factors in the endemic form of influenza are exposure to cold and lowered vitality from malnutrition, exhausting illness, or other debilitating influences.

The period of incubation is supposedly short, ranging from two to seven days. The chief complications are pneumonia, pleurisy, and endocarditis.

Two types of influenza are recognized: *epidemic influenza vera*, caused by the bacillus of Pfeiffer, and *endemic influenza vera*, which appears for several successive years after a pandemic.

Morbid Anatomy.—Our present knowledge of the pathological changes caused by influenza is very scant, for whatever alterations are produced by this disease promptly disappear at death. There is, however, an inflammation of the mucous membrane of the upper respiratory tract, and in severe cases the trachea, bronchi, and peribronchial tissues may also be affected. The remaining pathological changes are due to the complications, such as bronchopneumonia, otitis media, meningitis, and empyema. In infants there may be catarrhal inflammatory changes in the mucous membrane along the gastro-intestinal tract.

Symptoms.—The symptoms of influenza in children vary greatly, according to the severity of the attack. If mild, the onset is usually sudden, and the attack lasts about a week. Coryza, sneezing, and watering of the eyes are usually the first symptoms to make their appearance, and these are followed by chills, and coughing with profuse expectoration. The throat and nares are red and injected, and the tonsils are frequently covered with mucus, and studded with a yellowish deposit. The cough is loud, harsh, painful, and sometimes very persistent.

Muscular pain is more or less constant, and may often be the first symptom of the illness, manifesting itself in the back, extremities, and head, so that the child cries with pain when picked up or moved about. Vomiting may occur. The temperature range is from 101° to 103° F. There is usually associated pharyngitis and bronchitis, and prostration is always quite marked. In infants early involvement of the gastro-intestinal tract is shown by green stools, diarrhea, loss of appetite for breast or bottle, and colicky pains in the abdomen. Older children become nauseated and constipated, and complain of · epigastric pain.

In the more severe types of influenza the onset may be extremely sudden, and prostration great. Fever, though mild at first, may run exceedingly high, from 102° to 105° F. and even 106° F. At this time delirium may occur, and nervous symptoms become prominent. The child usually lies in a drowsy, stuporous state, and although convulsions are rare, musclar twitchings are not uncommon. The pulse rate is usually accelerated, and corresponds with the degree of temperature.

In children the attack may be preceded by severe vomiting, and sometimes diarrhea; but the symptoms and physical signs are not as severe as one would expect with the degree of fever, although prostration is profound. Follicular tonsillitis frequently accompanies this class of cases, and pneumonia is a common complication. The moderately severe cases terminate in six to ten days, but occasionally influenza assumes a chronic form, in which the temperature and other symptoms persist for several weeks.

In endemic grippe, especially, there is usually a slight trace of albumin in the urine, and the kidneys may become involved, actual nephritis being indicated by a decrease in the quantity of the urine, and the presence of blood and casts. There is no edema, but the persistence of albuminuria for weeks and months denotes a low-grade nephritis.

In very severe cases of influenza, a bacillus may be found in the blood stream, and meningitis and arthritis are not infrequent accompaniments. According to the predominating symptoms, the disease in the adult is classified as respiratory, nervous, gastro-intestinal, or febrile influenza; but in the child this distinction is not quite so definite for the symptoms are often irregular. In some children the gastro-intestinal, respiratory, and nervous forms may occur one after the other.

Respiratory Form.—In the respiratory type the entire tract becomes the seat of the disease. The onset is marked by coryza and evidences of acute catarrhal fever. In some cases bronchitis follows the catarrhal symptoms, there is a steady increase in the fever, delirium may appear, together with profound prostration. Pharyngitis, tonsillitis, and laryngitis are prominent features of the respiratory form of influenza, and latent pulmonary tuberculosis may again become active in consequence of the attack.

The expectoration is copious, and contains masses of purulent material; sometimes the sputum may be bloody and of a dark red color. The most serious complication is pneumonia, which is most frequently lobular in type. It may set in after several days of severe bronchitis and high fever, the smallest bronchi gradually becoming involved, or it may develop suddenly. In the majority of cases pneumonia is due to the pneumococcus, and clears up readily, but in some instances it rapidly proves fatal. The outcome depends to a great extent upon the virulence of the organism, and the resistance and vitality of the child. Pleurisy may occur, but is rare.

Nervous Form.—This type is marked by the absence of catarrhal symptoms, and manifests itself by pains in the back, chest, and extremities, intense headache, and extreme prostration. These children are very irritable and restless, but may become stuporous. Occasionally convulsions occur, but severe nervous manifestations are noted only in the worst forms of influenza. Rigidity of the muscles of the neck, muscular twitchings, paresthesia, and dizziness may be observed. Many cases in which nervous symptoms are conspicuous are accompanied by pneumonia. The chief complications are meningitis and encephalitis, but, in fact, almost any type of nervous disease may complicate an attack of influenza.

Gastro-intestinal Form.—In this type nausea and vomiting predominate, together with abdominal pain and diarrhea, all of which have a tendency to cause collapse. Malnutrition is severe and, together with the extreme prostration which accompanies grippe, results in exhaustion and emaciation which are often the forerunners of collapse. Gastro-intestinal symptoms usually predominate in very young children, and in infants there may be enlargement of the spleen.

Febrile Form.—Here fever may be the only clinical manifestation of the disease, and may be accompanied by chills, and be remittent in type; or, again, the fever may be continuous, and closely resemble typhoid fever.

Diagnosis.—Epidemic grippe is, in the majority of cases, easily diagnosed, but mild sporadic cases may present some difficulty until carefully studied. The profound prostration which is out of all proportion to the other clinical manifestations, together with the suddenness of onset and short duration of the fever, will usually point to the diagnosis.

The nervous symptoms may arouse a suspicion of meningitis, but true symptoms and signs of meningeal inflammation are lacking; and the onset of pneumonia in this class of cases is very significant of grippe.

In rare cases influenzal meningitis may be present and can be demonstrated by examination of the cerebrospinal fluid which contains the bacilli. Pneumococcic and cerebrospinal meningitis are, to a great extent, differentiated from the influenzal type by the bacteriological findings, for the symptoms are not widely different.

The high fever and cough with a history of chills may simulate

pneumonia, but careful examination of the chest will exclude this, except when pneumonia is present as a complication. At the onset of influenza both scarlet fever and measles may be simulated by the pharyngitis, coryza, injected conjunctiva, and bronchitis, and in these cases the differential diagnosis is occasionally rendered even more difficult by the appearance of a rash on the skin. An accurate history and careful study of such cases for two or three days will generally clear up the diagnosis.

In the subacute type of influenza the symptoms may to some extent resemble those of typhoid fever, and this is especially the case in prolonged attacks with continued fever, but the Widal reaction will be found negative, there are no rose spots, nor is there any marked enlargement of the spleen.

The presence of the influenza bacillus in the sputum may be determined microscopically early in the disease, but a bacteriological diagnosis of influenza, while very valuable, is too difficult to be resorted to in most cases.

Complications.—Pneumonia is by far the most serious complication of influenza, and may be associated with pleurisy and empyema. Endocarditis and pericarditis, together with irregularity of the heart—tachycardia and bradycardia—may also set in, and meningitis and encephalitis are sometimes sequels. Influenzal meningitis is marked by symptoms of meningeal inflammation; the cerebrospinal fluid is cloudy, and contains an increased number of polynuclear cells. Upon microscopic examination of the cerebrospinal fluid the bacilli are seen to be both intracellular and extracellular. This complication usually results in death. Acute otitis media is a frequent complication, and may result in mastoiditis and sinus thrombosis. Phlegmasia alba dolens, caused by venous thrombosis, has occurred in several instances. Cervical adenitis with involvement of the parotid and submaxillary glands is not uncommon.

Skin rashes may appear during the course of the disease, although no one eruption is characteristic of influenza. Among the various skin lesions which may be noted are herpes facialis, herpes zoster, erythema papulatum, urticaria, and a scarlatiniform erythema which, to a certain extent, resembles the rash of both measles and scarlet fever. Secondary anemia usually follows an attack of influenza, and some observers have noted an increase in the number of cases of appendicitis during epidemics of influenza.

Cystitis frequently follows grippe, especially in little girls, and influenza may either give rise to rheumatic symptoms or cause an acute exacerbation of rheumatism in children who already suffer from this disease. Influenza has a tendency to stir into activity any latent form of disease; this is particularly true of latent tuberculosis.

Prognosis.—The prognosis is usually favorable, provided the patient does not take cold during the attack, and thus bring on that dangerous sequel, pneumonia, as well as other complications. In infants who have influenza there is often serious involvement of the intestinal tract,

45

and as pneumonia is a common complication in the younger child, the prognosis is more serious in infants and very young children than it is in later childhood. Death may also be the result of toxemia and exhaustion, especially when a severe attack of influenza occurs in a child whose general health is undermined by malnutrition or some constitutional disease, such as tuberculosis or syphilis. Influenza attacks strong healthy children, as well as weaklings, and the mortality among children varies to a certain extent in different epidemics.

Treatment.—All cases should be isolated because of the contagiousness of the disease, and after recovery the sick-room should be fumigated. Children should immediately be put to bed, and remain there until after the attack has subsided, when they may be allowed to play about in one or two rooms from which the other members of the family are excluded. The bowels should be freely opened by the use of castor oil, 1 dram to $\frac{1}{2}$ ounce, or some other purgative, such as magnesium citrate, 2 to 6 ounces, or magnesium sulphate, 1 to 2 drams. If, in infants, there is diarrhea, they should also be given an enema, and no milk should be allowed until improvement takes place. In its place albumen-water, oatmeal gruel, rice- or barley-water, or beef juice may be given at frequent intervals, the amount depending upon the age of the child and severity of the symptoms.

Fever should be combated by sponging or by a tepid bath with the water at about 98° F. Following the bath the child should be well rubbed. An ice-bag may be kept upon the head, and in some cases it affords relief if applied to the chest, being moved about every half-hour.

The nervous symptoms accompanying the disease should be controlled by the use of phenacetin, $2\frac{1}{2}$ grains night and morning, or Dover's powder, $\frac{1}{2}$ to 1 grain, or codein sulphate, $\frac{1}{32}$ to $\frac{1}{20}$ of a grain. Should there be cardiac weakness nothing is quite so efficient as alcoholic stimulation, and 10 to 30 drops of whisky or brandy may be given every three hours, if necessary. Other cardiac stimulants which may be used are strychnine and digitalis, $\frac{1}{400}$ to $\frac{1}{200}$ of a grain of strychnine sulphate, and 1 to 3 drops of tincture of digitalis (or $\frac{1}{10}$ to $\frac{1}{2}$ grain of strophanthus) may be administered every three hours.

In older children the use of quinine sulphate, $\frac{1}{2}$ to 1 grain, is of decided benefit, particularly if combined with either phenacetin, 1 grain, or Dover's powder, 1 grain, and given every six hours. Its use in infants is not so highly recommended because of its tendency to upset the stomach.

The cough which accompanies influenza may be relieved by the use of codein sulphate, $\frac{1}{40}$ to $\frac{1}{20}$ of a grain, or heroin hydrochloride, $\frac{1}{50}$ to $\frac{1}{25}$ of a grain three times a day, the dose of these drugs depending upon the age of the child. Much can be done to prevent pneumonia and otitis, as well as other complications, by spraying the nasopharynx two or three times daily with a saturated boric acid solution or normal saline. When the cough lasts for some time after an acute attack, it is best combated by the use of cod-liver oil, to which is added 5 drops

of creosote to the ounce, and $\frac{1}{2}$ to 1 dram of this may be given two or three times daily.

The diet should be carefully regulated. All children who have any tendency to pulmonary disease, particularly tuberculosis, should be carefully guarded, and they should, if possible, if the attack occurs during the winter months, be removed to a mild and equable climate. Such winter resorts as Brown's Mills in the Pines, or Old Point Comfort, provide an ideal climate for convalescence in these cases.

In influenzal meningitis, the use of the specific serum which has now been placed on the market is strongly urged, since it bears the same relation to the disease, and is of the same significance, as is Flexner's serum in cerebrospinal meningitis.

SMALLPOX (VARIOLA).

Smallpox is an acute infectious disease, characterized by an eruption of the skin which passes through successive stages of papule, vesicle, pustule, and crust.

History.—It is believed to have existed in China at least a thousand years before the Christian era. It was imported into this country in the latter part of the sixteenth century by the Spanish. In 910, Rhazes wrote the first description of smallpox, and it was first distinguished from measles by an Arabian physician, Avicenna by name.

Etiology.—Natural immunity to the disease is rare, and those exposed, unless protected by vaccination, are almost certain to be attacked. In the majority of instances, one attack protects against a second, though second and even third attacks have been reported. It is exceptionally fatal in children under ten years of age, but may affect persons of all ages. A pregnant mother may contract the disease, and the fetus *in utero* be attacked at the same time. Males and females are equally affected. Negroes are particularly susceptible, and the mortality among them is greater than in the white race, the ratio being almost two to one. In temperate climates it is looked upon as a cool weather disease, and in tropical countries is regarded as a hot weather malady which ameliorates as cooler weather approaches. Once implanted in a community, the disease rapidly spreads, but the virulence of epidemics in different localities varies greatly. In the United States the disease is steadily on the decrease, as it is in all other countries where vaccination is practised.

Contagion.—There is no doubt that smallpox is the result of infection by a specific organism, and that this organism is present in the blood has been proven by inoculating a monkey with blood from a person suffering with the disease. It is also present in the pustules, the pus from these, when inoculated into a human being, producing smallpox. Unquestionably also it is present in the exhalations, and the infection may be carried by a second person or by the atmosphere. Clothing and other articles which have come in contact with the patient may also be a means of conveying infection.

The period in which smallpox is most contagious is during the stage of suppuration and early dessication. A case has been recorded by Austin Flint in which the contagion was spread by a cadaver. Domestic animals and insects may also transmit it. In most cases the disease is contracted by direct contact, and such contact need not necessarily be for any great length of time, inasmuch as the slightest exposure to smallpox is often followed by an attack. The severity of the illness in one person does not necessarily imply that other individuals, who contract the disease from having come into contact with him, will have a severe attack, for the disease when acquired from one suffering with it in virulent form, such as hemorrhagic smallpox, is often exceedingly mild, and the reverse may also be true.

The nature of the contagion is not definitely known, although Councilman has described a protozoön which gains entrance into the nuclei of the epithelial cells, and causes the formation of minute vacuoles about a central vacuole. The life-history of the organism has been further studied, and its relation to the skin lesions of smallpox give the impression that it is the actual cause of the disease.

Pathology.—In some cases the mucous membrane of the mouth and pharynx is the seat of pustules, and, indeed, there have been instances where the rash has extended down the esophagus and into the stomach. It may also involve the trachea and bronchi; and, although no true pocks are found in the bronchi, bronchitis, broncho-pneumonia, lobar pneumonia, and pleurisy are occasionally associated with it. The spleen becomes greatly enlarged, and the kidneys frequently are the seat of cloudy swelling; often minute areas of necrosis may be seen. The heart may be the seat of myocardial changes, chiefly of a parenchymatous or fatty nature. In the hemorrhagic form of the disease the serous and mucous surfaces and parenchyma of various organs become the seat of extravasations. At times there is hemorrhage in the bone-marrow.

Histologically the pustule begins in the rete mucosa, and consists of a central area of coagulation necrosis, surrounded by an infiltration of leukocytes together with serum and fibrin. In the more severe forms of pustule, infiltration extends into the papillæ of the skin, causing their destruction, which terminates in the formation of a pit.

Symptoms.—The types of smallpox described are:
1. Variola vera.
    (*a*) Discrete.
    (*b*) Confluent.
2. Variola hemorrhagica.
    (*a*) Purpura variolosa.
    (*b*) Variola hemorrhagica pustulosa.
3. Varioloid—smallpox modified by vaccination.

The period of incubation is usually from seven to fourteen days. The onset is sudden, accompanied by severe headache and backache, which in adults are sometimes preceded by a chill. In children the

only initial symptom may be a convulsion. Fever follows, often reaching 103° to 104° F. during the first twenty-four hours. The pulse becomes rapid and bounding, and at this stage there is frequently delirium. Constipation and persistent vomiting may become prominent. Usually a violent frontal headache, vertigo, and severe pains in the lumbar region are complained of.

The initial rash makes its appearance on the second day, either as a diffuse scarlatinous or a measly form, and covers a certain portion of the body surface, though it is usually confined to the abdomen, the inner surface of the thighs, and the axillæ. On or about the fourth day small red spots make their appearance, first on the forehead and wrists, followed by rapid extension over the face, abdomen, and extremities. Within another twenty-four hours these papules acquire a characteristic shot-like hardness, and at this time there is an amelioration of symptoms—namely, a drop in temperature and general relief from the violent headache and backache. On the sixth day the papules become vesicular and depressed, thus forming what is known as the umbilication, which is extremely characteristic of the smallpox eruption. The fluid in the vesicle increases in turbidity until about the eighth day, when it becomes intensely yellow and the umbilication disappears, thus forming a pustule.

At this time there is a return of fever and, because of the tension about the eruption, some degree of pain in these parts. On or about the eleventh day the pustules begin to dry, and this continues until the fourteenth day, when crusts are formed which fall off, leaving either a slight discoloration, as in the milder types, or an ulcer, or a pit, according to the degree of cicatrization. By this time the secondary fever has about disappeared.

Many variations of the eruption may be noted, the preceding description being that of the usual simple or *discrete* variety. When the pustules are in close proximity, they may unite, thus producing the *confluent* variety, and when accompanied by bloody infiltration the term *hemorrhagic* smallpox is used.

In the confluent type of the disease, the eruption usually makes its appearance earlier than in the discrete form, that is, on the third day. The symptoms are usually aggravated, and there is no abatement of the fever when the rash appears, as is the case in the discrete form. In the confluent form of the disease we may sometimes see enormous pus vesicles, some part of which may be dry as the result of early rupture here and there. In the more severe cases of this type extreme exhaustion makes its appearance on or about the tenth day, and frequently brings about a fatal termination.

The hemorrhagic form of the disease is even more severe than the confluent. Two types are described. The first is *purpura variolosa*, in which hemorrhage makes its appearance early in the form of a hemorrhagic rash, together with hemorrhage from the mucous surface which often occurs as early as the second or third day. Death usually soon follows. In the second form, known as *variola hemorrhagica*

pustulosa, the disease follows the usual course until the vesicular or pustular stage is reached, when hemorrhagic symptoms manifest themselves by the appearance of blood in the pocks.

VARIOLOID.

This variety, which is variola modified either by vaccination or a previous attack of smallpox, is characterized by the extreme mildness of its symptoms, and by early convalescence. The fever is less, and the eruption exceedingly light, sometimes failing to show its successive stages. Usually the interval between vaccination and the attack is the factor which determines the severity; that is, the longer the interval between the two, the more severe the disease is apt to be.

Abortive Types.—Various modifications of smallpox may be noted in different epidemics. In unvaccinated children there may sometimes be but a few pustules, and the disease may terminate uneventfully in a few days; again the vesicle may, instead of becoming filled with pus, dry and disappear, giving rise to the so-called *wart-pox*. Another form, known as *variolæ sine variolis* may be seen in which all the symptoms of the disease are present except the eruption; such cases are not numerous.

Diagnosis.—Vomiting preceded by a chill and accompanied by severe headache and backache should arouse suspicion, particularly so if there is a history of exposure to smallpox. When the eruption is typical, the diagnosis is easily made, for in no other disease do we find such a profuse pustular eruption as in smallpox; but in mild cases and those modified by vaccination the diagnosis is sometimes a matter of great difficulty. Much importance should be attached to a history of exposure, to the presence or absence of a recent and good vaccination mark, and to the visible signs of a preceding attack of smallpox. A positive diagnosis is scarcely possible before the appearance of the rash. The primary rash might be confused with that of scarlet fever or measles, but other symptoms of the two latter diseases ought to aid in the differentiation.

The constitutional symptoms of smallpox are, however, more severe than those of measles, and in variola the fever declines before the appearance of the eruption, while in measles the temperature is usually at its height at the time of the eruption. In smallpox there is no prodromal catarrhal inflammation of the upper respiratory tract, and the eyes are not inflamed. Koplik's spots are not seen in smallpox.

Scarlet fever may be differentiated from smallpox by the prodromal rash, sore throat, and the absence of severe lumbar pains. The symptoms of the two diseases are also essential points in the differentiation. In the malignant hemorrhagic type of the disease, death may occur prior to the appearance of the characteristic eruption, and in such cases it is extremely difficult to distinguish it from hemorrhagic scarlet fever and hemorrhagic measles.

The disease with which smallpox is most frequently confounded

is varicella, and this occurs most often when the disease assumes a mild form. The most valuable points in the differentiation of the two diseases are as follows:

In varicella the prodromal symptoms are not as intense as in small-pox. It is more apt to occur in children than in adults. The rash is most abundant about the trunk, and it appears in crops. The papules do not have a "shot-like" feeling, the vesicles are superficial, and ruptured by the slightest pressure. There is little or no infiltration about the pocks. Finally, the presence or absence of a good vaccination mark is a determining factor in making the differential diagnosis.

Pustular syphilid may be confounded with the disease, particularly if accompanied by fever; but the absence of, or merely a slight, rash on the face in syphilis, together with the history of a primary lesion in adults, or a Wassermann reaction, should be our guide.

Confusion may also arise in differentiating the hemorrhagic variety of the disease from cerebrospinal fever. The eruptions of acne, eczema, syphilis (pustular), and vaccinia have, in a few instances, been confused with the eruption of smallpox.

Complications.—Among these are laryngitis followed by fatal edema of the glottis, necrosis of the cartilages, and bronchopneumonia induced by the aspiration of particles into the lower air passages owing to the diminished sensibility of the larynx. Almost all of the fatal cases show evidences of bronchopneumonia.

Cardiac complications are also seen in the form of myocarditis and pericarditis, more rarely endocarditis. During the height of the primary fever a systolic murmur may sometimes be heard at the apex. Of complications in the digestive system, diarrhea is in all probability the most frequent, especially in children. Nephritis is rare, although albuminuria is frequent. Occasionally there may be inflammation of the ovaries and testes. The skin may be the seat of boils, acne, and ecthyma, and occasionally of small areas of gangrene. Violent conjunctivitis may result from neglect of the eyes, and there may be a diffuse keratitis. Otitis media may result from extension of the disease by way of the Eustachian tubes.

Complications of the nervous system may be serious. The delirium so common during the early stages of the disease may increase in intensity, and be prolonged indefinitely, until it results in fatal coma. Insanity occasionally develops during convalescence.

Arthritis may make its appearance during the stage of desquamation, and may terminate in suppuration.

Prognosis.—Age, race, and the type of the epidemic are the chief determining factors in the death-rate. The mortality is extremely high in the young. In the epidemic which occurred in Montreal, in 1885, 86 per cent. of the total number of deaths occurred in children under ten years of age. The average mortality is from 25 to 35 per cent. The mortality of varioloid is much lower, ranging from 10 per cent. in infancy to 5 per cent. in older children. Complications, such

as pneumonia and laryngitis, are always serious, and recovery from hemorrhagic smallpox is rare.

Treatment.—The child should be isolated, and, if possible, promptly removed to a smallpox hospital. If seen early in the attack it should be vaccinated at once. If the child remains at home, it should preferably be isolated on an upper floor, and all communication with the rest of the household cut off. Sheets saturated or dampened with a solution of carbolic acid should be hung at the doors. All unnecessary furniture and all carpets and hangings should be removed from the room. There should be separate eating utensils. The nurse in attendance should wear a gown that completely covers her ordinary dress, and should not come in contact with any one save the sick child.

The treatment is chiefly symptomatic. The violent pain in the back of the head is controlled chiefly by the use of sedatives, such as morphine sulphate, gr. $\frac{1}{40}$ to $\frac{1}{16}$ every four hours, in the more severe cases, and phenacetin, 1 to 3 grains every four hours, or antipyrin, 1 grain every four hours, in the milder cases.

The food should be liquid. When there is prostration stimulation should be resorted to, and for this purpose 10 to 30 drops of brandy, or strychnine sulphate, gr. $\frac{1}{400}$ to $\frac{1}{100}$, may be administered every three hours.

If vomiting is persistent, 10 to 20 grains of bismuth subnitrate, $\frac{1}{4}$ of a grain of cerium oxalate, $\frac{1}{2}$ to 1 dram of iced champagne, or simply cracked ice, may be given to check it.

Quinine sulphate, $\frac{1}{4}$ to $\frac{1}{2}$ grain, should be given every four hours when the fever runs high, and the temperature may also be reduced by sponging. In instances also where delirium is marked, baths should be resorted to. These should not be given cool at first; but, if a bath with friction at 95° F. does not bring down the fever, the temperature of the water may be gradually lowered from 95° to 80° F. Plenty of cold water should be given the child to drink, as in fevers of other types.

The treatment of the eruption is important, in order to prevent, if possible, the disfigurement which so frequently follows severe attacks of smallpox. Various methods have been tried, but none with satisfactory results. The application of a cold weak solution of carbolic acid or bichloride of mercury upon gauze is in all probability one of the most effective methods in use today. During the first seven or eight days of the eruption, tincture of iodine, either full or half-strength, may be painted once or twice a day over the face or any other portion of the body where the eruption is profuse. Many physicians claim most excellent results from this treatment. In order to prevent dissemination of the epidermis during the stage of crust formation, it is expedient to apply an ointment, such as vaseline or cold cream, freely over the skin surface.

Treatment of the complications requires consideration. In children, if diarrhea is severe, opium in the form of paregoric should be administered in 10- to 20-drop doses every six hours. Tracheotomy may be

necessary if the symptoms point to obstruction of the larynx by edema. Great care should be exercised to prevent the occurrence of bed-sores. The eyes should be kept absolutely clean with a solution of boracic acid, which will lessen the probability of keratitis. The nose, mouth, and throat should also be cleansed frequently to prevent, if possible, the formation of hard crusts. In addition, the mouth and nasopharynx should be sprayed with a 1 to 5000 solution of potassium permanganate, or a 1 to 5 solution of hydrogen peroxide, once or twice a day.

During the stage of convalescence great care should be exercised as to cleanliness and changing the position of the child, to prevent the formation of bed-sores or pulmonary complications. There should be daily baths followed by inunctions with olive oil. This should be continued until the whole skin surface is smooth, and all evidences of scab formation have disappeared, since these scabs are potent factors in the further dissemination of the disease.

VACCINIA (COW-POX).

Vaccinia is an eruptive disease of cattle, communicable to man only by inoculation (vaccination), in which event it produces one or more lesions, according to the number of points of inoculation. General vaccinia, in which the lesions are scattered irregularly all over the surface of the body, occurs in about 2 per cent. of all children who are vaccinated. The eruption is frequently the result of auto-inoculation from the original site of vaccination, and may be erythematous, urticarial, scarlatiniform, vesicular, bullous, pustular, or pemphigoid, although the most common type is the urticarial.

It usually appears from the fourth to the tenth day after vaccination, but in exceptional cases may occur weeks later. As a rule the eruption appears in successive crops, and the lesions are first papular, then vesicular, and finally become pustules, thus resembling the original sore. The eruption may be seen in all of its different stages of development upon the same individual.

There may be a variable degree of fever, this depending upon the number and extent of the lesions. In the scalatiniform variety there is usually great discomfort and a feeling of malaise, as well as moderate fever.

Auto-inoculation is common in children who have affections of the skin which cause a break in its continuity. These lesions become inoculated with virus from the original sore, and a profuse eruption of vaccinia may appear. The lesions may be discrete, but often become confluent, and bear some resemblance to the original vaccination mark.

Vaccinal ophthalmia may also be caused by transmission of the virus to the conjunctiva by means of the child's fingers or the indiscriminate use of towels, wash rags, and sponges. The crusts dry and fall off at the end of the third week, and, as a rule, the lesions of general vaccinia entirely disappear after this time, and there are no more new crops. Such inoculation confers protection against smallpox.

History.—While Edward Jenner was a medical student at Sodbury, he became acquainted with the fact that cowpox protected against smallpox. This he learned from a young country girl who, on hearing smallpox mentioned, exclaimed "I cannot take that disease, for I have had cow-pox." Jenner mentioned the fact to Hunter, who advised him to be inoculated, and thus prove the accuracy of the country girl's statement.

All this occurred in 1780, but it was not until 1796 that Jenner inoculated a boy, named James Phipps, eight years of age, with virus from the hand of a dairy maid, Sarah Nelmes, who at the time was suffering with cow-pox. As early as 1774, Benjamin Jesty, a Yetminster farmer, vaccinated his wife and children with matter taken from cows that were suffering with cow-pox. Subsequently his children were inoculated with matter from the lesions of smallpox, and did not take the disease. Nevertheless, the credit must be given to Jenner for establishing the fact that cow-pox did protect against smallpox, and this truth was established only after much study and experimentation. It was not until the year 1800 that cow-pox was introduced into the United States by Benjamin Waterhouse, Professor of Physics at Harvard, who at that time successfully vaccinated seven of his children. Two years later nineteen boys were inoculated, twelve of the number being subsequently inoculated with smallpox, and all showed immunity to the disease. Two unvaccinated boys were inoculated with the same smallpox virus, and both contracted the disease.

Jenner died on January 6, 1823, and twelve days prior to his death he wrote as follows: "My opinion of vaccination is precisely as it was when I first promulgated the discovery. It is not in the least strengthened by any event that has happened, for it could gain no strength. It is not in the least weakened, for, if the failures you speak of had not happened, the truth of my assertions respecting those coincidences which have occasioned them, would not have been made out."

Vaccine Virus.—Two types of virus exist, the humanized and the bovine type.

Humanized Virus.—Humanized virus is seldom used at present because of the possibility of the subject having syphilis, hereditary or acquired, or some other constitutional disease. When used it should be taken from a vaccine pock of the fifth to the eighth day, and such a vesicle should be primary and contain clear fluid. Should there exist any inflammatory condition other than that normally present, another individual should be sought. Usually the virus is obtained from young subjects because of the slighter possibility of their having transmissible diseases.

To obtain the lymph a vesicle is punctured and a capillary tube inserted in the puncture. The tube is then sealed, and kept in a cool place until needed for use, at which time the ends of the tube are broken, and the lymph is expelled by means of a small rubber bulb.

Bovine Virus.—Two types of this virus are employed: namely, lymph and vesicle pulp. The former is merely the clear lymph from a vaccine vesicle, while the latter is a combination of not only the lymph, but also the epithelial lining of the pock. This has been found to be more active than lymph alone. The lymph may be used in a dry form upon points of ivory or celluloid, which are known as dry points, or in the form of a glycerinated emulsion which is marketed in sealed tubes. The latter form is the one most frequently used.

Vaccination.—Nowadays the custom of vaccinating children between the ages of four and six months has become popular. Should there be an epidemic of smallpox, or a history of contact with smallpox, no matter what the age of the child may be or whatever else the child may be suffering from at the time, it should be immediately vaccinated. If there is no smallpox about, the physician ought to decide when the child should be vaccinated, and this should be when it is in good physical condition.

Technic of Vaccination.—The part to be vaccinated (preferably the left arm in boys and the calf of the leg in girls) should be thoroughly disinfected in the following manner: The part should be first washed with tincture of green soap and water, and this followed by an application of alcohol, then dried, cleansed with sterile water, and again dried with either a clean piece of gauze or a clean towel. The abrasion necessary for inoculation of the virus should be made either with a lancet or a needle, either of which must have been previously rendered sterile by immersion in alcohol. The needle is preferable in view of the fact that a new one may be used at each vaccination. Such an abrasion, if on the arm, is usually made at the insertion of the deltoid muscle, with cross-scarifications about one-third of an inch in length and extremely superficial in order to draw only lymph. If blood is drawn it has a tendency to prevent absorption, and if the scarifications are deep there is a possibility of severe inflammation subsequently. The virus should then be expressed upon the abraded area, and carefully rubbed in, which often precludes the possibility of failure. The lymph should then be allowed to dry by exposure to the air, after which it is covered with a sterile piece of gauze.

Shields should not be applied, as they have a tendency to produce congestion by pressure. To protect the vaccinated area it is advisable to keep it covered, either with sterile gauze or by sewing a small piece of clean gauze to the sleeve of the undershirt or to the inside of the stocking. Should the vesicle be injured, great care must be exercised to prevent its infection by other organisms.

Vaccination may also be performed hypodermically by the use of a hypodermic needle. In this method several punctures are made quite superficially, and the lymph is injected.

Stages and Symptoms of Vaccinia.—Practically no symptoms are manifest during the first and second days after vaccination. On about the fourth day, a redness appears about the site of inoculation which increases, then a papule forms, more flat than elevated, which becomes

vesicular on the fifth day, and attains its maximum size by the eighth. The vesicle formed is depressed in the centre, elevated at the margins, and contains a clear, thin, transparent fluid. On the tenth day an extensive areola surrounds the vesicle, the contents of which become purulent. At this time there is often marked induration, swelling and pain. By the twelfth day the areola begins to lessen, the fluid within the vesicle becomes opaque, and shows evidences of drying up. By the fifteenth day desiccation is complete, the vesicle dries up, and a hard crust is formed which usually falls off at the end of the third or the beginning of the fourth week, leaving a pitted red scar which later becomes pale.

Following vaccination there are usually constitutional symptoms which may be either mild or marked, according to the degree of inoculation. Fever, restlessness, anorexia, and irritability usually appear on the third or fourth day, and continue until the tenth or twelfth. If the vaccination is on the arm, the axillary glands are frequently affected, and become enlarged and tender. If the vaccination be on the leg, the inguinal glands may show the same disturbance.

It is generally conceded that people should be revaccinated every seventh year and whenever smallpox is prevalent.

Not infrequently certain irregularities are met with in vaccination, such as complete termination in a week, or the reverse—namely, a very slow development of the pocks. There may be ulceration or infection as the result of bruising or scratching, or a number of vesicles may appear in the neighborhood of the primary one, or secondary vesicles may form in other parts of the body. Vaccination may even be fatal in children in whom severe secondary infection takes place.

The arm may become quite sore, owing to a marked local reaction. In these cases moist boric acid dressings should be applied continuously, or a 1 per cent. picric acid solution or compound tincture of benzoin may be painted on the inflamed areola twice daily. The various other complications of vaccinia are, for the most part, surgical, and should be treated along these lines.

Complications.—Ulceration, sloughing, and cellulitis may result either from uncleanliness or from injury to the vesicle. Various skin disorders, such as erythema, urticaria, erysipelas, and impetigo, also glandular abscesses, either axillary or inguinal, may follow vaccination. Syphilis may be contracted by the use of humanized virus taken from some one suffering with the disease, either hereditary or acquired. Such instances are indeed rare, and are of little or no likelihood because of the almost universal use of animal lymph.

The possibility of transmitting bovine tuberculosis is very slight. Tetanus, too, is fortunately rare, because of the extreme precautions observed in the preparation of animal lymph; nowadays, if such a complication does occur, it is usually due to infection of the vaccine pock following an injury.

It has been thought that vaccination has a certain beneficial influence upon the course of some constitutional diseases, particularly syphilis

and tuberculosis. But the reverse is true, in that it has a tendency to light up or bring into activity a latent syphilitic or tuberdular taint. The general opinion prevails that if vaccination is not successful, provided it has been done carefully, it indicates a certain degree of immunity to smallpox.

Value of Vaccination.—In communities in which vaccination is systematically practised, smallpox is seldom, if ever, seen. A notable illustration of its efficacy is the fact that in Berlin, from 1795 to 1799, before vaccination was made compulsory, 65 per cent. of the deaths were due to smallpox. Following the introduction of vaccination, the figures for the ensuing five years were as follows: 7.5 per cent.; 6.4 per cent.; 0.7 per cent.; 1.3 per cent.; and 0.2 per cent. The mortality from smallpox in persons who have·previously been vaccinated ranges from 6 to 8 per cent.; in the unvaccinated it is at least 35 per cent., which shows the protective value of a single vaccination.

Contraindications.—In the absence of an epidemic of smallpox, it is not advisable to vaccinate an infant until after it is three months old. In older children the contraindications to vaccination are general or localized skin lesions, either syphilitic or tuberculous, or any other severe and recurrent skin disease, whether acute or chronic. Children who have just recovered from an exhausting illness, or are debilitated by other causes, should not be vaccinated until they regain their strength and normal vitality.

TUBERCULOSIS.

Tuberculosis is an infectious disease caused by the Bacillus tuberculosis, and characterized by the formation of tubercles or infiltrations which become caseous or sclerotic, and eventually either ulcerate or calcify. It may run an acute, subacute, or chronic course.

Etiology.—The tubercle bacillus was discovered in 1882, by Robert Koch. It is a rod-shaped organism, varying in length from 1.5 to 3.5 microns, and in breadth from 0.2 to 0.4 micron. The bacilli are usually slightly curved, and often beaded, which sometimes causes them to be confused with streptococci, though the beads of tuberculosis are merely vacuoles, the result of degeneration. The tubercle bacillus is immobile, and of very slow growth when artificially cultivated. It is acid fast, that is, after having been stained by means of a dye, it does not become decolorized when treated with an acid, but there are other organisms which possess this same peculiarity; namely, the Bacillus lepræ and the smegma group. The tubercle bacillus is readily killed when exposed to direct sunlight.

The culture media used for its growth are blood serum, glycerin agar, bouillon, or potato, and to promote their growth on these they should be kept at blood heat after being inoculated. ·The colonies appear as grayish-white masses on the surface of the culture media. Occasionally the organisms assume a branch-like formation, and may

be seen as minute oval or round bodies which take a deep stain; these are known as Schron's capsules.

In 1901, Koch announced that human and bovine bacilli were different, whereas prior to this time he had considered them as one and the same, although von Behring and Ravenal had demonstrated by experiments that the bacillus of human tuberculosis is capable of producing tuberculosis in cattle. The question today seems to resolve itself into a probability that the original parentage of the organism was one and the same, and that differences in its forms and characteristics were produced by its mode of life and its host. The disease, so far as is known, can be transmitted from man to cattle only when the individual is excreting organisms of the bovine type. On the other hand, it is possible for the human organism to become infected by the bovine bacillus.

Parke and Krumweide, in a study of 132 children between the ages of 5 and 16 years, found the bovine type in 33 cases which were classified as follows:

Tuberculous cervical adenitis	20
Abdominal tuberculosis	7
Generalized tuberculosis	3
Bone and joint tuberculosis	1
Tuberculosis of tonsil	1
Alimentary tuberculosis	1

In another study of 220 children under five years of age, 59 showed the bovine type of the disease, the lesions being distributed as follows.

Tuberculous cervical adenitis	20
Abdominal tuberculosis	13
Generalized tuberculosis	5
Generalized tuberculosis (of alimentary origin)	10
Generalized tuberculosis (including meningitis)	2
Generalized tuberculosis (including meningitis of alimentary origin)	8
Tuberculous meningitis	1

PERCENTAGE OF BOVINE INFECTIONS.

	Five to sixteen years.	Under five years.
Pulmonary tuberculosis	0	0
Adenitis cervical tuberculosis	37	57
Abdominal tuberculosis	50	68
Generalized tuberculosis	40	26
Meningitis tuberculosis	0	0
Bone and joint tuberculosis	3	0

Parke and Krumweide state that the bovine type of tubercle bacillus is a menace to the life of the young child, and causes from 6 to 10 per cent. of the total fatalities from this disease. The bovine infection is largely limited to children, but is fatal only in infants and very young children, in whom, in a large percentage of cases, it produces cervical adenitis and the rarer forms of alimentary tuberculosis. The bovine type, as a rule, causes a milder form of tuberculosis than the human type.

Distribution.—The bacilli are widely distributed, and are readily disseminated by an individual whose secretions contain the organisms. They are present in the body in all tubercular lesions save in the chronic types of the disease which involve the lymphatic glands and the joints. In some instances they are present in the blood, and in this manner gain access to various parts of the body. Outside of the body they are distributed chiefly by the sputum which has become dry and assumed the form of dust, whereby it is readily disseminated from place to place. In the tuberculous wards of hospitals, organism have been grown from the dust collected, and when inoculated into animals have been capable of producing tuberculosis. In the open air they are rapidly destroyed, either by direct sunlight or diffused daylight.

The bacillus of bovine tuberculosis is found in the secretions and excretions of animals suffering from the disease, in and about their stalls, and often in their blood and flesh. The milk of such cattle frequently contains the bacilli, whether or not they are suffering with tuberculosis of the udder.

Modes of Infection.—*Hereditary Transmission.*—The organism may be transmitted to the offspring either by the spermatozoa, by the ovum, or by the blood through the placenta. Transmission by the sperm has not been definitely substantiated; while possible, it seems improbable, even though the bacilli have been found in the semen. Transmission by the ovum is unquestionably possible, animal experimentation having confirmed it. Transmission by blood through the placenta is, no doubt, the most common method, whether the placenta itself be the seat of the disease or not. Transmission by either one of the above paths would account for the occurrence of congenital tuberculosis.

On the other hand, the organism may be conveyed to the offspring by the parents, and remain latent until such time as the child's powers of resistance have become so low as to permit the disease to develop rapidly. Nevertheless, it is evident that in both modes of transmission the organism in some way gains access to the body.

The chief factors in favor of hereditary tuberculosis are the following: (1) the comparative frequency with which placental tuberculosis has been found; (2) various experiments which tend to show how readily the offspring may become infected. The fact that the parents are tuberculous, or the mother tuberculous, does not necessarily mean that the offspring will be likewise, for children born of tuberculous parents frequently show no evidence of the disease. On the other hand, just as many manifest signs of the disease early.

Other predisposing factors are pathological changes which may have occurred within those tissues which are the most frequent site of the disease, such as repeated attacks of bronchopneumonia, pleurisy, bronchitis, chronic inflammatory conditions which affect the mucous membranes of the respiratory tract, and diseased tonsils and adenoids. In numerous instances, tuberculosis follows such

contagious diseases as measles, whooping-cough, and influenza, though, unquestionably, in such cases the tuberculous infection has been latent.

The organisms may gain access to the body either by the respiratory or the alimentary tract, and children living in homes where members of the family are suffering with the disease are apt to contract it by inhalation; particularly is this so if they are of the age when they can creep about the floor, and thus inhale whatever particles of dust may be on the surface of the floor.

The most common cause of tuberculosis in children is contact with persons who have pulmonary tuberculosis, which in not a few cases is transmitted by kissing. Next to its transmission by the sputum, tuberculosis is perhaps most often spread through contaminated milk. The clothing, handkerchiefs, carpets, hangings, and bedclothing of a patient with tuberculosis may all, for a short time, be carriers of the disease.

When taken into the alimentary tract the bacillus may cause a primary tubercular lesion of the intestine, resulting in involvement of the mesenteric lymph nodes; or it may penetrate the wall of the intestine, and gain access to other parts of the body, causing a generalized tubercular infection. Again, it has been proven that the organism may pass directly through the mucous membrane of the respiratory tract without inciting any tubercular lesion of the membrane itself. The same is true of the intestinal mucosa.

On the other hand, its passage through such membranes is greatly facilitated if, at the time, the membranes are the seat of inflammation, whether it be acute or chronic. The bacilli, having lodged on the surface of the mucous membrane, are taken up and conveyed by the lymphatics to the nearest lymph nodes, where they are either arrested or incite some tubercular change within the gland itself; or they may pass on through the lymphatics to involve other lymphatic structures. The glands so involved become markedly inflamed and swollen, and a varying degree of cell proliferation occurs, which eventually results in caseation. The bacilli may become encapsulated, and remain latent for a varying period of time. Later, through the weakening of the individual resistance, or by symbiosis with another germ, they may resume their activity and produce the disease. It may be seen, therefore, that the resistance, or *degree of bodily protective power*, is an important factor in arresting or promoting the process.

In younger children the bronchial lymph nodes are most frequently involved because of ready access to them by way of the bronchi. When affected, they readily undergo caseation and softening, and may rupture into one of the smaller bronchi or a bloodvessel, thus gaining access to the lungs. On the other hand, autopsies have frequently shown the lungs to be affected with little or no involvement of the bronchial lymph nodes. Consequently, it is not necessary that they should first be involved in order that the disease may extend to the lungs.

The tubercle, which is the pathological lesion produced by the organism, is an inflammatory circumscribed growth, varying in size from that of a pinhead, or less, to that of a pea or larger. It is grayish-yellow in color, and microscopically is seen to consist of an abundance of epithelial cells, in the centre of which are giant cells. The organisms themselves may be either intracellular or extracellular. The caseation and softening are the direct result of the activity of the tubercle bacillus. After the formation of a tubercle, the disease may spread by contiguity, in that other tubercles formed nearby may fuse and form a single tubercle.

Again, the infection may be conveyed by the secretions and excretions of the body, such as the lymphatic stream, which may convey infection from one lymph node to another, or, by the urine carrying the infection from the kidneys, the primary seat of the disease, to the bladder. The organisms may gain access to the blood, either directly from the lymph or by rupture of a tubercle into a bloodvessel, or by way of the thoracic duct into the venous circulation, thence into the arterial circulation. Consequently, in either way the disease may be generally distributed throughout the body. In children the disease is met with in increasing numbers as they advance in years.

The frequency with which tuberculosis occurs, as shown by autopsies, is given by Holt in a table as follows:

Institution.	Age of patient.	Number of autopsies.	Number showing tuberculosis.	Percentage of cases of tuberculosis.
New York Infant Asylum	Nearly all up to 2½ years	726	56	8.0
Babies' Hospital	Nearly all under 3 years	1000	168	16.8
New York Foundling Hospital	Nearly all under 3 years	1000	136	13.6
Müller (Munich)	Children of all ages	500	200	40.0
Hamburger (Vienna)	All ages up to 14 years	848	335	40.0
Hamburger (Vienna)	Including only children of 2 years and less	497	120	24.4

Lesions.—The lungs are the organs by far most frequently affected; next are the bronchial lymph nodes, the spleen, the liver, the pleuræ, the kidneys, the brain, the intestines, and the mesenteric lymph nodes.

The bronchial lymph nodes and the lungs are the most frequent seats of the disease in early infancy, and death as a result of the pulmonary process occurs most often during the first two years. In many of the remaining cases death is the result of involvement of the brain. At this age other forms of tuberculosis are rarely the cause of death. This shows that tubercular meningitis is most frequently met with in early childhood in conjunction with pulmonary tuber-

46

culosis. After the third year it more often occurs unaccompanied by pulmonary lesions, and is usually secondary to either lymphatic or bone tuberculosis. At this time of life, too, the intestines and peritoneum are more often affected than in earlier childhood.

In children under two years of age pulmonary tuberculosis is more apt to be diffused throughout the lungs, and it is not until the sixth or seventh year that the pulmonary lesions of childhood resemble those in adult life.

Pulmonary Forms.—The younger the child affected, the more diffuse is the process in the lungs apt to be. In children who have passed the seventh or eighth year, the pathological changes are more likely to resemble those found in adults.

Tuberculous Bronchopneumonia.—This is by far the most common form of tuberculosis met with in young children. Its course is usually subacute. The lesions consist of caseous areas which, in some instances, have undergone softening. Areas of consolidation are also present. As a rule both lungs are involved, more frequently the upper lobes than the lower. The lung, when sectioned, shows the presence of minute nodules of a grayish-yellow color, varying in size, and often containing pus, while others which have not undergone softening show microscopically giant cells and tubercle bacilli. The nodules are usually surrounded by an area of bronchopneumonia. Frequently a cavity is present, varying in size from a walnut to a hen's egg. A tendency to encapsulation of the tubercular foci is rare, for the softening usually continues until death.

Miliary Tuberculosis.—Miliary tubercles are present in the lungs in practically all cases of pulmonary tuberculosis. They appear as minute yellowish-gray tubercles upon the surface, in most instances in the neighborhood of an old tuberculous lesion. Except for the presence of these minute tubercles, the lung frequently appears to be normal, while in other cases the areas between the tubercles are congested and the seat of bronchopneumonia.

These tubercles are usually found within the walls of the smaller bronchi, or in the adventitia or intima of the bloodvessels. As a result of the presence of tubercle bacilli in these areas, the organisms are usually widely scattered throughout the lungs. The bacilli lodge also in various organs of the body, and produce a generalized tuberculosis, having gained access to the blood either by perforation of the lumen of a bloodvessel, or ulceration of a caseous mass which may be extra-vascular, or ulceration of a lesion within the bloodvessel wall. Where there is lung involvement the pleura usually shows the presence of tubercles on its surface.

Acute Miliary Tuberculosis.—This form of tuberculosis closely resembles an acute infectious disease. It may occur at any age, is seldom, if ever, primary, and usually follows some focus elsewhere in the body, most frequently lesions of the bronchial lymph nodes. It may be either active or latent in character, and readily develops after the entrance of the organisms into the blood stream, which may occur as the result of rupture or ulceration of the diseased lymph

node into a bloodvessel. Tubercles make their appearance throughout the body a few days after the organisms have gained access to the various organs by way of the blood stream. The serous surfaces, as well as the lungs, spleen, and other organs become affected. This form of the disease occurs most frequently between the ages of twelve and twenty.

Clinical Varieties.—Three clinical forms of acute tuberculosis are usually described:

1. The general or typhoid form, which gives rise to symptoms closely resembling an acute general infection.

2. The pulmonary form, in which the symptoms are chiefly pulmonary.

3. The meningeal form, in which the symptoms are cerebral and spinal.

1. General or Typhoid Form.—The symptoms in this form are similar in many ways to those of typhoid fever, for which it is frequently mistaken. The onset is usually gradual, with progressive loss in weight, weakness, and the appearance of fever which gradually increases in intensity, also an increase in the pulse rate. The lung symptoms may be extremely mild. As a rule there is a moderate bronchitis. The absence of nosebleed and of diarrhea, as well as irregularity of the temperature and a tendency to cyanosis, should aid in distinguishing it from typhoid fever. Excessive sweating, more excessive than in typhoid fever, is characteristic. The spleen is frequently enlarged, and there have been instances of hemorrhage from the bowel. In this form of tuberculosis, involvement of the choroid coat is frequent.

A negative Widal test, together with a negative blood culture, will also assist one in making a differential diagnosis, while leukopenia or negative leukocytosis is of no avail in that it is frequently observed in uncomplicated miliary tuberculosis.

Prognosis.—This is, as a rule, unfavorable, the disease terminating fatally sooner or later. The duration is usually from one to three months, but it may sometimes be but a week or ten days.

Treatment.—Treatment is usually symptomatic. The fever is abated by the use of such drugs as spirits of nitre or antipyrine. Cough sedatives are frequently necessary in view of the fact that persistent cough is sometimes one of the most annoying symptoms. Needless to say, food of the most nourishing character and an abundance of fresh air are essential in the treatment.

2. Pulmonary Form.—In this form the lung symptoms are usually severe from the beginning. In children it may follow the infectious diseases, such as measles or whooping-cough, and assumes the bronchopneumonic type. In older children there is copious mucopurulent expectoration, and there may be hemoptysis. The cough is more or less constant, the face usually flushed, and there may be a varying degree of cyanosis about the lips and finger-nails.

Owing to the areas of bronchopneumonia seen in children, the

physical signs differ somewhat from those found in adults. There may be areas of hyperresonance or *vice versa*. Upon auscultation at the base of the lungs, the breathing may be of high-pitched, tubular character. Râles are heard, either fine and crepitant, or sibilant and sonorous. The temperature in this pulmonary form of the disease usually rises to 103° or 104° F., the pulse becomes rapid and weak, and death may occur within a short period of time, or the disease may persist for a year or longer. In some cases the tubercle bacillus may be found in the sputum.

Rapid emaciation, together with persistent cough and fever, ought to arouse suspicion of tubercular disease, especially after one of the minor contagious diseases. Hemorrhage from the lungs is not uncommon, and sometimes may be the only suspicious symptom. In the bronchopneumonic type a differentiation from either bronchopneumonia or simple bronchitis is often difficult. In the true pneumonic form, which rarely affects children, an early diagnosis is practically impossible because of the close resemblance to true pneumonia in its earlier stages. Nevertheless, in pneumonia an abatement of fever is expected by the tenth or twelfth day and, should it not occur, one is led to think of the possibility of pneumonic phthisis.

Prognosis.—This, unfortunately, is unfavorable, the disease lasting usually only a few weeks or months.

Treatment.—In the acute stage, the treatment is chiefly symptomatic. The subacute and chronic stages are treated the same as chronic tuberculosis.

3. MENINGEAL FORM.—This is more common in children than in adults, occurring most frequently between the ages of two and five, and is usually secondary to some tuberculous lesion elsewhere, chiefly in the bronchial lymphatic glands. Rarely is it primary in the meninges, but the meninges about the base of the brain are frequently involved. Usually certain prodromal symptoms appear, such as progressive loss of weight, irritability, and loss of appetite. Symptoms referable to the meninges suddenly make their appearance; there are headache, vomiting, fever, and occasionally convulsions. The vomiting is characteristic in that it occurs at times other than when food is taken.

The fever rises to 103° or 104° F., the pulse, at first rapid, later becomes slow and irregular. Twitching of the muscles is fairly constant, and the pupils are contracted during the early stage of irritation. At this period the symptoms resulting from irritation usually subside, but there is marked drowsiness, and at times retraction of the head. The pupils become dilated, and the so-called tâche cérébrale appears. Finally the stage of paralysis sets in, the child becomes markedly comatose, and spasmodic contractions of groups of muscles may occur. The pulse becomes rapid and feeble, and there are symptoms of the so-called typhoid state—namely, delirium, dry coated tongue, and incontinence of urine and feces. The duration of the disease varies from two days to a month or more.

Diagnosis.—The diagnosis is usually not difficult, especially if a previously existing focus can be found elsewhere in the body, together with the characteristic train of symptoms A lumbar puncture may reveal the presence of the bacilli and an abundance of small mononuclear lymphocytes, also an increase in the cerebrospinal pressure, which may become as high as 48 to 52 mm. Hg. Of course the presence of the tubercle bacillus in the cerebrospinal fluid makes the diagnosis positive.

Meningeal tuberculosis is chiefly to be distinguished from meningitis due to causes other than the tubercle bacillus. The presence of a tuberculous lesion elsewhere in the body is of great aid. Meningitis resulting from syphilis is usually chronic in form, and rarely acute, and is apt to be localized to one side. The presence of tubercles in the choroid is diagnostic.

Prognosis.—This form of the disease is practically always fatal. In instances where cures have been reported, there has usually been an error in diagnosis. However, recovery has apparently occurred in a few cases.

Treatment.—Though the treatment is limited, every effort should be made to combat the symptoms, inasmuch as the disease frequently proves to be other than tubercular, and a cure may be effected. The headache, which is so intense, is often relieved by lumbar puncture, and narcotics should be used freely during the stage of irritation. Operations upon the skull have so far been of no avail.

Chronic Tuberculosis.—This form of the disease is usually met with after the fourth year. It is contracted either by inhalation or by way of the alimentary tract, or is secondary to a previously existing tuberculosis, chiefly tuberculosis of the bronchial lymph nodes.

Symptoms.—The onset is slow, and is accompanied by loss in weight and a cough which becomes more or less persistent, together with a rise in temperature which often goes unnoticed. In the early stage of the disease the pulmonary symptoms are either vague in character or absent altogether. Frequently the organisms may be found in the sputum prior to any lung manifestation. By auscultation râles may often be heard over various parts of the lungs. This is chiefly so in younger children, because the disease in them does not necessarily begin at the apices of the lungs, as it so frequently does in adults. Later definite physical signs render a diagnosis possible.

The chest becomes flattened, there are areas of dulness on percussion, and rales, frequently scattered, are heard over the chest. As the process continues cavities form, evidences of which are the character of the breathing, characteristic metallic rales, and a tympanitic note on percussion over the broken-down areas. At this time the loss in weight is marked, and the fever becomes irregular. Such a child is extremely susceptible to intercurrent infections.

The diagnosis of the disease in its early stages is extremely important, for upon this alone, in the majority of instances, depends the question whether or not a cure can be effected. In all cases in

which there is constant cough with loss in weight and fever of irregular type, suspicion should be aroused, and the chest frequently examined for signs of the disease, since on these depends the prognosis.

General Tuberculosis.—In many cases of tuberculosis in infants the early signs and symptoms are those of marasmus alone, and tuberculosis is not even suspected until toward the end of the illness. Not a few cases of tuberculosis in infants are first recognized at autopsy, because of the lack during life of any symptoms or physical signs referable to the disease.

As a rule these infants steadily lose weight and strength, and exhibit anemia for which there is no apparent cause. Cough and fever may be absent at first, although toward the end of the disease there is, as a rule, a daily temperature range of 100° to 102° F., the fever being either constant or intermittent.

Disturbances of the gastro-intestinal tract are the most prominent features of the disease, and there is usually indigestion, malassimilation, vomiting, and diarrhea, although these symptoms are rarely due to involvement of the stomach or intestines. Late in the disease
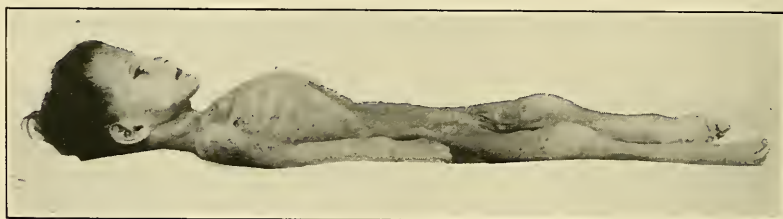
Fig. 71.—General tuberculosis in a child aged two and a half years.

a cough develops, the respirations are abnormally rapid, and the lungs show evidences of bronchitis which is followed by bronchopneumonia.

The terminal symptoms are dyspnea, cyanosis, marked asthenia, emaciation, and prostration, death resulting either from the pulmonary lesion, menigeal involvement, or general exhaustion.

Tuberculosis of the Glands.—Lymphatic Glands, Bronchial.—The glands most frequently affected in early infancy are the bronchial lymph nodes, comprising those about the trachea and its bifurcation, and those which accompany the bronchi into the lungs. Usually they are all affected at the same time. The changes which occur within the glands are identical with the changes elsewhere resulting from the presence of the organism. At times they may undergo suppuration, but not as frequently as do the cervical lymphatics when involved.

Suppuration of the bronchial glands is most common during infancy, but calcification of these glands rarely occurs at this age. When suppuration takes place, the escape of pus may cause a mediastinal or retropharyngeal abscess.

Other sequelæ of suppurative tuberculous bronchial lymphadenitis are compression and ulceration of the trachea and esophagus, acute miliary tuberculosis, and hemorrhage from the erosion of a large blood-vessel. When the glands which have undergone caseation become encapsulated, few of these dire mishaps occur, and the infection may be locked up in the glands for years, producing no symptoms, and doing no apparent harm.

Cervical Glands.—In 90 per cent. of all cases of tuberculous lymphadenitis the cervical glands are involved, and less frequently the other superficial lymph nodes of the body. Caseation of the external lymphatics in the neck, groin, axilla, and other regions of the body is usually followed by ulceration, and the discharge of pus externally.

Mesenteric Glands.—The same pathological changes occur in these glands as in other lymphatic glands. Softening may occur and, from leakage or rupture, produce a localized peritonitis. Enlargement of this group of glands may give rise to symptoms which are the result of pressure upon the vena cava, the portal vein, or thoracic duct. About 60 per cent. of all tuberculous cases in children show invasion of the mesenteric glands. They are rarely involved independently of the bronchial lymph nodes, and only occasionally is the condition sufficiently developed to be recognized independently of tuberculous peritonitis.

Symptoms.—The symptoms of abdominal tuberculosis are distention and pain in the abdomen, intestinal indigestion, flatulence, diarrhea, persistent elevation of temperature, and slow but progressive emaciation. Upon abdominal examination the glands may sometimes be palpated, if relaxation of the abdominal walls can be secured. Masses may be felt in both iliac fossæ, and quite a prominent group palpated in the region of the appendix, which forms a tumor.

Inasmuch as the symptoms somewhat resemble those of chronic appendicitis, a rectal examination may be necessary to determine whether or not there is enlargement of the appendix. The outlook in the cases which are sufficiently developed to be diagnosed is, as a rule, unfavorable. Recovery may take place, however, with surprising frequency, so that hope should never be abandoned. Still recommends laparotomy, with removal of the infected glands and the breaking up of adhesions, in addition to the routine treatment for all forms of tuberculosis.

Pleura.—Rarely does the pleura escape in tuberculosis of the lungs. In the generalized form of the disease it may be the seat of numerous miliary tubercles. Thickening of the pleura, together with adhesions over a portion of the lung involved, is quite frequent. Serous effusions which are often sacculated are not uncommon in infants and young children; empyema may occur, but is rare. The fibrous adhesions which form in tuberculous pleurisy may cause marked interference with pulmonary expansion.

Heart.—The pericardium is rarely affected save in acute general miliary tuberculosis, when the visceral surface may be dotted with a

few scattered tubercles. Tuberculous lesions of the endocardium and myocardium are practically unknown.

Brain.—In very early infancy and prior to the third year the brain is not uncommonly affected, but after this it is less frequently attacked. Minute miliary tubercles or caseous nodules may be present. About 70 per cent. of the cases of tuberculous meningitis are seen between the ages of one and five years.

Liver.—This organ usually becomes affected in general tuberculosis, numerous miliary tubercles forming on its surface and throughout its interior.

Spleen.—When involved, practically the same changes are found here as within the liver—namely, miliary tubercles on its surface and throughout its interior. It is rarely enlarged save in instances where nodules have formed.

Intestines.—In infancy involvement of the intestines is less common than in later childhood, but usually the small intestine is the seat of the disease. Minute nodules are deposited on the surface of the bowel. Ulcers are frequently found, especially in the neighborhood of Peyer's patches, usually extending only into the mucosa, but, if of long duration, they may involve other coats of the bowel. As a result of this ulceration perforation or cicatrization may take place and cause a narrowing of the lumen of the intestine.

Peritoneum.—Rarely is the peritoneum affected in early childhood, but in general tuberculosis of older children it may be the seat of miliary tubercles or tuberculous nodules.

Fig. 72. — Tuberculous peritonitis, tuberculous spondylitis, and tuberculous glands of the neck in a boy, aged seven years.

Kidney.—Miliary tubercles or nodules may be found in the kidneys accompanying generalized tuberculosis, although rarely. Tuberculosis elsewhere in the genito-urinary tract is most uncommon, although several cases of involvement of the testicle have been reported.

Prognosis.—The prognosis in tuberculosis of children depends to a great extent upon the location of the disease. Pulmonary lesions are always serious, and well developed foci usually prove fatal. Glandular tuberculosis is in many instances followed by recovery. The environment, climate, care, and treatment of a child with tuberculous lesions anywhere in the body also influence the prognosis to a great extent.

Prophylaxis.—The prevention of tuberculosis is a most important, and at the same time a very difficult problem, especially among the poor. It can be accomplished, however, if tubercle bacilli from every source are destroyed and needless exposure is avoided. Sputum from tuberculous patients should be carefully collected and destroyed, since this is probably the most frequent source of tuberculous infection. The living quarters of tuberculous patients should be disinfected before children or any other persons are allowed to enter them. A tuberculous mother should neither nurse her child nor kiss it upon the mouth, and the less she fondles it the better. No child should be allowed to associate in any way with tuberculous persons, for it has been demonstrated that tuberculous spray is projected several feet when a consumptive coughs.

Systematic school inspection is a great aid in differentiating tuberculous children from healthy ones. If the parents are tuberculous the utmost precautions are necessary in the home to prevent infection. The milk supply is an important consideration, and, if not obtained from a herd that has been tested with tuberculin, the milk should be pasteurized before giving it to children.

Since delicate children and those with chronic catarrh of the upper respiratory tract are especially liable to tuberculosis, they should be carefully protected from exposure and should lead an outdoor life, preferably in the country, fresh air and sunshine being the two great preventives of this disease. These precautions are especially applicable to children whose parents are tuberculous.

Tuberculin Tests.—In view of recent claims of benefit to be attributed to the use of old tuberculin as an aid to diagnosis, it seems expedient to give it brief mention. The preparation itself is a fluid, brownish in color, consisting of the filtrate from a culture of tubercle bacilli in glycerin bouillon after the bacilli have been filtered off. In short, it consists chiefly of the products of the bacillus. When used, it is diluted with water and injected subcutaneously, with all necessary aseptic precautions. The amount to be used is determined by the condition of the patient, and where there are evidences of tuberculous involvement should be extremely small.

The reaction following its injection manifests itself generally by a slow rise in temperature, for six to eight hours after the injection, followed by a rapid rise during the next few hours, and then a gradual decline. During the rise in temperature there may be such symptoms as headache, vomiting, and the like. A rise in rectal temperature to $99\frac{1}{2}°$ F. is considered positive.

It also produces a reaction in the tissues affected by the disease, causing them to become congested, and to evince changes reactionary in type. Lastly there is a reaction about the site of the injection, chiefly a hyperemia, which makes its appearance six to eight hours after the injection, and may last from a few to several days. Tuberculin is used cutaneously, subcutaneously, and in the eye in order to detect early, latent, or doubtful cases of tuberculosis. The

cutaneous test is, perhaps, the one most extensively employed, and the eye test least of all. In children these methods of determining tuberculosis are especially valuable.

Cutaneous.—The cutaneous test, or von Pirquet reaction, is practically a vaccination with tuberculin. The forearm is the site chosen, and after carefully washing the skin with alcohol or ether a small superficial scarification is made, preferably with a special scarifier, and a drop of undiluted tuberculin is then applied. It has been our practise to make two such vaccinations, and then to scarify an area between them to which no tuberculin is applied, this untreated area being used as a control.

The arm should then be held by the parent, or nurse, until the scarified areas are quite dry, so that there will be no contamination. In some instances the arm may be dressed with a piece of sterile gauze.

In active cases the reaction is noticeable within twenty-four hours, and consists of a red areola around the points of inoculation. A small red papule also forms, but disappears in the course of a few days or a week, the size of the inflammatory areola being an indication of the degree of the reaction.

The untreated scarification should heal with no sign of inflammation. The inflammatory areolas usually begin to fade the second or third day, but marked reactions may be visible for as long as ten days. Infiltration of the skin, and even induration, is sometimes observed, and in rare cases vesiculation.

This test is very valuable in infants and children under two years of age, and a positive reaction may be regarded as significant of tuberculosis in every case. A negative reaction indicates the absence of any tuberculous focus. These findings are invariably supported by physical examination and laboratory and autopsy findings, but no test for tuberculosis is as conclusive as is the detection of the tubercle bacillus in the cerebrospinal fluid, sputum, or elsewhere.

The reaction fails to appear in the eruptive stage of measles, and is very unreliable in cachectic conditions, scarlet fever, diphtheria, and typhoid fever. A second test is advisable when the first test is negative.

Subcutaneous.—The subcutaneous test, or stick reaction of Hamburger, consists in the injection of $\frac{1}{100}$ to $\frac{1}{1000}$ of a milligram of tuberculin beneath the skin of the forearm. Two reactions are observed, one at the point where the needle penetrates the skin, and a larger area where the tuberculin is injected. The reaction, which comprises swelling, redness, and induration, may appear in 24 hours, and last for five or six days.

Moro Test.—The Moro test consists in applying 1 grain of an ointment made of equal parts of old tuberculin and anhydrous lanoline to the epigastric or submammary region. The finger used for this inunction should be protected by a rubber cot, and the salve spread only over an area two inches in diameter, which has previously

been carefully cleansed. A control may be made by rubbing plain lanoline upon an area adjacent to the inoculated skin.

The reaction consists in a papular or vesicular eruption which appears in twelve to forty-eight hours, and persists for several days. In severe cases it is followed by pigmentation.

Eye Reaction.—The ophthalmic reaction, or Calmette test, is performed by dropping in the eye 0.5 per cent. solution of precipitated tuberculin in sterile salt solution. Within six to twelve hours there is swelling, redness, and injection of the palpebral conjunctiva with a slight or copious mucofibrinous secretion. The instillation should be done at night so that the reaction may be observed the following day.

The inflammation in the eye subsides in from one to three days, but only healthy eyes should be injected, and any sign of tuberculosis about the eye is a special contra-indication. The child's hands must be kept away from the eyes as long as they are inflamed. In older children, and in out-patient practice, this test is not recommended; and, as injury to the eye has occasionally occurred, the ophthalmic test is now very little employed.

Numerous modifications have been advanced since the von Pirquet reaction came into use, such as the application of ointment containing tuberculin, this being known as the salve reaction. None of these, however, has proven as positive and conclusive in its reaction as the von Pirquet.

The following statistics are given by Holt to show the relation of age to tuberculin tests in the child:

Age.	Number of cases.	Per cent. of tuberculosis.	Number of tests.	Per cent. of reactions.
Under 3 months	105	4	147	0
3 to 6 "	73	18	64	5
6 to 12 "	140	23	67	16
2 years	179	40	88	24
3 to 4 "	175	60	127	37
5 to 6 "	67	56	101	53
7 to 10 "	65	63	182	57
11 to 14 "	44	70	100	68
Over 14 "	..	..	112	90
	848	40	988	41

Treatment.—Of chief importance in the treatment of the disease are the hygienic and dietetic measures. Such a child should be kept out of doors as much as possible. It is frequently necessary to try a change of climate, such as sea air in the very early stages, and higher altitudes later. Foods of the most nourishing type, highly abundant in fats, are to be recommended. Milk is the best staple of the diet, and cream is almost as beneficial as cod-liver oil, if digested and assimilated in sufficient quantities. All alcoholic stimulants should be avoided, unless absolutely necessary. An abundance of sleep and rest is important, and moderate exercise should be taken. The administration of drugs should be delayed until it becomes absolutely necessary, and then they should be used cautiously in order to prevent any irritation of the stomach.

Tuberculin is recommended by some clinicians. It should be carefully used, beginning with extremely small doses, such as $\frac{1}{500}$ of a mg., and these increased when there is no reaction.

Other symptoms which necessitate consideration are the night-sweats which so frequently cause extreme weakness. The use of diluted vinegar, applied to the surface of the body in the form of a sponge bath, is frequently followed by relief. The fever should be combated as is best possible. Antipyretics, such as antipyrin, are sometimes of value. Hemoptysis is best treated by rest in bed and the use of morphin, either hypodermically or by mouth, in doses of $\frac{1}{40}$ to $\frac{1}{20}$ of a grain.

Cod-liver oil, if tolerated by the stomach, is very valuable for the upbuilding of the child's general health, and may be given in $\frac{1}{2}$ to 1 dram doses, three or four times daily. It gives the best results when taken after meals. The syrup of the iodide of iron is especially beneficial in glandular tuberculosis, given in 5- to 20-drop doses after meals.

In tuberculous adentitis, iodine may be used locally in the form of a 5 to 10 per cent. ointment, with lanoline as a base. A 10 per cent. guaiacol ointment is also valuable in some cases, and in children the local use of this drug is followed by better results than its internal administration. Fowler's solution is a good tonic, given in 1- to 3-drop doses three times a day combined with the syrup of hypo-phosphites or elixir of glycerophosphates.

Cresote may be given in 1- to 2-minim doses, in pill, in an emulsion with cod-liver oil, or by inhalation. Inunctions of cod-liver and other nutritive oils are advisable when internal medication is contraindicated by intolerance of the gastro-intestinal tract.

MALARIA.

Malaria is an infectious disease, due to a specific organism known as the plasmodium malariæ, and characterized by fever either of intermittent or remittent type, or by poisoning of a chronic type which results in anemia and splenic enlargement.

Etiology.—The parasite was discovered, in 1880, by Laveran, and in 1897 the mode of transmission was demonstrated by Ross. The plasmodium is distinctly a blood parasite, which gains access to the blood stream through the sting of mosquitoes, particularly the female Anopheles.

Malaria is common in infants and young children in regions where the disease is prevalent, and may occur *in utero*. One attack confers no immunity from future attacks.

Distribution.—The disease is diminishing everywhere, being now chiefly seen in southern Russia and Italy. It is quite prevalent in India, also in Africa, where it is of extremely pernicious type. In the United States it is steadily disappearing, though it still prevails in some

sections of the South, and during the spring and autumn months is very prevalent in the tropics.

The Parasite.—The parasite gains access to the stomach of the mosquito through the blood stream and forms minute cysts, the contents of which become transformed into crescent-shaped germs which, after the eruption of the cysts, pass to the salivary glands of the mosquito, and are readily transmitted to the human being by the bite of the mosquito. These young parasites enter into the human blood corpuscles, and there undergo a cycle of development.

There are two groups of organisms: (1) the large parasite, consisting of two types—the tertian and quartan organisms; (2) the small parasite of tropical fevers, known as the estivo-autumnal parasite.

Large Parasites.—The tertian is by far the most common of the large parasites, and passes through its cycle within forty-eight hours, giving rise to paroxysms on alternate days, provided there be but a single infection. Such paroxysms occur during segmentation, and last from twelve to fourteen hours. Should there be a double infection, a paroxysm occurs daily (quotidian fever), and upon examination of the blood two distinct groups of organisms will be found. In rare cases there may be more than two infections.

The parasite is non-pigmented and egg-shaped. Sometimes a small swelling is perceptible along half of the ring, giving it the appearance of a signet ring. It possesses in marked degree the power of ameboid movement, and thus undergoes frequent changes of shape and position. Occasionally the parasite undergoes another form of development, with ameboid processes different from the rings. The small tertian rings enlarge within twenty-four hours, obtaining their pigment granules from the hemoglobin of the corpuscles, which become much enlarged and fade. Within another twelve hours the parasites are converted into disks, bluish in color which, prior to the paroxysm, divide into minute egg-shaped bodies which are the young parasites. During this latter cycle the corpuscle becomes progressively paler, and finally loses its outline completely. Each segment separates individually as a hyaline organism, and gains access to other corpuscles.

Other forms of the tertian parasite are the extracellular varieties, which consist of two types, the gametocytes and the degeneration forms. The latter are merely those types which have escaped from their cells and have died. The gametocytes correspond to the crescents of the estivo-autumnal form, and occur after there have been frequent attacks of fever, appearing in the blood as extremely large organisms. Often they are double the size of a red corpuscle, and contain pigment which is more or less diffusely scattered. They never show segmentative changes, but are extremely active, and intended for sexual development.

Quartan Parasites.—This form differs from the tertian variety in that it requires seventy-two hours for its cycle of development instead of forty-eight hours, thus causing the paroxysms to occur on the fourth day. Should two groups be present, there will be paroxysms on two

succeeding days, followed by a day of rest. If three groups are present, the fever will be quotidian. In the early stages the parasite cannot be differentiated from the tertian variety, but upon the appearance of the pigment it is found to be coarser in type, and is capable of less activity.

Coincident with the growth of the parasite the corpuscle shrinks and becomes small, and its margin becomes irregular in outline. The parasitic protoplasm has a waxy appearance, consequently is more refractive than the tertian varieties. At the end of twenty-four hours the blood cell becomes crenated, brassy in color, and the pigment loses its activity. At the end of sixty hours the outline of the cell is completely lost, the organism is motionless, and pigment collects about the periphery.

Later the pigment migrates toward the centre, eventually collecting there, and segmentation begins, eight to twelve young parasites being formed. Here also gametes and spheres may be seen, but differ from those of the tertian type by their smaller size.

Estivo-autumnal Parasites.—This form, met with in the tropics, is the most dangerous of the three types of plasmodia. Early in the infection the grouping is distinct, but they soon separate, and at different stages of development are later found in the internal organs. The course and duration of the cycle is not always the same, therefore the fever is more or less continuous, loses its intermittency, and sometimes closely resembles the fever of typhoid.

In the very early stages, the development of hyaline forms differs but little from that of the tertian and quartan types. They are very refractive, and can be readily seen. The pigment within the bodies is of fine type. The blood cell is markedly enlarged by the parasite, frequently becoming crenated, and is brassy in appearance during the very early stages of development, but gradually ceases to exist in the peripheral circulation, and remains chiefly within the spleen. At times, the plasmodium may be found within the peripheral blood stream, in all stages of its development. Segmentation occurs precisely the same as in the tertian variety. Crescents later make their appearance within the blood corpuscles, become spindle-shaped, and eventually turn into spheres.

Lesions.—The lesions in children affected with malaria are practically the same as those in the adult. Chief among them are destruction of the red corpuscles and enlargement of the spleen which, in the chronic type, becomes hyperplastic and pigmented. Other organs, such as the liver, kidneys, and brain, may show evidences of pigmentation, depending upon the duration of the infection.

Clinical Forms of Malaria.—*Intermittent Forms.*—Two varieties of this type are seen—namely, the tertian and the quartan, the latter being the more common. Both are characterized by a cold and hot stage. The former begins with headache, lassitude, and sometimes by nausea and vomiting, which are soon followed by a chill. This may be so severe as to cause chattering of the teeth, shaking of the body,

and a cold, bluish appearance of the skin. The pulse becomes rapid, and there may be an increased quantity of urine voided. At this time the skin surface may be subnormal in temperature, whereas the temperature by rectum or axilla is very much elevated.

This stage may last from a few minutes to a couple of hours, and then the hot stage sets in. The skin temperature becomes elevated, the face is flushed, the pulse full and bounding. This lasts from one to several hours, and is followed by the sweating stage, during which perspiration appears all over the skin surface, and results in a profuse sweat. Coincident with this the headache, nausea, and other symptoms disappear, and the patient, overcome by exhaustion, sinks into sleep.

Marked variations in the course of the disease may be observed in very young children, the different stages being indistinct, and the disease assuming a remittent character. In very young children cyanosis takes the place of chills; there is restlessness, yawning, nausea, twitching or convulsions, coldness of the extremities, and sometimes diarrhea. The hot stage is almost invariably accompanied by fever of a higher degree than is seen in adults, often reaching 105° to 106° F.

The child is restless also in this stage; the face is hot and flushed, the eyes are injected; there is severe pain in the head, back, and limbs, the pulse is full and rapid, and the urine scanty and high-colored. This stage usually lasts an hour or two, after which the pain and fever subside, and the child breaks into a profuse sweat.

The child six years old or more has paroxysms similar to those in the adult, but very often the symptoms in younger children are so masked that malaria is not suspected at first, and a diagnosis of progressive anemia may be made because of the pallor and enlarged spleen. In the tertian form of malaria, the child may appear perfectly well in the interval between paroxysms.

The sweating stage is often entirely wanting, or may be so slight as to be unnoticed. The disease may make its appearance as a remittent fever, and eventually becomes intermittent. The quotidian type is the form most frequently observed in children. Next in frequency is the tertian variety, whereas the quartan form is extremely rare. Examination of the spleen usually shows some degree of enlargement, and in chronic cases the liver may be enlarged.

The course of the disease varies greatly. Recovery may occur after a few paroxysms with little or no medication. When malaria persists for some length of time, anemia and hematogenous jaundice may result from blood destruction, or the disease may eventually become chronic.

Irregular Form.—In young children the disease is apt to manifest itself by merely vague symptoms, instead of the train of symptoms which usually mark the onset in adults. Nervous phenomena are common, such as severe frontal headache accompanied by vomiting and lassitude. Pain in any part of the body, together with some tenderness, may be the first sign of a paroxysm. Again, a paroxysm may be ushered in with vomiting, high fever, embarrassed respiration,

cyanosis, and prostration.   Frequently moist rales may be heard over the lungs, and cause the disease to be mistaken for pneumonia.

Chronic Forms.—In many instances these forms can be recognized only by finding the malarial parasites in the blood.   Anemia, with fever and enlargement of the spleen, is the most constant symptom. The anemia is usually quite pronounced, the spleen in most instances sufficiently enlarged to be readily palpated, while the fever is often so mild that it is entirely unnoticed.   Other symptoms of minor importance which may manifest themselves are headache, occasional vomiting, weakness, constipation, muscular pains, and mild bronchitis.

When a child has suffered from repeated attacks of malaria, or the disease becomes chronic, marked cachexia may develop.   The child becomes emaciated, there are gastro-intestinal disturbances with anorexia and diarrhea, the features are drawn and pinched.

Pernicious Malaria.—Pernicious malaria is rarely seen in temperate climates; it is characterized by extreme intensification of all the symptoms.

Diagnosis.—This can readily be made on finding the parasites in the blood, especially if the blood is examined at the time of a paroxysm, and quinin has not been administered.   Often the blood if taken from the fingers will be negative, consequently, whenever possible, it should be removed from the spleen.   In view of the fact that conditions are not always favorable for making a blood examination, either because of lack of experience or equipment, the therapeutic test should be resorted to, namely, the administration of quinin.

Suspicion should always be aroused if, in a given case, the symptoms are more or less periodic in nature and the spleen is enlarged.   In the chronic forms of the disease the profound anemia and cachexia, together with muscular pains, fever of low-grade type, and splenic enlargement ought to differentiate the disease from tuberculosis, peritonitis, meningitis, lymphangitis, also from empyema, endocarditis, typhoid fever, and influenza.   Because of the fever and recurrent chills which accompany pyelitis this disease is frequently confounded with malaria.   Certain affections accompanied by enlargement of the spleen, such as leukemia, rickets, and hereditary syphilis, must also be differentiated.   These diseases can usually be excluded by careful examination, and the diagnosis of malaria confirmed by the therapeutic test, administration of quinine, and by examination of the blood.

Prognosis.—As a rule, the prognosis is favorable if the child affected by the disease does not reside in a malarial district.   In a very young child, a severe attack of malaria may so lower its power of resistance as to make it particularly susceptible to infections of other types. As a rule mild cases eventuate in spontaneous recovery, and in the majority of cases of malaria children recover promptly if the disease is correctly diagnosed and treated.

Treatment.—*Prophylaxis.*—In view of our knowledge of the mode of transmission of the disease, not only can its prevention be accom-

plished, but the disease can as well be ultimately exterminated. All houses in malarial districts should be screened by mosquito nets, and all parts of the body exposed to the bite of the mosquito should be bathed with lotions, which contain such drugs as menthol, turpentine, or pennyroyal, which are obnoxious to the mosquito. Pools of water should be drained, and when this is impossible petroleum should be sprayed on the surface. Children who have previously suffered from the disease should undergo treatment each spring and autumn for at least four or five years following an initial attack.

Prophylaxis is most important, especially in malarious districts. The child's sleeping quarters should be effectively screened, and it should be protected from mosquitoes in every possible way.

The treatment of the disease is more or less symptomatic; in the cold stage stimulation, and in the hot stage the application of cold to the body in the form of sponging, or an ice-bag to the head. Laxatives should be freely given early in the attack.

Administration of Quinine.—Owing to the disagreeable taste of this drug it is necessary to administer it in such forms as are willingly taken, and will cause the least irritation to the stomach. The bisulphate of quinine seems to have preference over other forms in that it is less likely to upset the stomach. When given by mouth an aqueous solution suffices, a fluidram containing 1 or 2 grains of the drug being administered four times daily, the last dose three or four hours before the expected paroxysm. Older children may be given 3 to 5 grains in the same manner. The administration of larger doses of quinine, that is, 2 grains every two hours, is often of advantage, if given for eight hours preceding the paroxysm, and 1 to 2 grains three times a day in the intervals between attacks. Massive doses may be gradually decreased as the symptoms subside.

When quinine is given by rectum a larger dose is necessary, and it should be injected in a starchy solution of some kind, such as gruel. It may be administered hypodermically in the form of the bimuriate with urea, bisulphate or hydrobromate, and the hydrochlorosulphate. Because of its irritating properties it seldom if ever should be used in this manner, as it almost invariably causes a marked induration at the site of injection, and necrosis of the tissues may result. Especially is this apt to be the case when it is administered in the above manner to very young children or to those whose resistance is lowered.

In older children the disagreeable taste of quinine can to a certain extent be disguised by the use of such vehicles as syrup of sarsaparilla, syrup of orange, etc., or it may be given in chocolate-coated quinine lozenges. Euquinine is easier to administer to young children because practically tasteless. The dose is the same as that of quinine sulphate. In still older children quinine may be administered in the same way as to adults.

In order to obtain the maximum results, quinine should be so administered that a relatively large dose is taken a few hours prior to the time of the expected paroxysm. This, however, is not always possible if

47

the stomach shows intolerance of the drug. Children will often be found to take relatively large quantities of quinine, 8 to 10 grains of the sulphate, and 10 to 12 grains of the bisulphate daily, without any evidence of disturbance. Under intelligent treatment with quinine, the spleen will diminish in size, and the paroxysms disappear.

Children, as stated, usually bear quinine well, and cinchonism does not occur as readily as in adults. Even after the child has apparently recovered, it is a good prophylactic measure to give 10 to 15 grains of quinine sulphate one day out of each week until the anemia has disappeared and the spleen is no longer palpable. It should then be given periodically at longer intervals. In the chronic forms of malaria, Fowler's solution and ferric chloride, each 1 to 3 drops, should be given in combination with quinine, and the children, when possible, removed to a different climate.

SYPHILIS.

Syphilis is a communicable disease which results from infection by a specific microörganism known as the Spirochæta pallida of Schaudinn. The organism is found in the primary lesion and the inguinal lymphatic glands, also in the liver, spleen, and cutaneous lesions of congenital syphilis.

In the acquired form syphilis is characterized by the appearance of a chancre followed by general lymphatic involvement, by eruption on the skin and the mucous membranes, then by infiltration of the body tissues, the bones, and their covering, eventually by the formation of gummata, which appear chiefly in the connective tissue. Both the acquired and hereditary forms of the disease are seen in infancy and childhood.

Acquired Syphilis.—This form of the disease is occasionally seen in children, though less commonly than the hereditary form.

Modes of Infection.—Infection occurs in various ways, being chiefly transmitted by the mother, who acquires syphilis after the child's birth, and conveys it to the offspring either by nursing, kissing, or other form of contact. It is possible for a child to become infected at the time of its birth, but this is rare, and is due to syphilitic lesions upon the genitalia of the mother. Infection may also be conveyed by wet nurses or by lesions upon the nipple.

The statistics of Fournier, showing the source of infection in a series of 40 cases, attributed the infection to parents in 19, to nurses in 8, to servants in 4, to sexual contact in 4, to vaccination in 2, to other children in 2, and to the physician in 1.

Symptoms.—The disease follows the same course in children as in adults. In from three weeks to a month after inoculation, the primary lesion, or chancre, appears at the site of infection, which is usually about the mouth or some other part of the face, rarely the genitals. This is followed by the appearance of secondary symptoms, chief of which are eruptions upon the skin and mucous membranes, later by

tertiary symptoms which may appear at any time from three to twenty-five years after the infection.

Prognosis.—The prognosis in acquired syphilis is much more favorable than in the hereditary form, and the course of the disease is usually mild. Even in infancy, there is no marked impairment of the general health, and the mortality is less than 10 per cent. of that in hereditary syphilis.

Diagnosis.—In the acquired form the diagnosis is made by the appearance of a chancre, by a history of infection, and by secondary manifestations. In hereditary syphilis there is no primary lesion. The infant has chronic rhinitis, and pemphigus or bullæ on the palms and soles which are not observed in the acquired form. Hutchinson's teeth are strongly suggestive of congenital syphilis.

Hereditary Syphilis.—This form of the disease is transmitted either from the father or from the mother or from both parents. Transmission depends to some extent upon the stage of the disease in the parents at the time of conception, and is more apt to occur during the secondary stage. In cases where the parents have been infected prior to the birth of any of their children, the first-born child is more apt to be the victim of the disease than the children born later.

Descent from the father, also known as *seminal transmission*, is more common than infection from the mother. In such cases, and provided no lesions exist along the genital tract, the seminal fluid will not cause a chancre if inoculated into a normal being, yet it carries with it the infection when fertilizing the ovum. Sometimes, even under such circumstances, an apparently healthy child may be born. Or a father who has acquired syphilis, yet at the time conception takes place is free from all evidence of it, may beget a child with a most virulent form of the disease.

Descent from the mother may occur in three ways:

1. Infection prior to conception.
2. Infection at the time of conception.
3. Infection following conception.

When infection occurs at the time of conception, the case is one of paternal heredity, due to the syphilitic spermatazoa of the father. If the mother alone has syphilis, the disease may be transmitted, but the chances of transmission are much less than when the father alone is infected. If, at the time of conception, the mother is the subject of tertiary symptoms of the disease, there is great probability that the child will escape.

Maternal infection exerts a more harmful influence than infection from the father, because of the dyscrasia which already exists in the mother, and produces lesions of the placenta which interfere with the maternal blood supply, and which, if these lesions involve a large part of the placenta, will usually cause an abortion. If, however, the placenta is only in part involved, the fetus is retained a longer period *in utero*, and may even be born dead at full term, or alive with the evidences of syphilis more or less marked. Infection during gestation

can be transmitted to the fetus up to the eighth month, after which time the chances of escape are highly probable.

Descent from both parents, also known as mixed heredity, is fairly certain if both parents are suffering from syphilis at the time of conception, and the virulence of the disease is greater than in hereditary disease of either maternal or paternal origin alone.

A healthy mother may give birth to a syphilitic child, this child not being capable of infecting its mother either by nursing or otherwise, and the mother remaining apparently free from the disease; yet such a child is capable of infecting a wet nurse. This is known as Colle's law.

Furthermore, it is possible for a syphilitic mother to give birth to an apparently healthy child, the child in all probability having acquired a certain degree of immunity from the disease while *in utero*, or having merely a latent form of the disease (Profeta's immunity).

Lesions.—*Skin.*—Save for their severity the lesions of the skin are practically the same as those of acquired syphilis. Depending upon the time at which they appear, they show a certain degree of variation, being most intense at birth, and then usually either pustular or ulcerative. When they appear some weeks after birth, they are usually erythematous and papular in type.

Erythematous Syphilides.—These develop usually during the third week of fetal life as minute spots which are of a pale red color, sometimes confluent, and which disappear upon pressure. Usually they are seen on the face and about the genitalia, in this way differing from the roseola of acquired syphilis. This eruption is apt to be confused with simple erythema; but the gradual transformation into papules, which become scaly on the soles and palms, and yield readily to antisyphilitic treatment, is a diagnostic feature.

Papular Syphilides and Mucous Patches.—These are more apt to appear on the face, palms, soles, and buttocks, but may sometimes extend over the entire body surface. The papules have a tendency to group and coalesce, forming large flat papules. When they occur about the mouth they frequently develop into fissures, which bleed readily and upon healing leave a scar. When situated upon warm surfaces, they become denuded of their epithelial covering, and emit an offensive discharge. They are most frequently seen about the anus or mouth. Papular syphilides situated on the palms of the hands and soles of the feet are apt to show evidences of exfoliation. The fusion of a number of such papules on the surface of the skin has a tendency to make the skin shed in large strips.

Vesicular Syphilides.—This form is usually associated with the papular type, and appears as small blebs. Sometimes the contents of the vesicles become purulent, but this occurrence is rare, and usually accompanies only the more severe types of infection.

Pustular Syphilides.—This type follows the papular eruption. In some cases the pustules may be seen at birth, in others they may not

manifest themselves for a long time after the initial syphilid. The severity of the infection influences their character. They usually appear on the face, hands, soles, thighs, scalp, and buttocks. At times the eruption may assume the form of impetigo, acne, or ecthyma. As a rule, the pustular eruption produces permanent scars, and may result in extensive skin destruction should it be complicated by cellulitis and gangrene.

Bullous Syphilides.—This form, also known as pemphigus, generally makes its appearance on the soles of the feet and the toes, the palms of the hand and the fingers, and the limbs. The eruption consists of blebs which are filled with a clear, turbid, or bloody fluid, are sur-
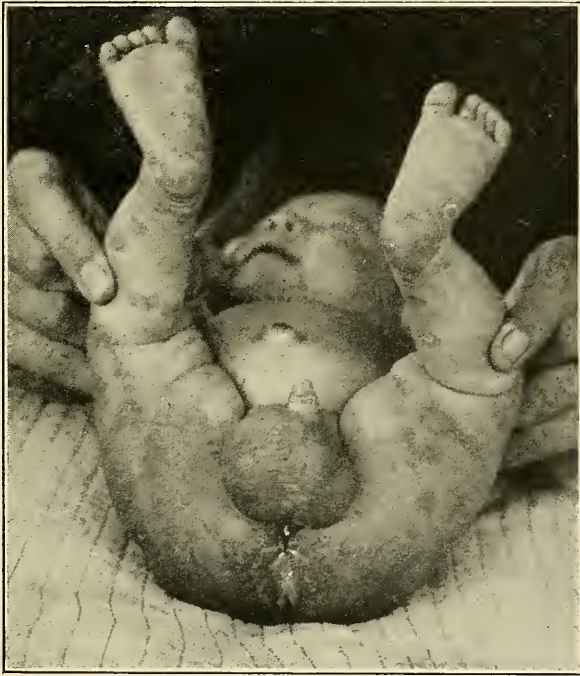
Fig. 73.—Inherited syphilis in a child two months old.

rounded by a slight areola on a base of reddish skin, and have a tendency to coalesce. The presence of such an eruption, together with lessened elasticity of the skin (because of a lack of subcutaneous fat), and a look of old age, should make one extremely suspicious of syphilis, particularly if the child is hoarse and has the snuffles.

Tubercular and Gummatous Syphilides.—These lesions may occur at any age, but more often between puberty and the thirtieth year. They may be either ulcerative or dry in type, and usually appear first upon the face and the anterior surface of the legs in the form of dull reddish infiltrations which are painless, gradually increase in size, eventually ulcerate, and become encrusted. When ulceration does not

occur, atrophic areas remain, whereas cicatrices which frequently produce marked deformities result from the ulcerative form.

Mucous Membranes.—Coincident with the papular and papulo-pustular eruption on the skin is the appearance of similar lesions upon the mucous membranes, which appear in the form of mucous patches in the mouth. The mucous membrane of the pharynx, the mouth, the larynx, the ear, and the nose are all subject to this eruption. One of the earliest and most significant evidences of the disease is coryza, which is manifested by a discharge, at first watery, which when dry forms crusts, underneath which are minute ulcers. This in turn clogs the air spaces, causing difficult respiration, and the characteristic snuffles. The hoarseness which is so typical is the result of inflammation or even ulceration of the mucous surface of the larynx. When there is marked infiltration, the air passages may be so narrow as to produce dyspnea and occasionally, though rarely, death.

Tertiary lesions of the disease manifest themselves usually at or about puberty, and are of the same nature as those seen in the adult. Infiltrations extend to various depths, and quickly break down, leaving marked degrees of ulceration. Such ulceration is apt to be found chiefly about the soft palate, the hard palate, the posterior pharyngeal wall, and the mucous membrane of the nose, resulting in marked deformity.

Bones.—Bone changes which are found in a syphilitic fetus, whether it be stillborn or dies after birth, show a marked degree of uniformity. The long bones are the ones chiefly affected, and the changes are found at the epiphyseal junction. In the early stages of the disease there is proliferation of the cartilage cells, followed by softening at the epiphyseal junction, and resulting detachment of the epiphyses from the shaft. As the disease progresses, certain degenerative changes occur, which, owing to infection by pus organisms, result in the formation of abscesses about the joint, or they may extend to the medullary cavity and result in osteomyelitis.

Liver.—Changes invariably occur in this organ. Usually there is a more or less diffuse interstitial hepatitis, which makes the organ distinctly larger.

Lungs.—Changes in these organs are more apt to result from hereditary than from acquired syphilis. They usually manifest themselves in the form of gummata or as a diffuse infiltration, the latter condition being known as white pneumonia. Microscopically the alveoli are found to be filled with fatty, degenerated epithelial cells.

Spleen.—The spleen is frequently the seat of a diffuse interstitial splenitis, and often increases to three or four times its normal size. When found during the first three months of life, it is of some diagnostic significance, especially if accompanied by other evidences of the disease.

Pancreas.—This also becomes the seat of a diffuse interstitial pancreatitis, but is usually not accompanied by sufficient symptoms to permit its detection.

Kidneys.—These, too, may undergo interstitial changes, and in a certain percentage of cases albuminuria is present.

Stomach and Intestines.—Ulceration and gummatous infiltration may attack these organs, but so rarely that such changes are of little or no importance.

Circulatory System.—Interstitial myocarditis and endarteritis are the usual lesions, and the latter, if severe, may result in thrombosis.

Lymphatic Glands.—Those most frequently affected are the anterior cervical group, as well as those groups which are subject to tubercular invasion, though the changes produced differ from tuberculous involvement in that the enlargement is slow and painless, and even though they attain a great size there is no suppuration.

Nervous System: Brain.—This organ, when involved, is usually the seat of multiple and diffuse lesions which produce symptoms of varying nature. Fortunately its involvement is not common in children, but hydrocephalus is sometimes seen in the newborn who suffer from syphilis, and gummatous infiltration may be present at birth. Some children manifest the disease by retarded development of the brain; in these cases speech is difficult, and there is a varying degree of weakness which causes a staggering gait. Meningitis, basilar in type, may manifest itself and become chronic. Involvement of the spinal cord results in paralysis of either the upper or lower extremities, according to the location of the lesion.

Eye.—Interstitial keratitis is one of the most constant lesions of hereditary syphilis, and usually makes its appearance between the seventh and fourteenth years.

Ear.—Otitis media may develop owing to extension of the inflammation from the Eustachian tube. It frequently produces deafness, and may involve the mastoid cells.

Teeth.—The first teeth exhibit no peculiarities of any importance, save that they soon decay. It is in the second or permanent teeth, chiefly the upper central incisors, that we find the characteristic evidences of syphilis. The so-called Hutchinson's teeth have a shallow crescentic notch in the cutting edge. All cases of hereditary syphilis do not, however, manifest these changes; in fact, the teeth may show varying degrees of deformity.

Early Syphilis.—The early diagnosis of this disease depends upon several factors, chief of which is accurate information given by the parents of the presence of syphilis in either parent, or a history of frequent miscarriages, abortions, or stillbirths, or a history of several children having been born apparently healthy, but who survived only a short time. Such knowledge is of great importance. A child may show little or no evidence of the disease, or there may be an eruption on the body of pustules and papules, or bullæ upon the soles and palms. The liver and spleen are usually greatly enlarged, and there may be evidences of interstitial pulmonitis or white pneumonia. Hydrocephalus also may be present.

In many cases the syphilitic fetus is dead at birth, and at postmortem the viscera, bones, and skin all exhibit signs of fully developed lues.

In other instances the infant is born alive, but is emaciated, and bullæ develop on the hands and feet. The bridge of the nose may be markedly depressed, and there is a persistent coryza, termed snuffles. Fissures and ulcerations (rhagades) appear about the lips and anus. The liver and spleen are enlarged.

Disturbances of nutrition soon arise despite careful feeding, and emaciation is progressive. The facial expression is that of a little old man. A diffuse papular and papulovesicular rash usually appears soon after birth, and the skin of the face may assume a copper color. These infants rarely survive.

In instances where the child at birth is to all appearances healthy, the symptoms of the disease usually do not manifest themselves until between the third and sixth weeks. Sometimes the first evidence is progressive emaciation, but usually coryza is the earliest symptom, and is frequently regarded as a simple cold. Coincidently there may be hoarseness of the voice, followed by the appearance of an eruption of papules and vesicopapules, some of which become pustular, and on bursting form crusts upon the surface of the skin. Mucous patches and rhagades also develop in these cases, but condylomata are not as common as in the early cases.

The skin on the soles of the feet, palms, knees, and nates is diffusely indurated, and the scaling may resemble eczema. Copper-colored areas may be seen on the thighs, and in some cases spread all over the body, giving the skin a mottled appearance. This is especially noticeable on the face, for these infants are usually pale and anemic, so that the copper-colored patches form a sharp contrast. The liver and spleen may or may not be enlarged. Pseudoparalyses may arise, and gastrointestinal disturbances are common.

The various joints of the body are tender as the result of acute epiphysitis. Syphilitic dactylitis may appear as early as the fourth week, and cause a fusiform swelling of one or more of the phalanges. It consists of gummatous infiltration of the periosteum and bone tissue; sometimes the overlying skin and soft tissues become involved. In neglected cases, necrosis of the epiphysis and destruction of the joint occurs, and may result in fistula formation. The loss in weight is steady, the child becomes marasmic, and may die.

The differential diagnosis between syphilitic and tuberculous dactylitis is very difficult; but there is usually more involvement of the soft tissues in the tuberculous form.

The Wassermann and von Pirquet reactions are valuable aids, and a therapeutic test may be made by placing the patient on antisyphilitic treatment. Craniotabes and bossing of the skull are bony changes which, if present, are always strongly suggestive of syphilis.

The spirocheta may be found in the blood, internal organs, skin, and the lesions in the mucous membrane; but it is difficult to demonstrate, and the Wassermann reaction of the blood is a much simpler method of determining the presence of lues. If, after the blood test, there is still doubt, the Wassermann or Noguchi reaction should be done upon the spinal fluid.

Late Hereditary Syphilis.—The disease may make its appearance at any time prior to the age of puberty. It may or may not be preceded by early symptoms of congenital lues. In some cases the evidences of syphilis are continuous with infantile lues, the child having always presented some stigmata of the disease, such as iritis or syphilides, but other children apparently enjoy good health with no eruptions or other symptoms until late in childhood, when one or more syphilitic manifestations appear. In all probability, however, these are cases in which the early symptoms were so mild as to escape attention.

The symptoms at this time correspond closely to those of the tertiary stage in the acquired form of the disease. There are evidences of arrested development, the complexion is of a peculiar leaden hue, the hair becomes brittle, and the nasal bone may be destroyed, thus producing a flatness of the bridge of the nose. The cornea may be hazy as a result of interstitial keratitis. The hard palate and nasal septum may be perforated, the teeth may show the characteristic crescentic notches, particularly the upper central incisors, and scars may form about the lips and nose. Enlargement around the epiphyseal junction, and deafness, either partial or complete, are characteristic evidences of the late form of the disease.

Dwarfism and infantilism are often caused by syphilis, and are due to lack of development of bone and muscle. At puberty there is but a scant growth of axillary and pubic hair, and the organs of generation are abnormally small. The face is characteristic, with its lustreless skin, prominent forehead, depressed bridge of the nose, and asymmetric skull. Hutchinson's teeth and interstitial keratitis often complete the picture.

The Hutchinson triad, *i. e.*, syphilitic deafness, interstitial keratitis, and notched teeth, comprise a symptom-complex which, whenever present, is conclusive evidence of syphilis. The bone lesions of late syphilis consist of osteoperiostitis of the long bones and cranium, also dactylitis.

Prognosis.—In every case this depends upon the extent of the lesions. In consequence of the changes due to the infection, such children are most susceptible to intercurrent affections. The age at which the disease makes its appearance, and the promptness with which treatment is instituted greatly influence the prognosis.

The breast-fed infant with syphilis has a far better chance for life than the bottle-fed baby, but infant-mortality due to syphilis is very high. More than one-third of syphilitic infants are born dead, and of those born alive more than one-third die before they are six months old. The longer the interval between birth and the first manifestation of the disease, the better the prognosis.

Treatment.—This should be instituted as early as possible, particularly in cases where there is a history of the disease in the parents, or of miscarriages, abortions, and stillbirths, or if a child exhibits any symptoms whatsoever of the disease. Mercury should be administered just as in the acquired form, and may be given either in inunctions, by mouth, or hypodermically; in children inunctions are preferable,

10 to 15 grains of mercurial ointment being used daily. The ointment should be rubbed in over a different area each day, in order to prevent erythema.

If given by mouth, either the bichloride or calomel may be given, of the former $\frac{1}{50}$ to $\frac{1}{60}$, and of the latter $\frac{1}{10}$ to $\frac{1}{20}$ of a grain, three or four times daily. When there are lesions of tertiary nature the iodides should be administered, either alone or in combination with mercury. In view of the fact that in children the iodides have little tendency to upset the stomach, relatively large doses may be administered with little or no derangement of digestion; usually 10 to 20 grains daily are sufficient.

The duration of the treatment should be regulated almost entirely by the symptoms. It is never advisable to continue it for long periods of time without an intermission. As there is usually some degree of anemia from the vigorous treatment, tonics in the form of iron, the saccharated carbonate, 1 or 2 grains, three times a day, should be administered. The syrupi ferri iodidi may be given in 10- to 20-drop doses. The child should have plenty of fresh air, and careful hygiene and hydrotherapeutic measures should be instituted to build up the general health.

The local treatment consists in the dusting of powders, such as bismuth and calomel, upon the ulcerated areas, and the cauterization with nitrate of silver of the ulcerations upon the mucous membranes. If rhinitis is severe, the nose should be irrigated daily with a 1 to 2000 solution of bichloride, and iodoform ointment may be applied to the inside of the nares. The mouth also should be washed daily with a 2 to 5 per cent. solution of potassium chlorate to prevent salivation while mercury is being given.

Salvarsan offers a speedy check to the active ravages of syphilis, and may be given to children in the dose of $\frac{1}{2}$ grain for every 60 kilos of body weight. This treatment should be carried out only by one thoroughly conversant with the technic, and the patient should be taken to a hospital. Mercurical treatment should follow the injection.

When epiphysitis is present the arm should be put in a splint with the elbow bent at a right angle, and the legs placed in straight splints if the lower extremities are involved. In these cases daily applications of unguentum hydrargyri should be used locally in addition to internal medication.

In both infants and older children, the nutrition should be carefully watched. An otherwise healthy syphilitic mother should nurse her child; if this is impossible, a syphilitic wet nurse may be obtained, or the child may be given a carefully modified milk formula. The diet of older children should also be regulated.

Where the treatment has been carried out continuously for a year, and there have been no active symptoms for six months, the disease may be regarded as well under control. Intermittent treatment, however, should be kept up for a year or two more before the case can be considered cured. During intermittent treatment the child should be given antisyphilitic treatment for six months of every year.

CHAPTER XXIII.

RHEUMATISM.

ACUTE ARTICULAR RHEUMATISM.

A DISCUSSION of acute articular, or acute inflammatory, rheumatism covers but a small part of the study of rheumatism, for in children of a rheumatic diathesis the manifestations of the disease may be numerous, varied, and peculiar. Arthritis, so typical of rheumatism in the adult, is often but an insignificant feature or may be wholly absent; but the skin frequently shows the effects of the toxins of rheumatism by the appearance of urticaria, erythema multiforme, erythema nodosum, or purpura.

The cardiac manifestations of this disease are by far the most serious and, unfortunately, are by no means uncommon. They include endocarditis, myocarditis, and, occasionally, pericarditis. The kidneys are less frequently affected, but nephritis is occasionally the sequel to an attack of rheumatism. Chorea is the most common neurosis associated with rheumatism, the relation between these two diseases being so close that chorea is now regarded as essentially of rheumatic origin. Tonsillitis of rheumatic origin is a well recognized clinical entity; and, inversely, many cases of articular rheumatism are now believed to be due to infection which has its source in the tonsils.

Torticollis may also be due to rheumatism, especially when its onset is sudden, and it is ushered in by a spasm of the cervical muscles caused by the irritation of the rheumatic toxins. Other muscles of the body may be affected in like manner; but rheumatism is rarely the diagnosis in these cases, the symptoms being usually referred to as "growing pains" when they occur in the legs, or as "a stitch in the side" when the intercostal muscles are affected.

Barlow and Warner, in 1881, described the rheumatic nodule, which is an oval, semitransparent, fibrous body looking like a boiled sago grain. These nodules most frequently form at the back of the elbow over the malleolus, and at the margin of the patella. Occasionally they may be found on the extensor tendons of the hands, fingers, and toes, or over the spinous processes of the vertebræ. They are composed of fibrin cells and fibrous tissue, and vary in size from a pin's head to a small pea, exceptionally attaining the size of an almond. They usually appear in crops, and may remain for months, but generally disappear within a few weeks.

Among the rarer manifestations of rheumatism in children may be mentioned mastitis, periostitis, and peritonitis. In many instances these diverse signs of a rheumatic diathesis are so slight and transi-

tory in nature that their rheumatic origin is overlooked; but, at one time or another, these children suffer from more or less typical attacks of acute, subacute, or chronic articular rheumatism and in these seizures clearly defined symptoms appear which reveal the true nature of previous vague symptoms of the disease.

Etiology.—Heredity is regarded as one of the most potent causes of rheumatism in children as well as in adults, and in such cases the family history often reveals rheumatic affections in older members of the family, as well as in the young brothers and sisters of the child.

Acute articular rheumatism may appear in typical attacks at any age from infancy to adult life; but, until the tenth year, it is usually atypical. Infants are rarely attacked; it is uncommon between the second and fifth years; but in older children it occurs more often, and is most frequently observed between the ages of ten and twenty. Girls are more prone to chorea than boys, but otherwise there is little difference in the incidence of the disease in the two sexes.

Rheumatism is most prevalent in the spring of the year, and is also precipitated by exposure to cold and wet. It is more common in the poorer classes, a fact which is probably explained by the damp dwellings, unsuitable and insufficient food, and unhygienic surroundings which are factors in its causation. One of the most unfortunate features of rheumatism is the influence one attack of the disease has in producing another; for, once a sufferer from rheumatism, there is always a strong tendency to future attacks.

Many cases follow attacks of tonsillitis, and occasionally rheumatism accompanies scarlet fever and influenza. Several theories have been advanced as to the actual cause of rheumatism; but in recent years the recognition of its infectious nature has steadily gained favor. Furthermore, the theory of its microbic origin has been strongly supported by the occurrence of epidemics, and by the discovery of a diplococcus by Triboulet, Wassermann, Pointen, and Payne. This organism is, in all probability, the specific germ, since it is capable of producing various manifestations of rheumatism when injected into the lower animals.

At one time a so-called nervous theory of rheumatism was in vogue, but this is now largely discredited. Its adherents contended that rheumatism was caused by a disturbance of the nerve centres produced by cold, and that a derangement of metabolism resulted from the primary disturbance of the nervous system. In consequence of poor metabolism, the nitrogenous bodies were concentrated into uric acid and deleterious substances instead of urea.

Other observers have claimed that rheumatism is due to defective assimilation, whereby are formed certain acids, among which lactic acid is held to be largely responsible in the production of the symptoms of this disease.

Pathology.—The pathological changes in acute articular rheumatism are slight and not at all characteristic. The synovial membrane of the affected joints is intensely hyperemic and inflamed, and there

is frequently an accompanying inflammation of other serous surfaces of the body. Within the joint is an effusion of turbid fluid which contains leukocytes and flakes of fibrin, and is usually sterile.

The periarticular tissues are also infiltrated with this exudate, causing a swelling about the joints, and there may be an associated inflammatory condition of the tendon sheaths. In protracted cases the cartilage may become eroded; but, as a rule, there are no permanent changes within the joint. Exceptionally the exudate in the joint cavity becomes purulent.

The characteristic changes in the heart are due to the effect of bacteria or the toxins of rheumatism on the endocardium. They consist in a hyperplasia of tissue which forms vegetations on the mitral valve and, as a rule, prevents perfect apposition of the valve leaflets and complete closure of the valve.

There are also pathologic changes in the myocardium, pericardium, pleura, and, rarely, in the peritoneum and meninges; but these practically amount to merely an inflammatory state, and are nowise pathognomonic.

The fibrin elements of the blood are increased, and fibrinous clots are found in the heart and great vessels. In addition, there is severe secondary anemia with a marked reduction in the number of red cells. Proliferative periostitis and subcutaneous fibrous nodular deposits are among the rarer pathological findings.

Symptoms.—The symptoms of acute articular rheumatism in the child differ greatly from its manifestations in the adult. The articular symptoms which form so prominent a feature in later life are much less distinctive, and fewer joints are involved. The pain and swelling in these joints are usually slight, and there is frequently no redness. In the ordinary case the fever is never very high, and is not of extended duration.

The onset is not acute, but is often preceded by a few days of malaise, with sore throat, anorexia, abdominal pain, and occasionally vomiting. The pains about the joints at this time are transitory and indefinite. After this there is commonly fever of 100° to 101° F., and the joint becomes swollen and tender; if the ankle, knee, or hip is involved, the child is unable to walk.

In mild cases there is merely stiffness in the affected joint, causing the child to limp if the leg is attacked. The knee- and ankle-joints are most often involved; next in frequency are the small bones of the feet, the elbows, and wrists. Muscular spasm is not uncommon. In some instances there is extreme tenderness about the muscles and tendon sheaths, the joint being but little affected; this gives rise to the so-called "growing pains" of children, which are often of much more serious nature than is generally supposed.

The urine in these cases is highly colored, concentrated and decreased in amount. The profuse acid sweats so characteristic of rheumatic fever in the adult are not as frequently observed in young children, the skin being more often hot and dry. While of mild type,

this atypical form of rheumatism in the child is apt to run a relatively longer course than in the adult, many of the cases being subacute, although a chronic affection is rare.

Owing to the predisposing influence of one attack of rheumatic fever upon another, recurrences are likely to happen from time to time throughout childhood, and even during adult life.

Rheumatic Symptoms in Older Children.—The atypical type of rheumatic fever just described is characteristic of most attacks observed in children up to the age of eight years, after which the symptoms of the disease conform more or less to the adult type. The attack comes on suddenly with acute inflammation of one or more of the larger joints. Pain is agonizing, and tenderness so exquisite that any attempt to palpate the joint causes the child to cry out in anticipation of the pain.

The skin over the joint is usually of a red and dusky hue, hot to the touch, and edematous. The temperature ranges from 102° to 104° F. on the first day, but there are marked variations in its course, and it finally declines to normal by lysis. Sweating is profuse, and its peculiar acid odor is characteristic. Sometimes the skin is hot and dry. The bowels are usually constipated, the tongue is dry and coated.

A distinguishing peculiarity of the disease is the order in which the joints are affected, the inflammation appearing in a fresh joint, or set of joints, as it subsides in the one originally affected, so that the disease is seemingly transferred from one articulation to another.

Cardiac symptoms, while not uncommon, occur less frequently in these cases than in subacute attacks, in which the joint symptoms are mild and relatively unimportant.

Extra-articular Symptoms.—In children, especially, involvement of the heart is of such frequent occurrence that it can be reasonably regarded as a sign or symptom of rheumatic fever. Either the endo-cardium, pericardium, or myocardium may be affected, and in severe cases all three are inflamed; but endocarditis is the usual finding. In many instances it follows an attack of rheumatism so mild that it is impossible to obtain a history of the illness from the parent.

There are no special symptoms which attend the onset of endo-carditis, and it is rarely fatal during its acute stage, but tends to develop subacute or chronic lesions which incapacitate or destroy life months or even years after the rheumatic attack. The mitral valve is almost invariably the site of the endocardial lesion; in most of the other cases the aortic leaflets show changes; occasionally both mitral and aortic valves are involved. This acute inflammation of the endocardium is usually of simple verrucose type, and is indicated by an apical blowing systolic murmur and by the reduplication of the cardiac second sound.

There may be, and frequently is, an increase in the height of the fever when endocarditis sets in, and also a certain amount of palpi-tation, dyspnea, and precordial pain. Even when there is no endo-

carditis, the pulse is increased to 120 or 130 per minute, and is soft and full; therefore in these cases, unless the pulse is decidedly irregular, tachycardia is of no diagnostic value in determining the presence of a heart lesion.

Pericarditis, while not so frequent as inflammation of the endocardium, is not extremely rare in older children, and may sometimes be seen in association with endocarditis. The inflammation is, as a rule, of a dry fibrinous type, the effusion consisting chiefly of organizable lymph, often in large amounts. This results in numerous adhesions and, on account of the tendency to recurrence which is characteristic of this form of pericarditis, the pericardial sac may eventually become almost obliterated.

The most important symptom is a to-and-fro friction sound, which is pathognomonic of pericarditis. A slight degree of myocarditis is often present with either endocarditis or pericarditis, but is more marked when there is endocarditis.

Skin Lesions.—These are seen more frequently in children than in adults, and usually manifest themselves by the development of purpura or erythema. The relation between the various forms of erythema,—simplex, urticaria, nodosum, multiforme, and marginatum, —is not as yet clearly understood. Erythema multiforme is the most common erythematous lesion caused by rheumatism; urticaria is not uncommon, but erythema nodosum is rare.

Owing to its frequent association with rheumatism, purpura in these cases receives the name of purpura rheumatica. But purpura, although not uncommon in rheumatic children, also accompanies other diseases; therefore it is probable that these extravasations under the skin are due to an accompanying infection rather than to rheumatism itself.

Sudamina and miliaria may appear as the result of overactivity of the sweat glands, and herpes also is occasionally observed in rheumatic children. Rheumatic subcutaneous nodules, which have already been described, while more common in children than in adults, are rarely seen in the eastern section of the United States; but when they appear in children, they may be regarded as a positive sign of rheumatism.

The Blood.—Examination of the blood in acute rheumatic fever usually reveals a rapidly developing and severe secondary anemia with a decided decrease in red cells and moderate leukocytosis. Blood cultures are usually negative, but diplococci have been isolated from the blood in severe attacks of rheumatic fever.

Muscular Symptoms.—The muscular symptoms of rheumatism consist chiefly of inflammation of the muscles and tendons about the affected joints, and there is usually considerable swelling and tenderness in these soft tissues. In protracted cases of rheumatism some degree of atrophy may follow.

Tonsillitis.—Sore throat, especially tonsillitis, often accompanies attacks of rheumatism in children, and sometimes precedes the articu-

lar symptoms. Some rheumatic children, however, who present no
articular symptoms, are also subject to attacks of tonsillitis. Follicular
inflammation of the tonsils is the common form in these cases, although
there may be a peritonsillar abscess.

The ordinary sore throat associated with rheumatism in children
usually lasts only three or four days, but its subsidence has no appre-
ciable effect upon the duration of the articular or cardiac symptoms,
or upon their severity.

The Respiratory Tract.—As a rule, the organs of respiration are
but slightly involved, but there may be a moderate degree of bron-
chitis, and occasionally pleurisy. Pneumonia is quite rare, and usually
assumes the lobular type. Inflammation of the pleura in these cases
is usually serous, but may be fibrinous. The chief symptom of pleurisy
is thoracic pain. Friction fremitus is rarely heard over a large area
of the chest wall. As a rule there is no decided rise of temperature
with the onset of pleurisy, and but little increase in the severity of the
constitutional symptoms. The left chest is most frequently affected.
In some instances the pleura becomes inflamed through extension
from the pericardium.

Although few cases of pneumonia can be clearly demonstrated
to be of rheumatic origin, yet in several instances a diplostreptococcus
has been isolated from the sputa in pneumonia, and also from the
pleural effusion.

Nervous Manifestations.—Chorea is the most common nervous
affection related to rheumatism, and may precede, accompany,
or follow a rheumatic attack. Sometimes it accompanies endocar-
ditis, and there are no articular symptoms. More than one-half the
children who are subject to chorea are of a rheumatic diathesis, while
those who suffer frequently from rheumatism are usually neurotic.
Headaches are common among these children, they are easily excited,
frequently suffer from night terrors, and display other evidences of
a nervous temperament.

These nervous phenomena are probably due to three causes—
nervous irritation and exhaustion, high fever, and profound toxemia.
In uncomplicated cases of rheumatic fever delirium is rare; but its
appearance may sometimes signify pericarditis, while in others it
may be due to an overdose of the salicylates. Convulsions are uncom-
mon, coma is rare, and meningitis is only exceptionally a complication
of rheumatism in children.

Diagnosis.—When typical acute articular rheumatism occurs in
children it is usually easy of recognition; but since cases are atypical,
especially in young children, and in some there is no appreciable
joint lesion, the disease is quite often unsuspected. In attempting
to arrive at a diagnosis of rheumatism, the family and personal
history of the child are important, as well as the articular manifes-
tations. Other signs, such as erythemata, tendinous nodules, growing
pains, chorea, sore throat, and pains in the epigastrium and chest,
must also be analyzed if the disease is to be recognized in its varied forms.

An examination of the heart in a suspicious case may often reveal the presence of endocarditis, which is a great aid to the diagnosis of rheumatism in those cases in which there is no joint involvement. The diagnostic points in a typical attack of inflammatory rheumatism in a child are as follows: a history of rheumatism in the family and of repeated attacks of sore throat; inflammation of one or more of the large joints with a tendency to shift from one joint to another; and signs of endocarditis.

In infancy rheumatic arthritis is so rare that many other conditions common at this time of life should be considered before such a diagnosis is made. In infants, scurvy is often mistaken for rheumatism, and the differentiation may be difficult until demonstrated by the characteristic state of the gums. There is, however, but little fever in scurvy; there is almost always pain and swelling in the lower extremities, the ends of the long bones being involved, as a rule, rather than the joints. Antiscorbutic treatment will demonstrate the presence of scurvy in a few days, and serves in these cases as a therapeutic test. Acute rheumatism must also be differentiated from scarlatinal arthritis; but in the latter disease there is a history of scarlet fever symptoms. Moreover, as the disease generally comes on in the second or third week, the child is usually desquamating. Scarlatinal arthritis shows a marked tendency to involve the wrists, while, as a rule, in acute rheumatism, the larger joints are attacked. True rheumatic arthritis may come on during an attack of scarlet fever, and here the diagnosis is extremely difficult.

Septic arthritis occurring in the course of pyemia, and the polyarthritis occasionally observed just after birth, may be confounded with rheumatic arthritis in its early stages; but, as a rule, suppuration accompanies these affections, and the constitutional symptoms of septic arthritis are much more severe than those of rheumatism. In gonorrheal arthritis there is usually a history of vulvovaginitis, the disease being most prevalent in girls; the knee-joints are the ones most frequently involved.

Acute osteomyelitis may produce symptoms much like those of rheumatism, and this affection should be excluded in every case, its early recognition being most important. The femur is the most common site of osteomyelitis, it usually attacks only one long bone, and involves only one joint. Both the local and constitutional symptoms are more severe than those of rheumatism.

Tuberculous arthritis may be difficult to exclude. It is strongly suggested when there is a slight attack of rheumatism in the hipjoint; but the course of tuberculous bone disease is essentially chronic, there is less induration about the joint, and the pain is greatest within the joint.

Pneumococcal arthritis is acute, but is the sequel to pneumonia, and diplococci may be found in the exudate within the joint. As a rule suppuration sets in, and simplifies the differentiation from rheumatism, since the rheumatic joint rarely, if ever, suppurates.

48

Epidemic cerebrospinal meningitis with extreme and unusual tenderness of the joints may suggest rheumatism; but, upon careful study, the reflexes will be found abolished, there is severe headache, and the cerebrospinal fluid shows acute meningeal inflammation.

Syphilitic arthritis is usually monarticular, but may occasionally be mistaken for rheumatism; there is, however, the usual history of syphilis in mother and father of the child; and, on careful examination, concomitant signs and symptoms may be found. If necessary, a Wassermann reaction will determine the existence of lues in these cases.

Intra-abdominal Complications.—These are rare, but peritonitis is occasionally seen together with inflammation of other serous cavities; sometimes these children suffer from a nervous diarrhea which brings on defecation just before or during a meal. Epigastric pain is a puzzling symptom more or less confined to children who suffer from rheumatism; it may either be due to a rheumatic state of the abdominal muscles or simply be caused by an associated gastric catarrh. As a rule, there is no tenderness over the epigastrium, and the pain usually disappears early in the rheumatic attack. There are no gross lesions of any of the abdominal viscera, although the spleen is often enlarged.

Epistaxis is not of rare occurrence in rheumatic attacks during childhood, and is rather an ominous sign because of its association with endocarditis. Iritis also appears in some cases, owing to the lodgment of rheumatic organisms in the fine capillaries of the iris.

Course and Prognosis.—In children the articular symptoms of rheumatism usually subside within a week or two; but exceptional cases may be protracted for several weeks or even months, while in some cases recurrences are so frequent that the children are practically never free from rheumatic manifestations.

Recovery from an acute attack depends to a certain extent upon the mode of treatment and the promptness with which it is instituted; but, if there are no complications, the outlook is favorable. Unfortunately, however, children are much more liable to grave complications, such as endocarditis and pericarditis, than adults; therefore, rheumatism in childhood is a serious disease. With each relapse fresh damage is done to the heart; and, while cardiac involvement in an acute attack rarely proves fatal, the damage to the heart eventually results in death. The appearance of rheumatic nodules is regarded as a particularly unfavorable sign in rheumatism, since they are usually associated with cardiac complications.

Treatment.—Rest in bed is the most important and essential measure in the treatment of acute rheumatism in children. By observing this precaution, much can be done to prevent the greatly dreaded cardiac complications of the disease. It is difficult to state definitely how long the child shall remain in bed, and also hard to keep a child in bed after the acute symptoms of rheumatism have subsided; but such grave consequences may follow if these children play about too

soon that, to be perfectly safe, they should be kept in bed for a month after an attack of acute rheumatic fever. The sick-room should be well aired, the temperature kept at about 70° F., and all draughts avoided. The child should wear a flannel night dress, and lie between blankets, to prevent any chilling of the surface of the body.

The diet should at first be liquid but nourishing, consisting for the most part of cereals, milk, and egg albumen; later bread, vegetables, and a little meat may be given, but it is wise to limit the amount of starches. An initial dose of calomel, 1 to 2 grains, should be administered, and may be followed by magnesium citrate, 2 to 6 ounces, or 1 dram of magnesium sulphate, after which the bowels should be kept regular by giving, when necessary, smaller doses of the citrate or sulphate of magnesium.

The joints may be wrapped in a layer of cotton wool and then loosely bandaged. In some cases the affected limb may be more effectually kept at rest by applying a light wooden or cardboard splint.

If pain is severe, it is sometimes necessary to use a bed cradle to keep the weight of the bedclothing off the limbs; in these cases, particularly, the local application of methyl salicylate, ichthyol, or of ice-cold or warm compresses soaked in a saturated solution of sodium bicarbonate will often give relief.

For high fever in rheumatism a tepid sponge may be given, and is less harmful than antipyretic drugs. The salicylates are specific in acute rheumatic fever, and should be given in full dosage, just as quinin is administered in malaria; for, thus taken, they may protect the cardiac valves and myocardium and pericardium from injury by shortening the attack of the disease. From 10 to 20 grains of sodium or strontium salicylate, according to the age of the child, should be given every three or four hours, and it has been demonstrated that if equally large doses of sodium bicarbonate are administered with the salicylates, the results will be better than if the salicylates are used alone. When given liberally, the bicarbonate of soda also minimizes to some extent the injurious effects of the salicylates upon the stomach, and aids in protecting the heart. If the pain is not relieved by the treatment outlined, acetanilid may be given in $\frac{1}{2}$- to 2-grain doses three times daily, or salol in 1- to 3-grain doses after meals.

In the more obstinate, or subacute forms, of rheumatism the salicylates are frequently of less value, and better results are sometimes obtained by the use of potassium iodide, 3 to 5 grains, three times a day, or the citrate or bicarbonate of potassium, 10 grains every four hours.

Lemon and lime juice in large quantities are very beneficial. These children should be encouraged to drink water freely, especially Seltzer and Vichy.

Morphine should be given only when pain is severe; but some of the latest preparations of the salicylates may be used to advantage

in children; among them aspirin, dose 3 to 7 grains, and novaspirin, 5 to 10 grains three times a day. These are especially beneficial when the stomach becomes intolerant to sodium or strontium salicylate. Ammonium salicylate may be given in 5-grain doses every three hours, and sometimes methyl salicylate internally in 5- to 10-drop doses, every three hours.

The heart should be examined carefully every day for any sign of dilatation of the left ventricle, for a systolic murmur, or imperfection in the first sound, inasmuch as the onset of valvulitis or endocarditis is not accompanied by any subjective or objective symptom.

When cardiac complications arise the child should be kept as quiet as possible, both mentally and physically, should use the bed-pan, and eat in a recumbent posture. If the heart is overactive, an ice-bag may be placed over it; if pericarditis is the cause of excessive cardiac action, tincture of aconite may be given in $\frac{1}{2}$- to 1-drop doses every four hours until free perspiration is induced. If myocarditis is present and the heart requires stimulation, strychnine sulphate, $\frac{1}{400}$ to $\frac{1}{200}$ of a grain; tincture of nux vomica, 1 to 5 drops, or $\frac{1}{2}$ to 1 teaspoonful doses of brandy may be given every three hours.

During convalescence children should take general tonics and receive special attention to prevent relapses. The anemia calls for iron, and 5 drops of the tincture of ferric chloride, or 5 to 20 drops of the syrup of ferrous iodide, may be given after meals. Sometimes iron causes indigestion and bodily pains, so that it must be discontinued; and cod-liver oil in teaspoonful doses, quinine in 1- to 3-grain doses, or elixir of glycerophosphates in teaspoonful doses, may be substituted.

The diet should be highly nutritious, but care should be taken not to overfeed, and also to prevent constipation. Milk, eggs, green vegetables, fish, and chicken are allowable if the digestion is good, but, as a rule, it is best to limit the amount of red meats.

If no heart lesions are detected, the child may be permitted to get out of bed two or three weeks after the fever has subsided and other symptoms have disappeared. If endocarditis is present, rest in bed is necessary for six or eight weeks after it is first noticed. In this case the heart should be carefully watched when the patient first sits up in bed, when he gets out of bed to sit in a chair, and when he walks; on the faintest sign of dilatation or undue strain, rest in bed should be again resorted to until the child can go about and take moderate exercise without apparently embarrassing the heart.

The prophylactic treatment of rheumatism in children and the after-care of children who have suffered from rheumatic attacks are often matters of great difficulty. These cases need a diet that is easily digested and is nourishing, containing a high protein content and a limited amount of starches. Flannels should be worn next to the skin the year round. If practicable, these children should be taken south for the winter and spring. As a rule, the seashore air

is too damp; therefore, unless there is endocarditis, mountainous regions or moderate elevations provide the most suitable climate.

Cold bathing is contraindicated; but a warm bath may be given each evening under proper precautions. When there is an endocardial lesion, violent exercise should be forbidden, games such as foot-ball, base-ball, and running races being very harmful. But moderate exercise is necessary, and the child should be encouraged to take short walks, and may amuse itself with croquet. Owing to the frequency with which rheumatism follows tonsillitis, the tonsils should be carefully examined, and every case of tonsillitis receive immediate and efficient treatment. Surgical tonsils should be removed as soon as possible.

MUSCULAR RHEUMATISM.

Muscular rheumatism is rare in infancy and childhood, but the acute and subacute types are occasionally observed.

Etiology.—The exact cause of the affection is unknown, but muscular rheumatism frequently follows exposure to cold or dampness. It is claimed by some authorities that disturbed metabolism exerts a predisposing effect upon this disease in causing the liquids of the body to contain a large quantity of toxic materials, which are precipitated into the muscle structure by cold or chilling.

Another theory is that rheumatism of the muscles is caused by a streptococcus much like the one supposed to be the cause of articular rheumatism. Among the exciting factors are indiscretions in diet, constipation, great physical strain or fatigue, improper or insufficient clothing, and damp weather. One attack predisposes to another. The disease is most common in children who are of a rheumatic diathesis.

Pathology.—The muscle fibers are swollen, and on cross-section show granular changes. Under the microscope in the acute form, which is really a myositis, we find round cell infiltration in the connective tissue and partial degeneration of the muscle fibers.

Symptoms.—Muscular pain and stiffness are the most prominent symptoms. Fever is rare. Cardiac complications do not occur as frequently as in articular rheumatism. The usual sites of muscular rheumatism are the cervical muscles (torticollis), the intercostal muscles (pleurodynia), and the lumbar muscles. As a rule, there are no constitutional symptoms.

Diagnosis.—This is usually easy to make, because of the location of the pain and the stiffness in the affected muscles; but articular rheumatism and neuritis must always be excluded.

Prognosis.—The prognosis as to recovery is good. There is little danger of cardiac complications, but recurrences are common.

Treatment.—When an attack of muscular rheumatism follows exposure, the child should have a hot Turkish bath, and then take 3 to 6 grains of Dover's powder, in three doses, one hour apart, and be put to bed. Aspirin, 3 to 7 grains, salol 2 to 5 grains, acetanilid,

gr. 1, or phenacetin in 1 to 2 grain doses, may be given three times a day. Hot compresses, a hot iron, or a hot-water bottle, placed over the affected muscles will often give relief. Chloroform liniment or a 20 per cent. salicylic ointment may also be applied.

A saline purgative, such as 1 dram of magnesium sulphate, or 20 to 30 grains of Rochelle salts, should always be given at the onset; and, if the urine is acid and highly colored, 10 grains of sodium bicarbonate in a glass of water may be administered three times a day. To prevent future attacks, these children should be protected from draughts and exposure, and should wear flannel or woolen undergarments the year round. Constipation should be corrected, and the diet carefully regulated.

CHRONIC RHEUMATISM.

Chronic rheumatism is rare in children, but may be the sequel of repeated acute attacks. It practically never occurs as a primary affection, chronic arthritic disease being usually due to some other cause, such as syphilis, tuberculosis, Still's disease, or rheumatoid arthritis.

Etiology.—Since most cases follow a series of repeated attacks of acute rheumatism, the etiology of chronic cases is essentially the same, dampness, exposure to cold, and heredity being the most significant causes.

Symptoms.—The symptoms are, for the most part, referable to the joints, which gradually become enlarged, painful, and immobile. As a rule, there are no constitutional symptoms. The course of the disease is protracted, although relatively shorter than in adults.

Diagnosis.—Two diseases, Still's disease and rheumatoid arthritis, closely resemble chronic rheumatism, and in every case should be excluded when making the diagnosis.

STILL'S DISEASE.

This affection is a chronic polyarthritis, which usually appears before the child is five years old. It is characterized by stiffness and enlargement of the joints, and enlargement of the liver, spleen, and lymphatic nodes.

Etiology.—The precise nature of the exciting factor in this disease is obscure, but it is believed to be chronic sepsis. Girls are affected in greater numbers than boys. Most cases occur before the eruption of the primary teeth.

Symptoms.—Acute exacerbations are accompanied by fever and by tenderness in the affected joints; but the most marked features of the disease are a gradually developing ankylosis of the joints and swelling due to pathological thickening of the soft structures. There are no exostoses and no degenerative bone changes; but muscular wasting is often extreme. The lymphatic glands about the joints and

throughout the entire body may be greatly enlarged, the liver and spleen hypertrophied, and the blood picture is that of severe anemia. Adhesions of the pleura and the pericardium are occasionally observed.

Prognosis.—Since there are no destructive changes in the joints, the prognosis is somewhat more favorable than in other chronic joint diseases; but complete recovery is rare.
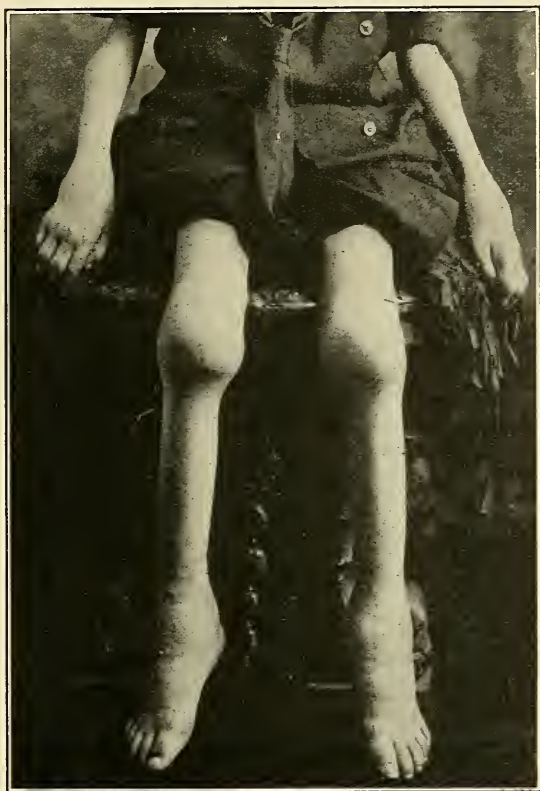
Fig. 74.—Still's disease.

RHEUMATOID ARTHRITIS—ARTHRITIS DEFORMANS.

Rheumatoid arthritis is extremely rare in children, and is at first usually mistaken for chronic rheumatism. It is marked by thickening of the synovial membrane, by enlargement of the articulating surfaces of the bones, and, occasionally, by effusions into the joints.

Etiology.—This disease is now believed to be infectious in origin, and the earlier theory that it originated in the nervous system has been largely discredited. It is usually the result of exposure to dampness and cold, and the infection is supposed to have its origin in decayed teeth, diseased tonsils, or other suppurating foci.

Symptoms.—In exceptional cases the onset of rheumatoid arthritis may be sudden, but, as a rule, it is slow. There is usually no fever, but the joints are painful and swollen, and gradually become worse. When the pain increases, motion becomes limited, and in protracted cases marked ankylosis and muscular atrophy occur. As the disease progresses, and additional joints are involved, pain on motion becomes more severe, and all movements of the body are restricted. The skin over the affected joints has a shining, waxy appearance, but there is no local heat, although the body temperature may rise a degree or so at intervals during the course of the disease.
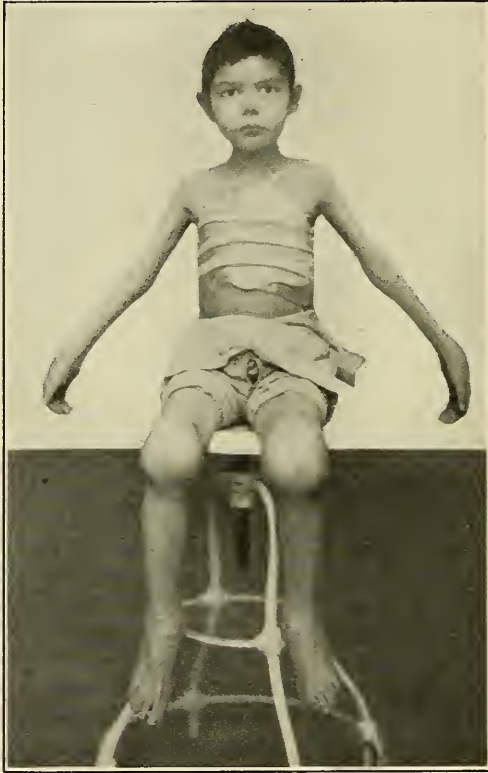
Fig. 75.—Still's disease.

Diagnosis.—The differential diagnosis between the several forms of chronic arthritic disease is difficult at the onset, but can usually be made after careful study. Chronic rheumatism is characterized by stiffness in the joints, little or no deformity, and a history of repeated acute attacks; while in Still's disease there are both deformity of the joints and stiffness due to thickening of the soft structures, and also glandular hypertrophy.

The chief diagnostic feature of rheumatoid arthritis is enlargement

of the articulating ends of the long bones, which results in ankylosis and marked muscular atrophy. Syphilitic arthritis may simulate chronic muscular rheumatism; but children with luetic changes in the joints usually show other symptoms of syphilis, such as Hutchinson's teeth and keratitis, and in doubtful cases a Wassermann should be made to clear the diagnosis.

Tuberculous arthritis may be differentiated by the absence of tuberculous foci elsewhere in the body, the absence of fever and of x-ray findings, and by the negative tuberculin reaction.
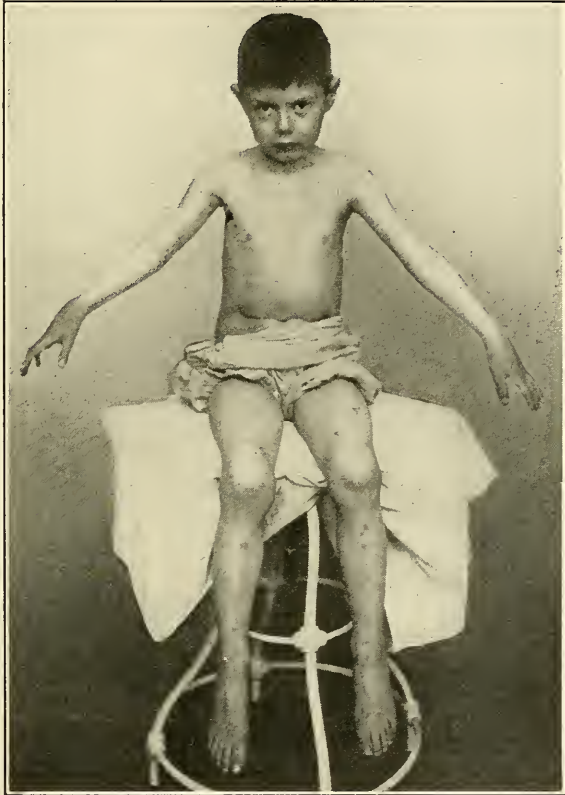
Fig. 76.—Still's disease. Same patient as in Fig. 75, but one year later.

Prognosis.—The prognosis is good as to life; but, as a rule, little can be done to relieve the condition of the joints.

Treatment.—The treatment of chronic rheumatism and of other forms of chronic arthritis is much the same. In the syphilitic types, antisyphilitic treatment is usually effective. Tuberculous joints are best treated by putting them in a cast, but rest is not indicated in rheumatoid arthritis, Still's disease, or chronic rheumatism, and children suffering from these diseases should be encouraged to use the affected joints as much as possible, stopping short of fatigue.

Warm bathing is very beneficial, and the affected joints should be massaged daily. Ichthyol is one of the best local applications, and a 25 to 50 per cent. ointment should be rubbed into the joint twice daily. In certain cases baking or dry hot air baths may be followed by marked improvement. Passive motion should be practised each day, and adhesions broken up while the child is anesthetized.

FIG. 77.—Rheumatoid arthritis in a girl aged twelve years.

If pain is severe, antipyrin or phenacetin may be given in 1 to 2 grain doses, three times a day, or potassium iodide 1 to 5 grains at a dose after meals. Children who improve under this treatment are sometimes enabled to walk by using suitable orthopedic appliances.

CHAPTER XXIV.

SCURVY.

Scurvy, or scorbutus, is a constitutional disease due to prolonged malnutrition. In about 50 per cent. of the cases it is associated with rickets, although these two diseases are essentially distinct. The chief characteristics of scurvy are a tendency to hemorrhage from the gums, the nose, and other mucous membranes, swelling and ecchymoses about the joints, especially the knee and ankle, and extreme tenderness and hyperesthesia of the lower extremities. There is also cachexia and marked anemia.

Scurvy in the infant is now recognized as the same disease which occurs in the adult, although it is modified by the different chemical and biological conditions found in the immature and rapidly developing organism of the infant or child.

The faulty nutrition which causes scorbutus is, in all probability, due to the lack of some food elements which are essential to normal metabolic processes and to growth. The exact nature of these constituents is not clearly understood, but it is believed that they belong to the group of vitamines.

Etiology.—Age is one of the chief etiologic factors, most of the cases being observed in infants between six and eighteen months of age. Before the sixth month scurvy is rare, although Kerley reports a case in an infant three weeks old. After the second year but few cases occur.

Infants of the middle and upper classes are most liable to this disease, and the great majority of cases have been observed in private practice; this offers a strong contrast to rickets, which is essentially a disease of the poorer classes. Hygienic surroundings have no influence on the development of scurvy, and there has been a history of previous gastro-intestinal derangement in such a small percentage of cases that the state of the alimentary tract seems to bear no relation to this disease.

The theory that it is of microbic origin is not supported by the clinical and pathological findings. It has apparently been proven that the most important etiological factor in the production of scurvy is some error in diet; but so far no single dietetic error has been demonstrated as the sole cause of this disease. Temporary faulty nutrition will not cause scurvy; but the malnutrition must be prolonged, and investigations seem to indicate that this dietetic error is a lack of some essential food element, rather than the presence of some abnormal food constituent.

In common with most other diseases of infancy, scurvy appears most frequently in bottle-fed infants, although it does occasionally occur in the breast-fed. The cases in breast-fed babies and in those fed on raw cow's milk form a very small percentage of the total number of cases of scurvy which have been studied, and some investigators believe that in these instances the milk which the infants had received had undergone some unexplainable chemical or biological change.

It is maintained that heating the milk removes from it something which is necessary for the prevention of scurvy, because of the great number of scorbutic children that have been fed on pasteurized, sterilized, or condensed milk, in comparison with the few cases of scurvy which develop in babies who are given raw milk.

In further support of the view that foods which are heated tend to produce scurvy, it has been demonstrated that many more cases of scurvy occur in children who are given sterilized milk than when pasteurized milk is used, and that when raw milk is substituted for sterilized milk many of these cases recover without further treatment.

The cooking, or heating, of an infant's food apparently destroys, changes chemically, or renders less digestible and assimilable, some principle of freshness within it, and results in metabolic disturbances which cause scurvy. By far the majority of cases occur when proprietary foods are given for a prolonged period, whether they are prepared with or without milk.

From these facts it is evident that the heating or cooking of food given to infants is of more importance in the production of scurvy than the use of, or failure to give any one particular food. The fact that scurvy sometimes develops in breast-fed babies and in those fed on raw cow's milk would appear to contradict the theory that the cooking or heating of the food is responsible for this disease.

Plantanza's observations show that although scurvy develops more frequently in babies fed on heated milk which is not used at once than on raw milk, yet the disease does not develop when fresh milk is heated and used immediately. Experiments on animals with raw and heated milk have failed to throw any light on this phase of the subject, for they have been too few to be conclusive.

The report of the American Pediatric Society's Collective Investigation of Infantile Scurvy, in 1898, brought out some important data with regard to the preceding diet in 379 of the cases. The summary of this report is given below.

> Breast milk, in 12 cases, exclusively in 10.
> Raw cow's milk, in 5 cases, exclusively in 4.
> Pasteurized milk, in 20 cases, exclusively in 16.
> Condensed milk, in 60 cases, exclusively in 32.
> Sterilized milk, in 107 cases, exclusively in 68.
> Proprietary infant foods, in 214 cases.

Morbid Anatomy.—The pathological changes in scurvy are most marked in the bones, the bloodvessels, and the blood. Hemorrhages may be widespread and multiple, taking place in the skin, mucous

membranes, serous membranes, internal organs, bone-marrow, and between the muscles. The subperiosteal hemorrhages are the most striking lesion, and may be very extensive.

The periosteum of the long bones is thickened and congested, and in some cases the hemorrhages break through the periosteum into the surrounding tissues, often infiltrating the cellular structures about the joints, although the joints themselves usually remain unaffected. These hemorrhages are much more common in the lower than in the upper extremities, and may appear in any area from the great trochanter to the knee, or from the knee to the ankle.

In the bone itself a rarefaction occurs which may or may not be due to intra-osseous extravasations. The diaphyses and epiphyses of these bones may separate, causing great deformity, although this usually occurs in fatal cases. The lower end of the femur or tibia is most frequently affected. The minute changes which take place in the bones are much like those seen in rickets, but these changes are probably a result of the accompanying rickets rather than lesions of scurvy.

Hemorrhages from the gums are very common in infants whose teeth have erupted, and the gums have a spongy appearance. The changes in the viscera are not constant, but there may be hemorrhages in the pleura, pericardium, and peritoneum. Bronchopneumonia and nephritis have been observed in quite a number of cases. Hematuria without inflammatory conditions in the kidneys is common, and hemorrhages may take place in the orbit or under the dura.

The alterations in the bloodvessel walls are, as yet, not understood; but since it has been shown by the capillary resistance test that there is a weakness of the vessel walls in scurvy, and studies of the blood have demonstrated that there is no deficiency of thrombin or blood platelets, and no excess of antithrombin, it is believed that the hemorrhages of scurvy are due to this alteration in the bloodvessel walls.

Symptoms.—There are no characteristic prodromal symptoms in scurvy, although a period marked by failing nutrition, irritability, and pallor may precede the first characteristic symptom, which is usually tenderness of the legs. Occasionally other parts of the body are hypersensitive; but in the majority of cases tenderness is first noticed in the leg, and this may be so slight that, although the infant cries out now and then when it is handled, it is quiet when in its bed.

This hypersensitiveness is often first noticed when the infant is being bathed or when the mother or nurse is changing its napkin; at the same time it may be observed that the child holds one or both legs still, but moves its arms, At first this tenderness is hard to locate, but it subsequently becomes constant and acute in one or more particular regions, usually about the knees and ankles, which are often swollen, these swellings being fusiform in shape, and due to subperiosteal hemorrhage.

In severe cases the tenderness becomes excessive, and may involve

all of the extremities, the affected limbs being rotated outwardly and maintained in that position, producing the pseudoparalysis of scurvy, which is caused by either the reflex or voluntary tenseness of the muscles. In severe cases the disability to move may be due to epiphyseal separation.

When the disease is marked, the sternum may become separated from the ribs and sink backward. The ecchymosis about the large joints and the fact that the child cries when touched, even when lying in its bed, often lead to the belief that it is injured. In advanced cases the infant becomes pitifully helpless, and lies perfectly motionless, screaming with apprehension whenever anyone approaches the crib, for fear it may be moved.

Fig. 78.—Scorbutus; same child as shown in *x*-ray plate.

While ecchymoses in the skin are not common, there are usually changes in the gums and mouth from the beginning of the disease. The gums become swollen and purple, and may almost cover the teeth, particularly the upper incisors. They bleed when touched, and hemorrhages may be spontaneous. Bleeding sometimes takes place in the vault of the pharynx and conjunctiva, and blood is frequently vomited or passed in the urine and feces. The stools are rarely normal in severe cases, for there is usually catarrhal colitis.

These symptoms all come on gradually with irregular intervals

PLATE IV
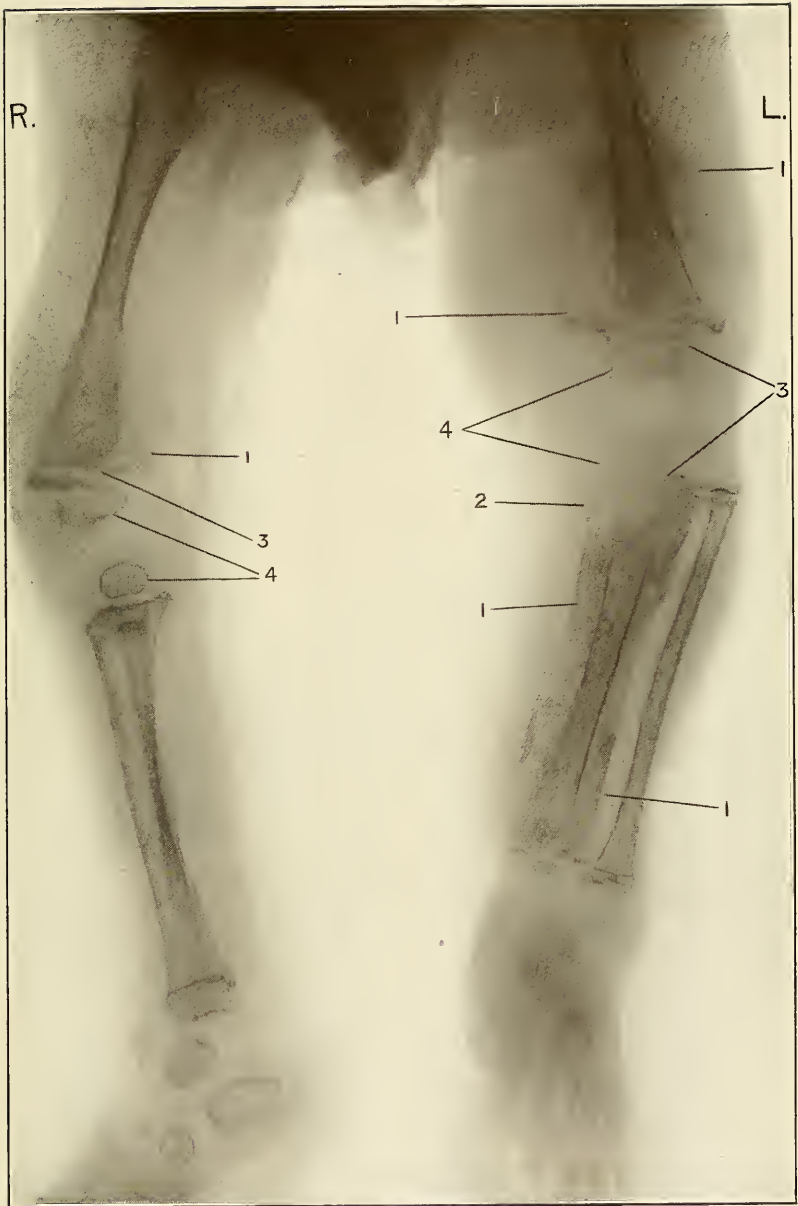
X-ray Plate of Child Shown in Fig. 78.

1 and 2 show subperiosteal hemorrhages and slight calcification of periosteum; 3, expanded ends of diaphyses; 4, practically normal epiphyses.

of apparent improvement, and no change in the child's general health may be noted for several weeks; but, sooner or later, evidences of malnutrition and anemia appear, and it becomes cachectic and emaciated.

In advanced cases there is usually irregular and inconstant fever, often due to complications; and, unless treatment is instituted, all of the symptoms grow steadily worse and the case terminates fatally in from two to four months by general asthenia, or some intercurrent infection or complication.

Diagnosis.—The diagnosis of scurvy can readily be made by those who have previously seen cases of the disease, and once it is suspected and its cardinal features looked for, there is but little trouble in recognizing it.

The history of an infant crying on being taken up, and a history of such feeding as might lead to the development of scurvy, together with such symptoms as spongy, swollen, bleeding gums, soreness and tenderness of the legs, and swelling about the large joints, should leave no doubt in the physician's mind as to the nature of the ailment. Further proof can be obtained by immediately placing the child upon an antiscorbutic diet.

Rheumatism is the disease most frequently simulated by cases of scurvy; but rheumatism is rare at the age when scurvy is most common, in scurvy there is no fever, and the pain is usually confined to the legs.

Poliomyelitis is sometimes suspected because of the pseudoparalysis; but acute pains in the limbs are rare, and there are no local inflammatory changes in poliomyelitis, while knee-jerks are absent in poliomyelitis, and present in scurvy.

Among the various other diseases with which scurvy is occasionally confounded are joint diseases, malignancy of the bones, spinal caries, trauma, and syphilis of the bone; but a carefully taken history and thorough examination, followed by the administration of orange juice as a therapeutic test, will, as a rule, differentiate between these conditions.

Prognosis.—This is excellent when the disease is recognized early and promptly treated. In most cases improvement is noticeable in from five to ten days, and complete recovery follows within two to three weeks. In neglected cases the prognosis depends upon the extent of the pathologic changes, the degree of malnutrition, the age of the infant, and the presence or absence of complications. Relapses are rare unless there is serious disturbance of the gastro-intestinal tract.

Treatment.—The prophylactic treatment consists in avoiding the continued use of any food which may have a tendency to produce scurvy, and whenever it is necessary to use such foods to supplement them by giving a certain amount of orange juice at stated intervals. When scurvy appears, fresh cow's milk should immediately be substi-

tuted for whatever food is being given, the physician prescribing a formula or milk mixture exactly suited to that particular infant.

This procedure alone will often cure the disease; but since the child usually suffers a great deal, fresh fruit juice, which is specific for scurvy, should be given at once. Orange juice usually agrees with an infant better than any other fruit juice, and from one to four ounces may be taken daily. A teaspoonful, slightly sweetened, may be given, if necessary, every two hours, preferably an hour or so before each feeding. Fresh beef juice and potato juice, as well as the juice of fresh vegetables, are also very beneficial especially for older children.

The syrup of the iodide of iron in 5- to 20-drop doses, and cod-liver oil $\frac{1}{2}$ to 1 dram, three times a day, are also beneficial when anemia and malnutrition are marked.

Scorbutic babies are very susceptible to intercurrent infections, and should be protected against exposure. They should be handled very little, and, if epiphyseal separation has taken place, splints should be temporarily applied.

CHAPTER XXV.

THE NERVOUS SYSTEM.

I. INTRODUCTION.

The central nervous sytem of the newborn is the most immature of all the organs of the body. This is true not only in regard to its morphology, but its function as well. The brain at birth is relatively large, yet its histological structures are poorly developed, and before it can functionate as in the adult substantial developmental changes are necessary.

Morphology.—Brain.—The brain at birth is relatively large and weighs approximately one-fourth of its ultimate weight. According to Ziehen, the weight of the brain at birth in proportion to the total weight of the body is 1:8 or 1:7, whereas in the adult it is 1:42 in the male, and 1:40 in the female.

According to Marchand, the brain weighs at birth 13 oz. (371 Gm.) in the male, and $12\frac{3}{4}$ oz. (361 Gm.) in the female. It increases very rapidly in size and, according to Pfister and Marchand, the original weight is doubled by the ninth month, and trebled before the end of the third year. Its ultimate weight is reached between the nineteenth and twentieth years in the male, and sixteenth and eighteenth in the female.

Spinal Cord.—The average weight of the spinal cord of the newborn varies between $\frac{1}{11}$ to $\frac{1}{10}$ of an ounce (3 to 3.4 Gm.) (Pfister), $\frac{1}{14}$ to $\frac{1}{5}$ of an ounce (2 to 6 Gm.) (Mies). Like the brain, its weight increases rapidly, is doubled by the fifth month, trebled by the end of the first year, and quadrupled by the beginning of the third year. At birth it is $5\frac{1}{12}$ inches (14 cm.) long, and very slowly reaches its ultimate length of 17 to $17\frac{3}{4}$ inches (43 to 45 cm.) (Pfister).

Histological Structure.—The central nervous system of the newborn is composed of immature embryonal tissue and cells which, by further growth and proliferation, develop into specific ganglionic cells and nerve fibers invested with myelin and neuroglia tissue. The most marked structural change is the investment of the nerve fibers with myelin.

According to Peritz, the most important centres necessary for the postembryonal life of the infant are first developed; namely, the gastro-intestinal and cardiorespiratory centres; next those which act as a defence to the body, primarily the sympathetic system and the spinal and cranial ganglia. At the same time are developed the centres for reflex and automatic instinctive movements (kicking, swallowing, sucking). The development of the sense centres follows later, then the speech centre and, finally, the higher psychical centres.

49

According to Thiemich, the myelinization of the tracts of the cord at birth is almost complete except that of the pyramidal tracts; in the brain stem and cerebellum numerous tracts are invested with myelin, whereas in the cerebral hemispheres but few tracts and centres have become myelinized. Pfister, Monakow, and others have demonstrated at birth some myelinized fibræ propriæ and medium-sized association fibers in the sense areas and tracts, in those for general sensibility, for the tactile, muscle, olfactory, and visual senses, as well as in the motor zone, in the pyramidal tracts, and certain segments of the corona radiata.

Myelinization progresses so rapidly that by the third or fourth weeks the long association fibers of the regio Rolando and regio calcarina are for the most part provided with myelin. By the end of the third month, most of the sense areas have been developed, and at the end of the first year the majority of the association fibers have been almost completely invested with myelin, whereas many years must pass before the tangential and the subcortical association tracts reach their full development.

Peripheral Nerves.—The myelinization of the peripheral nerves, according to Thiemich, has progressed but little at birth, and the areas developed show marked irregularities, being interrupted by areas which have not been myelinized. The development, however, progresses rapidly, and is complete by the end of the first year.

Physiological Development.—From what has been stated of the morphology of the central nervous system, it may be supposed that its functions likewise are most immature at birth, and only very slowly developed. This imperfect development is demonstrated by the electrical examination of the central and peripheral nervous system. The motor cortex, sensory, and motor peripheral nerves are so slightly irritable in the first few months of life that the infant's face is totally insensitive to strong electrical stimuli. Imperfect development on the part of the nervous system is accompanied by a greater need of sleep, about 20 hours daily being required during the first few weeks, and 13 to 15 hours by the end of the first year.

The development of the nervous system, however, is best shown by examining the reflexes. The tendon reflexes are present at birth, and are exaggerated from the second month to the second year. The superficial abdominal reflex is frequently absent at birth, but becomes very active in older infants. The Babinski and Oppenheim reflexes are present normally up to the sixth or even to the tenth month. The winking (optic) reflex appears from the sixth to the eighth week, whereas the conjunctival and reflex closure of the eyelids exist at birth and are active during the first few months. The pupillary light reflex is present at birth, and becomes especially active in the latter part of infancy. A reaction to accommodation has been observed by Pfister after the fourth week. The aversion to light disappears between the tenth and twentieth days.

Psychological Development.—Little is definitely known in regard to the psychical development of the child at birth. Stern thinks that, considering the number of tracts partially developed at birth, the child possesses a primitive consciousness, but that essentially the child is a subcortical being. Its acts are reflex movements brought about by stimuli, either from within or without, the centres for which are located within the cord and medulla. Thus, the various forms of the more vulgar type of sensations, such as thirst, hunger, and pain, call forth instinctively certain reflex responses, such as crying, sucking, kicking and swallowing, which are the protective vital manifestations of the newborn.

Sensibility.—In the first few days all sensory areas functionate sufficiently to call forth motor responses. The senses of touch, smell, and taste are all developed at birth; whereas the two higher senses, sight and hearing, are less highly developed. Sight is present in its most primitive form. The child reacts to the most extreme brightness, but has no conception of color, form, position, or distance. At about the third or fourth day it will momentarily follow a bright light, but will not fix its eyes distinctly upon objects before the fourth or fifth week, and not before the third or fourth month will the child follow objects, and begin to store up visual memories of what it sees.

Many infants are deaf at birth; stimuli, however, will call forth responses. The child may respond to a sudden noise by twitching the body or turning the head. Acoustic memories are gradually stored up; so that, by the end of the third month, the child has almost complete control of all its senses.

Development of Speech.—The child at first can utter involuntary sounds, thus gaining control of the speech mechanism. Gradually it learns by hearing to reproduce sounds, at first unconsciously, until finally conscious speech is acquired. By practice the child rapidly adds to its vocabulary which, according to Ziehen, at eighteen months should consist of 40 words, by the end of the second year of 200 to 300 words. With this rapid increase the child deepens his judgment, increases his associations, imitative instincts, his conceptions of personality, and gradually so develops that he will speak of himself in the first, instead of the third person.

In studying the morphology of the brain, its rapid increase in size within the first three or four years of life is held to be of the greatest importance. We now see why this is so. Functional development is commensurate with this gross increase for, by the end of the third or fourth year, the child is able to respond rudely to all stimuli for the sense organs, has its motor functions to a certain extent under control, is able to speak, to accomplish simple associations, and has a conception of personality (Craemer). We can, therefore, readily understand the great increase in brain tissue.

Peculiarities of the Nervous System of the Child.—Feeble inhibitory tone is the most important peculiarity of the child's nervous system. The child responds to stimuli with increased reaction which, in the

adult, would produce no result. This function of inhibition being the last function of the nerve cell to be developed is only imperfectly developed, owing to the very extensive areas of the central nervous system which are undeveloped. As the child grows older, and its nervous system develops, the inhibitory tone increases. According to Peritz, this feeble physiological inhibition is shown by the reactions of the child. It desires everything it sees; it is afraid of darkness, strange faces, and animals; it is easily frightened. Feeble inhibition is also observed in the child at play; it runs, jumps, kicks, being conscious of no restraint; all these are evidences of a lack of inhibition.

The lack of inhibitory tone is further shown by the development of the child's imitative instinct, as it soon imitates the movements of others. It is further shown in the flight of ideas observed in children. In relating stories, or in speaking, they ramble from one thought to another, just as these thoughts come to them; this is observed even in older children. Lack of tone is also shown by the effect upon the cardiorespiratory centre. The increase in heart rate, the variable quality of the pulse, and the respiratory changes show a lack of inhibitory development. The newborn is sympatheticotonic, not vagotonic.

Feeble inhibitory tone is shown pathologically in the ease with which sensory stimuli call forth motor responses. This is most clearly exhibited in the ease with which convulsions are produced in children by gastro-intestinal disturbances, intestinal parasites, fever, etc. The stimuli from the peripheral nerves are not inhibited, but are diffused over the motor cortex, and produce convulsions. Other examples are the reflex symptoms due to phimosis, eyestrain, and adenoids.

Another pathological evidence of feeble inhibition is the frequency of irregular muscular movements (choreic, choreiform) in children; whereas in the adult these choreiform movements, which are comparatively rare, are associated with definite cerebral lesions, in children this is not the case, the athetosis and choreiform movements which follow infantile cerebral palsies being independent of any local lesion. In the neuroses, the imitative instinct of the child is pathologically developed, especially in the monosymptomatic type of hysteria so common in children, in which the child imitates the movements of others. The sensory stimuli from without spread uninhibited over a whole motor cortical area, and evoke a response. The unusual development of the imagination so frequently observed in hysteria, often leading to lying, illusions, and hallucinations, is another evidence of this pathological lack of inhibition of the nervous system.

II. EXAMINATION OF THE NERVOUS SYSTEM.

The examination of the nervous system of the child is accomplished with considerable difficulty, for children cannot localize definitely their symptoms. Owing to fright, the symptoms may be exaggerated

or falsely localized. The examiner will do well first to gain the confidence of the child before beginning the examination. Naturally all painful tests, such as the electrical, should be made last. It is of the greatest importance in every case to take a complete history, especially the family history, before beginning the examination. In the general examination of a child much information can be obtained merely by inspection; this is also true in the examination of the nervous system, the shape, size, and symmetry of the head, the facial expression of the mentally defective, a flaccid or spastic paralysis, may all be noted.

Normal Reflexes.—The tendon reflexes are present at birth, become active after a few weeks and remain so until the second year. The more common deep tendon reflexes, such as the patellar, Achilles, triceps, and biceps are all evoked by holding the limb in such a position as to cause complete relaxation of the muscles, and then giving the tendon a sharp blow with a percussion hammer, whereupon the response will follow; in the patellar tendon reflex it is extension of the leg; in the Achilles, extension of the foot; in the triceps, extension of the forearm; and in the biceps, flexion of the forearm. The majority of the superficial skin reflexes obtained by irritating the skin do not appear at birth. The abdominal is sometimes observed about the third day, but is not constant before the fifth month. The cremasteric and gluteal reflexes appear at the third month, but are not constant before the end of the first year.

Abnormal Reflexes.—1. **Babinski Reflex.**—This reflex is obtained by irritating the outer margin of the sole of the foot by drawing some blunt object along it. Instead of the normal plantar flexion of the great toe, there is extension accompanied by a fan-like abduction of the other toes.

2. **Oppenheim Reflex.**—This reflex is also accompanied by extension of the great toe and other toes, and sometimes the foot as well; it is obtained by making firm pressure downward along the inner border of the tibia with the thumb or blunt end of a percussion hammer. In infants from six to ten months old this extension is a normal response, whereas a positive Babinski or Oppenheim reflex after this age is indicative of a pathological process—disease of the pyramidal tract.

3. **Ankle-clonus.**—An ankle-clonus is evoked by semiflexing the leg, and flexing the foot abruptly dorsalward, when a to-and-fro motion of the foot is produced.

4. **Patollar Clonus.**—This is a similar reflex produced by abrupt extension of the quadriceps femoris muscle and on pushing the patella abruptly downward. Both the ankle- and patellar clonus can be produced in very young infants. Peritz observed an ankle-clonus in a three-weeks-old infant suffering from meningitis. A pseudo-ankle- and patellar clonus can be elicited in neurasthenic and hysterical children. Thiemich states that an ankle-clonus can be obtained in fevers and irritable children up to one year of age without any pathological significance.

5. **Kernig's Sign.**—With the patient lying upon his back and the thighs flexed, complete extension of the leg at the knee-joint is impossible owing to spasticity of the flexor muscles. This is indicative of meningitis.

6. **Brudzinski's Sign.**—This sign is obtained, after passive flexion of the neck, with the patient on his back. The lower extremities will be drawn up, there being flexion at both the hip- and knee-joints. It is an early sign in meningitis.

The following are elicited in spasmophilic conditions:

7. **Trousseau's Phenomenon.**—This is produced by making pressure around the arm, best with an elastic band, thereby irritating the large nerves, and causing the hand and fingers to assume a tetanic position, the characteristic position in tetany

8. **Chvostek's Sign.**—This is obtained by gently percussing the cheek along the course of the facial nerve, best over Chvostek's point, which is midway between the zygomatic arch and the angle of the mouth. A clonic contraction of the muscles innervated by the facial nerve, the angle of the mouth, nose, eyelid, and forehead, follows. If the irritability is greatly increased, this same phenomenon can be elicited by stroking the cheeks (Schultz's phenomenon.)

9. **Erb's Phenomenon.**—This is electrically increased irritability of the nervous system. The CaCC is less than normal, and may appear at 0.1 MA. AnOC $>$ AnCC both being under 5 MA. CaOC less than 5 MA. is pathological.

Hypotonus.—By hopotonus is meant a condition of the muscles in which the normal tension is diminished; consequently greater excursions can be made by the extremities at the joints; thus, the legs can approximate the face; the heels, the buttocks; and the legs can be drawn apart to an angle of 190 degrees. In children up to four years of age this hypotonus is normal.

Sensory Examination.—Sensory examinations are not easily carried out in children, especially in those under five years of age, for they are not sufficiently intelligent to differentiate between the finer tests. Very young children are so sensitive to pin pricks that they begin to cry, making further examination useless. Only in children above nine years of age can we make sensory tests as in the adult.

Electrical Examination.—This is carried out the same as in the adult; but it requires considerable experience in order not to confuse the normal muscular contractions with those due to electrical stimuli.

Reaction of Degeneration.—The typical reaction of degeneration consists of a loss of excitability of both nerve and muscle to all faradic stimuli, and a loss of excitability of the nerve to all galvanic stimuli; whereas, galvanic stimulation of the muscle calls forth an increased response, *i. e.*, it reacts to a weaker current, or the contraction is changed in a typical manner so that, instead of a quiet, sharp contraction, there is a slow, vermiform, lazy contraction of the muscle. AnCC is excited more rapidly and appears before the CCC. This is

the typical reaction of degeneration, and denotes a lesion of either the anterior horn cells or the peripheral motor nerves.

Not infrequently, however, only a partial reaction of degeneration will be present, and may appear in various ways. There may be only diminished irritability of the nerve to both faradic and galvanic stimuli, or galvanic stimulation of the nerve and direct faradic stimulation of the muscle may cause a slow, lazy, muscular contraction. There may be only diminished irritability of the muscle to direct faradic stimuli, the contraction being prompt. In these various forms the AnCC = CCC or CCC > AnCC instead of ACC > CCC. These variations, together with the typical, slow, lazy, vermiform contraction of the muscle after direct galvanic stimulation, constitute a reaction of degeneration.

III. LUMBAR PUNCTURE.

Lumbar puncture, first described by Quincke in 1892, in the past few years has assumed such vast importance in the diagnosis and treatment of all nervous and mental diseases that a neurological study is no longer complete without a thorough investigation of the spinal fluid.

Technic.—Puncture must naturally be performed under the best surgical technic. Either the sitting or recumbent posture may be employed. In the sitting position the trunk is bent forward, the head flexed upon the chest, the arms allowed to hang loosely at the side. In the recumbent method, which in children is preferable, because puncture in the sitting posture is not free from danger to the little one, the child is placed upon its side, the knee and thighs well flexed, the head and shoulders bent forward, so as to separate the intravertebral spaces as widely as possible. A horizontal line is drawn from the crest of one ilium to the other, passing the vertebral column through the spine of the fourth lumbar vertebra. As the cord in the child is relatively long, in order not to injure it, the puncture should be made between the 4th and 5th lumbar or the 5th lumbar and 1st sacral vertebræ. One either punctures directly in the median line or somewhat to the side, preferably the former. The lower level of the upper vertebral spine is located with a finger of the left hand, and at this level the instrument is thrust through horizontally instead of piercing the centre of the intervertebral space. If punctured in the centre, then the needle should be directed upward; if punctured from the side, the direction of the needle should be upward, forward, and inward. One can readily tell by the sudden lack of resistance to the needle when one has pierced the spinal canal. The trocar is now withdrawn, and the fluid allowed to flow. The lumen of the needle may be occluded by fibrin or pus, or by the pressure of the fibers of the cauda equina against it. The former difficulty is generally overcome by reinserting the trocar, and in the latter by slightly withdrawing the needle. In some cases it may be necessary

to administer a few whiffs of chloroform or to freeze the skin with ethyl chloride. Following the puncture, the child should be kept quiet for twenty-four hours. For diagnostic purposes from 5 to 10 c.c. must be withdrawn.

Pressure of the Spinal Fluid.—Accurate measurement of the pressure with water or mercury manometers gives us very little information. A relative idea can be obtained by observing the flow—whether drop by drop, or in a constant stream. Generally speaking, the pressure is increased in all affections of the meninges, in brain and spinal-cord tumors, hydrocephalus, hemorrhages, abscesses, epilepsy, and eclampsia. On the other hand, meningitis may be present without increased pressure.

Laboratory Examination.—The normal spinal fluid is as clear as water, colorless, alkaline, and of low specific gravity, 1003 to 1007. Its composition is as follows:

Water, 98.74 per cent. Solids, 1.25 per cent. Albumin in the form of globulin and albumoses, 0.02 to 0.06 per cent. Dextrose, 0.4 to 1 per cent. Potassium salts, phosphate, and urea, 0.15 to 0.35 per cent.

The laboratory study of the fluid should be pursued along five different lines:

(1) Physical, (2) Chemical, (3) Cytological, (4) Serological, (5) Bacteriological.

Examination.

1. **Physical.**—The spinal fluid is either clear, turbid, opalescent, purulent, or bloody. In all the acute types of meningitis, such as the epidemic cerebrospinal, pneumococcic, septic, or influenzal, and in brain abscesses, one finds various grades of turbidity from a slight degree to purulence, depending upon the acuteness of the inflammation. On the other hand, in more chronic types, such as tubercular and syphilitic meningitis, the fluid is usually clear. In tubercular meningitis, the fluid, although usually clear in the beginning, may quickly become turbid, according to the degree of inflammation. In intraventricular hemorrhage, the fluid may be bloody.

2. **Chemical Examination.**—*Albumin.*—Nonne, Ross-Jones, Noguchi, Kaplan, and Lange have all described tests for detecting albumin. Only Kaplan's will be detailed here.

Kaplan's Test.—After boiling 5 c.c. of spinal fluid in a test-tube 1 cm. in diameter, 2 drops of a 5 per cent. butyric acid solution in normal saline are added, and it is boiled again. After boiling the second time 0.5 c.c. of a supersaturated solution of ammonium sulphate is underfloated by means of a pipette. After being allowed to stand for twenty minutes, an excess manifests itself in the form of a thick, granular cheesy ring at the point of contact. Kaplan gets a relative idea of the globulin increase by making tests of different dilutions: 0.5, 0.4, 0.3, 0.2, 0.1, and by adding distilled water up to 0.5 c.c. to the tubes containing less than 0.5 c.c. of spinal fluid.

Dextrose.—The reducing substance is sugar, which is detected by means of Fehling's test.

An increase in globulin generally accompanies any inflammatory condition of the meninges, and is also present in spinal cord tumors, without cellular increase.

Fehling's reduction is a normal reaction of the spinal fluid. It is also present in the more chronic type of meningeal inflammation as juvenile tabes, paresis, cerebrospinal syphilis, and sometimes as tubercular meningitis. It is not found in any of the acute forms of meningitis.

3. **Cytological Examination.**—Normally from one to five lymphocytes per c.cm. are present in the spinal fluid. Their study is of the utmost importance, inasmuch as there may be pathologically a great increase in cellular elements. There are three methods in use for the study of these cellular elements: (1) The French method; (2) the Fuchs-Rosenthal method; (3) the Alzheimer method.

The French Method.—This consists in centrifuging 5 c.c. of spinal fluid for from 20 to 30 minutes. After pouring off the supernatant fluid, the sediment is withdrawn by means of a fine capillary tube, placed on a slide, and stained for study. Only a relative idea of the cellular increase can be obtained by this method.

Fuchs-Rosenthal Method.—This consists in counting the cells upon a special counting chamber; that is, a modified blood-counting chamber whose area consists of 16 sq. mm., and depth 0.5 mm. This is the best method for obtaining an accurate estimate of the number of cells.

Alzheimer Method.—This method is employed for cytological study, and consists in embedding in celloidin the sediment obtained by centrifuging the spinal fluid, then cutting, mounting, and finally staining the sections for study. By this method the finer cytological studies can be made.

In all forms of meningeal irritation and inflammation there is cellular increase. Either the lymphocytes or polynuclear leukocytes predominate according to the acuteness of the inflammation. Consequently in all acute types of meningitis we have a predominance of polynuclear elements; whereas, in the more chronic type—spinal syphilis, juvenile tabes, and paresis—the lymphocytes predominate. In tubercular meningitis there is generally a lymphocytosis in the earlier stages; but subsequently the polymorphonuclear elements predominate, frequently forming as high as 90 per cent. of the total number of cells. In brain tumor (non-syphilitic), the endarteritic type of cerebral syphilis, hydrocephalus, and spinal cord tumors, there is a negative cell count.

4. **Serological Examination.**—In all neurological cases there should be a Wassermann reaction made of the spinal fluid, as well as of the blood serum. In cases of paresis, there is generally a positive Wassermann reaction in both fluid and serum; in tabes, the reaction in the fluid is positive in 60 per cent. (Nonne); it is generally negative in cerebrospinal syphilis (Plaut).

5. Bacteriological Examination.—Microscopic preparations should be made of all spinal fluids in order to examine them for bacteria. The Diplococcus intracellularis meningitidis is, as its name signifies, an intracellular diplococcus which does not retain the Gram stain. Tubercle bacilli are best found by making dry preparations of the web-like coagulum of fibrin which usually collects in the test-tube after standing for some time. The coagulum is well teased out upon a slide, and then stained for the tubercle bacilli. When a coagulum does not form the fluid should be centrifuged, so that the bacteria will settle in the sediment. It often requires long diligent search to find them, and repeated examinations from later punctures may have to be made before they are found. Cultures should always be made and, whenever in doubt, animal inoculations as well.

IV. CRANIAL AND CEREBRAL PUNCTURE.

(Neisser, Pollack, Pfeiffer.)

This is performed for both therapeutic and diagnostic purposes. It is easily done in infants, for the fontanelle membranes can be punctured without trephining. To avoid the sinus longitudinal superior, the needle is thrust 1 to 2 cm. laterally from the sagittal suture directly through the fontanelle membrane. The trocar is then withdrawn, when, if fluid be present, it will flow out. Clear fluid is indicative of external hydrocephalus, and bloody fluid of pachymeningitis interna hemorrhagica. Should one desire to aspirate the lateral ventricles, the needle is thrust in a few centimeters farther along in their direction. These procedures are made use of in the diagnosis and treatment of internal and external hydrocephalus and pachymeningitis interna hemorrhagica. In older children in whom the sutures and fontanelles have closed, the point of entrance must be trephined, and the whole procedure carried out under the strictest surgical technic.

DISEASES OF THE SPINAL CORD.

MALFORMATIONS.

Malformations of the spinal cord are frequently associated with those of the brain, with defects of the skull, and of the vertebral column, as well as with malformations in other parts of the body; viz., ectopia of the bladder and congenital hernia. Malformations of the spinal cord may be grouped as follows:

1. Amyelia—entire absence of the cord.
2. Atelomyelia—partial development of the cord.
3. Diastematomyelia and diplomyelia—the division and reduplication of the cord.
4. Heterotopia—the malposition of some of the gray matter.

All of these varieties are exceedingly rare, and of little interest.

5. Spina bifida.—By "spina bifida" is meant all congenital defects of the vertebral column, these being most frequently present posteriorly in the vertebral arches, but, more rarely, anteriorly in the body of the vertebra.

According to Marchand, there are two kinds of spina bifida: (1) the closed form, spina bifida cystica; and (2) the open form, rachischisis.

All of these malformations, excepting rachischisis, have been considered in Chapter VI on Congenital Malformations (see pages 100–103).

Rachischisis.—In rachischisis, which may affect either the whole or a part of the vertebral column, there is an open fissure which exposes the posterior surface of the body of the vertebra together with a part of the inner layer of the pia and spinal marrow. At the border of the cleft, the skin, fascia, muscles, bone, dura, and pia terminate abruptly. In embryonal life the spinal portion of the medullary groove remains patent.

MYELITIS.

Myelitis, other than acute anterior poliomyelitis and compression myelitis, is very rare in childhood. Adults and children react quite differently to inflammation of the cord. In the child poliomyelitis is the characteristic manifestation, whereas in the adult it is myelitis. The other types of myelitis, though rarely seen, are transverse myelitis, Landry's ascending paralysis, and disseminated encephalomyelitis. Vascular changes in the cord are also included under this heading, for anatomically and clinically they cannot be differentiated.

Myelitis may follow any infectious disease, and may complicate a syphilitic or tubercular infection. Certain poisons, such as gas and arsenic, are known to produce the disease. Numerous bacteria have been isolated from the cord, but none specific. According to Bruns, the disseminated encephalomyelitis is the most frequent type, the transverse myelitis and Landry's ascending paralysis being very rare. Disseminated encephalomyelitis and Landry's ascending paralysis are described under separate headings.

Transverse Myelitis.—This disease usually sets in slowly after some previous infection, with moderate fever, general malaise, weakness, pains or paresthesia, loss of appetite, and a slowly developing paralysis. On the other hand, the paralysis may develop suddenly, as an apoplectiform attack. As a result of the inflammation, there are sensory, motor, and trophic disturbances. Of the sensory symptoms there may be either pains—girdle pains—due to irritation of the posterior roots, or anesthesia, or paresthesia.

Motor symptoms may be as follows: (1) Clonic muscular spasms from irritation of the anterior roots. (2) A flaccid paralysis at the level of the lesion. (3) A spastic paralysis below the level of the lesion with exaggerated reflexes, ankle and patellar clonus, an absence of muscle atrophy and the reaction of degeneration, with positive Oppenheim, Babinski, and Mendel-Bechterew phenomena.

Of the trophic disturbances there are: (1) Atrophy of the muscles at the level of the lesion with an accompanying reaction of degeneration. (2) Bladder and rectal disturbances with secondary cystitis, cystopyelitis, and nephritis. (3) Decubital ulcers. The local symptoms vary according to the location and extent of the lesion. When situated in the lumbar portion of the cord, there is a flaccid paralysis of the lower extremities, with muscular atrophy, loss of reflexes, loss of sensation up to the level of the lesion, girdle sensations at the level, sphincter disturbances, and decubital ulcers.

When the lesion is situated in the thoracic region—its most frequent location—there is spastic paralysis involving the lower extremities, with exaggerated reflexes, ankle and patellar clonus, Oppenheim and Babinski phenomena, anesthesia up to the level of the lesion with girdle pains above it, sphincter disturbances, and decubital ulcerations. When the lesion is located in the upper thoracic and lower cervical regions, there may be a flaccid paralysis of the arms, with loss of reflexes, and muscular atrophy with spasticity of the trunk muscles and lower extremities. When situated in the upper cervical portion, in addition to spastic paralysis of both lower and upper extremities, there are oculopupillary disturbances, also interference with respiration due to involvement of the phrenic nerve.

A lesion may be so circumscribed as to give unilateral symptoms, leading to monoplegia. The typical Brown-Séquard syndrome may be present in rare cases. Occasionally a transverse myelitis may be so extensive as to give rise to a complete transverse lesion, in which event there is flaccid paralysis and loss of reflexes below the level of the lesion. The symptom-complex may be further complicated by the presence of multiple disseminated foci which extend over the whole central nervous system.

Prognosis.—The prognosis is unfavorable. Death is due either to involvement of the respiratory centre or to complications, such as intercurrent infections, cystitis, or decubital ulcers. However, marked improvement and even recovery may take place. This is usually observed in disseminated encephalomyelitis.

Treatment.—During the acute stage the patient should be kept in bed, and care taken to prevent bed-sores and cystitis. Electricity and all skin irritants are contra-indicated. Warm baths will relieve the spasmodic contractions of the muscles. Sedatives, such as sodium bromide, must be administered frequently. When convalescence sets in the galvanic current should be applied along the spinal column and over the atrophied muscles. When the patient begins to regain the use of his paralyzed muscles, moderate massage and gymnastic exercises should be prescribed, and Frenkel's reëducation movements taught. Improvement may follow the administration of potassium idodide. In all cases in which syphilis is the etiological factor, anti-syphilitic treatment should be instituted at once. In addition, certain orthopedic appliances may be necessary.

Compression Myelitis.—The spinal cord may be compressed by affections which involve the meninges of the cord, such as a tumor, or spinal meningitis, or the vertebræ themselves. By far the most frequent cause is caries of the vertebræ. Rarer causes are dislocations, fractures, tumors, syphilis, arthritis deformans, acute spondylitis due to rheumatic fever, osteomyelitis, and typhoid fever. The author has recently observed a case of beginning compression myelitis due to arthritis deformans in a little girl, six years of age.

CARIES—SPONDYLITIS TUBERCULOSA—POTT'S DISEASE.

Etiology.—Myelitis due to caries of the vertebræ is a disease of early childhood, beginning usually between four and eight years of age. It may, however, appear in early infancy, or may be delayed until puberty or adolescence. It is usually secondary to, and combined with tubercular disease elsewhere in the body, as the lungs, other bones, joints, or glands. It may develop spontaneously or following traumatism.

Pathological Anatomy.—Although usually secondary to tuberculosis elsewhere in the body, the primary vertebral focus of infection is in the body of one or more of the vertebræ. The tubercle bacillus causes the formation of spongy granulation tissue which, by disintegration of the bony tissue, leads to a secretion of thick material resembling pus. This may continue until there is complete softening of the body of the vertebræ when, on removal of the support, the overlying vertebræ sink in, and produce a characteristic angular projection of the spinous process—kyphosis.

Compression of the cord is not usually due directly to compression of the vertebræ, but to a narrowing of the spinal canal and compression by the thickened dura. The adjacent dura becomes inflamed, both as an internal and external pachymeningitis, and becomes greatly thickened. The pressure of this tubercular mass upon the cord causes edema by interfering with the free flow of blood and lymph, giving rise to anemia sufficient to cause different grades of degenerative changes in the cord. Generally this mass is located anteriorly, but may surround the cord. More rarely, sudden compression of the cord is due to dislocation of a vertebra.

The lumbar, dorsal, and cervical vertebræ are most frequently involved, in the order mentioned (Schmaus). Compression symptoms are present in 80 per cent. of the cases of the dorsal and cervical varieties, but are less common in the lumbar and sacral varieties (Schmaus). Microscopic examination shows degeneration of the tracts and nerve roots compressed. Following the edema there is swelling of the axis cylinder, later degeneration of the nerve fibers.

Symptomatology.—The symptoms may arise from the side of the bones, the nerve roots, or the spinal cord. The earliest symptom is pain over the involved vertebræ, this being increased by motion or compression. In children pain may be absent; and, as a result,

in order to produce fixation of the joint, there is reflex muscular contraction. Irritative root symptoms are not so extensive, but form an early symptom of the disease, the pain being sharp, lancinating in character, and distributed over the course of the affected nerve. The paralytic symptoms, the result of compression, depend upon the point of compression.

At first there is muscular weakness; but, as the pressure advances, the picture of transverse myelitis develops, differing from the latter in that sensory disturbances are much in abeyance. Touch and temperature may be affected; pain and muscle sense are usually intact. Sphincter disturbances are generally absent until late in the course of the disease. As the compression usually affects the thoracic region, there follows a spastic paraplegia of the lower extremities, with increased reflexes, ankle- and patellar clonus, Oppenheim and Babinski phenomena, and anesthesia of varying degree up to the level of the compression, where there is an area of hyperesthesia.

Severe girdle pains may appear as well as sphincter and trophic disturbances. (For the symptoms of compression on other parts of the cord, see chapter on Myelitis.) When the upper cervical vertebræ, the atlas, and the axis are involved there is usually marked bilateral occipital neuralgia. In addition bulbar palsies are frequent. Respiration is also endangered from involvement of the respiratory center.

Diagnosis.—The presence of a deformity, of localized pain, especially on percussion over the vertebræ, neuralgic girdle pain, rigidity of the spinal column, together with any paralysis, usually establishes the diagnosis. X-rays and the different tuberculin tests may assist. In the earlier stages, before the characteristic picture has been completed, since one or more symptoms may be lacking, it must be differentiated from tumors of the spinal cord and vertebræ, syphilis of the vertebræ, spondylitis due to the Bacillus typhosus, or an acute osteomyelitis, rickets, or rheumatic fever. Hysteria and neurasthenia may also be mistaken for this disease.

Course and Prognosis.—The course of myelitis due to Pott's disease is usually slow and chronic, extending over a number of years. The prognosis naturally depends upon the extent of tubercular involvement in the rest of the body, and upon the treatment. In many cases early symptoms develop long before there is paralysis, and if appropriate treatment—*i. e.*, fixation—is begun when the diagnosis is made, paralysis may be averted. Under proper fixation the paralysis which occurs early in the disease quickly passes away. When, however, paralysis develops during treatment, the progress is unfavorable. Relapses are frequent in later life, being accompanied by palsies, which do not occur in the earlier attacks.

Gibney's records show recovery in 50 per cent. of his cases, and death in 20 per cent., whereas Peritz estimated his at 30 per cent. of recoveries and 60 per cent. of deaths.

Treatment.—The treatment consists, first, of rest in bed, removal of the pressure, and fixation of the spine by proper orthopedic appliances or by bone transplantation; secondly, in the alleviation of the symptoms. Many of these, such as neurotic pains and muscular rigidity, are relieved by proper fixation. In addition, sedatives may be required. Hot and cold douches, and, when possible, packs applied to the spine, promote free circulation in the spinal cord.

General measures, such as plenty of fresh air, especially sea air, forced feeding, the administration of cod-liver oil and general tonics, are extremely important. Electricity and treatment with tuberculin have been ineffectual. The utmost care should be taken to prevent bed-sores.

LANDRY'S PARALYSIS—ACUTE ASCENDING PARALYSIS.

This rare disease was described by Landry in 1859. Following premonitory symptoms, such as general malaise, fever, pain in the extremities, and paresthesias, extending over a period of several days, paralysis appears in the toes and feet, and within a few hours extends over the entire limb. After a brief interval the paralysis spreads rapidly upward, involving the muscles of the back, thorax, arms, and neck. Deglutition and speech are interfered with, also the respiration, as shown by Cheyne-Stokes breathing. Usually there are short pauses in the progress before the paralysis is complete. Bulbar paralyses do not usually appear, owing to the rapidity of the process; although facial and ocular palsies have been reported. Paralysis may develop within twenty-four to forty-eight hours, and occasionally proves fatal in that time, or it may be two to three weeks reaching its maximum.

The paralysis is always flaccid. The reflexes may be absent, but are never exaggerated. Electrical disturbances are not usually found. Slight sensory disturbances, such as paresthesia and partial anesthesia, are often present. Muscular atrophy, and sphincter and trophic disorders do not appear. The consciousness remains clear until the end.

Instead of its usual ascending course, the disease may set in with bulbar paralysis, then rapidly extend downward over the arms and trunk. In this type, death may take place before the legs are involved.

In its nature the disease is an acute intoxication involving the medulla oblongata and spinal cord, and in some cases the spinal roots and peripheral nerves. Splenic enlargement is frequently observed. It has been described as a clinical form of acute poliomyelitis. The disease sometimes follows certain infectious diseases; viz., typhoid fever, diphtheria, influenza, anthrax, whooping-cough, and syphilis.

Pathological Anatomy.—Formerly the pathological findings were thought to be negative, but now finer methods of examination show a diffuse and disseminated myelitis with changes especially prominent about the bloodvessels, viz., thrombosis with softening, hemor-

rhages, and round-cell infiltration leading to changes in the nerve fibers, particularly to a swelling of the axis-cylinder. These changes are usually diffuse. In other cases, neuritis of the nerve roots and peripheral nerves has been observed. Numerous non-specific organisms have been found in the gray matter of the medulla and cord.

Diagnosis.—The diagnosis of the malady is usually easy, considering the rapidity of its course. Peripheral neuritis, acute poliomyelitis, and spinal infantile muscular atrophy must all be differentiated from it.

Course and Prognosis.—The course of the disease is most frequently fatal, death taking place within three to seven days, either from asphyxia or secondary aspiration pneumonia. Arrest of the disease may, however, be observed at any stage.

Treatment.—Great care must be exercised to prevent aspiration pneumonia. If the patient is not too weak, hot packs should be given. Counter-irritation with the cautery has been recommended. If syphilis is suspected, mercury should be begun at once. Ergotin, also, has been recommended; otherwise the treatment is the same as for myelitis.

ACUTE ANTERIOR POLIOMYELITIS—INFANTILE PARALYSIS.

Definition.—Acute anterior poliomyelitis is an infectious disease of the central nervous system, appearing either epidemically or sporadically, usually affecting very young children, and giving rise to a flaccid paralysis. This is followed by gradual improvement; but, as a rule, some permanent paralysis remains in certain muscles which undergo atrophy.

History.—The best early clinical description of this disease was by Heine, in 1840. Medin described it in 1890, and demonstrated that it occurred epidemically. Since that date numerous epidemics have occurred in this country, Norway, Sweden, France, Italy, Austria, Germany, and Australia. Formerly, as the name implies, the disease was thought to be one of the anterior horn cells; but since the epidemic in Sweden, in 1905, during which Wickmann demonstrated various clinical varieties of the disease, our conception of it has been broadened, and we now know that the lesion is not confined to the spinal cord, but extends to the pons, medulla, midbrain, cerebrum, and meninges.

Wickmann not only described the pathology and epidemiology of the disease, but was the first to describe the abortive type of poliomyelitis. In 1909, the disease was produced experimentally in monkeys by Flexner and Lewis, Landsteiner and Popper, and by Strauss. In the same year, working independently, Flexner and Lewis, Leiner and von Wiesner, and Landsteiner and Levadii, reproduced the disease in monkeys, transmitting it from one monkey to another. Finally, in 1913, Flexner and Noguchi cultivated the infecting organism, and have since established the fact that it is the cause of the disease.

Etiology and Epidemiology.—Numerous epidemics within the past decade have materially enriched our knowledge and conception of the malady. It is essentially a disease of early childhood, occurring most frequently within the first three years of life, although adults are occasionally affected. The time of greatest predisposition to it is during the latter half of the second year. The following table, in part taken from that of Frauenthal and Manning, shows the relative age of onset:

Ages.	Wickmann, Sweden, 1905. Per cent.	Manning, Wisconsin, 1908. Per cent.	Lovett, Massachusetts, 1909. Per cent.
0 to 5 years	40.6	49.8	71.5
6 years and over	59.4	50.2	28.5

Ages.	New York City, 1907. Per cent.	Rockefeller Institute, 1911. Per cent.	Müller, Hesse-Nassau. Per cent.
0 to 5 years	90.5	89.0	90.0
6 years and over	9.5	11.0	10.0

Both sexes are equally affected. Epidemics occur, especially during the summer months, reaching their maximum in late summer or early autumn. The disease predominates in the country rather than in the city. It has been known to follow practically all of the acute diseases, especially pneumonia and measles, but usually the children attacked have previously been perfectly healthy. Other exciting factors which have lowered the patient's resistance and made him more susceptible to infection have been exposure to cold and dampness, overexertion, and trauma. One epidemic usually renders a community immune.

The infectious nature of the disease has now been definitely established by the cultivation of the infecting microörganism by Flexner and Noguchi, and the experimental production of the disease in monkeys by inoculation of the cultures which had previously passed through a number of artificial media for a period of eighteen months. The microbic agent, or virus, consists of minute globular bodies which are stainable and visible under the high power of the microscope. It is a filterable organism, and is resistant to the action of glycerin, to freezing, to a 0.5 per cent. solution of carbolic acid, and to ordinary degrees of heat.

Mode of Infection.—From the work of Flexner and Lewis it seems well-established that the upper respiratory tract is the port of entrance of the disease. The lymphatics of the nasal mucosa which pass out with the filaments of the olfactory nerve are directly connected with the meninges, and carry the infecting organisms to the spinal fluid. The virus is also given off through the nasopharyngeal mucosa.

There are two diverse views as to the mode of infection: (1) By means of the stable fly (Stomoxys calcitrans), as expounded by Rosenau; (2) by personal contact. According to Flexner, from the evidence at

50

hand the virus is present in the nasal and buccal secretions. Those who suffer from the disease transmit it by implanting the virus upon the upper nasal mucosa of other susceptible persons who then develop an attack. Those who suffer from the abortive type of poliomyelitis spread it in the same manner. In addition there are (1) healthy carriers who have been in intimate contact with patients; also (2) chronic carriers who have recovered from an acute attack but who, even after the lapse of several months, are capable of transmitting the disease. Moreover, it may be conveyed by certain passive agents, such as clothing and dust, also by domestic animals, flies, and insects, for the virus is known to be very resistant.

One infection with poliomyelitis confers an active immunity upon the patient. Immune bodies are formed, which are readily demonstrated biologically by the neutralization of active virus by the blood serum of a patient who has recovered from the disease. This neutralized virus will not reproduce the disease experimentally in monkeys; that is, the active virus has now become absolutely inactive. The neutralization test is of the greatest importance in the diagnosis of the abortive type of poliomyelitis.

Pathological Anatomy.—That the virus gains access to the body through the upper respiratory passages, and is carried by the lymphatics along the filaments of the olfactory nerve to the cerebrospinal fluid, is shown by the earliest pathological changes observed in the central nervous system. There is hyperemia, also a mononuclear collection of cells in the perivascular lymph spaces of the bloodvessels of the pia-arachnoid which communicate with the cerebrospinal fluid. This cellular infiltration is conspicuous along the anterior surface of the cord, especially about the anterior fissure. The pathological process then extends to the cord and brain by way of the lymph spaces of the vessel sheaths as they enter the cord from the meninges. Here similar hyperemia and round-cell infiltration take place within the perivascular lymph spaces.

This cellular infiltration, extending along the course of the bloodvessels, may partially constrict the lumen and cause extensive edema. A few leukocytes may be present in the early stages, but they are replaced by lymphocytes, and by glial and proliferating endothelial cells. The proliferation and infiltration of cells extends into the adjacent tissues in the tissue spaces of the cord and neuroglia. In addition, hemorrhages are found, due either to toxic or mechanical injury to the intima.

As a result of these primary vascular changes—cellular infiltration, hemorrhages, edema—and the resulting anemia, there is degeneration of the interstitial tissue and nerve cells. Various degrees of degeneration are observed, from simple swelling to complete destruction and disintegration, and after this destruction polymorphonuclear neurophages enter and ingest the neurotic material. The lumbosacral and cervical enlargements of the cord are the portions most frequently involved.

At the same time similar changes are observed in the posterior roots, and to a less extent within the brain, pons, and medulla, especially about the cranial nerve nuclei, and in the gray matter about the fourth ventricle.

A microscopic cross-section made at this acute stage shows hyperemia, especially about the anterior horn, increase in and dilatation of the bloodvessels, hemorrhages, cellular infiltration of the vessel walls and throughout the gray and white matter, and nerve cells in different stages of degeneration. The ganglion cells may simply be clouded, or the nuclei may be indistinct or may have fallen out, or there may be complete disintegration of the cells together with its dendrites and axis-cylinder.

In addition to these acute changes within the central nervous system, there is also enlargement of all lymphoid tissue, especially of Peyer's patches in the intestines and of the mesenteric lymph glands, with cloudy swelling of all parenchymatous organs, such as the liver and kidneys.

Should the inflammation subside when but slight degenerative changes have taken place in the nerve cells, complete regeneration may follow; but, if continued until there is absolute atrophy, regeneration is no longer possible, and in consequence of the atrophy of the cells and neuroglia there is a contraction of the area about the anterior horn which is readily disinguished on cross-section. The ganglion cells have disappeared, and are replaced by paler glial tissue; the vessel walls are thickened; the division between gray and white matter is indistinct; and the anterior roots and peripheral nerves contain degenerated fibers. The affected muscles exhibit different degrees of atrophy, varying in color from salmon to a pinkish, grayish, or yellowish hue.

In extreme cases the muscle fibers have completely disappeared, and are replaced by fibrous and adipose tissue. The long bones are likewise affected, in that they are usually shorter, and the shafts are thinner than the normal ones. The affected joints and ligaments are relaxed. The demonstration of such a widespread reaction to the virus is wholly in line with recent clinical and epidemiological investigations which tend to regard the disease as a general infection and a generalized process which affect the parenchymatous organs, the lymphoid tissue, and, more especially, the nervous system (Peabody, Draper, Dochez.)

Symptomatology.—The clinical course of poliomyelitis resembles that of any other acute infection. We distinguish four periods: (1) A period of incubation; (2) a period of prodromal and initial symptoms; (3) a period of paralysis; (4) a period of retrogression. Frequently the different periods are not separable.

Incubation.—The incubation period usually lasts from two to ten days, although it may be less than twenty-four hours, or may extend to three weeks, according to the virulence of the infection and the resistance of the patient.

The prodromal period is characterized by certain general symptoms which continue from one to three days. Certain symptoms predominate in certain epidemics. The disease sets in usually with fever which varies between 102° and 106° F., and occasionally with a chill; there are general malaise, nausea, vomiting, loss of appetite, rapid pulse and respirations, profuse sweating, constipation, and retention or suppression of urine. There may be weakness in one or more limbs or groups of muscles, and in addition drowsiness, coma and delirium, but rarely convulsions. Certain skin affections—herpes, erythema, and a scarlatinal-like rash—are sometimes observed. Meningitic symptoms may be pronounced. There is rigidity of the neck, with irritability of the spinal column, severe pains in the back and extremities, hyperesthesia, basilar headache, Kernig's sign, and tremors; twitchings and convulsive movements and prostration may be marked.

In certain epidemics gastro-intestinal symptoms, expecially diarrhea, have predominated, while in others the disease has set in with bronchitis, or coryza, or with angina which simulated tonsillitis.

The fever may run to 104° or 106° F., exhibiting slight morning remissions, then dropping by degrees to 100° F. before the onset of paralysis; it may reach normal several days after paralysis has set in. The pulse rate varies between 120 and 200, the respirations between 40 and 60.

In some cases these prodromal symptoms may be so mild as to be entirely overlooked, the paralysis coming on overnight in a previously healthy child. The diagnosis of the paralytic stage of the disease is of the greatest importance; for, if an early diagnosis is made, proper measures can be instituted to prevent the spread of the disease, and early treatment begun before destruction of the nerve cells has taken place. In addition to the general symptoms, the examination of the spinal fluid is an important aid to the diagnosis. In the preparalytic stage the fluid is under moderate pressure, increased in amount, clear or opalescent in color. There is cellular increase, of which about 90 per cent. are polymorphonuclear cells, also a slightly increased or normal globulin reaction, with a normal reduction of Fehling's solution. On standing, a sterile coagulum may form.

Paralysis usually sets in several days after the onset. It may either appear suddenly—as overnight—or develop more gradually, there being first a weakness, paralysis following within several days. The paralysis is usually diffuse and progressive, extending from one limb to another, and reaching its maximum within several days to one week. Characteristic of the paralysis is its unsystematic distribution, for all combinations of paralysis of the extremities, trunk, and cranial nerves may be found. The following table, taken from the work of Frauenthal and Manning, shows the distribution of the paralysis, as recorded by Lovett and Sheppard in 1910:

DISTRIBUTION OF EARLY PARALYSIS.

	Cases.
One leg only	145
Both legs	146
One arm only	44
Both arms	12
One arm and leg, same side	50
One arm and leg, opposite sides	18
Both legs and one arm.	32
Both arms and one leg	8
Both arms and both legs	51
Ataxia (transitory)	7
Back	79
Abdomen	38
Neck	13
Respiration	39
Deglutition	12
Intercostal	1
Face	7
Right face	31
Left face	24
Strabismus	2
	759

From the above it is evident that the lower extremities are most frequently involved—twice to three times as often as the upper extremities. Paralysis of the diaphragm and the muscles of the abdomen, back, and neck is not rare. Bulbar palsies are frequently observed, the facial and hypoglossal forms, with resulting difficulty in deglutition and speech, being the most common. In addition, there may be ocular palsies from involvement of the abducens and oculomotor nerves.

In certain cases, such as have been described by Medin, bulbar palsies may be the only ones to appear. With the advent of these palsies, the prodromal symptoms diminish in intensity, the temperature, pulse rate, and respiration suddenly drop, and the gastro-intestinal and bladder disturbances improve, although retention of urine may persist when the lumbar segment is affected. On the other hand, meningeal symptoms progress until retrogression sets in.

Whereas, in the beginning, the paralysis is quite extensive, by the end of the second week it usually recedes and there is return of function. In exceptional cases this may be complete. Usually, cases of more or less extensive paralysis recover, except for one or more extremities or groups of muscles; then again, there may be no recovery from the paralysis. When the paralysis affects the legs, two types are distinguished, the upper and lower leg type. In the upper or thigh type, the glutei, iliacus, psoas, and antero-external muscles of the thigh, especially the quadriceps femoris, are involved; whereas, in the lower leg type, the peronei and anterior tibialis are usually the ones affected.

Likewise in the arm; instead of the paralysis being complete, two types—upper and lower arm types—are usually recognized. In the upper arm type the scapular muscles—the biceps, deltoid, and sup-

inator longus—are involved; whereas, in the lower arm type, all the muscles below the elbow except the supinator longus are affected. Complete and permanent paralysis of the four extremities, or of an arm, of the diaphragm, back, neck, and intercostal muscles, is rare.

Permanent paralysis of the abdominal muscles is more common. The paralysis is flaccid, and is quickly followed by a diminution in the size of the affected parts, with atrophy of the muscles unless the paralysis quickly recedes. A reaction of degeneration soon follows. In the beginning there is increased irritability to mechanical and faradic stimulation, but a loss of reflexes to faradic stimulation of both muscle and nerve rapidly develops. The muscle responds to galvanic stimulation by a slow vermiform contraction, and the ACC> CCA. Muscles that respond to faradic stimulation usually recover either entirely or in part; whereas muscles which exhibit complete reaction of degeneration improve but little, and never wholly recover.

The deep reflexes of the involved area are absent, but may return when the paralysis recedes. If the cervical enlargement be involved, there may be spasticity of the lower extremities with increased reflexes, ankle and patellar clonus, and positive Babinski and Oppenheim phenomena. The skin reflexes are either present or absent over the involved areas. Hyperesthesia is frequently apparent at the onset; otherwise, there is generally neither sensory nor sphincter disturbance. This period may continue for several days or several weeks; as a rule, improvement follows. Death may result, however, from some intercurrent infection or from paralysis of the respiratory centre.

When the paralysis does not recede, atrophy can be seen at the end of the first week, and within six to nine months the affected part may be mere· skin and bone. In some cases the atrophy is replaced by fatty tissue. Muscles which regain their function even as late as the sixth to the ninth month show much less atrophy. The reaction of degeneration goes hand in hand with the paralysis. Improvement, which is usually marked in the first few weeks, may progress throughout the first year, after which time the paralysis is generally permanent.

Trophic disturbances of the skin, tendons, bones, and joints are common. The skin of the affected limb is cold and cyanotic; the paralyzed limb inhibited in growth. The atrophy of the long bones, as well as that of the pelvis, thorax, and spinal vertebræ may be detected by the x-rays. The tendons atrophy from disuse. When the muscles about a joint are involved, the joint becomes relaxed from overstretching of the ligaments and capsule, and results in a looseness of the joint which may go on to dislocation.

Owing to the paralysis, secondary contractures develop early in the course of the disease, but are usually not complete until about a year and a half after the onset. These contractures are due partially to a loss of tone in the paralyzed muscles, but also to a predominance of the antagonists.

If paralysis of an extremity is complete, the paralysis will be a completely flaccid one. The most frequent deformities are those of

the foot—either pes equino varus, pes valgus, or pes planus. In addition there may be a claw-hand (*main en griffe*), scoliosis, lordosis or thoracic asymmetry.

In addition to the ordinary form of poliomyelitis, the bulbospinal, which is an involvement of the lower motor segment, there are two other forms which need description:

1. The cerebral type, associated with a lesion of the upper motor segment.

2. The abortive type.

Cerebral Type.—Associated with the bulbospinal type of poliomyelitis are rare cases of spastic paraplegia with increased reflexes and no atrophy, but accompanied by tremor, acute ataxia, athetosis, and clouded mentality, due to involvement of the upper motor segment, either of the motor cortex or of the conduction paths. Both the bulbospinal and cerebral types have been observed in the same individual as well as in several members of the same family.

Abortive Type.—The abortive type of poliomyelitis was first described by Wickmann. It is characterized by the usual prodromal symptoms. Paralysis does not develop, but in some cases there is muscular weakness with a corresponding diminution of reflexes. Recovery is rapid. This is a common form of the disease, representing from 35 to 50 per cent. of all the cases. As this type is equally as contagious as the paralytic type, an accurate diagnosis is of the utmost importance; and, in addition to the symptoms, the clinical laboratory furnishes valuable assistance in establishing the diagnosis from the following points: (1) The spinal fluid shows a cellular increase with a predominance of lymphocytes and an increase of globulin; (2) the patient's blood serum has the power of neutralizing active virus, making it inactive when injected into monkeys; (3) the virus detected in the upper respiratory passages will communicate the disease to monkeys. The two latter tests are as yet more or less impracticable to those who do not have access to an experimental laboratory.

Diagnosis.—The diagnosis of poliomyelitis is usually made without difficulty from the facts that its onset is acute, that the characteristic prodromal symptoms reach their greatest intensity within a few days, and that there is a flaccid atrophic paralysis, with absence of sensory, bladder, or rectal disturbances. The diagnosis in the preparalytic stage, now of the greatest importance, is usually not difficult during epidemics; but, owing to the various aspects of the symptoms, may be less readily made at other times. For instance, forms of the disease which exhibit marked meningeal symptoms have been confounded with cerebrospinal, tubercular, or suppurative meningitis, and with meningismus from other acute infections. The characteristic findings in spinal fluid examination, in conjunction with the clinical symptoms, will usually differentiate the types.

In poliomyelitis the spinal fluid is under increased pressure, clear at the onset, becomes slightly opalescent during the preparalytic

stage, and again clears at the height of the paralysis. During the preparalytic stage there is a predominance of polymorphonuclear cells, quickly changing to a predominance of lymphocytes when paralysis appears. There is also an increase of globulin, and the fluid reduces Fehling's solution.

Such diseases as tuberculosis of the hip-joint, osteomyelitis, rickets, scurvy, syphilitic pseudoparalysis, Parrot's disease, and syphilitic epiphysitis, accompanied by fever and pain in an extremity, may at times be confounded with poliomyelitis; but careful examination will usually reveal their true nature. Occasionally poliomyelitis must be differentiated from transverse myelitis and peripheral neuritis. Transverse myelitis is rare in childhood; it is accompanied by anesthesia, sphincter involvement, decubital ulcers, and spastic paralysis, all of which are absent in poliomyelitis. Peripheral neuritis, with the exception of the diphtheritic form, is rare in childhood. Its development is slower, usually step by step, the paralysis is more symmetrical, and is accompanied by sensory disturbances.

Acute cerebral palsies are distinguished by the hypertonia present, the increased reflexes, the absence of a reaction of degeneration, convulsions, athetoid movements, and involvement of the intellect.

Fig. 79.—Patient five years, four months old; had anterior poliomyelitis at the age of twenty-three months; shows wasting and shortening of right leg.

Birth palsies with flaccid paralysis, usually involving an arm and present at birth, may at times be confounded with poliomyelitis. Myotonia congenita may be distinguished by the absence of muscular atrophy and the absence of a reaction of degeneration. Hysterical monoplegias may exhibit atrophy, but the electrical reactions are normal.

Prognosis.—The death rate of epidemic poliomyelitis, variously estimated according to age and area of involvement, is between 15 and 24 per cent. In early infancy, adolescence, and adult life the

mortality is greater than in early childhood. As the area of cord involvement becomes more extensive, and bulbar symptoms develop from involvement of the respiratory centre, the prognosis becomes more grave. In fatal cases, death usually takes place within eight days following the onset, and children should not be declared out of danger before that time. The prognosis in the various stages—the preparalytic, progressive, and retrogressive—can be made by examination of the spinal fluid, which, as has been stated, is clear at the onset, opalescent in the preparalytic stage, and clears at the height of the paralytic stage.

In regard to the paralysis, spontaneous recoveries are frequent in the mild paralytic cases, being estimated by Frauenthal and Manning at 16 per cent., and by Zappert at 13.7 per cent. In about 80 per cent. of the cases, improvement takes place up to a certain point, and then the paralysis remains stationary. Under proper treatment, however, further improvement may take place in cases which have remained stationary for a considerable period; for partial recovery, together with return of response to faradic stimulation, has been recorded in muscles paralyzed for one to one and one-half years. After this period improvement is rare because of the contractures which have occurred by this time.

Prophylaxis.—Since all evidence favors the personal communication of the disease, measures adopted to prevent the spread of contagion should be carried out along these lines. A rigid quarantine should be established, and last from four to eight weeks. Children who have been exposed should also be isolated, and kept from other children, churches, schools, and public gatherings. For a short time urotropin should be administered. As the virus is excreted in the nasopharyngeal secretions, all of these should be disinfected and prevented from drying, since desiccation does not destroy the virus. In addition to the ordinary methods, a nasal spray of 0.5 per cent. of menthol, and a mouth wash of 1 per cent. of hydrogen peroxide should be used for several days. The urine, feces, clothing, and all linens used should be carefully disinfected. The attending physician and nurse should likewise take the utmost precautions to prevent the spread of the disease.

In addition there are also chronic carriers of the disease who are healthy, which further complicates the problem of prophylaxis. Healthy carriers are those who have come into intimate contact with a patient; as, for instance, the parents.

Treatment.—During the acute stage, and immediately after the diagnosis has been made, treatment should be begun. There should be absolute rest in bed in a comfortable position. An attempt should be made to eliminate the toxins from the body; (1) by forcing the drinking of water; (2) by an initial cathartic of calomel followed by magnesia; (3) by repeated colonic irrigations and gastric lavage; (4) by hot packs, or hot air, and electric baths. For the pain hot baths should be given for fifteen minutes, at a temperature varying

between 100° and 103° F., the temperature being gradually increased several degrees. An ice-bag should be applied to the head and spine, and the temperature controlled by cold sponging. All forms of counter-irritation are strongly contraindicated, as is also the use of antipyrin and ergot which were formerly resorted to.

Urotropin, 2 grains, every three hours, may be given for four or five days, as well as quinine sulphate for its marked antiseptic properties. Frauenthal advocates the use of tincture of echinacea, 5 minims to 1 dram every six hours, as an internal antiseptic.

Pain, either spontaneous or from passive motion, is an annoying symptom in this early stage, and demands treatment. Simple methods, such as removing the weight of the bed clothes from the affected limb, splinting, hot water or hot sand-bags, an electrical pad, or wrapping the limb in cotton-wool or blankets, will usually give relief. When these methods fail, lumbar puncture is often beneficial, and sodium bromide, grains 5, may be given to young children. In older patients, aspirin, codeine, phenacetin, and, occasionally, morphine may be necessary.

After the acute stage has subsided and retrogression has set in, we should aim to preserve the nutrition and function of the paralyzed limbs and prevent contractures by the use of massage, electricity, and hydrotherapy.

Careful massage, twice daily, should be begun as soon as the pain has subsided, and passive movements at the same time instituted. As soon as voluntary motion returns in the slightest degree, active reëducation movements should be taught. Passive movements can be carried out during the giving of hot baths. Active movements should be encouraged as much as possible.

Electrical treatment should be begun early, as it is the most valuable form of treatment. Frauenthal advises the use of the sinusoidal current, alternating every second day with a combined galvanic and faradic current. He recommends that the electrodes be placed over the origin and the insertion of the muscles. In the beginning the current must be mild, and gradually increased in strength; it should be kept up indefinitely, as it prevents muscular atrophy.

Since contractures develop early in the course of the disease, they should be guarded against during the acute stage by the use of sand bags or splints loosely applied so as not to interfere with any motion or nutrition of the extremity. When no deformity exists, a supportive apparatus, such as a walking chair, should be used.

In the chronic stage with loose joints, an apparatus should be employed to fix the joints, as well as mechanical appliances for the contractures. In addition to numerous useful mechanical appliances there are certain surgical procedures which should always be considered, such as shortening and lengthening of the tendons, transplantation of tendons and muscles, insertion of artificial tendons and ligaments, joint stiffening, and, finally, nerve transplantation and nerve grafting.

PROGRESSIVE MUSCULAR ATROPHY.

Formerly two distinct groups of muscle atrophy were distinguished; namely, the spinal, or neurogenous type, and the myogenous type, or muscular dystrophy.

This simple classification sufficed but a short time, for clinicians such as Charcot, Marie, Tooth, and Hoffman observed cases which maintain an intermediary and transitional position between these two forms, exhibiting symptoms of both types. At the present time we distinguish in children the following forms:

    I. Progressive muscular atrophy.
        A. Early infantile spinal progressive muscular atrophy (Werdnig, Hoffman).
        B. Progressive muscular atrophy, adult type (Aran, Duchenne).
        C. Amyotrophic lateral sclerosis.
   II. Neural Form of Progressive Muscular Atrophy (Charcot, Marie, Tooth, Hoffman).
  III. Muscular Dystrophy.

Early Infantile Spinal Progressive Muscular Atrophy (Werdnig, Hoffman).—This type of progressive muscular atrophy was discoverd by Werdnig in 1891, and reported by Hoffman in 1893. It is essentially a family disease, several members of a family being usually affected. It is rarely hereditary, although Hoffman observed it in several generations of one family. Isolated cases have been described. It is a very rare disease, as only 30 cases have been collected from the literature.

Pathological Anatomy.—There is degeneration of the anterior horn cells, motor roots, and nerves, with resulting marked atrophy of muscles; sometimes there is secondary fatty degeneration of muscle fibers,—*lipomatosis interstitialis.*

Prognosis.—The course is a rapid and progressive one, death usually taking place within one to four years either from paralysis of respiration or some intercurrent infection.

Symptomatology.—The disease is one of early childhood, usually appearing within the first year of life, and affecting hitherto healthy children. It is characterized by progressive weakness, which affects both sides symmetrically, first appearing in the proximal group of muscles. The muscles about the pelvis and thigh, the glutei, iliopsoas, and quadriceps femoris, are first involved, then it extends upward and involves the muscles of the back, neck, shoulder-girdle, and upper arm—the deltoid, serrati, rhomboids, supraspinalis and infraspinatus, the biceps and triceps. Finally, as the disease progresses, the muscles of the forearm, the legs, and the smaller muscles of the hands and feet are attacked. The muscles of the calf are affected late in the disease. Even in well advanced cases the distal muscles of the extremities are but little involved.

Paralysis and atrophy of the affected muscles quickly follow.

Atrophy about the calves and glutei is frequently masked by an excessive deposition of fat between the degenerated muscle fibers, the so-called pseudohypertrophy. Deformities of the body, hands, and feet are observed, and extreme scoliosis may be present. There is talipes equinus varus; the fingers and toes may be claw-shaped (*main en griffe*). Fibrillary twitchings are variable. There are no sensory disturbances. The deep reflexes are absent, the plantar response is normal. Electrical changes—that is, either partial or complete reaction of degeneration—are demonstrable. The cranial nerves usually escape. All organs of sense, speech development, intelligence, and sphincter control, remain unaffected.

Fig. 80.—Progressive muscular atrophy (*main en griffe*).

Diagnosis.—In cases exhibiting the characteristic symptoms and course, the diagnosis is easy. The disease may sometimes be confounded with the neural form of muscular atrophies, the myopathies, poliomyelitis, and myotonia congenita (Oppenheim).

Treatment.—No known treatment influences the course of the disease.

Progressive Muscular Atrophy, Adult Form (Aran, Duchenne).—This form rarely occurs except in older children, but when observed it is the same as in the adult. The feature which most clearly distinguishes it from the usual form in children is that, in this adult type, heredity plays no role, also that the disease usually begins in the distal portion of the extremities about the small muscles of the hands and feet.

Amyotrophic Lateral Sclerosis (Charcot).—This form rarely occurs in children, and its existence was even denied until Keehn and Nalf demonstrated it pathologically. It is a hereditary family disease, but isolated cases have been observed. The age of onset varies from early childhood to puberty.

Pathological Anatomy.—Among the findings is sclerosis of the lateral tracts of the cord, involving the direct pyramidal and Gower's tracts, together with atrophy of the anterior horn cells and the nuclei of the medulla.

Symptomatology.—The disease sets in with weakness in the muscles of the extremities, and difficulty in walking, the legs being stiff, and the child compelled to walk upon its toes. Other cases begin with bulbar symptoms—difficulty in chewing, swallowing, and speaking. Contractures of the arms and legs develop early. The deep reflexes are greatly exaggerated. Oppenheim and Babinski phenomena and both ankle and patellar clonus are present. At the same time there is atrophy of the small muscles of the hands. The reaction of degeneration is obtained in the affected muscles, and there are fibrillary twitchings, but no sensory, sphincter, or pupillary changes. Mental development is frequently defective.

Course.—The course of the disease is usually rapid and progressive; although it may remain stationary for a time, it is always fatal.

Diagnosis.—Progressive muscular atrophy in the adult must be differentiated from spastic paraplegia and diphtheritic paralysis. The combination of flaccid and spastic symptoms tends to clear the diagnosis.

Treatment.—Any treatment is unsatisfactory. Systematic massage and warm baths should be tried. Electricity benefits but little.

THE NEURAL FORM OF PROGRESSIVE MUSCULAR ATROPHY, PERONEAL TYPE.

(Charcot, Marie, Tooth, Hoffman.)

This type was described by Eulenburg in 1856, by Charcot and Marie in 1886, and in the same year by Tooth. Later, in 1889, it was also described by Hoffman.

Etiology.—It is essentially a hereditary family disease, observed in some cases through four or five successive generations. Boys are affected more frequently than girls. The age of onset usually extends from early childhood to adolescence.

Pathological Anatomy.—There are spinal and peripheral changes. There are degenerative changes in the posterior columns of the cord, in some of the anterior horn cells, in the anterior and posterior roots, and in the peripheral nerves.

Symptomatology.—In the neural form of progressive muscular atrophy the involvement of the distal portion of the extremities is characteristic. Muscular weakness and atrophy usually begin symmetrically in the peroneal group of muscles and the smaller muscles of the feet, causing a foot-drop in walking, the so-called characteristic "steppage gait." The muscles of the calf are as yet well preserved. The disease may remain stationary for from one to four years, and then may extend to the upper extremities, involving the smaller

muscles of the hands, the extensors of the forearm, and the extensors of the fingers and hand. In some cases the disease may spread out over the proximal portions of the extremities, involving the thigh, pelvic girdle, the fore and upper arms, shoulder-girdle and back.

Pseudomuscular hypertrophies are not present. Fibrillary twitchings are frequently observed. The Achilles reflex is always absent and the other deep reflexes are either diminished or abolished, depending upon the amount of atrophy. Sensory disturbances, such as pain, hyperesthesia, anesthesia, analgesia, and vasomotor disturbances, are common. Complete or partial reaction of degeneration is present. Diminished excitability may also be observed in muscles apparently normal. Sphincter disturbances do not occur. Secondary contractures are common. Club-foot (pes varus), or equinovarus, and clawed hands and feet are common.

Prognosis.—The disease is a slowly progressive one, extending over a number of years. It may be arrested at different stages, but exacerbations occur. Death usually is due to some intercurrent infection.

Diagnosis.—This form must be differentiated from the other forms of progressive muscular atrophy and dystrophy, and from multiple neuritis; but, when characteristically developed, the diagnosis is usually not difficult.

Treatment.—The treatment is only symptomatic. General hygienic and dietetic measures, massage, and galvanism should be employed. For the contractures, tenotomy and orthopedic appliances are necessary.

PROGRESSIVE MUSCULAR DYSTROPHY.

A characteristic feature of the myopathies and the dystrophies is that anatomically the primary affection appears in the muscles, and that the spinal cord and the peripheral nerves escape. Clinically we distinguish four different types of dystrophy, as demonstrated by Erb, but they cannot always be clearly differentiated. They are as follows:

    I. Pseudohypertrophic type (Duchenne).
    II. Juvenile type: Scapulohumeral (Erb).
    III. Infantile type: Facies-scapulohumeral (Landouzy, Déjerine).
    IV. Simple atrophic type: Hereditary (Erb, Leyden, Moebius).

Etiology.—This is a true, endogenous, hereditary, family disease, which may occur in several members of the same family throughout several generations. It is usually transmitted through the mother, she, herself, being rarely affected. Trauma, infections, and inanition may be accessory etiological factors. The age of onset varies considerably in the different varieties; it may occur at any time from early infancy to puberty, or may be delayed until adolescence has set in.

Pseudohypertrophic Type (Duchenne).—The usual age of onset is between two and seven years, and the disease develops slowly. The child may be late in learning to walk. At first there is awkwardness

in gait, also muscular weakness which is shown by the ease with which the child falls, and by the difficulty in rising. This type is characterized by atrophy and hypertrophy of the proximal group of muscles, with a predilection for certain groups; thus, the long muscles of the back or the abdominal muscles may be involved first. The muscles about the pelvic girdle and thigh, the glutei and anterior muscles of the thigh, the adductors, and then the flexor cruris, are involved. Later the muscles of the calf, the dorsal flexors of the foot, and, as
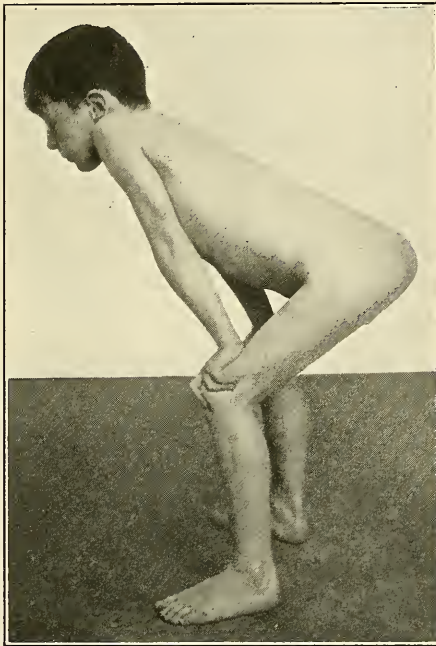
FIG. 81.—Pseudohypertrophic muscular paralysis. Boy is climbing up upon his legs.

the disease progresses, the muscles about the shoulder-girdle and upper arm become affected. There is also a tendency to hypertrophy of certain muscles; thus, in the lower extremity the gluteus, sartorius, and muscles of the calf; in the upper extremity the deltoid, infraspinatus, supraspinatus, and, sometimes, the triceps show beginning hypertrophy. The distal portion of the extremities is usually not involved. All this forms a characteristic picture.

Paralysis of the muscles of the back produces lordosis with prominent and protruding abdomen, and compels the child to throw its shoulders back and also to hold the head and trunk erect in order to maintain its equilibrium. Thus, a plumb-line, dropped from the scapula, falls behind the buttocks. The child's power of locomotion is greatly impaired.

There is a characteristic waddling gait, like that of a goose, or of a child suffering from congenital hip disease. The trunk is swayed

from side to side in order to get the centre of gravity over the hip-joints, and, also, because of the difficulty in raising the thighs.

The manner of rising from the floor is almost pathognomonic. The child first rolls over on his face and abdomen, draws his legs up under him, and extends his legs on the thighs. He then brings his hands near the feet, and, in order to elevate the trunk, he climbs up upon his legs with his hands; finally, by giving his head a swing, the

FIG. 82.—Pseudohypertrophic muscular paralysis, showing lordosis in the erect posture.

shoulders are thrown back, and his equilibrium is attained. In addition, there is difficulty in mounting stairs.

Another characteristic sign is the loose-winged scapula. For instance, if one attempts to raise the child after pressing his shoulders against his sides he "falls through" to his ears.

Certain contractures appear. There is a pes equinus, from contraction of the muscles of the calf. Contractures of the elbow- and knee-joints also develop. The mentality is, as a rule, normally developed,

although a number of cases have been reported which were associated with idiocy and epilepsy. Fibrillary twitchings and the reaction of degeneration rarely occur. The deep reflexes are present, and the plantar response and sensation are normal. There are no sphincter disturbances.

Juvenile Type, Scapulohumeral (Erb).—The type described by Erb begins late in childhood, and is characterized by atrophy, beginning about the shoulder-girdle. Some muscles are hypertrophied, while others are atrophied. Later, the muscles of the upper arm, then those of the back (with resulting lordosis), finally those about the pelvic girdle, the thigh and calf, and peronii muscles (with resulting talipes equinus), are involved.

Infantile Type, Facies-scapulohumeral (Landouzy, Déjerine).—This form begins within the first few months of life, affecting male and female babies equally, and involving the muscles of the face, especially the sphincters of the eyes and mouth. There follows a lack of facial expression—a mask-like appearance, with inability to raise the eyebrows, close the eyes, or puff out the cheeks. At the same time there may be involvement of the shoulder-girdle, or this may be the only symptom of the disease for years, after which the process may extend to the shoulders, thighs, and pelvis.

Simple Atrophic Type, Hereditary (Erb, Leyden, Moebius).—This form exhibits an unusual hereditary character, with no muscular hypertrophy.

These different forms are mere varieties of the disease, and all possible combinations of them may exist in the same individual.

Pathological Anatomy.—The pathological changes are similar in all the forms. The changes are essentially primary in the muscles, for none occur in the central nervous system. There is atrophy of most of the muscle fibers, with fatty degeneration, fatty and connective-tissue infiltration between the individual muscle fibers, and true hypertrophy of other muscle cells. On microscopic section, the striations of the muscle fibers have disappeared, the fibers are of different sizes, some large and other small, and they show fatty degeneration, vacuolation, and a contraction of the muscle fibers. Outside the sarcolemma sheath, there is deposited fat and connective tissue, showing numerous connective-tissue nuclei. The fat later becomes absorbed, and the connective tissue contracts, the atrophied muscle being finally replaced by contracted fibrous tissue. There is also true hypertrophy of other muscle fibers.

Diagnosis.—When fully developed, the diagnosis of this disease usually presents no great difficulty. It must be differentiated, however, from the different forms of muscular atrophy. As these two types are the antitheses of one another, it must be borne in mind that there are transitional forms which exhibit in part neurogenic, in part myogenic symptoms. At times, syringomyelia, diphtheritic paralysis, and beginning spondylitis may be confounded with progressive muscular dystrophy.

51

Prognosis.—The course of the disease is slow but progressive, extending over a number of years before the patient becomes bed-ridden unless death supervenes as the result of some intercurrent infection. The disease may remain stationary for a number of years; even recoveries from the juvenile type have been reported by Erb. The most unfavorable forms are those which begin in early childhood and are of the pseudohypertrophic type.

Treatment.—No treatment is effectual. Organic extracts of the thyroid, thymus, and pituitary glands have been tried, also injections of fibrolysin. Hygienic measures, such as fresh air, nourishing food, and gymnastic exercises, with moderate massage, and electricity, especially galvanism, to both affected and unaffected muscles, should be prescribed. Tenotomy and tendon transplantation may be necessary for the contractures, and orthopedic appliances to give support to the partially degenerated muscles.

MYOTONIA CONGENITA (THOMSEN'S DISEASE).

This rare disease is hereditary, occurs in several members of the same family, and is usually congenital. Boys are more frequently affected than girls.

Pathological Anatomy.—Schiefferdecker demonstrated hypertrophy of the muscle fibers, an increase in nuclei arranged in rows, and granulation of the sarcoplasm.

Symptomatology.—The chief symptom of the disease is a slow muscular contraction or myotonic spasm when voluntary movements are attempted. This tonic spasm may last from five seconds to half a minute and then relax, allowing the normal movements to be carried out. Different groups of muscles may be thus affected; but the muscles most frequently involved are those of the extremities, the legs more often than the arms. Next in order of frequency are the trunk muscles, finally those innervated by the cranial nerves, the sphincters of the eye, the masticators, the esophageal, and laryngeal muscles. Hence the infant may have difficulty when it attempts to nurse, older children on attempting to walk, the patient's feet being glued to the ground, also on shaking hands, the child not being able to let go until relaxation is complete. Fright and cold increase the spasm, whereas heat and alcohol relax it.

The disease is not apt to be recognized in early infancy and childhood, except in familial cases. The general appearance of the patient indicates a good athletic physique, although the strength is subnormal.

On examination we find two characteristic signs:

I. The muscles show an increased irritability to mechanical stimuli. At the point of stimulation there appears a localized swelling, due to muscular contractions, persisting from ten to fourteen seconds, and then gradually relaxing.

II. There is also increased irritability of the muscles to electrical stimuli. Mild faradic currents induce a muscle spasm, which persists

for a number of seconds after the current is cut off. Direct galvanic currents give rise to a reaction similar to the reaction of degeneration. This is Erb's myotonic reaction. There are no sensory or sphincter disturbances.

Diagnosis.—The condition should be differentiated from tetany.

Prognosis.—The affection is not fatal, but persists through life.

Treatment.—Massage and gymnastic exercises should be prescribed. Organic extracts have been tried, but without results.

MYOTONIA CONGENITA (OPPENHEIM).

This disease was described by Oppenheim in 1900. As its name implies, it is congenital, and is usually observed in the first few days of life. It is characterized by a congenital muscular weakness, usually a bilateral symmetrical flaccidity, which affects most frequently the lower extremities, less often the upper. The children lie motionless, unable to move their legs. Hypotonus is extreme, and the limbs will remain in any position in which they are placed. The neck, diaphragm, and facial muscles usually escape. There is no muscular atrophy. The deep reflexes—the knee-jerk and Achilles reflex— are greatly diminished or absent. The electrical irritability of the muscles is also diminished or absent, but the reaction of degeneration is not observed. There are no sensory or sphincter disturbances. The mentality is normally developed.

Pathological Anatomy.—Partial degenerative changes in the anterior horn cells and muscle fibers have been described, but these pathological changes are by no means uniform. Rothmann considers the disease a form of the early infantile type of muscular atrophy (Werdnig, Hoffman).

Prognosis and Treatment.—Recovery is possible, but the course is slow, and the majority of the children affected die from some intercurrent infection. Systematic massage and electrical treatment should be instituted, and the organic extracts, especially thyroidin, given a trial.

SYRINGOMYELIA.

Up to the age of puberty this disease is exceedingly rare, even its existence having been disputed. It is closely related to congenital malformations of the central nervous system, and has been described as accompanying hydrocephalus, spina bifida, meningocele, syringomyelocele, and reduplication of the cord. In some children the affection has an hereditary character.

Pathological Anatomy.—A cavity forms in the cord, being more frequently due to the disintegration of a previously formed mass of gliomatous cells which had infiltrated the normal tissue. This infiltration begins near the central canal and, as degeneration progresses, the cavity invades the surrounding spinal marrow, the posterior horns, posterior columns, and the anterior horns. The cavity is usually irregular in outline and varies in size.

Symptomatology.—The symptoms of this disease are essentially the same as in the adult. There is progressive muscular atrophy of the upper extremities from involvement of the anterior horn cells, and an accompanying spastic paraplegia of the lower limbs from involvement of the pyramidal tract.

The sensory disturbances are very characteristic. There is loss of pain sense and temperature sense on the same side as the lesion, while muscle sense and the sense of touch may be preserved. In addition, there are vasomotor and trophic disturbances of the skin and nails, hyperemia, anemia, bullous eruptions, ulcerations, abscesses, especially upon the fingers (Mowan's type), and arthropathies. Secondary contractures, claw-hand (*main en griffe*), and kyphoscoliosis are also observed. The symptoms naturally depend upon the position and extent of the lesion. Bulbar symptoms may be present.

Prognosis and Treatment.—The course of the disease is slow but progressive, and is uninfluenced by any known treatment.

HEREDITARY ATAXIA (FRIEDREICH'S ATAXIA)—HEREDITARY CEREBELLAR ATAXIA (MARIE).

We distinguish two distinct types of hereditary ataxia: (1) the classical spinal type, described by Friedreich in 1861; and (2) the cerebellar type described by Marie in 1893, known as hereditary cerebellar ataxia. Today these two types are considered to be merely two forms of the same disease, although clinically and anatomically they may appear as distinct entities; there are also mixed and transitional forms which clearly demonstrate their relationship.

Etiology.—Hereditary ataxia is a family disease, often affecting several members of a family, and the two sexes equally; it has also been observed in successive generations of a family. Sporadic cases, however, occur quite frequently. It appears early in life, most commonly between four and seven years of age, although it may begin much later, even at thirty. The cerebellar type (Marie) usually sets in later in life, at about twenty years of age, yet may begin in early childhood. There is a history of alcoholism, syphilis, epilepsy, or insanity in the parents. Not infrequently it follows certain infectious diseases.

Pathological Anatomy.—Either the spinal cord or cerebellum, or both, depending upon the form, whether spinal, cerebellar, or mixed, are diminished in size and show degenerative changes.

Of the degenerative changes in the cord there is sclerosis, which involves: (1) The posterior columns—the column of Goll completely, and the column of Burdoch only partially; (2) the lateral columns—degeneration of the ganglion cells of Clark's column, of the direct cerebellar tract, of Gowers's tract, and the crossed pyramidal tract in the lumbar region; (3) at times the anterior columns of the cord. In the cerebellum there are degenerative changes in the Perkinje cells of the cortex and of the cerebellar nuclei.

Spinal Form: **Symptoms.**—The most characteristic symptom is locomotor ataxia, beginning in the lower extremities. The child walks with its legs widely separated, taking small steps, and swaying to-and-fro like a drunken man. Ataxia of the upper extremities sets in later, frequently associated with a simple or an intention tremor which involves the entire body, especially the head and shoulders, causing a wobbling of the head and shoulders or whole body; in addition, there are at times choreic or athetoid movements. Static ataxia is frequently present; *i. e.*, ataxia of the body when standing erect or on extending a limb. A true Romberg sign does not usually appear.

Sensory disturbances are generally lacking. The deep reflexes are diminished or abolished, although the Babinski phenomenon may often be present. The other skin reflexes are normal. The sphincters usually escape.

Nystagmus is a characteristic symptom. The pupillary reactions are normal, and optic nerve atrophy and ocular palsies rarely occur. Speech disturbances are common, the speech being slow, awkward, and at times scanning.

Deformities are the rule. Scoliosis is frequently observed. The deformity of the foot is characteristic. There is talipes equinovarus, with the dorsum bowed, the big toe flexed dorsally, and the remaining toes assuming a claw-like appearance. A claw-hand (*main en griffe*) is also observed at times. These contractures are secondary to muscular atrophy. Trophic changes do not occur. The intelligence is normal.

The cerebellar type (Marie) differs in that it usually begins after puberty. The ataxia is of the cerebellar type. Optic nerve atrophy and ocular palsies are quite frequent, and the deep reflexes are exaggerated. Sensory disturbances are common. Between these two types numerous transitional forms have been described, with one or the other type predominating.

Differential Diagnosis.—The disease must be differentiated from multiple sclerosis, cerebellar tumor, juvenile tabes, and cerebral syphilis. Multiple sclerosis is very rare in early childhood, and in this affection nystagmus and intention tremor are generally more marked. From cerebellar tumor it can be distinguished by the absence of the general symptoms of brain tumor—headache, vomiting, optic neuritis—and by its more chronic course. From multiple neuritis it can be differentiated by the history. Juvenile tabes is very rare. It is characterized by lightning pains, headache, crises, pupillary changes, optic atrophy, and bladder disturbances. From cerebral syphilis or juvenile tabes Friedreich's ataxia is distinguished by the absence of a positive Wassermann reaction, both in the blood serum and spinal fluid, and by an entirely negative spinal fluid.

Prognosis.—The course of the disease is slow and progressive, but several years may elapse before the patient is bed-ridden. It may be arrested for a time, and may extend over a period of thirty years,

but is incurable. Death usually results from some intercurrent affection.

Treatment.—No drugs have any influence upon the course of the disease. These patients should have plenty of fresh air, sunshine, and good nourishing food. Massage and Frenkel's reëducation movements should be employed. When the patient is bed-ridden, care must be taken to prevent contractures.

TUMORS OF THE SPINAL CORD.

Tumors of the cord are rare in childhood, but two main types may be distinguished:

1. Extramedullary tumors, arising from the spinal meninges, of which we may further differentiate two varieties—the extradural and intradural.

2. Intramedullary tumors which form within the spinal marrow.

Extramedullary tumors are the more common, and among these are sarcoma, myxosarcoma, angiosarcoma, endothelioma, syphiloma, miliary tubercle, fibroma, mixed tumors, and, in infancy especially, associated with congenital malformations of the cord, also lipoma and teratoma. Of the intramedullary variety, there are solitary tubercles, gummata, sarcomata, and gliomata. Until ten years of age the tubercle is the most common intramedullary tumor, whereas lipoma and sarcoma are the most frequent extramedullary tumors.

Symptomatology.—This depends upon the nature of the tumor, whether intramedullary or extramedullary, also upon its position. The symptoms complained of result from pressure upon the spinal roots, spinal marrow, and vertebral column. The first is intense pain of neuralgic, lancinating character, due to irritation of the posterior roots. The location of the pain depends entirely upon the position of the tumor. Hyperesthesia and certain trophic disturbances, such as herpes zoster, may at times be present. These may be the only symptoms, but may extend over several years. In some cases the tumor involves the motor roots, and causes irritative symptoms—fibrillary contractions, or even true muscle spasms.

As a result of compression of the cord there is paralysis of both the sensory and motor nerves, which arise from the segments of the cord in which the tumor lies. There is also flaccid paralysis, muscular atrophy, the reaction of degeneration, loss of reflexes, and anesthesia as regards touch and pain.

Indirect spinal symptoms due to interference of the pyramidal tract give rise to spastic hemiplegia or paraplegia, according to the extent of the involvement—whether unilateral or bilateral—also marked spasticity, muscular weakness, exaggerated reflexes, the Babinski and Oppenheim phenomena, and trophic and sphincter disturbances. At the onset the lesion is usually unilateral, and frequently produces the typical Brown-Séquard syndrome. On the side of the tumor there is (1) paralysis which is flaccid at the level of the lesion, and spastic

below; (2) disturbance of the muscle sense while other senses remain normal, except for a hyperesthetic zone above the paralysis at the level of the tumor. On the opposite side there is either partial or complete anesthesia to pain and temperature.

Unilateral tumors very quickly become bilateral, when this classical syndrome will change to that of a transverse myelitis. The symptoms naturally depend upon the position of the tumor. Tumors in the cervical region give rise to a spastic paralysis of all the extremities, except that there is a flaccid paralysis of certain groups of muscles of the arm. If the upper portion of the arm be involved, the phrenic is often affected; if the lower portion, the forearm and hand exhibit a flaccid paralysis. Ocular symptoms may also appear. In the dorsal region the arm remains free with spasticity of the lower extremities, together with flaccid paralysis of the abdominal muscles, and loss of the abdominal reflexes. If located in the lumbar region there is general flaccid paralysis of the lower extremities. The most frequent location for a tumor in children is in the cauda equina. In these cases pain in the sacral region is very severe, and extends into the legs. The paralysis is a flaccid one, and bladder, rectal, and trophic disturbances are usually present.

As a result of pressure upon the vertebral column and erosion of the vertebræ by extramedullary tumors, one occasionally observes scoliosis and kyphosis. Pain on percussion of the vertebræ is frequently present. Oppenheim called attention to an impairment of the percussion note over the tumor.

Diagnosis.—Intramedullary tumors, owing to their unfavorable progress, must be differentiated from those of extramedullary type which are more amenable to treatment. The following table gives the chief points in the differential diagnosis; yet it must be borne in mind that the differentiation is usually not easy, and frequently is impossible:

Intramedullary Tumors.	Extramedullary Tumors.
Root symptoms usually absent.	Root symptoms usually present.
No pain on vertebral percussion.	Pain on vertebral percussion.
Become bilateral quickly.	Remain unilateral for a longer period: Brown-Séquard syndrome.
Remissions frequent.	Remissions rare.

The rapidity with which the intrameningeal tumor becomes bilateral tends to mask the real disease and obscure the diagnosis. If the anterior horn cells be involved, progressive muscular atrophy may be suspected; if both the anterior and posterior horn cells, syringomyelia is simulated; if of diffuse character, then transverse myelitis may be thought of. In addition, spinal syphilis, multiple sclerosis, and compression myelitis must frequently be differentiated from tumor.

Course and Prognosis.—The course of the disease is usually slow, extending over a period of ten to twelve years, and unless operated

upon, terminating fatally as a result of the extensive paralysis or by some intercurrent infection.

Treatment.—If extradural, the tumor should be removed surgically. Tumors associated with spina bifida, and brain, metastatic and extradural tumors are not amenable to operation. When syphilis is suspected, antisyphilitic treatment should be begun immediately.

DISEASES OF THE MENINGES.

PACHYMENINGITIS.

Pachymeningitis is an inflammation of the mucous membrane which lines the dura, and is a rare disease in children. We distinguish two forms of the affection: (1) Pachymeningitis externa; and (2) pachymeningitis interna.

Pachymeningitis Externa.—This may appear either in a chronic fibrous form, usually associated with a chronic inflammation of the pia arachnoid (leptomeningitis and meningo-encephalitis), or be secondary to lesion of the contiguous bony structures, such as fracture of the skull, or tubercular or syphilitic periostitis. Or it may be acute, and secondary to inflammation of the neighboring bony structures, such as otitis media, mastoiditis, caries of the middle ear, purulent rhinitis, or inflammation of the sinuses, especially the frontal; it may lead to an extradural abscess between the dura mater and bony structures.

Symptoms.—In many mild cases there are no characteristic symptoms; whereas, in the more severe type there may be violent headache with localized pain on pressure and percussion. In the most aggravated cases symptoms of intracranial pressure, *i. e.*, headache, projectile vomiting, slow pulse, stupor, and choked disk may appear. Focal symptoms are usually absent.

Diagnosis.—Pachymeningitis must be differentiated from the other complications of otitis media; namely, purulent sinus, phlebitis, brain abscess, and purulent leptomeningitis.

Treatment.—The treatment is purely surgical.

Pachymeningitis Interna.—Two forms of pachymeningitis interna are distinguishable: (1) Purulent; (2) hemorrhagic.

Purulent Pachymeningitis Interna.—The purulent type usually results from perforation in pachymeningitis externa, and is associated with inflammation of the pia mater (leptomeningitis).

Pachymeningitis Interna Hemorrhagica.—Rosenberg, of Finkelstein's Clinic, by his studies of pachymeningitis interna hemorrhagica, has extended our knowledge of the disease. It is by no means as infrequent as was formerly supposed; for Rosenberg collected 48 cases from this Clinic within four years. The disease can now be diagnosed, is amenable to treatment, and therefore of greater interest.

Etiology.—Pachymeningitis interna hemorrhagica is usually secondary to gastro-intestinal disturbances, malnutrition, lues, rickets, scurvy, or infectious diseases, especially to smallpox, typhoid fever, and, occasionally, to pneumonia and tuberculosis. Hemorrhagic rhinitis, both luetic and diphtheritic, occurring from two to four months before, has frequently been found by Rosenberg to be an etiological factor. Children cared for in hospitals and institutions are more susceptible on account of their greater exposure to infection.

Pathological Anatomy.—The lesions are bilateral, and are on the convexity of the brain, in the region of the anterior and middle fossæ, and over the region of the middle meningeal artery and superior longitudinal sinus.

In acute cases extravasations are observed upon the inner surface of the dura, enclosed in a fine fibrinous connective-tissue membrane containing leukocytes. The membrane later becomes translucent. It consists of fibrous lamellæ containing fibroblasts and newly formed vessels from the dura. The vessel walls are thin, being lined simply with endothelium. Hemorrhage takes place by diapedesis or by capillary bleeding, as a result of which hematoma are formed. Rosenberg attributes the changes to thrombosis of the sinus cavernosus.

Symptomatology.—The clinical picture is essentially the same as that seen in leptomeningitis, except that fever is usually lacking. Rosenberg distinguishes three forms of the disease, grouped as follows:

1. The latent form, which may show merely enlargement of the cranium, open and distended fontanelles, and a widening of the sutures. These may be the only symptoms.

2. This variety sets in with symptoms of acute cerebral pressure, giving rise to vomiting, headache, convulsions, rigidity of the neck, stupor, and increased reflexes.

3. A rapid form with severe symptoms arising in the nervous system, and simulating acute leptomeningitis. There are contractures of the extremities, rigidity of the neck, fever, coma, convulsions, strabismus, retinal hemorrhages, choked disk, and a positive Kernig's sign.

Diagnosis.—Clinically, the disease must be differentiated from sinus thrombosis, leptomeningitis, and brain tumor. An invaluable aid to the diagnosis is a study of the cerebrospinal fluid obtained by lumbar puncture. It is usually clear, and under increased pressure. When a communication exists with the subarachnoid space, owing to a torn pia, the fluid will be either hemorrhagic or of a lemon-yellow tint. When hemorrhage due to faulty technic can be excluded, these findings are pathognomonic.

A more reliable method of diagnosis is to puncture the large fontanelle, and withdraw some cerebrospinal fluid. (For the technic of cerebral puncture see page 778.) The fluid obtained is always hemorrhagic, but does not clot upon standing. After the cells have settled, it is seen to be of a light lemon color. Examination of the fluid shows no pathological cellular increase; there is, however, an increase in globulin, and a normal reduction of Fehling's solution.

Clinical Course and Prognosis.—Under appropriate treatment the prognosis is more favorable than was formerly supposed. In Rosenberg's series of cases there were 16 recoveries and 21 deaths; only 3 of these deaths could be attributed to the disease, the remainder being due to intercurrent infections. Healing follows the absorption of the fluid, after which the fontanelles close, the cranium again assumes its normal shape and size, and there is a corresponding amelioration of all the other symptoms. In untreated cases, however, death takes place within a few weeks.

Treatment.—There should be absolute rest in bed, and the nutrition of the child should be carefully watched. An ice-bag should be applied to the head, and lumbar punctures made repeatedly, removing from 50 to 100 c.c. of fluid at each puncture. Should improvement not follow, cranial aspiration should then be performed. After the removal of fluid, hemorrhages are likely to recur, and horse serum or human blood serum should be injected intramuscularly or intravenously as a prophylactic. If bleeding is due to syphilitic infection, appropriate antisyphilitic treatment must be immediately begun.

ACUTE SUPPURATIVE MENINGITIS.

Under this heading may be grouped all forms of meningitis except the tuberculous and cerebrospinal, which are considered elsewhere in this work. Acute suppurative meningitis is also called acute simple meningitis, acute purulent meningitis, and vertical meningitis, although it cannot be considered as a distinct disease, but should be regarded as a pathological condition due to infection by any one of a number of microörganisms.

It is customary to designate the forms of meningitis in this group according to the causative organism; e. g., influenzal meningitis, streptococcic meningitis, and pneumococcic meningitis. Clinically, these forms of the disease so closely resemble each other that the grouping of all the various types seems justified.

Etiology.—Acute purulent meningitis may be either primary or secondary, the primary form being most frequently of pneumococcic or influenzal origin, while secondary meningitis is generally caused by the staphylococcus. Meningitis may, however, arise from any acute systemic infection with bacteremia.

Among the rare causes of acute purulent meningitis are trauma and insolation. Meningitis may also develop from the extension of a suppurative process in the scalp, cranial bones, nasal cavities, the ear, the orbit, and the face, from the rupture of brain abscesses, or may be produced by more distant lesions, the infection being carried by the blood or the lymph stream.

The meninges are sometimes the site of infection by the colon bacillus, and meningitis may also accompany scarlet fever, measles, diphtheria, smallpox, rheumatic fever, typhoid fever, erysipelas,

pneumonia and influenza, and may occur as a terminal infection in many chronic infectious diseases.

The bacillus pyocyaneus and, more rarely, the gonococcus are isolated in some cases, while in others there may be more than one variety of organism, the process being due to mixed infection. Children of all ages are attacked; as a rule, the previous state of health seems to have no influence on the occurrence of this meningeal inflammation. Most of the cases occur sporadically, and are probably more frequent during the late winter and spring months than at other seasons of the year.

Pathology.—The dura mater, the pia mater, or both may be affected; usually the pia mater is most extensively involved, the inflammatory process being a general one; but it may also be more or less localized to a limited area. Thus, the spinal cord, the cortex of the brain, the ventricles of the brain, or all of these may be the site of inflammation; or, as in the cases where there is infection of the ear, for example, the lesions may be unilateral. The vessels of the pia are congested, minute hemorrhages take place, and serofibrinous or purulent fluid bathes the parts. The convolutions may be flattened, and the pia arachnoid, or even the ventricles, may be quite distended. As a rule there is associated involvement of the spinal cord.

When the inflammatory process is confined to, or is most extensive over, the vertex, it is sometimes termed vertical meningitis. In pneumococic meningitis the bulk of the exudate and many adhesions are found in the upper portion of the brain. Not uncommonly, the anterior half of the brain may be encased in pus. Microscopic examination of the affected tissue reveals marked congestion of the bloodvessels and round-cell infiltration in the inflammatory areas.

Symptoms.—In the majority of cases of acute purulent meningitis the onset is sudden and the symptoms violent; although in some instances the first symptoms of meningitis may be masked to a considerable extent by the primary disease, especially if this be erysipelas, septicemia, or pneumonia. Drowsiness and stupor, with irregular respirations and pulse, may be the first indications of meningitis. In primary cases, due to the pneumococcus and influenza bacillus, vomiting and convulsions usually usher in the attack. As a rule there is an initial chill, followed by a sudden rise in temperature, severe headache, increasing delirium, photophobia, rigidity and retraction of the neck, aimless movements of the legs and arms, general signs of irritation, dilatation of the pupils, and finally coma. A bulging of the anterior fontanelle is often apparent in young infants, and Kernig's sign is present in all cases.

Diagnosis.—As a rule, the diagnosis of acute purulent meningitis can readily be made from the symptoms; but in order to determine the particular type with which we are dealing an accurate history must be taken, the symptoms thoroughly investigated, and a bacteriological examination of the cerebrospinal fluid made. Although the symptoms of the various forms of meningitis may be somewhat dis-

similar, and while we may suspect from the history of the case that a certain type of the disease is present, neither of these circumstances is of any practical value, and the only conclusive evidence of a certain infection is the finding of the specific organism within the spinal fluid.

Pneumococcic Meningitis.—Of the various forms of meningeal inflammation grouped under the heading of acute suppurative meningitis, this is the one most frequently met with in young children. It is practically always associated with general pneumococcic infection and pneumococcemia, and in most cases there is a demonstrable lesion in the bronchi, lungs, pleuræ, pericardium, or peritoneum. Although in occasional cases no pneumococcic lesion is evident, yet at postmortem the pneumococcus can invariably be isolated from the heart's blood.

The meninges usually become involved during the height of an attack of pneumonia, but meningitis occasionally precedes or follows pulmonary consolidation.

As a rule, in any series of cases the majority will be found to occur in infants under one year of age. At autopsy the gross changes resemble those of cerebrospinal meningitis, but there is little, if any, involvement of the cord. The fibrinous and purulent exudation is more profuse than in any other type of meningeal inflammation.

Symptoms.—The symptoms closely resemble those of cerebrospinal meningitis, but in many cases there is entire absence of rigidity, cervical opisthotonos, Kernig's sign, and hyperesthesia, while the pulse and respirations may not be appreciably altered. The onset is sudden, the course of the disease is short and violent, being, as a rule, from two to eight days, rarely longer. A positive diagnosis can be made only by examination of the cerebrospinal fluid, which is cloudy, under increased pressure, and contains an excess of polynuclear leukocytes and many pneumococci grouped in short chains.

Septic Meningitis—Streptococcic Meningitis—Staphylococcic Meningitis.—This form of meningitis is usually secondary, and is a complication of septicemia, otitis, mastoiditis, trauma, erysipelas of the scalp, sinus thrombosis, infection of the umbilicus, or of spina bifida. The symptoms are not quite as severe as in pneumococcic meningitis; but the disease is usually fatal, especially the streptococcic type, which is more severe than that due to the Staphylococcus pyogenes.

Examination of the brain reveals diffuse inflammation of the pia and a profuse purulent exudate, but not the excess of fibrin so characteristic of pneumococcic meningitis. The cerebrospinal fluid is turbid, is under increased pressure, and contains numerous pus cells and streptococci or staphylococci, the streptococci being usually grouped in long chains.

Influenzal Meningitis.—This form of acute purulent meningitis occurs in association with influenzal infection elsewhere in the body, but is very rare. Infants are more liable to attack than older children. Most of the reported cases have been secondary to involvement of the nose, throat, or bronchi. The postmortem findings are the same

as in pneumococcic meningitis, and the influenza bacillus may be found in the heart's blood and in the primary lesions, as well as in the cerebrospinal fluid.

Symptoms.—The onset of this form of meningitis is not quite so sudden as in the pneumococcic type, but the symptoms are acute and violent. In the great majority of cases, an attack ends fatally within a few days after the appearance of the initial symptoms. The mortality in influenzal meningitis is not quite as high as in the preceding forms of meningeal inflammation in this group, but recovery is unusual.

Diagnosis.—The diagnosis can be made only by microscopic examination of the cerebrospinal fluid and by making cultures therefrom, since the influenza bacilli are by no means as abundant as are the infecting organisms in other forms of purulent meningitis, and in some cases they cannot be found in smears.

The cerebrospinal fluid is turbid, and contains many polynuclear leukocytes in addition to influenza bacilli. In some cases organisms other than the influenza bacillus are present, which indicates a mixed infection.

Typhoid Meningitis.—Occasionally, during the course of typhoid fever in children, a meningitis which is purulent in character will develop, the typhoid bacillus being the causative organism. The prognosis in this typhoid meningitis is slightly more favorable than in the other forms of purulent meningitis, although recovery occurs only occasionally.

Differentiation of Purulent Meningitis from Other Diseases.—Meningismus, or toxic irritation of the meninges, occurring during the course of any illness, may simulate meningitis; but the pulse and respiration do not become irregular, as in meningitis, and examination of the spinal fluid is negative. Typhoid fever is sometimes simulated at the onset of meningitis; but in meningitis vomiting is more persistent, there is rarely diarrhea, as in typhoid fever, there is no marked enlargement of the spleen, and the Widal reaction is negative.

Pneumonia is sometimes mistaken for meningitis; but when physical signs become appreciable in the chest, the meningeal symptoms usually subside. Uremia may be excluded by urinalysis, and eclampsia by an accurate history of the case and by study of the patient for a day or two.

Brain tumors may be excluded by the absence of localizing symptoms, such as optic neuritis, paralyses, and other evidences of a definite local lesion.

Prognosis.—The prognosis in acute purulent meningitis is most unfavorable, and in all the various types of the disease a large majority of the children perish. The mortality varies from 65 to 85 per cent., and is lowest in those cases where operation is resorted to for relief.

Treatment.—Acute purulent meningitis, secondary to infection of the head, ears, or face, is sometimes greatly modified by free drainage of the infected area. Antimeningococcic serum is of no value in this

form of meningitis, but repeated lumbar punctures will relieve the pressure symptoms. In influenzal meningitis a specific serum has been used by Wollstein, of the Rockefeller Institute, and others with good results. In the staphylococcic variety, homogeneous vaccines may be of service if given early in the course of the disease.

The general management of acute purulent meningitis is the same as that of other forms of meningeal inflammation, although in many cases little can be done, except to provide nourishment for the child and make it as comfortable as possible. Warm baths or a hot pack may be given three or four times a day, and ice-bags applied to the head and along the spine. The child should be kept in a quiet, dark, and well ventilated room.

If restless, 2 to 5 grains of sodium bromide, with $\frac{1}{2}$ to 1 grain of chloral hydrate, may be given every two or three hours to infants, and twice this dose to older children. If rectal administration is necessary, the dose should be double that given by mouth.

In very severe cases it may be necessary to administer morphine sulphate, $\frac{1}{100}$ to $\frac{1}{50}$ of a grain hypodermically, or codeine sulphate, $\frac{1}{80}$ to $\frac{1}{40}$ of a grain at 4- to 6-hour intervals, or hyoscine hydrobromate, $\frac{1}{800}$ to $\frac{1}{300}$ of a grain.

The bowels should be kept regular by giving small doses of calomel, $\frac{1}{10}$ to $\frac{1}{4}$ grain, during the day, or by the administration of a daily dose of $\frac{1}{4}$ to $\frac{1}{2}$ teaspoonful of cascara sagrada.

Feeding is often attended by much difficulty. A liquid diet is preferable, and milk is the best food; but, in many cases, the child refuses food, while in others it is unable to swallow. Nutrient enemata may be resorted to, but the bowel soon becomes irritable and unable to retain them, and gavage is then necessary.

The nose and mouth must be kept absolutely clean. In feeding the child who cannot swallow there is always danger that food may pass into the air passages. Bed-sores soon develop unless the child's skin is kept clean and dry, and precautions are taken to prevent pressure.

Stimulation is rarely of any permanent value; but, when required, brandy, 10 to 30 drops, strychnine sulphate, $\frac{1}{400}$ to $\frac{1}{200}$ of a grain, atropine sulphate, $\frac{1}{800}$ to $\frac{1}{400}$ of a grain, or camphorated oil, one to three drops, may be given every two or three hours.

CEREBROSPINAL MENINGITIS—EPIDEMIC MENINGITIS.

Definition.—This is a specific infection of the meninges of the cerebrospinal tract, caused by the diplococcus intracellularis meningitidis of Weichselbaum, and occurs both epidemically and sporadically.

Etiology and Epidemiology.—Epidemic cerebrospinal meningitis is a disease of both winter and spring, reaching its height between February and May. No country has been free from its ravages, although countries of the north temperate zone, especially those of Central Europe and our own northeastern States have suffered most.

Age is of considerable importance, since it chiefly affects those under three years, the incidence diminishing as the age increases. Males and females seem equally susceptible. Occupation and hygienic conditions play important roles in the etiology; thus, soldiers housed in barracks, miners, and inmates of prisons and almshouses are especially susceptible.

The diplococcus is almost invariably present, especially in the early stages of the disease, in the nasopharynx, particularly the upper part, and in the posterior nares. By making a swab, and then microscopic preparations, properly stained, these diplococci can be found. Albrecht and Shon demonstrated that healthy persons entirely free from the disease may harbor the cocci in the nasopharynx; and, according to Horder, the number of these healthy carriers varies from ten to thirty for every case of cerebrospinal meningitis occurring during an epidemic. Usually the diplococcus persists for three to four weeks after convalescence has set in. With regard to the carriers the cocci may be isolated even after several months, sometimes disappearing for a time, and then reappearing.

All evidence points to direct contact as the means of dissemination. Owing to the low degree of vitality of the organism, dissemination takes place through minute droplets of the secretion in coughing, speaking, or sneezing.

Pathogenesis.—The portal of entry of the infecting organism is the mucous membrane of the nasopharynx, where it sets up a pharyngitis, and from a study of recent epidemics it would seem that the cocci reach the meninges through the lymph and blood streams—lymphohematogenous—this being the most frequent and the earliest manifestation of meningococcic septicemia.

Pathological Anatomy.—In the acute fulminating cases leading quickly to death, there are either no visible changes or only hyperemia of the meninges. In the ordinary acute cases a purulent exudate, which is usually thick and yellowish green, is deposited in the meshes of the pia, chiefly at the base, especially around the optic chiasm, and extending to the cranial nerve-roots, cerebellum, spinal meninges, and to the convexity of the brain. The ventricles are distended with a turbid seropurulent exudate containing flakes of fibrin; sometimes it is purely serous, and leads to hydrocephalus. The choroid plexus and ependyma are always involved. Everywhere Gram-negative diplococci can be found microscopically. At a late stage of the disease, when the infection has subsided and convalescence has set in, the exudate has disappeared, and is replaced by fibrous thickening of the pia arachnoid.

Symptoms.—Although prodromal symptoms of indisposition and slight headache may be present for three to four days, usually the onset is very sudden, with fever, with or without a chill, intense headache, vomiting, rigidity of the neck, and general malaise. The temperature varies between 102° and 104° F., with a corresponding increase in pulse rate. The headaches are usually occipital, and very

severe. Vomiting may be almost continuous in young children, but less persistent in small infants and young adults. There may be slight stupor, or even delirium, during the initial period, which usually lasts from two to four days, following which symptoms of meningeal irritation develop.

The vomiting persists, there is loss of appetite, obstinate constipation, and the abdomen becomes markedly scaphoid. The pulse is rapid and irregular; the respirations are also irregular. The fever usually runs a variable course, or may be intermittent, with days of normal temperature. Following the initial stage, the sensorium usually clears during the first week. In severe cases the spleen is enlarged. Albuminuria is a frequent accompaniment; at times there is slight glycosuria. Herpes is quite common. Other skin rashes, such as large rose spots, small petechiæ, and rashes simulating measles, scarlet fever, and urticaria are observed.

Nervous Symptoms.—The most important symptom of irritation is rigidity of the neck, which may be marked; on any attempt to move it, the patient will resist and complain of severe pain. Bruidzinski's sign (flexion of the lower extremities on acute flexion of the head) is positive. Opisthotonos may likewise be extreme. The legs are drawn up. Kernig's sign is positive, and a positive Babinski sign may also be present. Tonic convulsions of the muscles of the extremities, abdomen, and face, are frequent; likewise clonic convulsions, especially involving the face. Convulsions simulating epilepsy, at times Jacksonian, are observed.

Hyperesthesia and hyperalgesia are most pronounced. In addition there is photophobia and susceptibility to noises. The vasomotor system is also affected, giving rise to the familiar tâche cérébrale. Cranial and peripheral palsies are rare. The reflexes are variable; the deep reflexes are usually increased, although they may be normal, diminished, or absent. The superficial reflexes are likewise quite variable; they are usually increased, but later disappear. The pupils may be dilated or contracted, and unequal. The reactions are usually sluggish or absent.

The stupor is not so deep as in other forms of meningitis, for the patient can usually be aroused. There may be delirium and marked insomnia. In infants the fontanelles are bulging.

Clinical Forms and Course.—Horder distinguished the following forms of cerebrospinal meningitis:

1. The ordinary, or acute, type just described.
2. Superacute type.
3. The fulminating, or malignant, type.
4. Mild type.
5. Postbasic meningitis of infants—cervical opisthotonos of infants (Gee and Barlow).
6. Aberrant forms:
    (*a*) Abortive.
    (*b*) Intermittent.
    (*c*) Cases simulating other diseases.

In the superacute forms the symptoms are more intense, and the patient usually succumbs within two or three days. In the fulminating type death may take place within twelve hours. In milder forms, the symptoms are less severe. This is common during epidemics, and unless a careful examination is made and a lumbar puncture performed, it may go unrecognized. The abortive cases are to be recognized only during epidemics; they exhibit slight fever, vomiting, and stiffness of the neck, all of these symptoms clearing up within several hours. The post-basic meningitic form is characterized by extreme retraction of the head, marked opisthotonos (the head frequently approximating the buttocks), vomiting and extreme emaciation.

Course in Acute Type.—The disease either ends in recovery, interrupted by recrudescence, or becomes chronic, and the emaciation extreme. Incontinence and bed-sores develop, and muscular rigidity becomes marked. The disease may continue for months and, although recovery may take place and leave no residual defects, yet usually mental deficiency, blindness, deafness, or palsies follow. Death may ensue at any time during the course of the disease.

Diagnosis.—The characteristic signs and symptoms, which are especially noticeable during an epidemic, will usually arouse one's suspicions of meningitis. An absolute diagnosis can be made only by thorough examination of the cerebrospinal fluid, the findings in which are as follows:

(1) The spinal fluid is both increased in amount and under increased pressure. (2) The fluid shows turbidity which varies, being slight in the invasive stage, and marked during the active stage, later clearing as the inflammation subsides. (3) The globulin is always increased, the amount depending upon the stage of inflammation. (4) The fluid does not reduce Fehling's solution. (5) Cytology: During the invasive stage the lymphocytes predominate, but on the advent of acute inflammation the polynuclear cells predominate, ranging between 70 and 80 per cent. Later, when the inflammatory stage subsides and chronic hydrocephalus becomes more pronounced, there will be a predominance of lymphocytes. (6) Bacteriology of the fluid: Intracellular meningococci can usually be seen some time during the first ten days. The fact that they are not perceptible in smears does not necessarily indicate that they are absent, and the fluid should always be cultured on appropriate culture media.

Complications.—Joint complications, either monarticular or polyarticular involvement, are observed. Inflammations of the eye—panophthalmia, iritis, iridocyclitis, usually unilateral, and leading frequently to blindness—also inflammation of the inner ear, generally bilateral, giving rise to deafness, are all observed. Empyema, pericarditis, and endocarditis are rare complications. Chronic hydrocephalus frequently follows this disease.

Prognosis.—Since the use of Flexner's serum the death-rate has fallen from 70 to 80 per cent. to about 30 to 40 per cent. The mortality is highest in young infants, especially in the early stages of

52

the disease. The prognosis cannot be made by examination of the spinal fluid alone; but the greater the number of intracellular cocci the better will be the outlook, and *vice versa*.

Prophylaxis.—Since we now know that the mode of dissemination of the cocci is through the buccal secretions, care must be taken to prevent direct contact. Strict quarantine should be established, just as for other infectious diseases, and the same precautions observed in regard to the disposition of the urine, feces, and infected fomites, as well as thorough fumigation after recovery.

Even more important is the treatment of the carriers, as it is by them that the disease is really spread. They, likewise, should be placed under quarantine until the nasopharynx is free from cocci, which, under the use of antiseptic gargles and sprays, is usually in about ten days. All healthy persons should avoid the predisposing causes—exposure to severe cold, catarrhal affections, and overcrowding.

Treatment.—Several immune specific sera have been made. The one in general use in America is that of Flexner. It must be administered by the intraspinal route, the method being as follows:

A lumbar puncture should be made, and the turbid fluid withdrawn in an amount always equal to, or greater than, the quantity of serum to be injected. The serum should then be slowly injected, either with a syringe or by the gravity method, the time allowed for the injection being about ten minutes. The usual dose is 20 c.c., and a dose larger than this must be given with extreme care, even though a large quantity of spinal fluid has been withdrawn. This procedure is repeated daily until the spinal fluid has been sterile for several days, and the patient clinically shows great improvement.

If the case is progressing favorably the spinal fluid clears, and lessens in amount and pressure, the leukocytes rapidly diminish, the meningococci disappear, and the power to reduce Fehling's solution —of considerable importance in the prognosis—reappears. If there is a relapse, the same treatment is repeated. Autogenous vaccines have also been employed, but as yet without convincing results.

If meningococcic septicemia develops, the serum should be administered intravenously; in hydrocephalus should be injected directly into the lateral ventricles; and in severe joint affections it should be introduced directly into the joint.

The general treatment is of the utmost importance. The patient must be kept quiet, preferably in a dark room, and must be given plenty of fresh air. An ice-cap should be kept on the head. Hydrotherapeutic measures, such as warm baths, will allay the nervous symptoms. Hexamethylenamin should be given routinely—five grains three or four times a day to a child five years old. Sedatives, preferably some preparation of opium, may be necessary on account of the pain and delirium. Morphine, gr. $\frac{1}{12}$, may be given hypodermically to a child of five years, and if necessary the dose may be repeated. Every precaution must be taken to guard against bed-sores and, if possible, the patient should lie on a water bed.

The nourishment of the child should receive most careful attention. When necessary, feeding by gavage is usually more satisfactory than by rectum. Attention should be given the bladder, bowels, eyes, skin, and ears, and, should any complications develop, they should receive appropriate treatment.

TUBERCULOUS MENINGITIS.

Tuberculous meningitis, the most common form of meningitis, is a tuberculous inflammation of the cerebrospinal meninges, developing secondarily to some tuberculous focus elsewhere in the body or as a part of a general tuberculosis.

Etiology.—Tuberculous meningitis is a disease of early childhood, occurring most frequently between the second and sixth years. It has been observed as early as the third month, and after the sixth month has been found quite frequently. After the sixth year, cases of the disease rapidly diminish. The sexes seem equally affected. The disease is most common in the early spring, from March to May. Breast-feeding offers no immunity to this infection.

The disease is always caused by the tubercle bacillus. The cerebrospinal meninges are never involved primarily; but the infection arises from some latent tuberculous focus elsewhere in the body, such as the caseous bronchial, cervical, mediastinal, or mesenteric lymph glands, lesions in the lungs, pleuræ, bones, joints, and intestines, or tuberculous meningitis may be merely a part of a general miliary tuberculosis.

Pathogenesis.—The pathogenesis of tuberculosis in children is considered in the chapter on Tuberculosis. It seems well established by the studies of von Pirquet, Hamburger, and, more recently, by those of Dunn that in the great majority of cases infection takes place through the respiratory tract, that the bacilli lodge in the lung where they set up a primary focus of infection, and that the regional lymph glands become secondarily involved through the lymph vessels.

From the primary focus of infection the bacilli invade the bronchi, thence pass to the nasopharynx and its regional lymph glands. By the swallowing of the bacilli in the sputum they reach the intestinal tract, thence the mesenteric lymph glands, and peritoneum. Occasionally, through drinking milk infected with the bovine type of bacillus, the intestinal tract is the primary seat of the lesion.

The most frequent mode of infection of the meninges is through the blood stream, either directly from the primary focus in the lungs or from some secondary focus, such as a caseous lymph gland, which perforates into a vein, and causes the dissemination of the bacilli to all the organs of the body, setting up a miliary tuberculosis. Peritz attributes the frequency with which meningitis develops in the child to the increased demand for blood on the part of the rapidly growing brain.

In addition there are a number of factors which have an exciting influence upon these tuberculous foci, and lead to tuberculous menin-

gitis. Thus certain infections, especially measles and whooping-cough, predispose to tuberculous meningitis, possibly owing to diminished resistance due to these infections. Trauma, such as injury to the head, tuberculous glands or joints, or operations for the removal of glands, may cause a dissemination of bacteria, either by direct injury to the active focus, or by a lessening of the resistance of the arachnoid membrane, such as is caused by a blow upon the head. Overexertion, mental excitement, and strain have precipitated tuberculous meningitis.

In rare cases the meninges may be infected by tuberculosis of the contiguous structures, such as caries of the inner ear, vertebræ, or cranial bones.

Pathological Anatomy.—Tuberculous meningitis is primarily a basilar meningitis. The most important pathological changes are the miliary tubercles and the inflammatory exudate which are found especially at the base of the brain around the circle of Willis and optic chiasm, extending into the Sylvian fissure, and out over the pons, the base

Fig. 83.—Opisthotonos in tuberculous meningitis; a boy six years of age.

of the medulla oblongata, the cerebellum, and spinal meninges. The exudate is.either gelatinous, serofibrinous, or greenish-yellow and of fibrinopurulent character; owing to its consistency, it is adherent to the pia. The amount of exudate is relatively small compared with that in other types of meningitis.

The tubercles, either transparent or of a greenish-white color, are especially numerous about the base of the cerebrum. They are often observed arranged in rows along the vessels of the Sylvian fissure after the removal of the pia. The convexity of the brain is less involved, although there may be cloudiness, and an infiltration of the pia with tubercles. The ependyma and choroid plexus of the lateral ventricles are also invaded by tubercles with resulting internal hydrocephalus. The ventricles become filled and distended with a clear serous fluid, or it may be bloody. As a result of pressure the walls may be softened, the convolutions flattened, the fontanelles bulging, and the sutures separated.

The dura may at times exhibit a mild pachymeningitis interna hemorrhagica, or may be infiltrated with tubercles. The cerebral

cortex is always involved. Tubercles may invade the cortex and caseate, giving rise to a meningo-encephalitis.

Symptoms.—The onset of the disease is usually slow and gradual, occurring most frequently in weak, anemic, and poorly nourished children. On the other hand, children presenting the very picture of health may likewise be affected. The disposition of the child slowly changes, and it loses interest in its play, becomes irritable, fretful, peevish, easily tired, moody, wishes to be let alone, cannot be pleased, and is drowsy and sleepy. Its sleep is restless, interrupted by slight delirium, or there may be insomnia. Headache, which at first is mild and periodic, rapidly becomes more severe and persistent. Symptoms referable to the gastro-intestinal tract develop. There is loss of appetite, vomiting, pain in the abdomen, and severe constipation. The vomiting is cerebral in character, projectile, and is unassociated with nausea or time of eating.

These prodromal symptoms last from several days to weeks, or even months, in which time, however, symptoms of cerebral irritation have gradually developed. There is hyperesthesia of the skin and sense organs. The patient is susceptible to harsh voices or bright lights; merely the slightest touch is painful. In addition there are certain vasomotor phenomena, such as transient flushing of the cheeks, face, and body. The well-known tâche cérébrale is almost always present. This is an irritation of the skin, manifested by a red streak which appears when the finger-nail is drawn over the skin of the abdomen.

There are also slight symptoms of motor irritation. The child performs certain stereotyped movements; it picks at its lips, genitals, and bed-linens, winks its eyes, grinds its teeth, performs sucking and chewing motions, and from time to time takes deep sighing inspirations.

At this stage, although drowsy, the child may answer questions coherently. The temperature is usually elevated in the evening to 100° or even 102° F., the pulse is usually between 70 and 80, and exhibits marked arrhythmia. The patulous fontanelles may be distended and pulsating. The pupils are usually contracted, and react promptly. Exaggerated reflexes are, as a rule, present, although they may be unequal. Slight rigidity of the neck, back, and lower extremities may give rise to a positive Kernig's sign, also to Bruidzinski's sign (on passive flexion of the head upon the chest, the thighs are drawn up upon the abdomen).

The symptoms of cerebral pressure and irritation grow worse. The drowsiness increases, and the child sinks into a stupor from which, however, it can be aroused. Acute irritative symptoms may set in, with severe headache, delirium, convulsions, and finally deep stupor. The delirium is usually mild. The child lies almost asleep, with its eyes half-open, and performs stereotyped movements, utters incoherent sounds, and throws itself back and forth on the bed or frequently utters piercing shrieks—the hydrocephalic cry. Stupor follows the delirium, or they may alternate.

When the sensorium again clears, severe headache and dizziness are complained of. There is increased susceptibility to strong light —photophobia—and to loud noises; the symptoms of motor irritation are increased; the rigidity of the muscles of the neck, trunk, and extremities also increases. Tetanic contractions of the muscles of the jaw and face may take place, while contractions of the flexor muscles of the lower extremities give rise to a characteristic posture; *i. e.*, the patient lies upon the side, with the thighs flexed upon the abdomen, and the legs upon the hips. The abdomen is usually retracted, which gives it a "boat-shaped" appearance. As a result of the extreme emaciation the intestines can be readily palpated.

Paralyses are quite frequent, and cranial nerve palsies common, owing to the extensive basilar involvement. There are ptosis, dilatation, and inequality of the pupils, strabismus, and, occasionally, complete ophthalmoplegia. The pupils react slowly to light or are inactive. Nystagmoid movements are often seen. Examination of the eye-ground frequently shows a choked disk and the presence of miliary tubercles in the choroid. Facial paralysis is also often observed. Paralyses of the extremities, such as monoplegia or hemiplegia, may follow convulsions, which may be either partial or complete; if unilateral, they may simulate a Jacksonian epileptic seizure.

The reflexes are usually increased. A positive Babinski phenomenon may or may not be present. As unconsciousness deepens the reflexes are lost, the temperature rises, and the breathing becomes Cheyne-Stokes. Remissions in this state are not rare; they are only temporary, however, and after several days of improvement the patient falls into the final stage of coma.

In this stage there is complete unconsciousness, a relaxation of the hitherto rigid extremities and neck, the reflexes are gone, the pupils are dilated and show no reaction, the vomiting and the outcries cease. There may be either retention of urine or incontinence of both urine and feces. The pulse always becomes rapid and feeble, frequently 170 to 200 or more per minute; the temperature rises, and during the last few days may reach 106° to 107° F.; it may, however, be subnormal. Death usually follows, or may occur during a convulsion. This final stage extends over a period of three to ten days, while the duration of the whole disease after definite symptoms have appeared is from two to four weeks.

Atypical Cases.—*Acute Cases.*—In a few cases the onset is very acute, the disease setting in with convulsions, after which the patient quickly falls into coma, which terminates in death within a few days.

Meningo-encephalitis.—Following convulsions, monoplegia or hemiplegia may appear, due to involvement of the motor area.

Spinal.—In rare cases, lancinating pains are complained of, due to involvement of the spinal roots.

Chronic Form.—Such cases have been described, extending over a period of months, in which remissions lasting weeks and months have taken place.

Diagnosis.—The diagnosis during the prodromal period may be very difficult; although the vomiting, severe headache, drowsiness, change in disposition, and irregular pulse and respirations should arouse one's suspicions of some form of meningitis.

An absolute diagnosis of tuberculous meningitis can usually be made by examination of the cerebrospinal fluid, which also enables one to differentiate between the various types of meningitis. The fluid removed by lumbar puncture is under great pressure, and is at first usually clear or slightly turbid, later opalescent or turbid, or may even be purulent (owing to mixed infection). There is generally a relatively slight cellular increase, in which the mononuclear cells predominate. As the spinal fluid becomes secondarily infected, the polynuclear elements predominate.

As a rule, there is a great excess of globulin. There may or may not be a reduction of Fehling's solution, depending upon the type of cells that predominate; if the mononuclear cells are in excess the solution reduces, whereas if the polynuclear elements increase this reducing power diminishes.

An absolute diagnosis of tuberculous meningitis can only be made by isolating the bacilli from the spinal fluid. This can be accomplished in practically every case, although it usually requires a long, diligent search. Repeated punctures are frequently necessary. The technic of the procedure is as follows:

From 10 to 20 c.c. of spinal fluid are put in an ice chest and allowed to stand twelve to twenty-four hours, during which time a fine fibrinous coagulum, resembling a spider web, usually forms. This coagulum is carefully floated out upon a glass slide, then teased out, and finally stained for the tubercle bacillus. In other cases centrifuging may be necessary, and a preparation made of the precipitate. In still other cases superimposing drop upon drop, obtained by scraping the side of the test-tube with a platinum loop, may give the desired result. Should it be unsuccessful, then an intraperitoneal inoculation of the spinal fluid should be made in a guinea-pig, in which case the diagnosis will be confirmed only after the death of the patient.

In addition other means of diagnosis should be employed. Tubercle bacilli can often be isolated from the sputum. Tuberculin reactions —those of von Pirquet, of Moro, and of Monteau—assist by establishing the presence or absence of tuberculous infection. In a series of cases reported by Koch, from von Pirquet's Clinic, 84 per cent. gave a positive reaction during the second week before the child died, dropping to 65 per cent. in the last week of the illness. The tuberculous reaction diminishes during the last stage.

Other evidences of tuberculosis should also be sought for. By a physical examination enlarged bronchial glands or active lung foci may be determined, and be confirmed by the x-rays; at times the primary focus of infection in the lungs is shown by the x-rays, and should always be looked for. The clinical symptoms, combined with the laboratory findings, usually establish the diagnosis.

Prognosis.—Tuberculous meningitis is almost always fatal. There are, however, a few authentic recoveries recorded, in which tubercle bacilli were present in the spinal fluid; yet, considering the length of the remissions, it is necessary to keep these patients under observation for a long period of time before pronouncing them cured.

Prophylaxis.—The child should be carefully guarded against any possible tuberculous infection. This is considered more fully in the chapter on Tuberculosis. If it becomes infected, then the greatest care should be exercised to prevent mental excitement, overexertion, and to protect the child from the infections—measles and whooping-cough—which so frequently precipitate meningitis.

Various procedures have been advised for the treatment of this disease, but none have been effectual. Drainage of the subarachnoid spaces, also ventricular puncture, have been suggested. Irrigation of the spinal canal has also been performed. The least harmful procedure is lumbar puncture, which should be repeated as often as necessary for the relief of symptoms. Improvement usually follows this procedure, but death ensues later.

Treatment.—Our field for treatment lies in prophylaxis; since, after the disease has been established, the treatment can be only symptomatic.

DISEASES OF THE BRAIN.

CEREBRAL PALSIES.

In the symptom-complex of cerebral palsies are included a variety of clinical manifestations, the remains of former cerebral diseases, of various anatomical lesions, and etiological factors, leaving behind certain permanent anatomical lesions and clinical disabilities. From a single symptom it is impossible to make a definite anatomical diagnosis.

Etiology.—Cerebral lesions giving rise to cerebral palsies may occur as follows:

1. *Causes Prior to Birth.*—Malformations of the brain, porencephalia, microcephalia, and cysts are frequently associated with malformations elsewhere in the body, as in the kidneys, heart, etc. Fright, anger, sorrow, or trauma may give rise to hemorrhage or thrombosis. Alcoholism in the parents is of some importance; also syphilis which leads to tissue changes about the vessels, and gives rise to endarteritis.

2. *Injuries at Birth.*—Trauma from forceps, asphyxiation from a long and difficult labor, or premature birth may all bring about meningeal hemorrhage. Usually, however, intra-uterine trauma or maternal syphilis precipitates premature birth.

3. *Causes After Birth.*—Direct trauma to the head may cause hemorrhages. The most common postnatal cause, however, is

encephalitis secondary to acute infections, such as measles, scarlet fever, whooping-cough, typhoid fever, tonsillitis, chorea, and endocarditis.

We distinguish two main types of cerebral palsies:

(1) Infantile hemiplegia. (2) Cerebral diplegia, or Little's disease. In general it may be said that a difficult labor, premature labor, and asphyxia give rise to cerebral diplegia, whereas infections lead to hemiplegia.

Pathological Anatomy.—The initial lesions are: (1) hemorrhage; (2) embolism; (3) thrombosis; (4) acute, and sometimes chronic, encephalitis.

At a later stage, and as a result of these initial lesions, the following conditions are frequently seen: (1) porencephalia; (2) microgyria; (3) cysts; (4) patches of glial and connective-scar tissue; (5) localized sclerotic patches (tuberous sclerosis); (6) unilateral sclerosis of a portion of one cerebral hemisphere—cerebral atrophy.

When the condition is the result of an inflammatory process, the meninges are usually thickened.

Infantile Hemiplegia.—This form of palsy usually comes on after birth, either in consequence of a syphilitic taint or an encephalitis, but may occur either before or during birth. After the fifth year it is rare.

Symptoms.—Although prodromal symptoms, such as headache, fever, vomiting, and general malaise may precede the onset, yet the disease usually sets in suddenly with fever, convulsions, and sometimes coma, followed by hemiplegia, which, however, may not appear for a week or more. Convulsions are usually on the same side as the paralysis.

The paralysis in the beginning is a flaccid one, involving the face—its muscles and inferior branches—and the arm and leg.

After eight to ten days the acute stage subsides, and the paralysis gradually recedes, and becomes spastic. Improvement takes place, especially in the face and leg. The face muscles may almost wholly recover merely showing weakness by mimetic and emotional contortions. The leg, too, may almost wholly recover, for on examination the only evidence of the former lesion may be exaggerated reflexes.

The arm becomes spastic, and, in less favorable cases, the leg likewise. The position that the arm assumes is characteristic. The upper arm is adducted, the forearm flexed and pronated, the wrist flexed, the thumb adducted, and the fingers flexed upon it. The leg, too, shows a spastic condition; it is rotated inward, and extended at the hip- and knee-joints, with plantar flexion of the foot—pes cavus. In walking the leg is dragged, and there is a circumduction like that seen in an adult hemiplegic. When it walks the child makes accompanying movements of the arm by raising it, and the faster the patient walks the higher the arm is lifted.

Although the reflexes may be absent at the onset, they are always exaggerated. The Babinski, Oppenheim, and Gordon reflexes are

present. Ankle and patella clonus may also be present. Aphasia may appear associated with either a right or left-sided lesion, but it usually soon disappears. Ataxia and intention tremors accompanied by voluntary movements are present, also involuntary, posthemiplegic, choreiform, and athetoid movements, as a result of which muscular hypertrophy takes place.

Sensory disturbances and cranial nerve palsies are rare. At times there is atrophy of the optic nerve, hemianopsia, and rigid pupils, the latter usually associated with syphilis.

Trophic disturbances of the muscles, skin, and especially of the bones, are common. The skin is cool and livid. There is muscular atrophy from lack of function. The changes in the bones are most important, the growth of the cranial bones being sometimes so affected as to produce marked asymmetry of the head and face. The long bones are especially involved, there is shortening of the affected arm and leg, and the growth of the bone is affected, especially its length, and the shortening is more at the distal than the proximal end. The circumference is also lessened, especially in the central portion.

Epilepsy is a frequent complication, developing in more than one-half the cases. After the initial convulsion, the patient may remain free for days, months, or years before epileptic seizures appear.

The mental development of the child may be normal; usually, however, some disturbance of intelligence is observed, and this may vary from mere feeble-mindedness to absolute idiocy.

Cerebral Diplegia—Spastic Paraplegia—Little's Disease.—This type usually occurs either before birth or during delivery as the result of a difficult labor. Asphyxia neonatorum is the most frequent cause for hemorrhage. Trauma from forceps and hereditary syphilis are likewise important.

Symptoms.—In severe cases the disease is usually noticed soon after birth, the child's body and extremities, especially the legs, being rigid and spastic. In less severe cases this may not be observed until the time when the child should begin to walk, when, on putting it on its feet, marked rigidity of the extremities is noticed. The thighs are rotated inward, the knees cross and touch from contractions of the adductors, and the child stands upon its toes with its foot in complete plantar flexion. The contractions in the thigh involve the adductors and flexors, whereas the gastrocnemei are most involved in the lower leg. The arms are less affected, although the spasticity may be marked.

When the upper extremities are involved, the arms are adducted to the body, the elbows are bent, the wrists flexed, the thumbs adducted, and the fingers flexed upon them.

The muscles of the body generally are involved, giving rise to lordosis, kyphosis, and scoliosis.

The gait is quite characteristic—the so-called "*scissors gait.*" On attempting to walk the legs are crossed, the knees are pressed together and cannot be drawn apart, and the child walks on its toes.

Tremors and ataxia are common. Choreiform and athetoid movements are less frequent in this form than in the hemiplegic type.

The deep reflexes of the involved area are greatly exaggerated. Ankle- and patellar clonus are present, as well as the Babinski, Oppenheim, and Gordon reflexes. On the other hand, the extremities may be so rigid that no reflexes can be elicited. Trophic disturbances of the skin, muscle, and bones are usually not extensive.
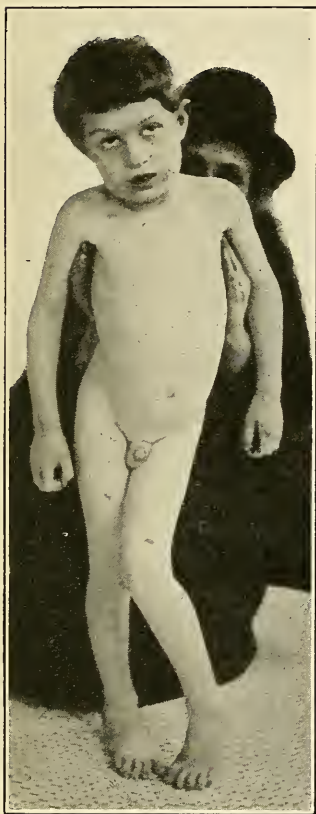
Fig. 84.—Feeble-minded, spastic paraplegia; male, aged six years.

Fig. 85.—Idiocy, spastic diplegia; male, aged four years.

There may be cranial nerve palsies, nystagmus, strabismus, atrophy of the optic nerve, and inequality of the pupils. The face may be involved, and have a mask-like appearance. Dysarthria and bradylalia are common, and are usually accompanied by impairment of intellect. Esophageal spasm is sometimes observed, and causes difficulty in swallowing.

Convulsions in the first few days after birth are common; but

epilepsy, which develops later, is not as frequent as in other types of palsy.

The mental development of the child may be normal, although there is usually a defect in mentality varying from feeble-mindedness to idiocy.

Diagnosis.—The diagnosis of cerebral infantile palsies is purely a clinical one, and can be made only by considering the history with special reference to the question whether or not the palsy is progressive, and there are any progressive brain symptoms.

Cerebral hemiplegia, setting in acutely as an encephalitis, must be differentiated from meningitis, infectious fevers, infantile eclampsia, and poliomyelitis. The differentiation from the latter disease is no longer of much importance, for we now know that both the cerebral and spinal forms may coexist in the same patient, being known as encephalopoliomyelitis.

Cerebral palsies must be differentiated from brain tumors of slow growth, from cerebral syphilis, multiple sclerosis, hydrocephalus, and Friedreich's ataxia.

Prognosis.—Improvement may go on for years. The arms may almost completely recover, except for a slight permanent spasticity. Improvement also takes place in the lower extremities, for these children learn to walk, even if tardily; the mental calibre, too, often somewhat improves. They are always predisposed to intercurrent infectious diseases, to which they readily succumb. In the hemiplegic form death may take place during the initial stage. Improvement is usually as great as in the diplegias.

Treatment.—This is purely symptomatic, in order to maintain the proper nutrition of the parts involved, and depends upon the intelligence of the child, the extent of the paralysis, and the amount of deformity.

The galvanic current should be applied to the affected muscle. Hydrotherapeutic measures, such as warm baths, tend to relax the muscle spasm. Massage, combined with passive movements, gymnastic exercises, and Frenkel's movements are of special benefit in cases in which there is not too great impairment of the mentality.

Braces may be necessary to prevent contractures, and various orthopedic operations—tenotomy and tendon transplantation—have been devised for their relief.

Trephining of the skull with removal of the primary foci has been performed for the relief of epilepsy. The mental training of the child should not be neglected.

IDIOCY.

Synonyms.—Mental Deficiency—Imbecility—Feeble-mindedness.

Children who have mental diseases may be divided into two classes: those who have physical defects in addition to the mental condition, and those in whom there are no physical defects.

It is impossible to make a sharp differentiation between the various

types of mental defects in children, as the different forms blend into one another; but cretinism, Mongolian idiocy, and amaurotic family idiocy are types easily recognized.

Imbecility and feeble-mindedness are terms applied to the minor forms of idiocy in which there is no extensive cerebral lesion. The backward child should not be placed in this class, for its retarded mental development is often due to some abnormal physical condition which can be corrected, such as defective sight or hearing.

Etiology.—Idiocy may be either congenital or acquired. In the majority of cases which present physical defects, idiocy is congenital, and may be associated with defective brain development, as in porencephalia and agenesis corticalis, or with lack of development of the brain as a whole.

Another class of cases is associated with internal or external hydrocephalus, and microcephalic children furnish a certain number of
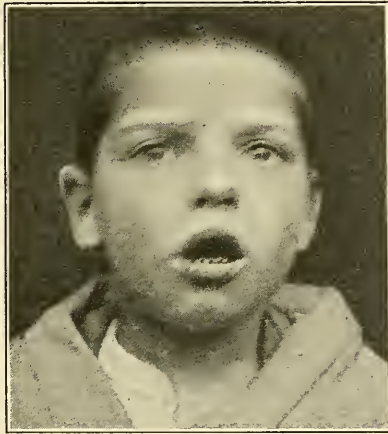
Fig. 86.—Idiocy, male, aged five years.

these cases. Consanguinity, injuries at birth, and syphilis are not uncommon causes of idiocy in children, and in many cases in which paralysis appears there is a history of meningeal hemorrhage or cerebral hemorrhage of traumatic origin.

Cretinism, cerebrospinal meningitis, and anterior poliomyelitis may also give rise to idiocy, and a certain proportion of epileptics become idiots as a result of brain lesions due to repeated epileptic seizures.

As a rule, mental defects unassociated with physical abnormalities are not apparent until the second period of childhood, melancholia, mania, katatonia, and dementia being occasionally observed in older children.

Although no definite time limit can be established for the manifestation of the various phases of mental function, yet in the normal infant there should be evidences of control of the senses of hearing and sight by the fourth month, later appreciable signs of memory, percep-

tion, and power of attention, and the child should be able to recognize objects and familiar faces and be able to express pain and pleasure.

At nine months some degree of understanding should be manifested when familiar words are spoken, and at this age the normal infant should begin to imitate spoken words, and attempt to creep about.

When any of these functions is considerably delayed, the child should be carefully studied in order to determine accurately the extent of mental impairment. The family history and lives of the parents should be investigated for some possible etiologic factor, and an accurate history of the child's life from birth be secured. Special note should be made of any insanity, alcoholism, chorea, or hysteria in the family, and in the child's history convulsions shortly after birth, injuries at birth, eclampsia, and asphyxia are important points.

Symptoms.—Both mental and physical symptoms accompany idiocy, although an idiot may resemble a perfectly normal child.

Mental Stigmata.—Mental deficiency is rarely suspected in early infancy, except in the case of the Mongolian idiot, in whom we find abnormality of the features. When physical defects are present, they are usually noticed before mental abnormality is suspected, and in these cases the infant is brought to the physician because it cannot sit up, walk, or, perhaps, talk at the proper time.

Upon examination it becomes evident that the child has no control of its limbs; saliva usually dribbles from its mouth; it has a vacant stare, or moves the eyes aimlessly, taking no particular notice of anything. The eyes should always be examined in these cases to determine whether or not there are any visual defects, such as microphthalmia, rederemia, coloboma iridis, lesions of the vitreous, congenital cataract, strabismus, or partial or central blindness of central origin.

The sense of hearing may be defective or entirely absent from congenital or acquired causes, and in idiots there is always imperfect development of the senses of taste and smell. The appetite is usually ravenous, but, as a rule, there is no particular preference as to food. There is also a remarkable degree of insensibility to pain, heat, and cold, and this in some cases may be so great that bodily injury, and even mutilations, self-inflicted, are not uncommon.

Even when the sight is perfect, the idiot may not recognize its mother, shows no instinctive understanding of the nursing bottle, and at the age when it should be talking—one to two years—utters strange sounds and shrill cries, and makes no attempt to imitate spoken words.

At the age of three or four years, most cases of idiocy can be recognized, for the children do not talk, their vacant expression is apparent to all but the mother, they are unclean in their habits, in defecating and urinating, and in temperament they are either amiably stupid, or irritable, excitable, and uncontrollable.

Convulsions are not rare in idiots, and in the majority of cases they signify the presence of definite organic lesions of the central nervous system, such as are found in meningitic and syphilitic idiocy.

Physical Manifestations.—The head may be either excessively large (hydrocephalic) or very small (microcephalic). The hydrocephalic head is familiar to all, with its rounded ball-shape, widest at the temples, and presenting a sharp contrast to the small face and tiny features. These children are usually dull and stupid, but not irritable, and they have a timid, shrinking, and sad appearance.

The microcephalic idiot has a small head with low forehead, a poorly developed occipital prominence, closed fontanelles, and prematurely closed and ossified sutures; but, strange as it may seem, the small skull is in many cases independent of the poorly developed brain, and a child whose skull is normal may have a microcephalic brain.

Even the microcephalic skull is usually larger than the brain requires, for there is a lack of development of either the whole brain or of the occipital, frontal, or parietal lobes. The eyes are small, the ears project, and the nose and lower jaw are large, so that the face seems quite large in proportion to the skull. In some cases the rest of the body may appear to be perfectly normal, while in others there is paralysis, either flaccid or spastic.

True microcephalic idiots are obstinate, vulgar, and brutal. If the brain lesions are not extensive, they sometimes live past middle age, but the hydrocephalic infant rarely survives until the end of the first year.

The syphilitic idiot usually presents the characteristic signs of syphilis, such as the bossed skull, saddle-nose, Hutchinson's teeth, rhagades, and congenital deafness.

Howe, in a summary of 517 cases of idiocy, found the following physical defects: Deafness in 12, blindness in 21, and defects in the nose or mouth, such as hare-lip and high palatal arch, in 23 cases. The condition of the extremities varies in the different types of idiocy. Howe found in 54 cases of this same series, deformities of the hands and feet, and in 96 cases there was paralysis of one or more limbs.

Many hydrocephalic infants suffer with spastic paraplegia, the legs being affected, as a rule, while the upper extremities are but little involved. The paralytic idiot has either hemiplegia or diplegia, and contractures may become quite marked. In cretinism, microcephalic idiocy, and syphilitic idiocy there is manifest weakness of all the extremities.

Among the other physical peculiarities which idiots occasionally present may be mentioned supernumerary or deficient fingers and toes, asymmetry, malformations, congenital dislocations, ankyloses, and numerous minor abnormalities.

Diagnosis.—The diagnosis of idiocy is a matter of great importance, but can be positively arrived at only after accurate history taking, which in many instances will reveal the etiological factor, also after careful physical examination for the stigmata of degeneration, and an investigation of the mentality in which the patient's intelligence is compared with that of a normal child of the same age. Mentality

slightly lower than normal may mean merely backwardness, but great deviations from the normal are in the majority of cases due to idiocy.

Prognosis.—So far as complete recovery is concerned, the prognosis in idiocy is unfavorable; but surprising improvement often takes place, so that a case must not be pronounced hopeless unless there is some congenital defect of the brain, or there has been a hemorrhage at birth which caused paraplegia or diplegia. In cases which follow meningitis or encephalitis, the outlook is somewhat better than in those just mentioned, and even in cretinism and syphilis prompt therapeutic measures are not without good results.

Treatment.—In all but the mildest forms of idiocy the children are far better off in institutions which will educate and train them by special methods than in their own homes; and, moreover, they should be kept apart from normal children. Special schools and training are necessary, too, for the backward child, if the best results are to be secured. The idiot who is tractable may be kept at home until the sixth or seventh year if there are no younger children to whom he may set a bad example.

Moral degenerates and vicious and unclean idiots should be placed in institutions just as soon as they can be admitted. The problem of the mentally deficient child is an economic one, and experience has proven that many of these unfortunates may become self-supporting instead of being always a cost to the family or the State.

AMAUROTIC FAMILY IDIOCY.

This disease, which was first described by Warren Tay, in 1881, and later given its name by Sachs, of New York, is not a rare one. It is characterized by arrested cerebral development, by blindness, and by changes in the macula lutea, together with progressive impairment of the functions of the muscles. The affection shows a marked predilection for the Hebrew race, nearly all of the recorded cases having occurred among Jewish people.

Etiology.—The etiology is most obscure, and it has not been definitely determined whether the arrested development and degeneration are due to antenatal or postnatal causes. Many observers believe the affection to be toxic in origin, and it has also been attributed to syphilis and alcoholism in the parents. It is not uncommon for two, three, or four children in the same family to be affected.

Pathology.—Postmortem investigations have shown degeneration of the ganglion cells throughout the entire nervous system. The cellular structures lose their identity, their nuclei being scarcely demonstrable, and the protoplasm markedly degenerated. Blindness in these cases is due to degeneration of the ganglion cells of the retina and of the fibers of the optic nerves and tracts.

Symptoms.—The infant is apparently normal at birth, and may remain so until the sixth, or even the tenth month, when the parents usually notice that it makes no progress in development. At this

age the child does not hold up its head, moves about very little, takes no interest in its surroundings, and does not even follow objects with its eyes. But, unless an ophthalmic examination is made, blindness may not be suspected, although the eyes have a peculiar and fixed stare. Nystagmus may be present but is not pathognomonic.

The child now begins to retrograde, instead of progressing, and by the end of the first year optic atrophy and paresis may be complete. The muscles are absolutely without power, and the little one can neither sit up nor hold up its head. At first there is flaccidity, later rigidity and spasticity of the muscles, and occasionally convulsions.

Mental retrogression is also noticeable, and the child fails to recognize familiar objects and faces, but is unusually susceptible to sounds, starting violently at each sudden noise, such as the slamming of a door or clapping of the hands. Eventually it becomes dull, apathetic, totally indifferent to its surroundings, and has no power to change the position of its limbs. Progressive emaciation renders the child helpless, and a pitiable object of mere skin and bone.

In some cases swallowing becomes impossible, and gavage is necessary. Death usually occurs in about a year after the onset, and is due to marasmus, exhaustion, or hypostatic pneumonia.

Prognosis.—The disease comes on slowly, and its course is marked by gradual but progressive mental and physical degeneration, and an invariably fatal termination.

Diagnosis.—As a rule the diagnosis is readily made from the signs and symptoms. The findings on ophthalmic examination are pathognomonic.

Treatment.—Treatment is never curative, but under judicious management the life of these infants may be prolonged for a year or more, although death is inevitable.

MONGOLIAN IDIOCY.

The Mongolian type of idiocy occurs only in the Caucasian race, and is characterized chiefly by a Mongolian, or Chinese, cast of face, also by a microcephalic skull and retarded growth of the bones.

Etiology.—This form of idiocy is congenital, and in the majority of cases it will be found that both parents were past middle age when the child was born, but nothing further is known of its etiology. The disease appears with equal frequency in all classes of society. There is usually but one case in a family.

Pathology.—The brain is smaller and lighter in weight than normal, the fissures are defective, and there is evidence of faulty development of the cortex. Usually there are abnormalities of the palate, ears, and fingers. Malformations of the heart, such as incomplete ventricular septum and patent ductus arteriosus, are not uncommon, although malformations of other viscera are rare.

Symptoms.—In early infancy, the facial expression and backwardness in physical development are the only noticeable signs. The

53

Fig. 87.—Mongolian idiocy; child aged twenty months.

Fig. 88.—Mongolian idiocy; patient aged nine months.

head is flattened from before backward, the fontanelles remain open longer than usual, the nose is broad and flat, and the eyes are more widely apart than normal, are prominent, and slant obliquely, so that the palpebral fissures extend upward and elevate the outer canthus.

The peculiarities of the face in these cases, together with mouth breathing and the abnormally large tongue which protrudes from the open mouth, often make one suspect cretinism; but the skin is soft and velvety in the early stages, and the hair neither dry nor brittle.

Adenoids are sometimes thought of as a cause of the mouth breathing; but this is due to narrowing of the nasopharyngeal vault produced by anteroposterior narrowing of the skull and prominence of the upper cervical vertebræ, all of which can readily be appreciated by making an examination for adenoids. The hands are short, the little finger is also short, and frequently curves inward over the ring finger. Abnormalities of the bones of the hands and wrists are demonstrable by x-ray examination.

The muscles of the body are flabby, and the joints all show evidences of preternatural mobility. Growth is very slow, and mental development markedly retarded. Dentition is delayed, the teeth usually not appearing until the fourth or fifth year when the child can walk and talk.

Prognosis.—There are mild, moderate, and severe cases of Mongolian idiocy. The severe cases usually die in early childhood and before they are three years old; those moderate in degree may live past puberty, and do fairly well in institutions; while a few of the mild type gradually improve mentally until they show a fair amount of intelligence, and may reach adult life.

Diagnosis.—The facial expression of the Mongolian idiot is so characteristic that a mistake in diagnosis is most unlikely. Other diagnostic points are the shortness and incurvation of the little finger and the extreme flexibility of the joints. Mongolian idiocy may sometimes be mistaken for cretinism; but thyroid therapy has no effect on the Mongolian idiot, and close examination of these children reveals other differential points, such as the slanting eyes, the condition of the hair and skin, and the absence of myxedema.

Treatment.—There is no medical treatment that exerts any influence on the affection. Massage, fresh air, and careful regulation of the diet, combined with the best possible hygienic conditions, are of benefit in maintaining the child's health and strength.

DISEASES OF THE NERVES.

MULTIPLE NEURITIS.

Multiple neuritis, except the postdiphtheritic form, is a very rare disease in childhood. It is either secondary to certain infections,

diseases and poisons, or may occur as an idiopathic disease. Its most frequent cause is diphtheria, which gives rise to some form of neuritis in from 5 to 12 per cent. of all cases. In addition, multiple neuritis has developed after typhoid fever, and, more rarely, after scarlet fever, measles, malaria, influenza, erysipelas, chicken-pox, pneumonia, and tonsillitis. Of the poisons, alcohol, phosphorus, arsenic, and lead, all produce neuritis, but these forms are seldom seen in childhood.

Diphtheritic Paralysis.—Diphtheritic paralysis is a frequent complication of diphtheria, and is estimated variously at between 5 to 12 per cent. of all cases. This proportion is thought to have been increased by the use of antitoxin. It must be remembered, however, that the death rate of the disease has diminished, and that many children who previously would have died from a severe form of diphtheria are now saved by antitoxin, and these cases may later show paralysis. Unless the serum is administered very early it will have little effect, as it does not seem to give the same protection to the nervous system as to the rest of the body. Neither does the severity of the infection modify greatly its frequency; for, although most commonly observed in the severe types, yet it often occurs after mild infections. In fact, the paralysis may sometimes be the first sign of diphtheria, the throat symptoms having been so mild as to have escaped notice. Paralysis usually sets in two to three weeks after the disease has subsided, although it may develop during its course.

Pathological Anatomy.—The most extensive pathological changes are found in the peripheral nerves. These changes may be both interstitial and parenchymatous, the latter being the most pronounced. There are degeneration of the parenchyma and inflammation and proliferation of connective-tissue cells which cause cylindrical and fusiform swellings on the trunks of the nerves, appearing most frequently on the smaller nerves in the muscles and skin. Similar degenerations have been observed in the cranial nerves, as well as inflammatory changes in the gray matter of the spinal cord, and hemorrhages and hyperemia in the white matter and spinal ganglia.

Symptoms.—The paralysis is quite variable. It may either be limited to isolated groups of muscles or more generalized, involving numerous spinal and cerebral nerves.

Three different forms of the affection may be distinguished. In the early and milder form, paralysis of the soft palate develops first, often making its appearance during the acute stage of the disease following the angina; or it may not appear until one to three weeks have elapsed. The speech becomes nasal in tone, and unintelligible. On attempting to swallow fluids, some is regurgitated through the nose, and the ingestion of solid food is markedly impeded. Upon examination the soft palate is seen to be flaccid, immobile on phonation, and exhibiting with difficulty a reaction of degeneration. Most frequently there is anesthesia of the mucous membranes, with corresponding absence of the pharyngeal reflex.

The paralysis may extend, and involve the deep pharyngeal and

laryngeal muscles. In consequence of the failure of the epiglottis to close during deglutition, of the anesthesia of the mucous membranes, and of the regurgitation of food through the nose, there is great danger of aspiration of food into the larynx, giving rise to fatal bronchopneumonia. With involvement of the recurrent laryngeal nerve there is hoarseness, or even aphonia.

The vagus fibers supplying the heart may be suddenly affected and cause a slow pulse (rare), or, more frequently, arrhythmia and a rapid pulse rate, due to paralysis of the depressor fibers. Sudden death may ensue from this cause. In other cases the paralysis may extend to the eyes, involving bilaterally the ciliary muscles, causing inability to accommodate the eye, to read, or to do any fine work. The reaction to light and to accommodation is, however, usually present. Other ocular palsies are rare. The abducens paralysis is the most frequent; the oculomotor or trochlear the least common. The deep tendon reflexes of the lower extremities may be lost early in the disease. In a few cases they may be exaggerated with an ankle-clonus.

In the second form—the more severe type—the involvement is more general, extending to the extremities either as ataxia or as true palsy with sensory disturbances. Ataxia appears about four weeks after the paralysis of the soft palate. The gait becomes ataxic, and Romberg's sign can be elicited. The deep reflexes are abolished, but in this form there is no paralysis in the extremities; in fact, the muscular strength may be perfectly normal. Paresthesias are complained of; but sensory disturbances, except loss of muscle sense, are usually not pronounced.

In the third, or most severe form, there are extensive atrophic flaccid palsies with a positive reaction of degeneration. The muscles of the neck, the trunk, the intercostals, and the diaphragm may likewise be involved. Paralysis of the phrenic nerve gives rise to cyanosis and severe dyspnea which may suddenly cause death. The breathing becomes Cheyne-Stokes in type, accompanied by rapidly increasing edema of the lungs. This is the most common cause of death, the mortality being 76 per cent. in 33 cases collected by Ross.

A facial palsy has also been observed; sensory disturbances are quite marked; hyperesthesias and anesthesias are extensive. Bladder and rectal disturbances are rarely present. Trophic disorders of the skin may appear. In grave cases, marked mental depression has been observed.

Course and Prognosis.—The course of the disease is usually a favorable one, and ends in complete recovery. Paralysis of the vagus and phrenic nerves, aspiration pneumonia, and general inanition are to be feared, and may be rapidly fatal; but all danger to life is generally past after six to eight weeks. The paralysis of the soft palate and accommodation recedes, and at the end of three or four months the symptoms have usually all cleared up. Occasionally some remains of the paralysis, especially of the extremities, will persist for from eight

months to a year. Complications, such as nephritis and endocarditis, make the prognosis more grave.

Treatment.—In the majority of cases of postdiphtheritic paralysis, the treatment consists of general tonics and palliative measures. When the paralysis has progressed beyond simple paralysis of the soft palate, absolute rest in bed should be ordered, and all possible strain upon the heart prevented. The paralysis may be so extensive, and the danger of aspirating food into the larynx so great, as to necessitate feeding by gavage. The tube can be passed either through the mouth or the nose. Usually from 8 to 10 ounces of food can be given at intervals of four to six hours. If sufficient nourishment cannot be administered in this manner, and the nutrition suffers, nutritive enemata may be required. Cracked ice should be given to relieve the thirst.

Medication.—Of drugs, strychnine is the most beneficial. It should be administered hypodermically at three-hour intervals in doses of $\frac{1}{300}$ of a grain for a one- to two-year-old child; from $\frac{1}{200}$ to $\frac{1}{150}$ of a grain for a two- to four-year-old child; from $\frac{1}{150}$ to $\frac{1}{100}$ of a grain after the fourth year. If strychnine can not be used hypodermically, tincture of nux vomica may be given in corresponding doses. For rapid heart action and restlessness, small doses of tincture of strophanthus combined with codein are indicated. To ward off threatened cardiac paralysis, morphine should be administered hypodermically together with strychnine. When respiratory paralysis seems imminent, artificial respiration should be instituted together with the giving of strychnine. In these severe cases, large doses of diphtheria antitoxin have appeared to have some beneficial results.

In the acute stage all hydrotherapeutic measures are contraindicated. When, however, the paralysis begins to recede, electricity, massage, and mechanotherapeutical procedures should be begun. Mild galvanic stimuli should be applied to the extremities and the soft palate. Frenkel's exercises should not be begun until after the heart has fully recovered.

Multiple Neuritis, Postinfectious (Non-diphtheritic).—Other infections which, as we have seen, may give rise to paralysis, are typhoid fever, in rare cases scarlet fever, measles, influenza, malaria, erysipelas, chicken-pox, pneumonia, tonsillitis, tuberculosis, and syphilis. Neuritis following these infections is characterized by paraplegia, either limited to the peroneal group of muscles or, in some cases, to the thigh, or it may extend to the arms, giving rise to paralysis of both arms and legs, in which, however, there is no predilection for the group of muscles supplied by the radial nerve.

Palsies of the vagus, so characteristic of the diphtheritic form, rarely occur. As the disease progresses in the peroneal group of muscles, there is resulting weakness which subsequently produces foot-drop. On walking there is the typical "steppage-gait." The paralysis may be confined to this group of muscles or may extend to the thighs and arms. Involvement of the muscles of the trunk is rare. There is

resulting loss of reflexes, both superficial and deep, in the involved area, a positive reaction of degeneration, and muscular atrophy. Sensory disturbances are marked. Hyperesthesia and anesthesia are complained of. Trophic disturbances are sometimes observed.

Multiple Neuritis, Toxic.—Alcoholic Neuritis.—This is exceedingly rare in children. Only a few cases have been collected from the literature. The course of the disease is similar to that in the adult.

Lead Neuritis.—Neuritis due to lead poisoning is more frequently observed in children than alcoholic neuritis. The children become exposed to lead through drinking cups and toys. The course of the disease differs but little from that in the adult, except that the peroneal muscles of the legs are first attacked, and this gives rise to foot-drop. The arms are later involved, the distribution being that of the radial nerve, then the extensors of the fingers and hand. The paralysis is a flaccid one, with loss of reflexes, a positive reaction of degeneration, and muscular atrophy. There are no sensory disturbances. Bladder and rectal disturbances are negative.

As in the adult, lead colic is frequently complained of. Anemia and the lead line are often present. Encephalitis with convulsions, hemiplegia, and optic neuritis have all been observed.

The prognosis is good if the poison can be completely eliminated.

Arsenical Neuritis.—This type of neuritis has occasionally followed the administration of Fowler's solution in the treatment of chorea and other maladies.

It is characterized by symmetrical atrophic palsy of the arms and legs which develops rapidly, and is accompanied by neuralgic pain and hyperesthesia. In the legs the peroneal group of muscles are especially apt to be involved; whereas in the arms it is the muscles supplied by the radial nerve. The nerves and muscles are painful on pressure. There are muscular atrophy, an absence of reflexes, and a reaction of degeneration. Trophic disturbances of the skin—hyperidrosis, glossy skin, and pigmentation—are quite characteristic.

FACIAL PARALYSIS.

The facial nerve may be affected anywhere along its course, either within the cranium, within the bony canal, or after its exit from the cranium. Obstetrical facial palsy, which results from injury to the nerve at birth, either by pressure of the forceps or some obstacle to the passage of the head through the pelvis, has already been described (see page 111).

Other etiological factors involving the portion of nerve peripheral to the exit from the cranium are cold and exposure to dampness as seen in the rheumatic type, the pressure of enlarged lymphatic glands, mumps, and trauma, such as a severe blow on the ear, or following operations on glands or tumors at the angle of the jaw. A common cause is an inflammation of the nerve within the Fallopian canal produced by disease of the middle ear. This is seen most frequently

after chronic otitis media, especially when there is caries of the petrous portion of the temporal bone, due very commonly to tuberculosis.

Intracranial diseases, such as basilar meningitis, tumor, or a fracture of the skull, may occasionally give rise to facial palsy. The paralysis following these intracranial lesions is usually complicated by other basal palsies and cerebral symptoms. The auditory nerve is also likely to be affected.

Symptoms.—The symptoms depend upon the portion of the nerve affected. If the involvement is peripheral, there is paralysis of all the muscles on one side of the face, including those of the forehead and those about the eye. The affected side of the face becomes flaccid, in consequence of which the mouth is usually drawn toward the unaffected side. The face is smooth; the nasolabial fold is obliterated; the child is unable completely to close the eye (lagophthalmus). Any voluntary movement of the side of the face involved is impossible.

There is inability to wrinkle the forehead, to pucker the lips, to contract the eyebrows, to whistle, or to puff out the affected cheek. Nursing and mastication are interfered with. If the paralysis is complete there is difficulty in deglutition and articulation. Inequality of the face is evident when the muscles are brought into action, as in laughing or crying. Sensory disturbances do not appear. The electrical reactions depend upon the extent of injury, varying from diminished electrical irritability to a reaction of degeneration.

If the involvement be within the bony canal, and is the result of a previous middle-ear disease, there is usually a history of discharge from the ear and some deafness. The symptoms are the same as those mentioned above from peripheral involvement. As the chorda tympani is given off within the canal there is, in addition, a disturbance of the sense of taste in the anterior third of the tongue, together with a diminution of the salivary secretion. If the involvement be intracranial, due either to a basilar meningitis, a tumor, or a fracture, the auditory nerve is usually similarly involved, and in addition there are cerebral symptoms. If the lesion is central and above the nuclei of the seventh cranial nerve, the superior branch innervating the muscles of the forehead usually escapes, the electrical reactions are normal, and there are generally paralyses of the extremities.

Prognosis.—In the rheumatic form, as a rule, recovery takes place in several weeks or months. In palsies due to middle-ear disease the outlook is less favorable, and permanent paralysis is likely to result in muscular contractions which give rise to spasms and twitchings. With respect to palsies which are caused by intracranial involvement, the result depends upon the etiological factor.

Diagnosis.—In the majority of cases, the diagnosis of facial paralysis offers no difficulty. To get a clear conception of the case, its cause, the location of the lesion, and the extent of the involvement must be

determined. It is also highly important to differentiate between a peripheral lesion and a central one above the pons.

Treatment.—The treatment depends upon the causative factor. In the rheumatic form, hot applications, local bleeding from behind the ear, and blistering are extremely beneficial, in conjunction with the administration of the salicylates. When due to ear disease, appropriate local treatment should be begun, and massage and electricity resorted to in all stubborn cases.

CHOREA.

Synonyms.—St. Vitus' Dance—St. Anthony's Dance—Sydenham's Chorea—Chorea Minor.

Definition.—Chorea is an affection of the nervous system, characterized by incoördination and paresis of the muscles of the body, and by a tendency to cardiac complications. Involuntary muscular movements, twitchings, and emotional instability are among the prominent features of the disease. It is now regarded as infectious, and may occur sporadically or in epidemics.

Many varieties of chorea have been described, grouped according to the prominence of one or more special symptoms. Among these may be mentioned chronic progressive chorea, chronic adult chorea, chorea major, congenital and senile chorea, posthemiplegic chorea, chorea gravidarum, and choreic insanity. Dubini's disease, or electric chorea, is a form marked by sudden spasms.

Etiology.—The specific organism which produces chorea is unknown; but because of the frequent association of this disease with rheumatism, and the tendency to cardiac complications which it exhibits, it is believed to be closely allied to the microörganism which causes rheumatism. This close association of the two diseases is shown in the following summary, given by Fischer, of cases that have been reported.

Of Steiner's 252 cases, 4 suffered from rheumatism; of Sachs' 70 cases, 6; of Sinkler's 279 cases, 37; of Crandall and Holt's 146 cases, 63; and of Fischer's 100 cases, 25.

There is also in these cases a marked tendency to tonsillitis, and in a large majority of instances of chorea we find hypertrophied and diseased tonsils, all of which serves to link rheumatism and chorea closer together, and to lead to the suspicion, or belief, that the infecting organisms which produce these diseases gain entrance to the system *via* the tonsils.

Fright and nervous shock have been thought to play an important part in the etiology of chorea; but it seems more probable that these should be regarded as immediately exciting causes rather than predisposing factors. In epidemics, especially those occurring in institutions, imitation may be responsible for a certain number of cases, but only as an exciting factor.

Among other causes leading to chorea may be mentioned overwork at school, reflex irritation from pruritus, adenoids, polypi, phimosis,

eye strain, intestinal parasites, and menstrual disorders. Heredity is another important predisposing factor, and in many cases the child's neurotic tendencies can be traced to neuropathic parents, or we find a family history of gout, rheumatism, tuberculosis, or other constitutional dyscrasia. Chronic malaria, anemia, and chronic gastro-intestinal disturbances are common findings. Masturbation is practised by many of these children, and the habit is frequently a forerunner of the disease.

One of the most marked characteristics in the etiology of chorea is sex, for girls are affected twice or even three times as often as boys. In a series of cases reported by Sinkler, 232 of 328 were in females. Chorea is especially common from the seventh to the fifteenth year, is rare before the third year, is practically never seen in infants, and in adults, as a rule, is observed only in pregnant women.

In the eastern part of the United States the majority of cases occur in the spring months, and fewest in the early winter, the seasonal curve corresponding to that of rheumatism. Chorea is most common among Russian Jews, and rare among negroes.

Pathology.—The pathology of chorea has not been determined, and there is at present great diversity of opinion on this subject. It is obvious that there can be no extensive or permanent lesions in the nervous system, because complete recovery usually ensues a few weeks after the onset of the affection. The process is probably toxic in nature, and affects the central nervous system, especially the motor cortex about the Rolandic area.

It is reasonable to assume that in those cases associated with rheumatism, and in which there is cardiac involvement, the same toxin which affects the nervous system also damages the heart. In fatal cases, the diplococcus of Poynton and Paine has been isolated from the pia mater, and in those fatal cases associated with endocarditis, capillary emboli were found in the brain. Few cases, however, show any pathological lesions, and no constant or permanent changes have been found in the cases which have been studied postmortem.

Symptoms.—The manner of onset in chorea is very variable, and an attack may be preceded by a prodromal period of several days or even a week. As a rule the disease comes on gradually, the child being at first a little more "nervous," irritable, and fretful than usual, and crying on the slightest provocation. Restlessness at night and slight twitchings during the day may be observed, also awkwardness and clumsiness, especially in handling objects. Spoons, pencils, and books are unaccountably dropped, and the child is scolded for carelessness. It becomes increasingly difficult, and later impossible, for the patient to use its tumbler, knife and fork, pencil and pen, or to dress, most of all to execute fine movements, such as buttoning the clothes. Other delicate movements, such as threading needles or sewing, are also impossible, and the child frequently trips and stumbles.

Following these symptoms the unmistakable signs of the disease

are usually recognized, the boy or girl exhibiting awkward involuntary and irregular muscular movements, which are intensified by any effort on the part of the child to control them. These incoördinate jerking muscular contractions, which may involve all or any part of the body, constantly take place while the patient is awake, but involve alternately various groups of muscles, and cease only when the child is sound asleep. The muscles of the hands, arms, legs, and face are the ones most commonly affected, and the tongue may become involved to such an extent as to interfere with speech.

These movements are never rhythmical or symmetrical, and one extremity may be affected to a greater extent than its fellow. The usual order of involvement is first the right arm, left arm, right leg, and left leg. When the shoulders are affected they jerk up and down, the arms rotate from side to side or swing backward and forward, the hands are flexed, extended, pronated, and supinated in turn, while the fingers are rigidly contracted and bent, rendering the child unable to hold anything.

The lower extremities may be involved to such an extent that the child can neither sit, stand, nor lie still, and in severe cases it must be restrained to prevent it from injuring itself. The face may assume a number of expressions, and is constantly distorted. A systolic heart murmur is frequently heard, and at the height of an attack the pulse may be arrhythmical, and the respirations irregular.

As a rule the choreiform movements extend to all parts of the body, but in 25 to 33 per cent. of the cases they are limited to one side of the body, the other manifestations of the disease being just the same as when all of the muscles are involved. Hemichorea is, however, usually regarded as more serious, because it is often associated with paresis of the affected extremities, also with psychical complications, such as melancholia and hallucinations.

In all cases of chorea there is weakness of the muscles, yet exhaustion is rare, and the child does not complain of being tired. This paretic state of the muscles can readily be demonstrated by asking the patient to grasp the examiner's hand. To test for incoördination the child should be told to extend the arms outward, and then to touch the tip of the nose with each index finger alternately, or to bring the tips of the index fingers together quickly after the arms have been extended outward.

The facial muscles may be so affected that the brow cannot be wrinkled, the eyes kept shut, or the tongue held out for more than a few seconds. In severe cases the child is unable to walk, talk, chew, or control the bowels and bladder; it appears to be paralyzed, and presents a pitiful spectacle.

There is, as a rule, well-marked secondary anemia in chorea. The urine usually contains an excess of uric acid. Herter has demonstrated the presence of hematoporphyrin in the urine—in both chorea and rheumatism. Heart murmurs are frequently heard; and, while some of them may be of anemic origin, the majority are, unfortu-

nately, due to intercurrent endocarditis, being diastolic or apical-systolic in time. Pericarditis may also appear as a complication of chorea.

There is marked disturbance of nutrition, and the appetite is usually impaired. Pain is not uncommon, and in many cases is of rheumatic origin. The mental state of these patients varies. At the onset there is hyperexcitability and irritability, later the children become emotional, laughing or crying upon slight cause. In very severe cases, melancholic or maniacal symptoms may develop. Unless complicated by rheumatism or endocarditis, there is no elevation of temperature in chorea.

Diagnosis.—The diagnosis is readily made, and is based upon the characteristic, sudden, irregular, involuntary, and spasmodic movements of the body and on the abnormal movements of the tongue. Habit spasm may sometimes simulate chorea; but in habit spasm the movements are to some extent under the control of the will, while any attempt to control choreiform movements will only exaggerate them. Hemiplegia may be simulated by the pseudoparalysis in chorea; but chorea may be ruled out by the absence of spasticity and increased reflexes.

Choreic movements are sometimes associated with infantile cerebral palsies; but true chorea can here be excluded because of the increased reflexes and spasticity which accompany infantile cerebral palsies. Imitative choreiform movements are of short duration, and in hysterical chorea the movements are to a certain extent purposeful, not so irregular as in true chorea, and other symptoms of hysteria are demonstrable.

Course and Duration.—Chorea is a self-limited disease, and, if untreated, usually ends in recovery in from six to ten weeks. The active symptoms yield in about four weeks; but the condition of the child's nervous system influences both the course and duration, and in a few cases chorea may last for six months, or become chronic and persist indefinitely. In other cases only certain local spasms may continue for a long or short time. The intensity of the attack has apparently no effect on its duration, and many severe cases under careful treatment recover within the usual time.

Prognosis.—In the cases without cardiac involvement, the prognosis is uniformly good, and complete recovery the rule; but relapses are quite common, and render the prospect of permanent recovery rather unfavorable. Relapses are most common in the spring following the first attack, and some children have an attack every year for three or four years in succession. The outlook is serious in all cases associated with delirium and prostration, as well as in those cases complicated by cardiac lesions.

Treatment.—Since chorea is known to occur in epidemics, it is wise to isolate the child affected from other children, and especially is this advisable in boarding schools, asylums, hospitals, and other institutions where the inmates are closely thrown together. This measure

is particularly important in the case of girls between the ages of seven and fifteen, when they are most susceptible to the affection.

The management of a case of chorea depends to some extent upon the severity of the attack; but in any case the general care of the patient is of quite as much, if not of more, importance than the administration of drugs. Rest in bed for two to four weeks is necessary in severe cases, and restraint may be required to keep the patient from falling on the floor.

The child's room should be sunny and airy. All excitement should be avoided, and no visitors should be permitted in the room; but the patient may be allowed to play with a few toys, and the attendant should endeavor to amuse and entertain it in a quiet way. After the coarser movements have ceased, the child may be allowed to sit up and go about the room for a little while each day, gradually increasing the time spent out of bed as improvement sets in.

If there is any evidence of cardiac involvement, rest in bed should be enforced for a longer period, and the patient should not be allowed to sit up until the physician feels assured that the lesion is a permanent one. After the child is out of bed, a reasonable amount of outdoor life should be advised, but the mother or nurse must be careful not to let the patient become fatigued, either physically or mentally. It is a good plan to have these children take one or two naps during the day.

Even in very light cases the child should be taken out of school, and it should never be subjected to ridicule or any punishment. Playing with other children should be prohibited until some time after convalescence. Exciting books, games, and pictures should be forbidden, and an effort made to surround these children with a quiet but interesting environment. Sometimes it is necessary to take the child away from home for awhile, particularly an older child.

In severe cases, hospital treatment and care are usually more beneficial and satisfactory than home management. Cold douches or warm baths and massage daily are of advantage in chorea, and some authorities recommend a daily colonic flushing.

The diet should be a bland one, the food depending upon the age and digestive capacity of the child. Milk is one of the best articles of diet for these children, and cereals, weak broths, chicken, cooked fruits, and fresh vegetables may be given. Tea and coffee, rich soups, sweets, pastries, and other indigestible foods should be prohibited.

The two best drugs in chorea are iron and arsenic; but in the rheumatic cases the salicylate of soda, in 10-grain doses, combined with an equal dose of bicarbonate of soda, three times daily, is most effectual.

Arsenic is given three times a day in the form of Fowler's solution, beginning with 2 or 3 drops in plenty of water after meals, and increasing the daily dose by 1 drop until 9 to 15 drops are taken three times a day, unless gastro-intestinal disturbances, puffiness under the eyes, and headache appear, when the arsenic should be imme-

diately stopped for a few days, then resumed in small doses as before. It is never wise to give more than 15 drops three times a day, and this maximum dosage should be kept up for only a few days, and then gradually diminished to 5 drops three times a day.

In many cases arsenic lessens the severity of the symptoms and shortens the attack; but, if there are no appreciable results after two or three weeks of its administration, it might as well be discontinued. The urine should frequently be examined while Fowler's solution is being given, and, should albumin appear, the arsenic should be immediately stopped.

Iron may be given in the form of the citrate, $\frac{1}{2}$ to 1 grain, or in one of the various preparations, such as the peptomangan (Gude's), 1 dram, or $\frac{1}{2}$ grain of ferri sulphatis, three times a day after meals. Chloral, grains 1 to 3, or veronal or trional, grains 3 to 5, may be given once or twice daily to older children for their sedative effect.

In severe cases morphine sulphate, $\frac{1}{20}$ to $\frac{1}{10}$ of a grain, hyoscine hydrobromate, $\frac{1}{200}$ to $\frac{1}{100}$ of a grain, or chloral hydrate, 5 to 10 grains, may be necessary to quiet the patient. Antipyrine, 15 to 20 grains daily, and strychnine sulphate, $\frac{1}{60}$ of a grain three times a day, are useful in some cases. Some children respond well to the sedative effect of sodium bromide, 3 to 5 grains every three hours. The bowels should be regulated by dram doses of magnesium sulphate, or $\frac{1}{2}$- to 1-dram doses of aromatic fluid extract of cascara sagrada.

In cases of rheumatic origin it is wise to confine the treatment to antirheumatic lines, giving sodium salicylate or aspirin in 5-grain doses four times a day, and restricting the diet accordingly.

The treatment which a child receives during convalescence from chorea is of the utmost importance because of the marked tendency to relapse, especially during the spring months. Work in school should not be resumed for several months after recovery, and these children should be sent to the country or seashore to recruit.

The diet should be very nutritious, the warm or cold baths should be continued, and mild exercise indulged in. The anemia should be combated by the use of cod-liver oil, $\frac{1}{2}$ to 1 dram, or the syrup of ferrous iodide, 5 to 20 drops, three times a day after meals.

Any source of reflex irritation, such as eye strain, hypertrophied tonsils and adenoids, or phimosis, should be removed. For children who are subject to recurrent attacks of chorea, courses of the salicylates or of Fowler's solution are advisable during the periods of apparently complete recovery. Rheumatic cases subject to relapses sometimes remain free from attacks indefinitely if removed from a damp, cold climate to a warm, dry one.

TETANY.

Tetany is a motor neurosis, marked by persistent or intermittent, and more or less painful, spasms of the muscles of the upper and lower extremities. It is a condition rather than a disease, and is believed

to be a symptom of several different affections. Most cases occur in the spring.

Forms of the Disease.—Tetany may occur in either adults or children, and in Australia it has been observed in epidemics. There is also a surgical form. In attempting to classify the malady according to symptoms, we find one group of cases in which the muscular contractions are persistent, and another in which they are intermittent.

Etiology.—The affection is not so common in America as it is in Austria and Sweden, and some other parts of the world; but it is probably more frequently met with in this country than is generally supposed. The majority of cases occur in infants between the third month and the second year. It is less common during later childhood, and is rarely seen in adults. Children of the poorer classes, whose surroundings are unhygienic and unhealthful, furnish most of the cases, and in more than 50 per cent. of all instances of tetany there is an associated rachitis which is believed to be an important etiological factor.

Beyond this the etiology of tetany is very obscure. It is usually preceded by some depressing or debilitating condition, and never affects healthy children. Among the different etiologic factors which have been mentioned are gastro-intestinal disturbances, infection and infestation of the intestines by parasites, the transmissible diseases, poisoning by alcohol, lead, and ergot, extirpation of the parathyroid glands, or deficient parathyroid metabolism. In some cases tetany is supposed to be a manifestation of hysteria.

Many observers believe tetany to be due to a deficiency of calcium salts or a disturbance of calcium metabolism, and artificial tetany has been relieved by the injection of calcium. In cases of the disease there is a diminution of calcium salts in the brain, an increase of calcium phosphate and ammonia in the urine, and an increase of ammonia in the blood. Tetany is also believed to be of toxic origin.

Pathology.—Few pathological changes and no characteristic lesions have been observed in the small number of cases of tetany which have come to autopsy. Dercum mentions as the conditions to be found postmortem proliferation of the neuroglia, atrophy in the ganglion cells and nerve fibers, serous exudation into the cervical cord and ventricles of the brain, spinal extradural hemorrhage, and sclerotic changes in the nervous system. Other observers have reported periarteritis, phlebitis, bronchopneumonia, chronic gastro-enteritis, tuberculosis of the brain, and hemorrhages into the parathyroid glands.

Symptoms.—The most important symptoms of tetany are the result of mechanical or reflex excitability of the spinal cord and peripheral nerves, and they may appear suddenly or gradually. In some cases there is a prodromal period, during which the child may be depressed, dull, stupid, it may complain of headache or dizziness, and of pain in the extremities. This is quickly followed by a tonic spasm of the muscles, most marked in the arms, which are held close to the chest, and flexed at the elbow, wrist, and finger-joints. The rigidity is some-

times so pronounced that it is impossible to overcome the resistance
of the contracted muscles.

The muscles of the leg are less commonly affected, and those of
the face, neck, and trunk are rarely involved. The hand assumes
a characteristic position, known as the "accoucheur's" or "writer's
hand," the fingers being flexed on the metacarpophalangeal joints,
while the phalanges are extended, the thumbs adducted, and the wrist
is acutely flexed with the hand turned to the ulnar side. The hands
may also take the position seen in driving.

The foot may be extended or hyperextended, and the toes flexed.
Often the feet take the position seen in talipes equinus. In rare
cases, all of the muscles of the body may be involved (Fig. 119).
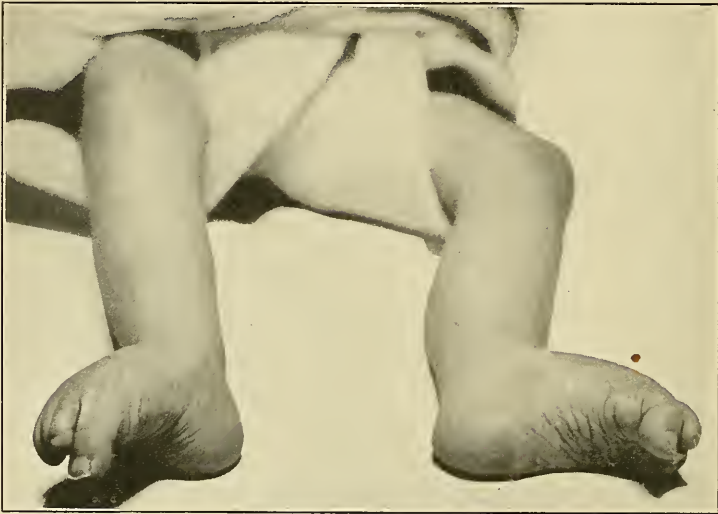
Fig. 89.—Tetany.

During the periods of latency the contractures lessen, and may
disappear to such an extent that the only noticeable symptom is
hyperexcitability of the muscles and nerves. The affection may
last but a few hours or for several weeks, or the child may have a suc-
cession of periodic attacks. During the latent periods in which there
are no active symptoms, certain phenomena may be elicited which
will enable the practitioner to determine that he is dealing with
tetany.

Trousseau's sign is a spasm of the muscles of the forearm and
fingers produced when the arm is compressed by an elastic band.
Chvostek's sign is manifested by a contraction of the muscles of the
face or forehead when they are tapped; sometimes, as a result of the
tapping, other muscles of the body also contract. Erb's sign is evoked
by increased electrical excitability of the peripheral nerves, muscular

contractions being produced by very weak currents. Schultz's sign is elicited by stroking the skin over the zygoma, whereupon contractures appear, similar to those which constitute Chvostek's sign, but Schultz's phenomenon is observed only in the most severe cases, therefore is seldom demonstrated.

Pain is usually present during the spasm, and may be so severe as to make the child cry out. Any attempt to overcome the spasm greatly increases the pain, and pressure on the contracted part is also painful.

Among the other symptoms of sensory disturbance are tinnitus aurium and vertigo. There is no loss of consciousness unless convulsions occur; the urine is rarely affected; the pulse is but slightly accelerated; there is no elevation of temperature unless it is caused by some associated or underlying condition, or by the toxemia usually present. Laryngospasm is a common occurrence.

Diagnosis.—This offers no difficulty, being based upon the bilateral contractions of the hands and feet which are so characteristic of the disease, also upon evidences of increased excitability of the peripheral nerves, as expressed by the various signs above mentioned. A history of preceding gastro-intestinal disease, the existence of rickets, and the fact that there is no loss of consciousness, are all important points which must be taken into consideration.

Tetanus may be excluded by the location of the contractures, the absence of trismus, and the intermittency of the spasms. Meningitis may be ruled out by the lack of mental symptoms, while lesions of the brain may be dismissed from consideration by the fact that there are no local symptoms, and that the contractures in tetany are bilateral and symmetrical. Trousseau's sign is demonstrable even during the latent periods, and enables one to diagnose the affection when spasms do not occur.

Prognosis.—Tetany is not a fatal disease unless associated with general convulsions, although death may ensue as a result of the underlying condition; therefore the prognosis depends, to a great extent, upon the cause of the affection in each individual case. As a rule, cases of tetany due to malnutrition or intestinal toxemia tend to recover under proper treatment, while tetany caused by organic lesions in other organs or due to eclampsia is more serious, and the outlook not so favorable.

Treatment.—The treatment of tetany should be directed chiefly to the underlying cause which must be eliminated as quickly as possible. Rickets, if present, should be promptly treated by regulation of the diet and the administration of remedies suggested in the discussion of the treatment of that disease. Furthermore, since intestinal toxemia and malnutrition are important factors in the production of tetany in children, any disturbances of the gastro-intestinal tract should be corrected, and the diet and nutrition regulated.

An initial purge of castor oil, 1 to 3 drams, should be given, and at intervals of five days or a week these children should take 1 or 2

54

grains of calomel in divided doses. An enema of salt solution or soap-suds is often productive of good in acute cases.

These infants should be kept upon breast milk whenever practicable, and artificially fed infants should be given a milk mixture carefully modified to meet the dietetic conditions.

In addition to this regulation of the diet, the physician should see that the child's surroundings are as hygienic as possible, and that it gets plenty of fresh air and sunshine. During an attack various measures may be employed to relieve the spasm. The child must be kept quiet and warm, and all disturbing noises should be prevented. A hot bath at 110° F. may be given twice or three times daily for its relaxing effect during the attack. In the interval between attacks, a salt bath should be given each evening, followed by an inunction of olive oil.

The most valuable sedatives are calcium bromide, sodium bromide, and chloral hydrate. Calcium bromide, which may be given in 5- to 10-grain doses every four hours, not only counteracts the hyper-excitability, but also supplies to the system the calcium which it requires. Sodium bromide may be given orally in 1- to 3-grain doses every two hours, or may be combined in 5- to 10-grain doses with chloral hydrate, grains 1 to 3, and be given by rectum.

In these cases parathyroid extract usually has no perceptible effect, and the administration of calcium lactate, 2 to 5 grains at a dose, is followed by no change whatsoever in the condition.

During convalescence, cod-liver oil, 20 to 60 drops, and iron and ammonia citrate, ½ to 1 grain, or syrupi ferri iodidi, 5 to 10 drops, may be given after meals. Outdoor life in the country is a valuable aid in promoting speedy recovery.

CHAPTER XXVI.

PUBERTY.

BECAUSE puberty lies just on the boundary line between childhood and adolescence, this subject is treated fully neither in classical text-books on pediatrics nor in treatises on general medicine. This lack of comprehensive study probably accounts for the fact that, with many physicians, vague notions transmitted by hear-say or empiric writings take the place of exact knowledge of the physiological and pathological changes which occur at this critical period.

What is puberty? The name, derived from pubes or pubis (hair), would apply physiologically to the time of the appearance of hair on the genitals (pubes). This coincides approximately with the establishment of the procreative faculty, which is characterized, according to sex, by ovulation or the maturing of spermatozoa, and usually, but not necessarily, with the growth of pubic hair. Here, however, local conditions only are considered, while the profound changes which actually occur involve the whole organism, body as well as mind.

In trying to time this evolution we speak, as always when growth and development are concerned, only of the average. It is a well-known fact that some girls begin to menstruate at the age of eleven, ten, or even less, and others not before their fifteenth or sixteenth year, while conception has been reported as early as the eighth year. In boys, too, manifestations of puberty are sometimes observed at twelve years of age, in others not before, or even later than, eighteen years. Equally true is it that while in some children the processes of evolution are complete in eighteen months, or even a year, in others eight to ten years may elapse before maturity is attained.

Summarizing, we designate as puberty the whole period of development between twelve and fifteen years in girls and fourteen to eighteen years in boys, and this development comprises the series of changes, physical and psychical, which transform the juvenile organism into the mature one. These great changes, let it be understood, are influenced by many diverse conditions. They may be premature or retarded, rapid or slow, intense or attenuated, according to temperament, race, climate, nutrition, heredity, sex, and social conditions.

PERIOD PRECEDING PUBERTY.

Body.—Following the steadily progressive growth of later childhood up to about the age of ten in girls and twelve in boys, the general development of the body seems for a year or two to be reduced to the minimum, as if, during this time, Nature were trying to gather strength

for the increased rate of growth which is to follow. According to the available statistics of different nations, this limitation affects the height, weight, and bony system (thorax, skull, pelvis, extremities), as well as muscular development. Limitation of growth seems to be the general law for this period, and probably holds equally true concerning the internal organs, although this has not been definitely proven except in the case of the liver.

Toward the close of this phase of life a rapid lengthening, especially of the lower extremities, takes place. The growth of the trunk is retarded, while that of the thorax is hardly perceptible; therefore the respiratory capacity and the size of the heart as compared with the height are less than at any later time. As the weight, too, increases but slowly, boys and girls of this age are apt to look ungainly, long legs, a short trunk, and leanness being their most striking physical characteristics. This puerile type, as A. Delpeuch calls it, seems essentially to be identical with that of the so-called tuberculous diathesis. One cannot but wonder whether those who subsequently contract tuberculosis retain their puerile proportions, and are thereby predisposed to the disease, or whether the failure to acquire the adult conformation is not really the effect of latent tuberculosis.

Mind.—Emerging from a period of mere imitation, the little boy or girl toward puberty begins to exercise the power of reason. The knowledge which, before this time, has been merely stored in the memory mechanically is now utilized by the mind in forming ideas, in comparing, and in generalizing. Reason and judgment, hitherto impersonal, become original, more correct, and more definite. The attention is more concentrated, reflection is clearer, imagination is curbed, and, as the sexual instinct awakens, the affections become sweeter and more intense. The feelings, less impulsive, are better controlled by a will which now asserts itself. All these different mental activities produce a more stabile equilibrium. Discipline is more readily borne; behavior, the power of application to work, and work itself are improved. The teacher congratulates himself upon the changes brought about, and parents see with satisfaction the happy modifications in the character of their child.

PUBERTY.

Body.—This preparatory period is followed by the most critical and interesting phase of life, *i. e.*, puberty. Up to this time the child has shown only racial instincts, but he now begins to develop individual characteristics, and family traits become accentuated. A rapid development of the organism as a whole sets in, not gradually, but by leaps and bounds. Height and weight increase, the muscles develop, the general bony system enlarges, and all the viscera seem in haste to acquire adult formation. Coincident with this period is the sudden development of all the organs of generation, and the forcing of their functions, some of which now appear for the first time.

There are no fixed rules as to the order in which these changes take place; in fact, they appear in almost endless variety, according to the influences of climate, race, sex, heredity, and environment. For this reason it does not seem quite correct to reckon puberty from the development of a single system of the body—for example, the reproductive. Ovulation or the forming of mature spermatozoa does not alone make a mature woman or man, but that combination of physical and psychical development which Nature at this time brings about, and which is designated as puberty proper. The length of the period is variable; but in this country the time between the twelfth and fourteenth years in girls can be accepted as the average. Boys pass more gradually into adolescence, usually between the ages of fourteen and sixteen.

As to the details of physical development, we find that growth in height usually precedes by a few years the pronounced increase in weight which takes place during puberty. After a time of slow growth during late childhood, girls grow more rapidly from their eleventh or twelfth year to their fifteenth year, while increase in weight, marked between the thirteenth and fifteenth years, sets in and continues, although much more slowly, until the age of twenty. Boys increase decidedly in height between fourteen and seventeen, with the maximum around the fifteenth year, but no remarkable gain in weight occurs, as a rule, until sixteen or seventeen. Speaking generally, we may say that until eleven years of age boys, and from then until fifteen years of age, girls exceed the other sex in weight and height; after fifteen or sixteen years of age girls again fall behind.

Corresponding with the general growth the shoulders broaden and the bony thorax enlarges markedly—as much as five inches in three years—making room for the increasing pulmonary development which oftentimes raises the maximum capacity by 500 c.c. In girls, even in those who wear no corset, it is chiefly the upper thoracic portion which enlarges, the lower costal region developing much more slowly; therefore the girl's breathing tends to the costal type while the young man's respiration is usually of the abdominal type.

The circumference of the head keeps pace with the general development; in girls between the eleventh and thirteenth years it is greater than in boys, before and after this age smaller. The bony pelvis widens more in girls, its greatest growth taking place between eleven and fourteen years, as compared with thirteen to sixteen in boys. Yet even at fifteen or sixteen the female pelvis surpasses that of the male in size, and is evidently prepared for possible gestation. Muscular development, naturally greater in boys, reaches its maximum between fourteen and sixteen years in girls and fifteen to eighteen in boys. The flesh is firmer and harder in the latter, while in the former it is infiltrated with fat, which serves to give to the body a rounded and graceful contour.

Larynx.—In *girls* the voice rarely breaks, but, on the contrary, becomes fuller and more resonant, the larynx enlarging chiefly in its

vertical diameter. In *boys* the transverse diameter increases most, the vocal cords are lengthened, and the voice, after a period during which it "cracks," is permanently pitched an octave lower than before.

How intimately this and other developmental changes are related to the inner secretion of the organs of generation (testicles) is demonstrated by boys emasculated before puberty. Their voices remain high, they are slender and narrow chested, their pelves are broad, and there is little growth of hair except on the scalp.

Thyroid Gland.—No less important is the decided influence which the thyroid gland exerts upon the growth of the whole bony system, and especially on the genital system of both sexes. In about 15 per cent. of girls this gland becomes swollen at puberty, and in 60 per cent. is temporarily enlarged when menstruation appears.

Hair.—When hair appears on the previously smooth body puberty is imminent. In girls a sparse growth begins to show a few months before menstruation, first on the pubes, but gradually covering the genitalia and axillæ. In boys from fourteen to sixteen years of age it may also appear over the chest and back, the extensor surfaces of the extremities, and upon the upper lip, cheeks, and chin. The growth of the beard is, however, not coincident with puberty; in fact, in some races this is altogether lacking.

Circulatory System.—The sudden acceleration of development at puberty may cause undue strain on the heart, and, perhaps, a disproportionate development of body, heart, and bloodvessels. Hence, in spite of the fact that cardiac growth is greater during puberty than during preceding years, irregular and feeble pulse, palpitation, shortness of breath, and vertigo are frequently complained of, while vasomotor instability is evidenced by blushing, angioneuroses, and functional albuminuria.

The Genitalia.—Decided changes now take place in the genital system. The organs become more vascular, rapidly increase in size, and mature within two years, whereas three or four years must elapse before adolescent weight and height are attained. In girls the labia majora hypertrophy and completely cover the vagina; the clitoris and the anterior part of the labia minora also increase in size, the former becoming erectile, and the vagina turgescent and more capacious.

From a cylindrical shape the uterus changes to the adult form, with larger and flattened fundus; its muscular walls and mucous lining thicken considerably, and numerous glands develop.

The ovaries double their weight from $\frac{1}{2}$ to 1 dram, and twelve to fifteen Graafian follicles gradually approach the surface, ready to enlarge and burst in turn at Nature's call. More or less coincident with these transformations is enlargement of the breasts which causes transient pricking sensations, sometimes even painful tension and twinging pains along the ribs.

Abdominal heaviness and a sense of pressure are felt in the pelvis,

also tenderness on palpation over the hypogastrium; occasionally there are flashes of heat, congestion of the face, and not infrequently, epistaxis, general lassitude, and sacral, iliac, or lumbar pains which radiate to the thighs.

Gastro-intestinal disturbances, such as anorexia, dyspepsia, and constipation, as well as nervous palpitations, insomnia, accelerated respiration, and increased perspiration, may all be noted. The girl is often irritable, extremely emotional, and feels unfit for work.

In this period between the twelfth and sixteenth years, when the whole genital apparatus is turgescent and the vital functions at high tide, the first menstruation occurs, lasts sometimes but a few hours, usually for a day or two, and ends with the liberation of the first ovum and a loss of blood less than that normally lost by the mature woman (3 to 6 fluidounces). After a few months the menses become regular and the procreative faculty is fully established.

Genital development takes place a little later in boys than in girls. After the first curly hair appears on the external genitalia, the scrotum considerably increases in size and in pigment, and its sudorific secretion becomes more noticeable. Cowper's glands, the prostate, the seminal vesicles, and the testicles rapidly approach the adult size. At this time nocturnal emissions occur, and may sometimes be painful. At first they may contain no spermatozoa; after the fifteenth year, spermatozoa are present, and recur at more or less regular intervals.

Boys pass into adolescence more gradually, and always later, than girls, other conditions being equal. Many factors influence the beginning of puberty. With few exceptions, it occurs earlier the higher the mean atmospheric temperature. An increase in heat, even when it lasts but a short time, causes more rapid growth, and a retardation of development has been observed after a fall in temperature. For instance, growth during the winter months is only slight, during spring and summer it is more rapid, particularly in height, while autumn brings a more decided gain in weight.

A sanguine or nervous temperament, large stature, strong constitution, hereditary tendency to precocity, city life, an environment of affluence, the use of alcohol, and premature sexual relations—all induce early puberty.

Latin races mature earlier than Anglo-Saxons. Those who grow up in poverty lag behind in weight and stature, the period of limited growth just before puberty being prolonged and puberty retarded, but full development is attained at about the same age as among the well-to-do.

Mind.—At puberty not only the body, but also the mind, passes through a crisis, the accompanying psychical phenomena of which are, unfortunately, often regarded with indifference or skepticism, whereas, in the interests of sound and harmonious development they should receive the most careful consideration. The period is characterized by general restlessness and a certain instability of mind, vague

aspirations rather than concrete thought dominating the mind. There is an ardent desire for knowledge. Rhythm and music, dramatic roles and poses, give exquisite pleasure, but the critical faculties seem to be more or less dormant. It is the age of ideals, of hopes and tender sentiment, of folly and imitation, of self-consciousness and over-sensitiveness. There is a proneness to exaggeration. Feelings change like the pictures of a kaleidoscope; to use Marfan's apt expression, we see a mind in revolt. Girls no longer mix so indiscriminately as before, sexual characteristics reveal themselves, and inherited traits, good or bad, become more conspicuous. A trait wholly absent at this time is not likely afterward to appear.

The girl's natural grace is enhanced, her love of dress intensified, she studies the art of pleasing. At the same time her inborn qualities become more pronounced, timidity merges into love of solitude— even shyness—natural kind-heartedness becomes unduly tender, even eager for unnecessary sacrifice. Early pride turns into haughtiness, indolence into laziness, the innocent ruse of former days buds into deception, a good entertainer becomes an incessant chatterbox. Highly sensitive, capricious, full of temper, tricks, and pranks, now exuberant, now cast down, passing from laughter to tears more quickly than sunshine to rain on an April day—such a girl is difficult to manage in school or house, and taxes the patience of a saint. Yet, after all, her faults are of passive type, as compared with those of boys.

Just as his physical development is more or less out of proportion during the period of his most active growth, so the average adolescent boy is, in a psychical sense, an inharmonious personality. He is wiser than his teacher, more experienced than his parents—more gritty, more witty—in fact, a superior being. He readily becomes hypersensitive, argumentative, contrary, overbearing, authoritative, contradicting others and himself at every instant. His chief characteristics seem singularly accentuated, almost to the point of being caricatures. He has more than the ordinary self-respect, self-reliance, self-consciousness. If naturally benevolent, he makes Utopian plans to help the whole world. If a good mixer, he is liable to become an inveterate talker, or Münchhausen. His natural cheerfulness borders on hilarity, and a lively disposition may become impulsive, violent, or even brutal.

General Health.—The general health at puberty is subject to many variations for the simple reason that, in addition to the diseases due to ordinary etiological factors, the physiological changes which now occur may become pathological if the general development is too sudden, or so disproportionate as to leave certain organs too weak for the increased amount of work now demanded of them. While statistics show that in middle-grade schools the morbidity among boys is 30 per cent., and as high as 50 per cent. in girls, the mortality is comparatively very low. Nevertheless, disturbances (functional as well as organic) should receive the closest attention, because neglect at this time may be responsible for sequelæ that will persist throughout life.

Therefore the physiological and psychical development of growing boys and girls should be carefully watched that we may guide them safely through the Scylla and Charybdis of puberty. Insufficient nourishment, uncleanliness, unhygienic surroundings, including bad light as well as bad air, a sedentary life, and faulty posture, are no doubt now, as at any other period of life, baneful conditions. Moreover, it is certain that physical overwork which lowers the general resistance, the overtaxing of the functional activity of immature organs (such as the heart, lungs, or kidneys), and mental strain upon the nervous system, the heart, respiration, muscular strength, and nutrition, are in a special sense predisposing, provocative, and aggravating etiological factors at puberty. The necessity of protecting young boys and girls against them at a time when, stirred by new sensations and ambitions, they are apt to go beyond their strength in competition with others on the athletic field or in the class-room, cannot be sufficiently emphasized.

Schlossmann and Pfaundler noted that of 600 cases, more or less closely connected with puberty, 20 per cent. suffered from disturbances accompanying menstruation, 15 per cent. from cardiac neuroses, 10 per cent. from goitre, 15 per cent. from periodical headache, 13 per cent. from neurasthenia, 13 per cent. from hysteria, 6 per cent. from epilepsy, 4 per cent. from chorea, and 5 per cent. from the albuminuria of adolescence.

The mortality from tuberculosis, according to Kirchner, increases in boys from 10 to 16, and in girls from 18 to 26 deaths to 100 total deaths. As disturbances of special organs are treated under their respective headings, we shall merely enumerate them rapidly, according to the system principally affected:

Skeleton.—Faulty position and excessive unilateral exercise, as, for instance, in playing the violin or in tennis, may cause curvature of the spine, especially when the osseous skeleton is insufficiently firm, and the muscles are weak from lack of nutrition and harmonious exercise. Genu-valgum may develop from long-continued standing. So-called "growing pains" are probably in most cases due to infection, overexertion, or slight trauma. Osteitis and periostitis are infrequent.

Skin and Muscles.—Nevi, previously unnoticed, may enlarge at puberty. Eczema, urticaria, seborrhea, and acne seem to be more frequent as the general growth becomes more rapid. As regards muscles, myositis, torticollis, and the juvenile forms of muscular atrophy or dystrophy may occur.

Circulatory System.—Cardiac disease, especially mitral stenosis, often previously unsuspected, manifests itself at puberty, while systolic murmurs over the apical and pulmonary areas, palpitation, arrhythmia, tachycardia, vertigo, and syncope may appear transitorily, and disappear with progressive development. It is sometimes difficult to distinguish these murmurs from permanent changes, therefore the prognosis should always be guarded.

The vasomotor system is ill-balanced; and disproportions between

the heart, bloodvessels, and body may now be observed. Girls are especially predisposed to chlorosis; according to some writers this deficiency of hemoglobin in the circulating blood is the result of Nature's effort to prepare for a possible pregnancy by storing in the liver iron from the blood. This form of anemia, as well as others following tuberculosis, kidney affections, excessive exercise, and repeated severe hemorrhages, or due to undernourishment, should receive prompt treatment so that harmonious development be not retarded.

Respiratory System.—A peculiar relationship exists between the genital organs and the Schneiderian mucous membrane which finds expression in the so-called nasal asthma and coryza of puberty. Contrary to former belief, adenoid growths may persist, and should be removed in every case. Attacks of angina, also of tonsillitis with its train of symptoms, are common. Spasm of the glottis, aphonia, and croupy cough appear in laryngeal affections. If the voice breaks, the vocal cords need a period of rest and should not be unnecessarily strained by shouting or efforts at loud singing. At puberty the asthmatic attacks which accompany bronchitis during childhood often absolutely cease.

Digestive System.—Disturbances of digestion are common at puberty, especially when there is a tendency to overeat or to bolt the food. Dyspepsia due either to excessive or deficient secretion of hydrochloric acid, and even of the atonic or nervous type, is rather frequent, particularly in girls. Constipation, colic, and mucomembranous enterocolitis are quite common. If the contents of the bladder and rectum are habitually or abnormally long retained (sometimes from false modesty), anteflexion or retroversion of the uterus may result; while faulty corsets and tight skirt bands are, no doubt, causative factors of visceral ptosis, especially when there is a tendency to relaxation of the abdominal viscera.

Diseases of the Urinary Organs.—Diseases of the urinary organs are rare. Orthostatic albuminuria usually disappears with adolescence, enuresis in girls with the establishment of menstruation, and in boys when the prostate has attained such size as materially to assist in the retention of urine.

Nervous System.—The nervous system of a healthy young person at puberty may present changes which at other times would be considered distinctly pathological, and parents and teachers need keen insight and great powers of forbearance at this critical period of rapid physiological development. Evidences of inherited disease, as, for instance, general nervousness, hemicrania, hysteria, latent syphilis, and general paresis, or the early mental weakness of dementia precox, may be noticed for the first time. Epilepsy rarely decreases, but is almost always aggravated to the adult type, or it may now appear.

Pressure sensations, languor, pain in the back, sleeplessness, and the easily induced exhaustion of neurasthenia may often be overlooked at a time when neuralgia, cephalalgia, a state of apathy, and

even indolence, are so common. Disturbances of sight manifest themselves in many ways, but probably do not depend upon pubescent changes. The senses of smell and of taste (pica) are sometimes perverted, and often aural noises are complained of temporarily.

The *phenomena of puberty* may come on *prematurely* as regards the body or mind; for instance, menstruation and pregnancy have been reported in girls eight and nine years old. In boys sexual and physical development are largely coördinate, although in exceptional cases repeated seminal emissions have occurred in early childhood. *Delayed* menstruation—that is, true amenorrhea (as differentiated from atresia of the uterovaginal canal)—may be caused by congenital heart disease or dystrophy of the ovaries and uterus, the latter being usually found in conjunction with undeveloped breasts and lack of pubic hair.

A functional form of amenorrhea with anemia, neuroses, and psychoses, is not infrequent after an acute infectious disease, or after sudden changes of climate, while vicarious menstruation may occur periodically from any mucous membrane. Before, during, and after menstruation itself, there are often colicky pains, gastro-intestinal disturbances, headache, vertigo, angioneuroses, flashes of heat, and urticaria. Leucorrhea, malposition of the uterus, or inflammation of its appendages may become manifest or aggravated at puberty. Other disturbances, such as dysmenorrhea, metrorrhagia, or inflammation of the mammary glands, are not uncommon.

In boys, also, at this phase of life the breasts may become affected, and occasionally a sensitive swelling appear in one or both, possibly accompanied by pain and redness, which disappear spontaneously within a short time. Circumcision may become necessary for phimosis, which leads to enuresis, masturbation, or balanitis from accumulation of smegma. Urethritis and orchitis—the latter, as a rule, of tuberculous origin—are not rare. Undescended testicles may in time from pressure, especially when arrested in the inguinal canal, cause painful sensations, and, unless they can be restored to their proper place either by operation or a suitable pad, their removal should be considered on account of the danger of atrophy or malignant degeneration.

Glands.—The consequences of absence of testicular secretion, as illustrated by young eunuchs; the neuroses and psychoses of ovarian dystrophy, acromegaly, gigantism, and infantilism observed in connection with disturbances of the infundibular gland; the influence of the thyroid upon physical and mental characteristics; the exaggeration of convulsive phenomena in suprarenal insufficiency—have all been so forcibly brought to light in recent investigations that their mere enumeration impresses us with the immense importance of the internal secretions. The glands producing them seem to be especially active at puberty; therefore, a disturbance of their function at or before this time must exert a far-reaching influence upon development in general.

This chapter would be incomplete without mention being made of masturbation, a pathological condition doubtless chiefly due to inherited weakness of the will. The inclination for it is marked in the mentally deficient, in epileptics, and in idiots. Occasionally epidemics of it break out in schools. It is true that with the awakening of the sexual impulses, phimosis, vulvitis, eczema, accumulations of smegma, vesical calculi, constipation, and similar conditions, by irritating the genital nerves, may be the exciting cause. Such local conditions should, therefore, in due time receive proper attention. Harm does not necessarily follow occasional self-abuse; but, if habitually indulged in, general nervousness, listlessness, exhaustion, cardiac palpitation, migraine, absent-mindedness, loss of memory, disinclination for work, and anemia may result, and, what seems worse, still further weakening of the will. Boys more easily fall victims to this habit than girls. Plain, non-stimulating diet, plenty of physical exercise, the avoidance of all incentives to sexual passion in literature, art, and association, with the correction of the physical factors mentioned, and a plain heart-to-heart talk with a sensible sympathetic teacher or parent will be of more benefit than criticism or open rebuke.

INDEX

A