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Diseases of Children.

By GEORGE M. TUTTLE, M. D., Attending Physician to St. Luke's Hospital, Martha Parsons' Hospital for Children and Bethesda Foundling Asylum, St. Louis, Mo.

Fig. I.

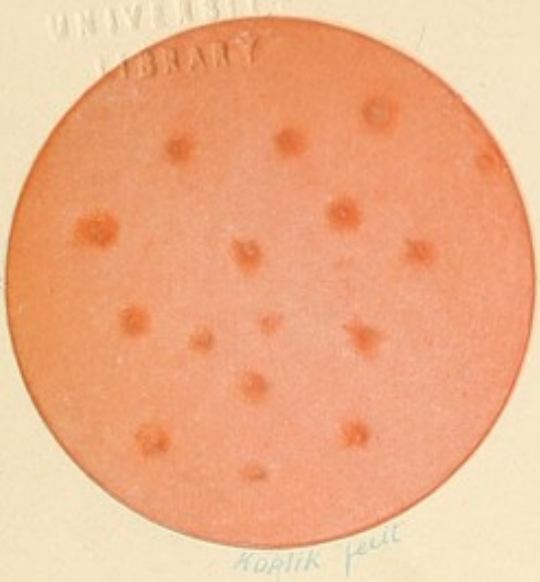


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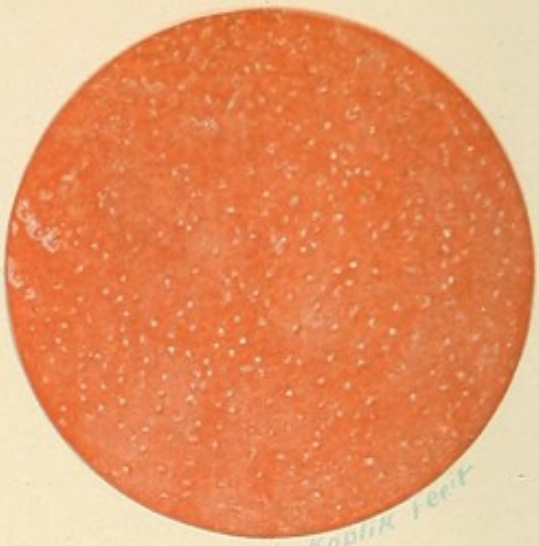


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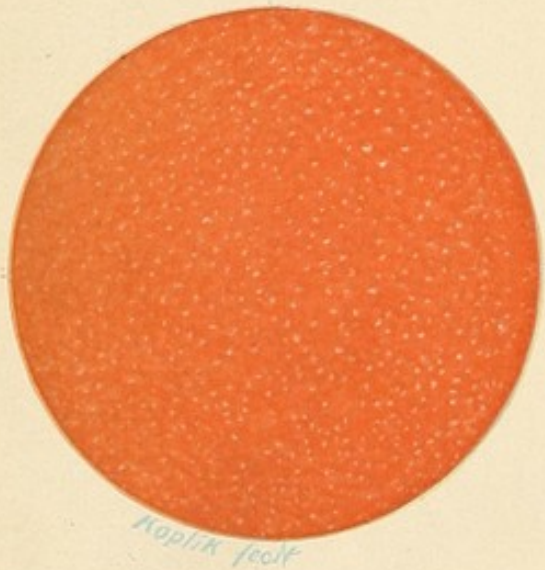
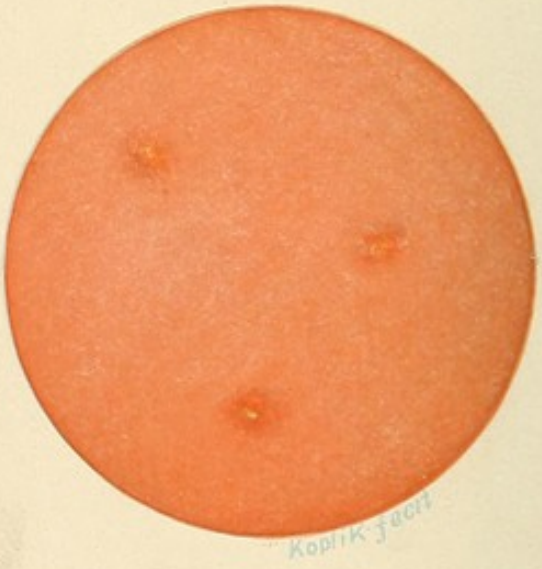


Fig. IV.



The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4. Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in line. Minute *yellow points* are surrounded by a red area. Always discrete.

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DISEASES OF CHILDREN.

A MANUAL FOR STUDENTS AND PRACTITIONERS.

BY

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PREFACE.

In preparing this manual for publication the author has had especially in view the requirements of the beginner in the study of pediatrics. His design has been to cover the subject fully yet in a concise form, dealing more largely with the *physiology of infancy* and with *artificial feeding* than with the pathological states found in childhood. Such diseases as in no wise differ from the same conditions as seen in adult life have been but briefly described. For more extended information on such subjects, or on those coming under the head of the so-called "specialties," the student is referred to more extended treatises.

No attempt at originality is professed, and acknowledgment is hereby made of free reference to the standard text-books on the subject of pediatrics, the treatise of Holt having been most frequently consulted.

The aim has been to present the subject in a systematic, orderly form, and in as few words as possible, both of which conduce to ease of study and reference.

GEORGE M. TUTTLE.

St. Louis, Mo.

374622

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DISEASES OF CHILDREN.

CHAPTER I.

THE INFANT AT BIRTH.

RESPIRATION.

How established: In beginning extra-uterine life breathing is a child's first new function. Previously its lungs have been impervious to air and to other than nutritive blood. Now the placental circulation is stopped and the blood must pass into the lungs to be oxygenated. By the reflex stimuli of comparatively cold air on the skin and of venous blood feeding the respiratory center in the medulla respiration is begun, usually with a cry. Each breath is deeper than the preceding one, until the whole lung gradually passes from a state of physiological atelectasis to complete expansion.

External signs: This change can be beautifully watched by observing the chest-wall, which, being very elastic and yielding, will stay depressed, in inspiration, over *unexpanded* lung, while those parts over *expanded* lung will rise and fall with the motions of the chest in breathing. To be satisfied that respiration is fully established, one should see that no part of the chest-wall sinks during inspiration.

Rate: The child should now breathe about forty-five times to the minute, but the respiratory rhythm is likely to be irregular.

As **aids to effecting natural respiration**, slapping the buttocks, blowing on the skin, or alternate applications of hot and cold water, are usually all that are necessary.

CIRCULATION.

In this another **marked change** occurs. The umbilical vein and ductus venosus, leading from the umbilicus to the in-

ferior vena cava, are closed, dwindling to a fibrous cord, the round hepatic ligament, in two to five days. The Eustachian valve, which guided the blood from the right auricle to the left auricle, disappears at once; the foramen ovale, through which the blood passes from the right auricle to the left auricle, closes in ten days, by a fold of endocardium growing over it from below; the ductus arteriosus, which carried the blood from the pulmonary artery to the descending aorta, becomes an impervious cord in four to ten days; and the umbilical arteries, which carried the blood from the internal iliacs to the navel, become the anterior vesical ligaments above, and remain as the superior vesical arteries below.

The **pulse** at birth varies from 140 to 120 per minute, and is frequently irregular. The higher rate is found in girls and small infants; the lower, in boys and large infants.

TEMPERATURE.

At birth the **temperature** is about 99° F. It may sink rapidly directly after birth; but in a healthy child it soon regains normal. The new-born infant is naturally very sensitive to cold, having lived so long in a temperature of 98° F., and great care must be taken to prevent chilling.

SKIN.

Vernix caseosa: The child at birth is covered, to a greater or less degree, with a slippery white substance, the vernix caseosa, a mixture of epithelium, down, and secretion from the sebaceous glands.

Lanugo: In addition to the vernix caseosa, the skin shows an abundant growth of very fine downy hairs, the lanugo, which soon disappears.

Desquamation: Shortly after birth the skin is frequently very red, and later marked desquamation may occur.

LENGTH.

The average **length** is about twenty inches. Below eighteen inches is evidence of prematurity.

WEIGHT.

Boys average seven and a half pounds; girls, about seven pounds. Below five and a half pounds shows a very low vitality and suggests prematurity.

HEAD.

Measurements and fontanelles: The head measures about thirteen inches in circumference in the occipito-frontal line. The *anterior fontanelle*, at the junction of the coronal and the sagittal sutures, is open one and a half by one-half inch. The *posterior fontanelle*, formed by the sagittal and lambdoid sutures, is also plainly to be felt.

Caput succedaneum: The head is apt to be much misshapen from pressure during its passage through the pelvis, and on one place is a thick boggy swelling, the *caput succedaneum*, formed by serous infiltration into the tissues of the scalp over the part presenting in the birth-canal, and hence escaping pressure. The caput disappears in a few days, and the shape of the head rights itself in a few weeks, both without assistance.

THORAX.

Measurements: The average circumference of the chest at the nipple-line is thirteen inches, the same as that of the head, the antero-posterior and transverse diameters being about equal. With the addition of the shoulders, we have a decidedly larger sized body than the head, a point of importance in connection with the birth of the shoulders over the perineum.

ABDOMEN.

Here the average circumference is about fourteen inches.

INTESTINES.

Meconium: At birth the intestines contain a very dark green, pasty substance called meconium, made up of bile, epithelium, intestinal mucus, and lanugo. This is voided in considerable quantities directly after, and sometimes during

birth, and, under the influence of the colostrum, for the next four days. It is found to be absolutely sterile and free from bacterial life.

KIDNEYS.

Urine: Constantly after birth the bladder empties itself of a small quantity of limpid urine of low specific gravity.

Uric acid and urates: In the kidneys themselves there are deposits of uric acid and urates, the so-called *infarcts*, crystals of which pass down the ureters and are found as purplish stains on the diapers. During this passage they may cause sharp pains. This is a frequent cause of crying in infancy, and shows the lack of sufficient water in the system.

UMBILICAL CORD.

Pulsation: The umbilical cord will be found to be strongly pulsating at birth, and it is best to wait for this to stop before ligating. An exception to this rule is made in congestive asphyxia, where the withdrawal of some blood from the child is desirable. It has been found that when the cord is not tied until after pulsation is stopped the infant gains on an average of three ounces of blood, and its initial loss of weight is lessened.

Ligation: The cord should be tied two inches from the navel, very tight, with narrow aseptic bobbin, and cut beyond with aseptic scissors. Before leaving, the stump should be examined, as it is quite common to find it bleeding a little. Dress the stump with dry sterilized gauze or cotton, dusting with powdered bismuth. Sometime within a week the cord should drop off by a process of dry gangrene, leaving a healthy ulcer at the base.

BATHING.

After tying and cutting the cord the infant should be *greased* thoroughly with vaseline, and laid aside in a warmed woollen blanket, with a soft towel next the skin, until the mother is entirely attended to.

Then the baby should have a *general bath*, in a warm room, before an open fire, if possible, in water of a temperature of 100° F. Castile soap, a soft sponge, and a soft towel should be the adjuvants. After drying, all the flexures and creases in the skin should be dusted with talcum, or some similar dry powder.

EYES.

Infection: There is always danger of some form of *infection* to the conjunctivæ from the uterine and vaginal secretions. It is therefore wise to follow the *prophylactic treatment* introduced by Crédé, of first washing the eyes with clean water, or saturated boric acid solution, and then instilling a couple of drops of a 2 per cent. silver nitrate solution into each eye, being sure that the lids are open so that the silver reaches the whole conjunctiva, bulbar and tarsal. The baby should be kept in a darkened room the first few days.

Strabismus: It is well to remember that co-ordinate movements of the eyeballs are imperfectly performed in infancy and hence *strabismus* is frequently present.

MOUTH.

The **mouth** should be freed from mucus, liquor amnii, or whatever may have gotten into it during birth, by sweeping the finger, covered with a piece of soft dry gauze, carefully and gently over the whole cavity, going well back into the pharynx.

EXAMINATION FOR ABNORMALITIES.

This should next be made, with special attention to the *skin*, *mouth*, *head*, *spine*, *hands*, *feet*, *genitals*, and *anus*.

CLOTHING.

This should be light, warm, unirritating, and allow free movement of the body and limbs. A broad abdominal flannel band should be worn the first four months. The other essentials are a woollen shirt, a flannel petticoat, a

diaper, and a dress. For night, the dress is exchanged for a night-gown. Safety-pins only should be used in fastening the clothing.

FOOD.

Colostrum: Nature prepares no real food for the first three or four days, but during this time the breasts of the mother secrete *colostrum*, a thin bluish fluid, showing under the microscope the presence of large corpuscles in addition to a small number of the usual fat-globules. This colostrum is *laxative* in its action. Eight or ten hours after birth the baby should be put to the breast, and, after this, four times daily until the *milk* comes. This accustoms the baby to sucking, reflexly stimulates the uterus to contract, and hastens the discharge of the meconium.

If there really seems to be need of any food, as in weakly infants, a *5 per cent. sugar solution* is the best form until lactation begins. Excessive crying, as if from hunger, in a healthy baby can usually be stopped by simply giving *boiled water*.

SLEEP.

The child should *sleep* almost continuously during the first few days. From the very start, *training in good sleeping-habits* should begin, the baby being taken up only to wash and to nurse, and being taught to go to sleep when laid down, and allowed to sleep in a crib by itself. No walking nor rocking it, nor other bad habits, should be permitted.

PREMATURE AND DELICATE INFANTS.

Viability: Probably no child of less than twenty-seven or twenty-eight weeks of intra-uterine life can survive. Each week after this increases its chances considerably. In these babies the vitality is very low, the organs of digestion, circulation, and respiration are very imperfectly developed, and, above all things, the heat-producing function is very slight.

In **treatment** the two most difficult problems are to maintain the animal heat, and properly to nourish them. They should not be clothed, but rather wrapped up in cotton and

then in blankets, and surrounded constantly by hot bottles, and kept in as nearly as possible a uniform high temperature. This can be best accomplished in an *incubator*, if one can be obtained. The skin should be thoroughly greased with sweet oil, and no bathing should be allowed.

Breast-milk from the mother, or a wet-nurse, is the very *best food*, and should be given in small quantities, from one-half to three-quarters of an ounce, every one and a half hours. This may be given by a large medicine-dropper, or by gavage, without removing the baby from its "nest." If breast-milk cannot be obtained, a carefully modified diluted cows' milk is the next best food.

CHAPTER II.

NORMAL DEVELOPMENT OF THE INFANT.

WEIGHT.

During the **first year** the child should be weighed every week, and during the second year every other week. The gain or loss in weight from one weighing to another is the very best indication we have of the general welfare of the child. Frequently the first signal of beginning trouble is the absence of the regular weekly gain, or the presence of a slight loss.

Physiological loss: During the first three days of life there is a so-called physiological loss in weight, averaging about ten ounces. This is due to loss of urine, meconium, and vernix caseosa, and the lack of food the first few days. With the advent of milk in the mother's breasts the normal gain begins, and, by about ten days, the birth-weight should be regained.

At **six months** the birth-weight is about doubled, and at the end of a year about trebled. During the first six months the gain should average four to eight ounces per week; and from two to four ounces during the second six months. From four to six pounds per year is about an average gain for the next ten years.

Sickness of any kind, and particularly digestive disturbances, stop the normal gain, and usually substitute a loss. Even the physiological process of *dentition* is accompanied by a diminished weekly gain.

HEIGHT.

During the *first year* the growth is about eight inches, an average of two-thirds of an inch per month, the increase being somewhat greater during the first quarter. During the *second year* the growth is about four inches, and during

the *next ten years* it averages about two inches per year. This growth, in infancy, takes place more rapidly in the extremities than in the trunk, although at birth the trunk was relatively longer than the limbs.

HEAD.

During the first year the **occipito-frontal circumference** of the head should increase about four inches, and about one inch more during the second year. A gain of an inch and a half more should take place by the fifth year, at which time the head attains its adult size.

The **cranial sutures** ossify by the sixth month, the posterior fontanelle closing by the second month, but the anterior not till the eighteenth month, having increased some in size during the first nine months. *Early closing* fontanelles and sutures suggest premature ossification, or cerebral defects; while fontanelles and sutures *remaining open over time* are suggestive of rickets or hydrocephalus.

THORAX.

The **thorax** grows more transversely than from before backward, keeping pace with the growth of the head, and not until the third year is it distinctly larger than the head. A chest of a three years' child not larger than its head points to extreme delicacy.

ABDOMEN.

The **abdomen** at birth is larger than both head and chest; at two years it should be about the same size, and afterward the smallest of the three.

MUSCULAR ACTS.

Voluntary, but inco-ordinate **muscular movements** are made about the fourth month. The head is held erect about the same time. The child sits erect about the seventh month. The first attempts at walking are made about the twelfth to the fifteenth month, some children learning to creep three or four months earlier.

PULSE.

The **force** and **rhythm** in infancy are the important factors rather than the rate. During the first year the frequency varies from 110 to 140, reaching 100 by the end of the second year. By the eighth year the adult rate is present. The rate is markedly influenced by very trivial causes, such as crying, struggling, or excitement, making its value unimportant. It is helpful to remember that the pulse can be easily counted in the anterior fontanelle.

RESPIRATION.

Respiration is chiefly *abdominal*, and the rapidity very variable. During sleep it averages considerably less than during wakefulness. The *rate* in the *first months* is thirty-five to forty; during the second year it falls to twenty-eight; during the third, to twenty-five; and by puberty it reaches the adult rate, eighteen to the minute.

In health, the rhythm—inspiration, expiration, pause—is unchanged. The ratio between pulse and respiration is about three and a half to one.

TEMPERATURE.

After the more or less **marked fall** succeeding birth the temperature immediately assumes the adult range, the main point to be kept in mind being the extreme fluctuations under slight causes, the heat-centres being very unstable. In the chronic wasting diseases a subnormal temperature is quite common.

DENTITION.

At **birth** the teeth are enclosed in the dental sacs in the alveoli of the jaws, and their growth is upward by calcification of their roots, this growth beginning at birth.

The **milk** or **deciduous teeth** are twenty in number, and are cut in the following order, although quite wide variations are frequent:

1. Two lower central incisors, at six to nine months; 2.

Four upper incisors, at eight to twelve months; 3. Two lower lateral incisors and four anterior molars, at twelve to fifteen months; 4. Four canines, at twelve to fifteen months; 5. Four posterior molars, at twenty-four to thirty months.

Early teething usually means early ossification of the cranial bones; and late teething usually means rickets, or other form of malnutrition.

In **a healthy child** dentition is usually not accompanied by any constitutional disturbance, the tooth very gradually forcing its way through the overlying gum by a process of pressure-absorption, first showing its presence as a lump under the reddened mucous membrane, this growing whiter as the tooth nears the surface.

If any **symptoms** are present, they are only restlessness, fretfulness, disturbed sleep, salivation, a disposition to put objects into the mouth, and possibly a little fever.

In previously **unhealthy children** more serious *symptoms* may coincide with dentition, such as vomiting, diarrhœa, fever, earache, eczematous eruptions, and possibly convulsions; but there is always the question whether the unhealthy condition as a predisposing factor is not more of a cause of the symptoms than the teething. The more carefully other causes are looked for, the fewer cases due to dentition only, even in these susceptible infants, will be found. Rarely, a case calling for lancing the gums will be discovered.

The **second** or **permanent teeth** are cut as follows: 1. First four molars, at six years; 2. Eight incisors, at seven to eight years; 3. Eight tricuspids, at nine to ten years; 4. Four canines, at twelve to fourteen years; 5. Second four molars, at twelve to fifteen years; 6. Third four molars at seventeen to twenty-five years. Except for the first four molars the order is about the same as for the first set. In growing, the second set cause atrophy of the roots of the first set until they loosen and fall out.

URINE.

For obvious reasons accurate results as to quantity of urine in infancy are hard to obtain. During the first few days of

life the daily quantity is three ounces; by two months it increases to eight to ten ounces; by six months to ten to sixteen ounces; by two years to twenty ounces; by five years to twenty-six ounces; by eight years to forty ounces; and by fourteen years to forty-eight ounces. The specific gravity averages from 1005 to 1010. The reaction is acid. Traces of albumin or of sugar may be found during certain periods, but rapidly disappear.

THE FÆCES.

These partake of the characteristics of *meconium* for four or five days, when they change to *milk-stools*. These are yellow, of the consistence of semi-solid butter, smooth, of acid reaction, and inodorous. The *gases* present are hydrogen and carbonic acid. The fæces amount to about three ounces per day in quantity, and are passed from two to four times daily. Chemically they consist of 85 per cent. of water, and of fat, proteids, lactic acid, salts, and bile-elements. Bacteria are present in large variety, especially the bacillus coli communis. Stools resulting from cows' milk are whiter, from the presence of more curds. When the child begins a mixed diet the stools assume the adult type.

SPECIAL SENSES.

Sight: This is present from birth, but the eyes are very sensitive to light, and not till the fourth month do they seem to be used voluntarily.

Hearing: Deafness is present for a day or two. This is probably due to the absence of air from the tympanum, but when respiration is well established the hearing begins, and later becomes quite sharp. The infant does not seem to locate sounds before the fourth month.

Touch: Sensation is present, but is dull for three months. It is highly developed in the tongue and lips, where the temperature-sense is also acute.

Taste: This sense is highly developed from birth.

Smell: We know scarcely anything about this sense.

Speech: This function is very variable in its development. Very much depends on efforts at training, as institution-children always talk much later than those brought up at home. By the end of the first year the child should begin to say a few words, and by the end of the second year should form a few sentences. If there has been no effort at speaking by the end of two years, probably some mental defect is present or "deaf-mutism" may be suspected.

CHAPTER III.

EXAMINATION OF THE CHILD.

History: First obtain a careful account of the family history, and then the personal history of the child. Then gather together systematically all the facts about the present illness, willingly listening to all the ideas and observations of the mother or nurse about the child. Always inquire about the food and as to any drugs which may have been taken.

Inspection: A careful observation of the child is next necessary, trying to disturb it as little as possible. Notice the *position*, whether it is natural and easy; the character of the *sleep;* the *respiration;* the *skin*, its color and whether dry or moist; the *facial expression;* the *nostrils*, whether quiet or moving; the *cough*, and if present its character; the *cry;* the *mental condition;* the *mouth*, whether it is kept open or closed in breathing; the condition of the *eyes* and *pupils;* the presence or absence of enlarged *lymph-glands;* the presence or absence of *muscular movements* or *twitchings;* the presence of any *discharges*. Compare any of these as found with the condition in a normal infant.

Physical Examination: First take the *temperature*, by rectum if possible, as it is very hard to get by mouth, and the axillary temperature is uncertain. Then take the *pulse*, usually at the same time, remembering the comparative unimportance of its rate. Next strip the child, and examine the *skin* carefully for rashes. Auscultate and percuss the *heart* and *lungs* carefully. Palpate the *abdomen*, remembering that normally the liver reaches below the free border of the ribs, and that the spleen cannot be felt. Inspect the *genitals*. Examine the *muscular development*. Examine the *bones*, then the extremities and the ribs. Investigate the development of the *nervous system*, and the functions dependent on it. Examine the *fontanelle*, and if open see if it is bulging, flat, or depressed. Lastly examine minutely the *ears*, the *mouth*, and the *throat*.

CHAPTER IV.

DISEASES OF THE NEW-BORN INFANT.

ASPHYXIA.

Forms: There are two distinct forms of asphyxia after birth—(1) the congestive, and (2) the anæmic. In the first, the child is livid in color, appears swollen, and has good muscular tone and a full, strong pulse. In the second form the skin is pale, the muscles flaccid, and the pulse inappreciable.

Prognosis: The prognosis in the congestive variety is far better than in the anæmic.

Asphyxia—treatment: This is simple in the *former;* first allowing a little blood to escape from the cut cord to reduce the internal congestion, and then alternately dipping the baby into hot (110° F.) and cold water. The reflexes being still present, the response is marked.

In the *latter* variety allow the child to get as much blood as possible from the placenta, and then perform artificial respiration, for a very long time if necessary, at the rate of ten to fifteen times per minute. Sylvester's method is undoubtedly the best, as the baby can be kept in a tub of hot water during the manipulations. Be sure that the mouth and pharynx are clear of any foreign substance.

In some cases where artificial respiration fails, *direct inflation* of the lungs, by the mouth-to-mouth method, or by passing a catheter into the larynx, succeeds. In these cases, after respiration is established attention is necessary for some time to prevent relapses and to be sure that atelectasis is overcome.

ICTERUS.

This common affection, of still unknown origin, appears from the second to the fifth day and lasts about a week. It *occurs* in about one-third of new-born infants. It is prob-

ably hepatogenous in origin, and due to resorption of bile-pigment. It requires *no treatment*, disappearing easily.

In some cases an early jaundice *increases in intensity* and presents constitutional symptoms. In these cases the *prognosis* is bad, and there is usually either a septic infection from the umbilical vessels or a congenital malformation of the bile-ducts.

OPHTHALMIA.

Definition: This is an *infectious conjunctivitis*, in the majority of the cases due to the gonococcus, less frequently to the ordinary pyogenic germs. All grades, from a mild conjunctivitis to chemosis, corneal ulceration, and sloughing, may occur.

Symptoms: Redness, swollen lids, copious purulent discharge, pain, and photophobia.

Ophthalmia—treatment: Prophylaxis, as already described, is most important. If the disease is started, frequent and thorough cleansing with a saturated boric acid solution, or a 1 : 5000 bichloride of mercury solution, is necessary. Twice a day a few drops of a 1 per cent. silver nitrate solution should be instilled in the eyes, and in the intervals ice-cold compresses, frequently changed, should be kept on the lids. It is always wise to call in the services of an oculist.

CEREBRAL HEMORRHAGE.

Source: A quite frequent accompaniment of birth is some form of bleeding in the skull, which is regularly from the congested vessels of the *pia mater*. These hemorrhages are mostly mechanical, and are seen in prolonged non-instrumental births quite as frequently as after the use of forceps. The hemorrhage is situated usually on the base, and nearer the back than the front of the brain. They occur in both breech and vertex cases.

Cerebral hemorrhage—symptoms: If large, the baby is often stillborn. If smaller, asphyxia, torpor, irregular respiration, feeble, slow pulse, and convulsions are the regular symptoms. The pupils are variable, but oscillatory move-

ments of the eyeballs are frequent. According to the size and location of the clot, localized paralysis or complete hemiplegia is present. In the surviving cases permanent damage to the brain and its supplied parts is left, the symptoms depending on the location of the lesion, such as idiocy, epilepsy, monoplegia, diplegia, or hemiplegia.

Cerebral hemorrhage—treatment: This is almost entirely preventive by shortening long labors by the judicious use of the forceps. For the changes left behind by these accidents very little can be done.

CEPHALHÆMATOMA.

Definition: This is an extravasation of blood under the pericranium, and due to some traumatism during labor. The tumor is always limited by the attachment of the pericranium, never passing over a suture or fontanelle. It appears from the first to the fourth day, and grows gradually for about a week, when it slowly disappears. It is fluctuating, and shows none of the signs of inflammation. As it grows older there is a raised ridge around the margin due to organization of the effused blood, and giving the impression of a depression of the skull inside this ridge. If the scalp is lacerated, infection is likely to occur, followed by abscess. The whole tumor will disappear in two or three months by a process of absorption, unless some accident happens to it.

Cephalhæmatoma—treatment: Care should be exercised to protect the part from injury, and to keep aseptic any abrasions of the skin in the neighborhood. Non-interference is otherwise the proper course. If an abscess forms, open and pack.

OBSTETRICAL PARALYSES.

The **two commonest forms** of this condition are (1) *facial*, and (2) *brachial*, paralysis. The *former* (1) is due to pressure on the facial nerve by one of the blades of the forceps. The eye on the paralyzed side remains open, and in moving the face this side is found to be smooth, and the mouth is drawn to the sound side.

Facial paralysis—treatment: None is needed, as the condition usually disappears within a week. In those cases where no improvement appears within a month regular electrical treatment should be given.

In (2) **brachial**, also called **Erb's, paralysis** one or more groups of muscles of the upper extremity are paralyzed. Here also the cause is usually traumatism from the forceps or from pulling on the arm, or in the axilla. The muscles most often affected are the deltoid, biceps, brachialis anticus, supinator longus, and the supra- and infra-spinatus. The arm hangs flaccid, and after some time the muscles atrophy.

Brachial paralysis—treatment: Most of these cases also recover spontaneously in a few months, but this can be hastened by electrical treatment. Strychnine is a useful drug in both forms.

SEPSIS.

Sepsis in the newly born is present in a great variety of manifestations. The infection by pyogenic bacteria commonly takes place through the umbilical wound, but any abrasion of the skin or mucous membranes presents an avenue of invasion.

Sepsis—symptoms: These vary from those of a slight local infection to a general severe or fatal septicæmia; from an omphalitis, an umbilical phlebitis, or a peritonitis, to a streptococcal inflammation localized in almost any, or more than one, of the important organs, such as the lungs, pleura, peri- or endo-cardium, meninges, bones, joints, stomach, intestines, or the cellular tissues. The *general symptoms* are irregular fever and its concomitants, rapid, feeble pulse, wasting, stupor alternating with restlessness, diarrhœa, and vomiting. Hemorrhages are frequent, and it is in these cases that the severe forms of *jaundice* occur.

Sepsis—treatment: Prophylaxis is most important in the aseptic care of the navel and of any abrasions of the skin. After the disease has started any local symptoms should be treated as usual, and in addition a general supportive and stimulating treatment should be followed—in other words, proper food and whiskey.

TETANUS.

Definition: This is an infectious disease, caused by a specific germ, and is characterized by tonic muscular spasms affecting early the muscles of the jaw, but later all the muscles of the body. The infection usually occurs by the navel, but other sources may allow its entrance.

Tetanus—symptoms: These begin usually about the time of the separation of the cord, with rigidity of the jaws and consequent difficulty in nursing. Later, the body becomes rigid at intervals, complete relaxation taking place between spasms. This spastic condition is easily brought on by any external irritant, such as noise or jarring, the reflexes being markedly increased. Later, general tonic spasms with opisthotonos supervene. The temperature varies from 101° to 106° F. Death is caused by exhaustion or stoppage of the respiration.

Prognosis: It is a very fatal disease, the mortality being from 90 to 95 per cent.

Tetanus—treatment: Prophylaxis by surgical cleanliness in the care of the cord, or any wounds, is most important. In actual cases the patient should be kept deeply under the influence of chloral and bromide, they being pushed to their physiological limit. Absolute quiet of the patient should be enforced. Feeding by a catheter through the nose may be necessary. Of late years a tetanus antitoxin has been used with seemingly good results, when given early enough.

EPIDEMIC HÆMOGLOBINURIA.

Winckel first described this disease in 1879. Its essential **symptoms** are hæmoglobinuria, with cyanosis and icterus, occurring in epidemics. The disease begins three or four days after birth, and death takes place in about two days. It is very fatal, and should probably be classed under the head of Sepsis. **Treatment** is of no avail.

MELÆNA.

Definition: By this term is meant hemorrhage from the stomach or intestines, a not infrequent occurrence shortly after birth, but of varying degrees of intensity.

Symptoms: These gastro-enteric hemorrhages begin usually during the first few days of life. The blood is light colored when passed fresh, or dark when partially digested. Associated with this hemorrhagic diathesis there may be bleedings under the skin, into the kidneys, or from the vagina or navel, or any of these forms may occur independently.

Prognosis: This is serious, but not always fatal, depending on the extent of the bleeding and the vitality of the child. Of late there is some evidence of this condition also being an infection, and entering through the digestive tract.

Melæna—treatment: Ergot is the drug recommended, but its efficacy is doubtful. Special attention to the general nutrition is more important, and washing the bowels and stomach with a cleansing astringent solution should be tried. External hemorrhages are treated by local astringents.

PEMPHIGUS.

This form of **bullous eruption** is often found on new-born babies.

There are *two* general varieties which are very important to differentiate:

First, the form occurring as one of the manifestations of *congenital syphilis.*

The **diagnosis** rests on the presence of other lesions of the disease, on the presence of bullæ on the palms and soles, and on the history of the parents.

Second, there is a form consisting almost entirely of *bullæ* located on various parts of the body, the trunk especially, which seems to be infectious in its nature. This latter form heals well under the application of drying antiseptic powders and cleanliness.

GRANULOMA OF UMBILICUS.

This is a little **lump of granulations** left after the sloughing of the cord, and causing a purulent discharge. At times there may be associated with it a *patent urachus* leading to the bladder, and hence a few drops of urine may ooze from the navel.

Treatment: It is best snipped off by scissors and the base touched with solid nitrate of silver.

UMBILICAL HERNIA.

This form of **rupture** usually appears through the opening in the linea alba for the umbilical vessels, and before the vessels, and before the third month of life. It varies in size from a simple convexity of the navel to a tumor large enough to become strangulated.

Umbilical hernia—treatment: A mechanical application is usually all that is required, the main care being to prevent the formation of a rupture by wearing an abdominal band during the first four months. If the hernia is present, it is best held back by rubber strapping around the abdomen with an ordinary wooden button over the ring, acting as a pad to prevent protrusion. If this is worn constantly for three to six months, the hernia is regularly cured.

MASTITIS.

A slight degree of **inflammation of the breasts** is fairly common in new-born infants of both sexes.

Symptoms: These are pain, tenderness, and secretion of small quantities of a milky fluid.

Etiology: Traumatism and lack of cleanliness.

Treatment: Apply cool, clean compresses, and do not rub nor squeeze the breasts. They may be painted with tincture of belladonna if the inflammation is severe.

SCLEREMA.

This is a condition of **hardening of the skin** and *subcutaneous tissues* in circumscribed areas, or more generally. It occurs in feeble, badly nourished babies, and is usually fatal. The condition is associated with marked lowering of the cutaneous temperature of the body, and is probably the result of the hardening of the subcutaneous fat, due to this low temperature. There is no pitting on pressure.

Treatment: Artificial heat and proper nutriment are all we can do for these cases. Use of the incubator is indicated.

HYDROCEPHALUS.

Varieties: This condition, of *water on the brain*, is of two varieties, *external* and *internal*.

The *former*, in which the fluid is between the brain and dura mater, in the arachnoid, is quite rare. The *latter*, with the fluid in the cerebral ventricles, is the common form. The condition may be congenital, or more rarely acquired.

Hydrocephalus—etiology: The cause is a mechanical one, producing exudation by pressure, or is due to a chronic inflammation of the lining membrane of the ventricles. The fluid resembles cerebro-spinal fluid in character, and may be present in quantities up to two quarts or more. The ventricles are dilated, and the brain is thinned by pressure, at times to a mere shell.

Hydrocephalus—symptoms: The *cranial sutures* are widely open, even those at the base in marked cases. The *fontanelles* are open and bulging, the head is enlarged, the forehead is high, and the face seems small. *Fluctuation* and even *translucency* can be obtained in marked cases. The head cannot be held up, the *mental condition* is dull, and there is a general lax state of the muscles, although there may be localized rigidity. *Nystagmus*, *strabismus*, and *inability to close the upper lid* over the eyeball are often present. *Convulsions* may occur.

When the head enlarges greatly in utero birth may be impossible without perforation.

Prognosis: Recovery is rare. When the process ceases and the child grows up some mental defect is usually present.

Hydrocephalus—treatment: This is very unsatisfactory. Iodide of potassium to cause absorption may be tried. Various operative procedures have been used, and aspiration is probably the best of them; but all are of doubtful value.

MENINGOCELE.

Definition: This means a *protrusion* of some part of the *membranes* of the brain through a hole in the cranial wall, usually in the location of a suture or fontanelle. The tumor

is filled with fluid communicating with that in the brain, and frequently brain-elements are also present.

Symptoms: The tumor fluctuates, and is most often seated in the occipital region or at the root of the nose. The child usually has other deformities associated.

Meningocele—treatment: Aspiration and the injection of tincture of iodine should first be tried. A plastic operation may be necessary.

SPINA BIFIDA.

Definition: This is a protrusion of the spinal meninges through the unclosed laminæ of one or more of the vertebræ. In the sac of membranes is usually some portion of the spinal cord. The tumor is covered by skin to near the apex, when the covering becomes parchment-like and easily ulcerates.

Spina bifida—symptoms: The tumor is present at birth, and usually is situated in the lumbar region. It fluctuates; and if the baby cries, as the intracranial pressure is increased it enlarges. If the cord-elements are in the sac, paralysis of legs and of the bladder and rectum is present. The tumor has a tendency to grow, and eventually to ulcerate and burst, when death from infection usually follows.

Prognosis: If no paralysis coexist, complete recovery may occur. If paralysis is present, the prognosis is bad.

Spina bifida—treatment: Protect the tumor from pressure and trauma. Attend to the rectum and bladder. Aspiration with injection of tincture of iodine is the best operative procedure. A plastic operation may be made.

HARELIP—CLEFT PALATE.

Definition: This abnormality is due to imperfect closure of the maxillary and intermaxillary processes in embryonal life. The fissure may be single or double, and may involve the lip only, or the intermaxillary bone, or the soft or hard palate, or all. The deformity is unsightly and prevents the infant sucking with ease.

Harelip and cleft palate—treatment: *Medical* care is to feed with a spoon, or dropper, or gavage if necessary; to keep the mouth scrupulously clean. *Surgically* the fissures may be

closed by operation; or the cleft palate by a dental plate. In operating for harelip the second or third month is the proper time, and for cleft palate the third or fourth year.

OTHER DEFORMITIES.

Clubfoot: This deformity, usually of the equino-varus variety, is often congenital. It belongs to the province of the orthopedic surgeon, but is mentioned here to call attention to the importance of immediate treatment, the earlier the better. By massage, manipulation, and a simple apparatus the parts can usually be brought back to their normal relations in the early weeks of life, when later complicated apparatus and surgical operations would be necessary.

Imperforate rectum: This anomaly is fairly frequent, and is, of course, recognized by the failure to pass the stools. The rectum may be imperforate at the anus or higher up. The low cases are easily operated on by breaking through the septum with a director. If the lower end of the bowel is too high to reach from below, or cannot be found on dissection, the only recourse is an artificial anus in the groin.

Hypospadias: This abnormality of the *urethra* is the commonest of the congenital defects of this region. The urethra, instead of being continued forward to the glans penis, opens anywhere on the under surface of the penis, from the sulcus just behind the glans to the peno-scrotal juncture. When the opening is far back in the perineum the scrotum may be divided into two halves, giving rise to the condition known as false hermaphroditism. The farther forward the urethra opens the less disagreeable to the patient the condition becomes. There is nothing serious to be feared from it, but in adult life it may be a cause of sterility.

Treatment: This is purely surgical, the operation being plastic, for the formation of a new urethra from the end of the natural one to the glans penis.

Epispadias: This is a much rarer condition of urethral abnormality, the urethra here opening somewhere on the dorsum of the penis. This is also of no importance from the

standpoint of the patient's life, but may be a source of great inconvenience and also of sterility.

Treatment: A plastic operation is the only method of cure.

EXSTROPHY OF THE BLADDER.

Description: This is one of the most distressing deformities of the new-born infant. The anterior wall of the bladder, in larger or smaller amount, is wanting, due to incomplete closure of the abdominal plates. The ureteral openings are usually in plain sight, and the urine drips away continuously, wetting the clothing, excoriating the skin, and producing the disagreeable odor of fermenting urine. The patient is objectionable both to himself and to all around him. This abnormality, which is really only an exaggerated condition of epispadias, is also harmless as far as life is concerned.

Exstrophy of the bladder—treatment: Only a complicated plastic operation can cure these unfortunates. Operation should always be undertaken, and the results are at times fairly good. There is no reason why the operation should be postponed later than five or six months of age.

CRYPTORCHIDISM.

Description: The testicles should descend, from their abdominal location of embryonal life, into the scrotum by the end of the eighth month. Frequently one or both testicles may remain in the abdomen, or descend only into the inguinal canal by birth. Ordinarily, in the course of a few weeks, without interference, they will finally reach the scrotum. If they remain in the abdomen, no harm can come to them; but if they are caught in the inguinal canal, they are subject to traumatism, and may become inflamed and be the source of great pain.

Cryptorchidism—treatment: If the testicles remain in the abdomen, nothing should be done. If they are in the inguinal canal, they should be protected from injury as far as possible. By gentle manipulation they can be assisted in descending to their proper place. If they become inflamed,

they should be treated by rest and cold applications. If they will not descend, and become the source of much pain, it may be necessary to remove one or both of them. This is not objectionable, as the individual will probably be sterile anyway. *Orchidopexie—i. e.*, loosening up the testicle and suturing it in its proper place in the scrotum—has been done.

CHAPTER V.

FEEDING OF INFANTS.

WOMAN'S MILK.

Characteristics: All observers agree that a healthy woman's milk is the best food for a child during its first year. This being the case, we must make a careful study of the properties of healthy human milk.

Woman's milk is a secretion from the mammary gland, formed by the metamorphosis of the cells of the acini. It consists of a large proportion of water, holding in solution and in suspension four different kinds of solids—proteids, fat, sugar, and salts. It is a thin, sweetish fluid, of alkaline reaction, and with a specific gravity varying from 1029 to 1032.

Chemical composition: The average proportion of the different components is as follows: proteids, 1 to 2 per cent.; fat, 3 to 4 per cent.; sugar, 7 per cent.; salts, .2 per cent; water, 87 to 88 per cent.

Of course, considerable variations in any one milk occur from day to day, and from week to week; and the milks from different women are never precisely alike; but the tendency to approach the above standard is always marked. In the first two weeks and in prolonged nursing (over ten or twelve months) marked differences from the above proportions are found; but during the intervening time these averages may be expected.

Woman's milk—proteids: These are the *albuminous* constituents of milk, the so-called curd. There are two main bodies of this class, *caseinogen* and *lactalbumin*. The *casein* is only half as much as the *milk-albumin;* but the exact chemical properties of the whole proteid class are as yet not very perfectly worked out.

They are the ingredients furnishing the nitrogen to the child for the growth of the nitrogenous tissues of the body, and until the child is on a mixed diet they are its only source of nitrogen. They are hence very necessary for life; and if deprived of the proper quantity, the infant will become anæmic, with weak and flabby muscles and loss of flesh and strength. If in excess in the milk, they are the most frequent source of gastro-enteric disturbance.

Fat: The *fat* of milk is the familiar *cream*, existing as an emulsion of minute droplets in the alkaline fluid. If milk is examined under a microscope, the fat-globules should be the only elements visible, and they are seen as quite thickly aggregated, highly refractive granules, of a fairly uniform size.

The *fat* is of use mainly as a source of heat, and in storing up fat around the various body-tissues. It is also needed as a special food for the nerve-tissues, and has some unknown connection with the growth of the bones. While fat is not so essential to life as the proteids, still deprivation of it is followed by rather definite symptoms of general malnutrition and anæmia, and among special symptoms constipation and a tendency to the development of rachitis are seen.

Woman's milk—sugar: This is the most stable and least troublesome ingredient of the milk. It is the carbohydrate element, and is present as *lactose*, or milk-sugar, in solution in the water.

An excess of sugar does not often cause digestive derangement, but does lead to a rapid increase in the weight and fat of the infant; such children are not strong and resistant, however, and rapidly fall victims to acute disease. Sugar's function is mainly to produce the animal heat of the body, and to assist in the storing up of fat around the tissues.

Salts: The *salts*, or "ash," are the mineral ingredients of the milk, consisting mainly of calcium phosphate, potassium carbonate, and sodium chloride. They are the great source of nutriment for bone, and supply also the mineral ingredients demanded by the other body-tissues.

Woman's milk—water: This is necessary in large amounts in the food to hold the solids in solution, and so make easy

their digestion, and to supply the water needed by all the tissues of the body.

Colostrum: During the first three or four days after the baby's birth the breasts secrete the so-called colostrum, which differs decidedly from the true milk. It is thicker and yellower, has a much higher specific gravity, and is very rich in proteids and salts. The fat and sugar are less in quantity than in true milk, while there is present a considerable number of large irregular granular bodies, known as colostrum-corpuscles. These are probably cells from the acini of the gland in which metamorphosis is incomplete.

It is secreted in comparatively small amounts, and its function seems to be as a temporary food, and as nature's laxative to remove the meconium.

Woman's milk—quantity daily: After the colostrum-period is finished the two breasts secrete daily somewhat the following average quantities of milk. These amounts are rather difficult to fix exactly, and are estimated from a large number of experiments in pumping the breasts and measuring the results; in finding the quantity taken by a baby by weighing him carefully both before and after nursing; and in estimating the capacity of infants' stomachs at different periods of life.

Daily quantity of milk secreted: During second week, 13 to 18 ounces; during third week, 14 to 24 ounces; during fourth week, 16 to 26 ounces; during second and third months, 20 to 34 ounces; during fourth and fifth months, 24 to 38 ounces; during sixth, seventh, and eighth months, 30 to 40 ounces.

Average quantity at each nursing: During first week, $\frac{5}{8}$ to $1\frac{1}{2}$ ounces; during second week, 1 to 3 ounces; during third week, $1\frac{1}{2}$ to 4 ounces; during fourth week, $1\frac{1}{2}$ to $4\frac{1}{2}$ ounces; during second month, 2 to 5 ounces; during third month, $2\frac{1}{2}$ to $5\frac{1}{2}$ ounces; during fourth month, 3 to 6 ounces; during fifth month, $3\frac{1}{2}$ to $6\frac{1}{2}$ ounces; during sixth month, 4 to 7 ounces.

Of course, the above daily amounts and the quantities at each nursing vary markedly, depending on the amount of real breast-tissue, the general condition of the mother, and the size and strength of the baby.

Rules for maternal nursing: If the mother is able to nurse her child, it should be given the breast regularly, at certain definite intervals, according to the following scheme:

It should be allowed to nurse about twenty minutes on each occasion, being at once awakened if it goes asleep at the breast before finishing its meal, and it should nurse from the two breasts alternately.

Before and after nursing the nipple should be washed with boric acid solution, and after nursing the baby's mouth should be washed out with the same.

As soon as the meal time is finished the baby should be put back into its own bed, and left to go asleep without rocking or other aid. If it is not awake by the time for its next meal, it should be awakened and fed.

By beginning from the first, and following the rules absolutely, the baby is quickly trained to accustom itself to this proper way of feeding, and the parents are relieved of the greatest trials connected with the care of their child. These regular nursing-habits are rapidly learned, and are productive of many good results—good digestion, good assimilation, lack of colic, lack of crying, prevention of gastro-enteric disease, regularity in sleeping, and insure regular resting-hours for the mother. It is surprising to discover what a machine a baby can be made into: he will learn to awake almost by the clock, to go asleep at once after feeding, or to lie awake quiet and contented. It is far more difficult to introduce these reforms in the life of a baby who has been taught bad habits of feeding, than it is to teach them to a new baby from the first.

Feeding scheme: For *two months*, every two hours, omitting two feedings at night; *e. g.*, 7, 9, 11 A. M.; 1, 3, 5, 7, 9, 11 P. M.; 3, 7 A. M. Total, ten.

For the *third* and the *fourth month*, every two and one-half hours, omitting two feedings at night; *e. g.*, 7, 9.30, 12 A. M.; 2.30, 5, 7.30, 10 P. M.; 2.30, 7 A. M. Total, eight.

After the *fourth month*, every three hours, omitting all night-feeding; *e. g.*, 7, 10 A. M.; 1, 4, 7, 10 P. M.; 7 A. M. Total, six.

These latter intervals are adhered to till *ten* to *twelve*

months, by which time some solid food is allowed in addition to the breast-milk, and taking the place of one or more of these feedings.

A woman's milk as the food, and the above rules in taking it, are the ideal or standard that should be aimed at in feeding every infant. Unfortunately, every mother cannot nurse her baby; some mothers will not nurse their babies; every baby does not have a mother to nurse it; and some mothers do not furnish a food that is suitable for her particular baby. Hence many infants have to be raised on *artificial food*. Under such circumstances the problem is to furnish the infant the best substitute in quality for woman's milk, and to feed this substitute in accordance with the same rules. The nearer we can copy maternal feeding in quality of food, in quantity given, and in regularity of feeding, the larger proportion of artificially fed infants will be kept from gastro-intestinal and nutritional disorders.

Contraindications for maternal nursing: There are a few justifiable reasons why a woman should not nurse her own child, even when she has sufficient milk. Among these are excessive nervousness, chorea, epilepsy, tuberculosis, puerperal hemorrhage (excessive), nephritis, eclampsia, or septicæmia; or where after careful observation in previous lactations it has been proved that her milk is distinctly prejudicial to her child.

Cracking of the nipples or *inflammation of the breast* may be so severe and painful that nursing is impossible; but there are many devices for remedying these evils, and often nursing can be begun again after a temporary cessation.

Such devices are the nipple-shield and breast-pump, and therapeutical care of the nipples and breasts as laid down in the text-books on obstetrics. In these two conditions, in the absence of other contraindications, always make the effort to have lactation continued if possible.

Menstruation supervening during lactation frequently causes a temporary gastro-intestinal disturbance; but the cause is all over in a few days, when the milk again becomes a suitable food for the child.

If *pregnancy* intervene, it is wisest to stop nursing, as the milk rapidly deteriorates, since the strain on the mother of nourishing herself, the child at her breast, and the embryo, is prejudicial to all three. Further, her baby has by this time had the advantage of its mother's milk for some months, and *weaning* is of less importance to the child now than it would have been earlier.

Insufficient food: The physician is often asked whether he thinks the child is getting enough food, and as a matter of fact almost any ailment is ascribed to "being hungry." It is quite important to know the signs of this condition, and it is always well to try thoroughly to rule out other possible causes before deciding that the symptoms complained of are due to insufficient food. There is no doubt that more children are over-fed than starved; and because a child will take more food, if offered it, is no reason why he needs that food for his nutrition.

The **main signs** to be relied on of insufficient feeding are: lack of the regular weekly gain in weight, and very soft, flabby breasts, from which only a very little milk can be gotten at the nursing-time. Less positive, but confirmatory evidence is obtained by the baby napping only a short time after feeding, instead of sleeping quietly until his next nursing-interval; fretfulness and crying when awake, it being certain that other causes are excluded; irregularity in the bowels, diarrhea and constipation alternating; and either an excessively long or a very short time being taken by the child in emptying the breast.

If an insufficient amount of food is supplied for any *prolonged time*, marked evidences of *malnutrition*, as wasted muscles, flabby skin, sunken fontanelle, anæmia, and delayed dentition, are present. Lack of a proper quantity of any of the ingredients of the milk for a considerable time is followed by special symptoms, as described under their special chemical components.

If by the presence of the above signs we are suspicious of the milk as being the source of trouble, we must examine into both its quantity and its quality before making any

change, so as to discover the exact cause of the infant's symptoms.

Methods of clinical analysis of mother's milk: In investigating the causes of insufficient feeding, and in watching the results of efforts made to change the different milk-constituents, soon to be described, frequent chemical analyses of the milk are necessary. Only a skilled chemist can make an accurate analysis of milk, and frequently we will have to go to one; but Holt has produced a very simple and useful method and apparatus for ordinary clinical work, which, he claims, gives as satisfactory results for milk, as the usual urinary analysis does in connection with urine.

Holt's test for clinical examination of milk: This consists of a graduated tube holding 10 c.c., a small specific gravity float, and a cylinder.

A little more than 10 c.c. of milk is required. First take the specific gravity of this in the cylinder; then pour the milk into the tube up to the 10 c.c. mark exactly, and let it stand about twenty-four hours at a nearly uniform temperature of 70° F.; at the end of this time read off the percentage of cream on the graduated tube. The *fat* is three-fifths of this amount.

Now having the specific gravity and the fat, and knowing that the sugar is very constant, we can reach a fairly good estimation of the percentage of *proteids* by remembering that the average specific gravity is 1031 and the average fat-percentage is 3.50, and that fat decreases the specific gravity and proteids increase it. Thus, if the fat is high and the specific gravity high, the proteids are very high; if the fat is high and the specific gravity normal, the proteids are high; if the fat is low and the specific gravity high, the proteids are normal; if the fat is low and the specific gravity normal, the proteids are low; while if the fat is low and the specific gravity is low, the proteids are very low. While not giving the exact proportion of proteids in figures, this does give a general idea of the amounts of the milk-constituents.

Diet for nursing women: This should be essentially the same as for *her* in health. It should be nutritious, easily

digestible, probably a little larger in quantity than under normal circumstances, and consist of a good variety of the different forms of food-stuffs. Milk is the best staple article, being taken in large quantities, and, if anything is to be taken between regular meals, nothing is better. The use of beers, malts, and alcoholics is not of much special advantage, none of them equalling milk as a milk-producer.

Exercise for nursing women: Exercise is very important and necessary for a woman who wishes to nurse her infant. It is one of the best regulators of the quality of the milk, and is indirectly of value in lactation, by its well-known power of keeping the various functions in good condition.

Drugs for nursing women: *Drugs* are eliminated through the milk much less constantly than is generally supposed. Still at times the milk does contain them when taken by the mother, a fact which must always be kept in mind when prescribing for a nursing woman, and advantage of which can be taken at times therapeutically, especially in dealing with syphilis. This method, though, of treating a nursling through its mother's milk is very inexact, and cannot be relied on.

The list of drugs that have been found in milk is large; some of the more important ones are: the purgative principles of rhubarb, senna, and castor-oil; the metals, as antimony, arsenic, iodine, lead, iron, and mercury; volatile oils, as copaiba, garlic, or turpentine; the iodides and bromides, and opium.

Methods of changing the ingredients in woman's milk: If after chemical analysis we find any marked changes either in the quantity or the quality of woman's milk, by adopting certain procedures in the way of diet and exercise we can often correct these abnormalities and bring about the production of a milk more nearly approaching the normal standard, and one that will consequently be more suitable for the child's digestion and nutrition.

Rotch gives a condensed table for these changes as follows:

To *increase the total quantity:* Increase the liquids in the mother's diet, especially milk (malt extracts may be helpful),

and encourage her to believe that she will be able to nurse her infant.

To *decrease the total quantity:* Decrease the liquids in the mother's diet.

To *increase the total solids:* Shorten the nursing-intervals, decrease the exercise, decrease the proportion of liquids, and increase the proportion of solids in the mother's diet.

To *decrease the total solids:* Prolong the nursing-intervals, increase the exercise, and increase the proportion of liquids in the mother's diet.

To *increase the fat:* Increase the proportion of meat in the diet.

To *decrease the fat:* Decrease the proportion of meat in the diet.

To *increase the proteids:* Decrease the exercise.

To *decrease the proteids:* Increase the exercise up to the limit of fatigue for the individual.

It is wise in all cases of *disturbed lactation*, whether in maternal or wet-nursing, to make efforts in accordance with these rules to produce a milk that is suitable for an infant who is not thriving, before changing to any other method of feeding.

Wet-nursing: Theoretically the milk of another woman is the best substitute for mother's milk, and there are infants who will thrive only on woman's milk. The advantages of wet-nursing are the substitution of the exact ingredients of the milk, without any previous preparation, in a sterile condition, and the feeding of this food in Nature's way.

In practice, wet-nursing is far from satisfactory, and its difficulties are many. The *disadvantages* are trouble and expense in getting a proper nurse of good character, good habits, and ability to nurse her foster-child.

Good moral qualities are necessary for many reasons. Her relations with the child and the family will be so intimate that of necessity the growing child will be influenced by its association with her, and the peace of mind of the family will be dependent on her. If of bad temper, or free from a sense of responsibility, she soon learns that her services may be

indispensable, and then she will take advantage of this fact on the least provocation. She may leave suddenly at a time when such a change to the child is distinctly dangerous. If intemperate, or dissolute, she may get intoxicated at any time, or may contract some venereal disease, after which occurrences she is distinctly dangerous to the child.

In this country the average wet-nurse is the mother of an illegitimate child, or a married woman in extreme poverty. Neither can be thoroughly depended on, but any other kind is very difficult to obtain. No woman with an illegitimate child other than the first should be taken, as in other cases the moral qualities will be undoubtedly such as to make the woman positively unfit to nurse.

Physical qualities: A good wet-nurse should be between twenty and thirty-five years of age, of healthy appearance, not anæmic, and absolutely free from present or past taint of tuberculosis or syphilis. The physician should examine carefully her mouth, teeth, and throat, her subcutaneous glands and skin, her legs, the tibiæ especially, and the genitals, if suspicious.

The breasts should be firm and conical, and present evidences of abundant gland-tissue rather than be large sized; while the nipples should be large, protruding, and free from fissures or erosions.

Before engaging her it is well to have a chemical analysis of her milk made.

Age of milk: The milk should be somewhat of the age of her foster-child; although a difference of four months is of little moment, except if she is to nurse a very young infant, when her milk should not be more than two months old. The very best evidence as to the quality of her milk can be obtained by observing its effect on the digestion and nutrition of her own child.

Change of nurse: Even after the most critical examination of the nurse and her own child her milk may not be a satisfactory food for her foster-child, and we may have to change, and try again until a nurse is found whose milk is fitted to the foster-child's stomach. Before doing this though, always

apply the rules for changing the ingredients in a woman's milk.

Causes affecting quality of milk: Often in obtaining a wet-nurse the marked change in her diet, and the diminution in the quantity of exercise she has been accustomed to take, are responsible for the milk being unfit. In our country a wet-nurse usually puts her own baby, if she has not lost it, in an institution, or boards it out with some woman who makes a business of such work, as the wet-nurse is expected to give all her time and all her milk to the foster-child. Her own child frequently gets sick, and often dies as the result of this marked change in its way of living. The worry over this is another great cause of change in her own milk, making it unfit for the foster-child. If physicians and well-to-do people, in hiring a wet-nurse, would take pains to remedy this misfortune, they would prevent one of the causes which interfere with the happiness of the wet-nurse and consequently with the quality of her milk. In wet-nursing, precisely the same rules as to frequency and regularity of feeding should be followed as in maternal nursing.

MIXED FEEDING.

By this is meant a **combination of breast and artificial feeding**. It is frequently helpful to accustom a baby fairly early to take water, or some little food once a day from a bottle. This prepares him for its use in case some sudden emergency should arise when the ordinary breast-milk could not be given.

In the **later stages of lactation** it often happens that the mother is unable to furnish enough milk for a large, vigorous baby, and then one or more bottles a day can be well used to supplement the maternal supply.

When a baby is **wet-nursed** it is still wiser to use a bottle a day, as frequently a given wet-nurse must be dispensed with either voluntarily or involuntarily, in which case the infant is not left entirely in the lurch.

In any case the proposition that partial breast and partial artificial is always to be preferred to complete artificial feeding, is to be remembered.

In giving a child artificial food when he is on breast-milk mainly, it is always well to have a chemical analysis made of the breast-milk on which he is thriving, and to compose the artificial food in conformity with this. If any one element in the breast-milk is lacking, this can often be supplied, with advantage, by an increased quantity of this element in the artificial food.

WEANING.

Indications: Nature often indicates the time that this change in feeding should be brought about by either an evident insufficiency in the mother's milk or by the continued absence of the regular weekly gain in the child's weight. The regular weighings show one of their marked uses here. These two points, combined with the presence of six or eight teeth, and with the season of the year, are of more importance in deciding on the time for weaning than the mere age of the infant. So long as the mother's milk holds out well, and she herself is not suffering from the drain, and so long as the child is gaining in weight and is healthy, be in no hurry to wean.

The **presence of teeth** shows that the starch-digesting functions are ready, makes sucking somewhat painful for the mother, and is an indication that the time for additional food to the breast-milk is arriving. Other things being equal, choose any time of the year than hot weather for weaning a baby.

Methods: Weaning had best be done *gradually* by the addition of more and more artificial feedings, and the lessening of the breast-feedings, until the latter are replaced. Here comes in the value of the early training to mixed feeding advised in a preceding paragraph. Weaning in such a case is easy, and usually without incident. The average time for weaning is, in this country, in the neighborhood of the ninth month.

COWS' MILK.

Practically the **only substitute** for permanent artificial feeding of infants is **cows' milk** so modified as to make it agree

chemically as nearly as possible with human milk. The proximate principles in cows' milk are the same as those in woman's milk, but in different proportions. There is undoubtedly some indefinable chemical or physical difference in these proximate principles, in quality also; but for practical purposes we have to ignore this, and use them as being the same until further investigation discovers what these differences are, and teaches us how to modify their quality as well as their quantity.

Cows' milk—chemical composition: Fresh cows' milk is of neutral or slightly acid reaction, the specific gravity varies from 1028 to 1033, and it consists of water holding in solution and in suspension four kinds of solids—proteids, fat, sugar, and salts. The average proportions of these are as follows: proteids, 4 per cent.; fat, 4 per cent.; sugar, 4.5 per cent.; salts, .7 per cent.; water, 87 per cent.

Comparison of cows' and woman's milk: Comparing these proportions with those in the table for woman's milk, we find that there is a marked excess of proteids and salts and a deficiency in sugar, while the fat and the water are comparatively alike.

Cows' milk—proteids: The proteids differ from those in human milk not only in quantity, but in the relative proportions of caseinogen and lactalbumin, the caseinogen here being four times the lactalbumin, instead of one-half of it as in human milk. This explains the large firm clots formed by cows' milk when coagulated by the stomach acids, as compared with the small curds from woman's milk, and hence the difficulty experienced by infants in digesting the proteids of cows' milk. In fact, the indigestibility of cows' milk for the average infant is entirely due to the proteids.

Cows' milk—fat: The fat is similar in quality as well as quantity, and presents nothing of interest from a digestive standpoint. The fat of milk is represented by the well-known cream, which by its lighter specific gravity rises to the top when milk is left to stand.

Sugar: The sugar of milk, or lactose, is the same in both kinds of milk; but being in smaller quantity in cows' milk makes this far less sweet to the taste, and, what is of more

importance, of far less nutritive value from the carbohydrate standpoint. This lack of sweetness sometimes explains the reason why babies object to cows' milk at first.

Salts: These are three and a half times as abundant in cows' milk, with a marked excess of calcium phosphate.

Cows' milk—germ life: Not only should the difference in reaction be noted, but most important is the fact that human milk as sucked from the breast is sterile, while cows' milk, as a commercial article, always contains bacteria in varying numbers.

Sources: It makes little difference what breeds of cows the milk comes from, but there are a few points worthy of mention in this connection. Contrary to the general idea, mixed milk from a herd is preferable to that from one cow, as one cow's milk is far more apt to vary in its proportions from time to time than the mixed product of a herd. Further, if this one cow should be diseased, the infant is far more exposed to infection than when the milk of one diseased cow is diluted by the milk from a healthy herd.

Milk from the Jerseys and Alderneys is much richer in fat than that of any other breed, but unfortunately these varieties are more prone to tuberculosis than the commoner breeds. Tuberculosis is a very common disease in cows, and should be always guarded against in a milk-supply. All herds used for dairy purposes should be tested at frequent intervals for tuberculosis, and the suspicious animals destroyed, and no new cows should be added to the herd until they are proved free from tuberculosis.

Cows' milk—its care: The cows should be milked with aseptic precautions, the udder and teats being washed before milking, the milker's hands being thoroughly cleansed, and the receptacles for the milk being sterile. In this way a milk with as few germs as possible is obtained, and the chances of its undergoing change by the development of toxins are reduced to a minimum. The milk should be used as fresh as possible; every hour it is kept after milking before use adding to the possibility of its developing some change making it less fit for food. Milk which is cooled directly after milking keeps the best of any on the market.

Cows' milk—sterilization: By these methods a milk is obtained as nearly sterile as possible, or what might be called "aseptic milk." But with the greatest precautions—and these are hard to effect in the ordinary dairy—all milk contains more or less bacteria. As a consequence the process of sterilization is much used, especially during hot weather, to destroy the bacteria in milk.

By this process is meant the prolonged heating of the milk at 212° F., by the action of steam or boiling water. It is usually subjected to the action of heat at this temperature for twenty minutes or more. This accomplishes the destruction of the ordinary germs upon which the diarrhœal diseases depend, as well as the tubercle bacillus, the germs of diphtheria, typhoid, and others.

Such milk keeps well, and does not cause digestive troubles; but certain other and undesirable changes take place at the same time: the taste of the milk is changed to that of boiled milk, which is disagreeable to some; the sugar is partially burned, giving the milk a brownish appearance; and the casein is rendered more difficult of digestion. Children fed on this for any length of time are likely to be constipated, and there is much authority for the statement that prolonged use of sterilized milk causes rickets and at times scurvy, from the destruction by the heat of "the vital principle" of the milk. This latter point, however, is still an open question.

Sterilization—methods: Milk may be sterilized by putting the receptacle containing the milk in a vessel of water and boiling this water for the required time. The Arnold steam-sterilizer is a practical apparatus for accomplishing this by the action of steam circulating in a confined chamber around the vessels containing the milk. Milk is best sterilized in the bottles it is to be fed from.

Cows' milk—pasteurization: Since the profession has appreciated that the sterilization of milk has its drawbacks, efforts have been made to find a method of destroying the bacteria in milk without producing these objectionable changes in the milk-constituents. The method now much used with this end in view is called *pasteurizing*, and is based on the idea of using the least amount of heat that will destroy the ordinary bac-

teria that are injurious in milk. This amount of heat has been found to be 167° F., and milk brought to and retained at this temperature for twenty minutes is found to be free from bacterial growth, and to show far less of the objectionable changes than when subjected to a temperature of 212° F. for a prolonged period. Such milk will not keep so long as sterilized milk, and cases have been reported in which *pasteurization* seemed insufficient to prevent the growth of the ordinary bacteria of diarrhœa. Neither sterilization nor pasteurization is perfect.

Pasteurization—methods: Pasteurization is best accomplished by means of Freeman's pasteurizer, an apparatus containing enough boiling water to bring a certain amount of milk in bottles immersed in it to 167° F. by its latent heat, and to keep the milk at this temperature for twenty minutes. Without some such apparatus it is difficult to keep water at a uniform temperature of 167° F. with a continuous heat-supply.

The important point to be remembered in pasteurizing is quickly to cool the milk after it has been kept at the requisite temperature of 167° F. for twenty minutes, by placing it on ice and keeping it there.

Either pasteurization or sterilization is necessary during the hot weather of summer; but may frequently be dispensed with in cool weather. Remember that they are used as preventives of digestive disorders, and not to cure them when once started.

ARTIFICIAL FEEDING.

We have seen that cows' milk contains the same proximate principles as woman's milk, but in different proportions. The problem of preparing cows' milk for use as an infant-food consists in so changing these proportions by addition and subtraction as to make a milk in which the proximate principles are virtually the same in quantity as those in woman's milk. Knowing the average percentage of the constituents of human milk, and the exact analysis of the cows' milk we are to modify, it becomes a matter of comparative ease to produce an infant-food such as is proper for the average child. In individual cases circumstances may be such as to require some far

different proportion of the proximate principles than the average child needs. These changes in individual cases, although a little more difficult in their technique, can still be produced by a little intelligent thought and care.

Artificial feeding—preparation of cows' milk: We have cows' milk giving by analysis: proteids, 4 per cent.; fat, 4 per cent.; and sugar, 4.5 per cent.; and wish to modify this to analyze: proteids, 1.5 per cent.; fat, 4 per cent.; and sugar, 7 per cent.

Proteids: By adding two parts of sterile water to one part of such milk we bring the *proteids* to 1.33 per cent., about the desired amount; but this reduces the fat also to 1.33 per cent. and the sugar to 1.5 per cent. Manifestly such a milk is markedly deficient in two of the main food-constituents, and although the child would probably digest such a food well, nutritive derangements would soon follow.

Sugar: To bring this diluted milk to the proper *sugar*-percentage, we add 5½ per cent. of milk-sugar, the amount for any definite quantity of milk being easily computed. This is about a teaspoonful of milk-sugar to four ounces of milk.

Milk-sugar is preferable mainly because it is the natural sugar of milk; but cane-sugar may be used with impunity if good milk-sugar is difficult to obtain. The results of infant feeding while using cane-sugar seem equally as good as when milk-sugar is used. In using cane-sugar a somewhat smaller quantity, as a teaspoonful to six ounces of milk, is necessary.

Fat: To bring the *fat* to the desired amount, cream of a known percentage may be added; but this introduces some proteids and some sugar with it. The best and simplest way is to take five times as much milk as it is desired to dilute for twenty-four hours' feeding of the baby, and let this stand about six hours on ice in a glass jar. At the end of this time skim off the top fifth. This is *top-milk*, so called, and analyzes about 12 per cent. of fat, with proteids and sugar each about 4 per cent. If this top-milk is diluted with two parts of sterile water, we have the proteids 1.33 per cent., the fat 4 per cent., and the sugar 1.5 per cent. Now only the addition of the 5.5 per cent. of lactose is required.

Example: As an example, suppose we had a baby who needed thirty ounces of food a day. One-third of this, or ten ounces, would be milk, so we would set aside five times this, or fifty ounces, to cool on ice for six hours. At the end of this time we would take off the top ten ounces of this milk, which would contain 4 per cent. of proteids, 12 per cent. of fat, and 4 per cent. of sugar. To this we would add twenty ounces of sterile water in which was dissolved 5.5 per cent. of thirty, or a little more than an ounce and a half of milk-sugar. This makes thirty ounces of milk analyzing: proteids, 1.33 per cent.; fat, 4 per cent.; sugar, 7 per cent.; salts, .23 per cent. The only change left is due to the cows' milk being acid in reaction, which is overcome by the addition of bicarbonate of sodium, one grain to the ounce, here adding thirty grains; or of lime-water, one to twenty, here adding an ounce and a half. In alkalinizing with lime-water we must remember that the water further dilutes our milk, and must leave out that much of the diluting water.

At times, instead of diluting the top-milk with sterile water, *barley-* or *oatmeal-water* is used as a diluent. Either may be made by boiling an ounce of barley or oatmeal in a quart of water to a pint and then straining for use. They are somewhat pasty, and are supposed to assist in the digestion of the casein; and the oatmeal-water to be of value in constipation.

After the *total quantity* of food is modified to the right proportions it is *separated* into the requisite number of nursing-bottles for a day's supply, each bottle holding the proper quantity. These are then stoppered with ordinary non-absorbent cotton, and are sterilized or pasteurized if required; or if the process of preparation has been carried on aseptically, are simply set aside in the refrigerator. When ready to use, the bottle is warmed to about 98° F., the temperature for eating, the cotton is removed, and a clean nipple applied over the neck.

Artificial feeding—rules for feeding: The average-sized child should be fed certain definite quantities of this milk at certain definite intervals, a scheme being constructed using the same intervals as in breast-feeding, and calculating the

amount at a feeding by the average quantity obtained from a breast.

Scheme for Artificial Feeding during the First Year.

(*The Feedings begin at 7 A. M.*)

Age.	Intervals (hours).	No. of feedings in twenty-four hours.	Omitted No. of night feedings (11 P.M. to 7 A.M.)	Amount at each feeding (ounces).	Total in twenty-four hours (ounces).
1 week	2	10	2	1	10
2 weeks	2	10	2	$1\frac{1}{2}$	15
4 weeks	2	10	2	2	20
6 weeks	2	10	2	$2\frac{1}{2}$	25
2 months	$2\frac{1}{2}$	8	2	$3\frac{1}{4}$	26
3 months	$2\frac{1}{2}$	8	2	$3\frac{1}{2}$	28
4 months	3	6	2	$4\frac{1}{2}$	27
5 months	3	6	2	5	30
6 months	3	6	2	$5\frac{1}{2}$	33
7 months	3	6	2	6	36
8 months	3	6	2	$6\frac{1}{2}$	39
9 months	3	6	2	7	42
10 months	3	6	2	$7\frac{1}{2}$	45

The bottle should be finished in *twenty minutes;* if not, take it away. Do not warm the remnant and give it again to the baby.

By the time the *tenth month* is finished the average baby begins taking some other form of food as a substitute for one or more of the milk-feedings.

In *large, robust children* somewhat larger quantities than the above averages are better; while in small, delicate children smaller quantities are prescribed. The stomach undoubtedly bears some direct ratio in size to the weight of the infant, a point which should be remembered in deciding on the proper quantity of milk to be given a particular infant at a nursing. The regular weekly weighings are here of equal value as in breast-feeding as an index of whether the baby is being properly nourished.

Artificial feeding—care of bottles and nipples: The best bottles are graduated, for ease in measuring the food; and have wide neck and sloping shoulders, for ease in cleaning. They should be kept scrupulously clean of all evident signs of milk or foreign matter, and before being filled with the milk should be boiled.

The best *nipples* are of black rubber, and large enough to fit over the neck of the bottle and to be turned inside out for cleaning. Those with a rubber tube connecting them to the bottle and running into the milk should not be used, as it is almost impossible to keep them clean. The nipple should be cleaned thoroughly after use, and kept in a borax solution when not in use. The hole in the nipple should be large enough to allow the milk to drop out, but not to run in a stream.

Milk laboratories: There have been established in very recent years in many of the large cities of the United States milk laboratories for the exact modification of cows' milk to fit it for infant feeding. At these laboratories the milk is ordered by prescription exactly as drugs are ordered by prescription at the apothecary's. Each laboratory is a branch of the original Walker-Gordon laboratory founded in Boston under the supervision of Rotch. Each laboratory has its own herd of cows, which are fed and housed with great hygienic care, and are tested for tuberculosis regularly with tuberculin. The milk is milked and shipped to the laboratory under aseptic precautions. At the laboratory it is separated in a centrifugal machine and a 16 per cent. cream obtained. The skimmed milk is used for the proteids, and a 25 per cent. solution of lactose for the sugar. The chemist at the laboratory knows the quantity of proteids and sugar in the 16 per cent. cream, and the quantity of proteids, fat, and sugar in the skimmed milk. With these and the definite solution of milk-sugar he can form modified milk of any definite strength of proteids, fat, and sugar.

The physician in ordering the milk knows the requirements of his particular case, and on a prescription-blank prepared for this purpose writes the order for the modified milk to be delivered ready for feeding.

The form of **prescription-blank** is as follows:

℞ Proteids,	%.	Alkalinity,	%.
Fat,	%.	No. of feedings,	.
Sugar,	%.	Amount at each feeding,	℥.
		Heat to	° F.

This being filled out by the physician as required, is sent to the laboratory, and each day thereafter, until changed by the physician, a basket of the required number of bottles for twenty-four hours is delivered, each bottle containing the exact quantity of milk of the proper proportions ready for feeding. This should be kept in a cool place, and when a bottle is to be fed it should be warmed to the proper temperature for feeding (98° F.), the cotton stopper removed, and the nipple applied.

Milk laboratories—their advantages: Food prescribed through the medium of the laboratory has the advantage of accuracy of percentage of constituents, of exact quantity at a feeding, of sterility of the milk, and of ease of changing the percentages gradually and exactly as conditions demand. Further, it is next to impossible for the family to make any change in the quality or quantity of this milk without the knowledge of the physician, a fact of value in dealing with difficult cases of digestion or nutrition.

Artificial feeding—home modification: The great disadvantage of the milk laboratory is its expense, thus putting such milk out of reach of the masses of the people. With patients who cannot afford this milk, and in localities where there are no laboratories, home modification is our only resource. Modification at home can be done with some little trouble, and although exact percentages cannot, of course, be gotten, comparative accuracy can be attained and a good substitute for laboratory-milk produced. Frequently, in cases requiring much care in feeding, laboratory-milk may be used temporarily until improvement begins, when home modification can be substituted, thus using the expensive milk for a short time only.

Home modification—Coit's decimal method: This is the

simplest and easiest worked method of home modification yet suggested. It is based on the metric system, and all the calculations are made in decimals.

Three solutions are required: 1. A decimal (10 per cent.) cream, or super-fatted milk for introducing the fat; 2. A saccharated (10 per cent.) skimmed milk for introducing proteids not carried by the cream; 3. A standard (10 per cent.) sugar solution for introducing the lactose not carried by the cream or the skimmed milk. Solutions 1 and 3 only are required when the proteid percentage is small. As the child grows older, and a higher proteid percentage is necessary, solution 2 is required also.

Decimal cream is produced by allowing a quart of ordinary fresh milk from a mixed herd to stand on ice for fifteen hours, and at the end of this time one-fifth of it is taken from the top. This averages 15 per cent. of fat, and loses about $\frac{1}{2}$ per cent. each of sugar and proteids. If to this we add one-half its volume of water, a decimal cream is obtained, analyzing: 10 per cent. of fat, 2.33 per cent. of proteids, and 2.66 per cent. of sugar. From this the following formulæ, showing the amounts of proteids and lactose coincidently introduced with any definite fat-percentage, are easily deduced:

Decimal cream in introducing 4 per cent. of fat, also introduces 1 per cent. of proteids and 1 per cent. of lactose. Decimal cream in introducing 3.5 per cent. of fat, also introduces .8 per cent. of proteids and .9 per cent. of lactose. Decimal cream in introducing 3 per cent. of fat, also introduces .7 per cent. of proteids and .8 per cent. of lactose. Decimal cream in introducing 2.5 per cent. of fat, also introduces .6 per cent. of proteids and .7 per cent. of lactose. Decimal cream in introducing 2 per cent. of fat, also introduces .5 per cent. of proteids and .5 per cent. of lactose.

Saccharated skimmed milk depends on the fact that skimmed milk analyzes 4 per cent. of proteids and 5 per cent. of sugar. Five per cent. more of lactose is added simply for convenience of calculation. This means adding one ounce by weight of lactose to twenty ounces of skimmed

milk. Our solution then analyzes: proteids 4 per cent. and lactose 10 per cent. If we wish to add 1 per cent. of proteids, we use one-fourth of the total food required from solution 2; if .5 per cent. of proteids, one-eighth, etc., always remembering that we introduce coincidently two and one-half times as much sugar. The formulæ here deduced are also plain:

Amount of food in c.c. $\times\frac{1}{8}$ (saccharated skimmed milk) adds proteids .5 per cent. and lactose 1.25 per cent. Amount of food in c.c. $\times\frac{1}{4}$ (saccharated skimmed milk) adds proteids 1 per cent. and lactose 2.5 per cent. Amount of food in c.c. $\times\frac{3}{8}$ (saccharated skimmed milk) adds proteids 1.5 per cent. and lactose 3.75 per cent. Amount of food in c.c. $\times\frac{1}{2}$ (saccharated skimmed milk) adds proteids 2 per cent. and lactose 5 per cent.

Standard sugar solution is prepared by dissolving 10 per cent. of lactose in sterile water, or two ounces by weight in twenty ounces of water.

In calculating formulæ four facts only are necessary: the quantity of food required; the percentage-formulæ required; that the standards, except the proteids, are 10 per cent.; and the quantity of other constituents introduced with the standards.

With these facts in mind, all that is necessary further is to reduce the quantity expressed in ounces to cubic centimetres by multiplying by thirty, and to multiply this product by one-tenth of the constituent to be introduced. Examples with and without the introduction of extra proteids will be given:

Single feeding:

	Per cent.		
	Fat.	Proteids.	Sugar.
Quantity, ℥2. Formula desired,	2.	.50	6.
℥2 × 30 = 60 c.c. × .2 = 12 c.c., decimal cream, adds,	2.	.50	.50
Leaves,	0	0	5.50
℥2 × 30 = 60 c.c. × .55 = 33 c.c. sugar solution, adds,			5.50

Working formula—12 c.c. decimal cream.
33 c.c. standard sugar solution.
15 c.c. water.
60 c.c.

One day's food:

	Per cent. Fat.	Proteids.	Sugar.
Quantity, ℥35. Formula desired,	4.	1.	6.50
℥35 × 30 = 1050 c.c. × .4 = 420 c.c. decimal cream, adds,	4.	1.	1.
Leaves,	0	0	5.50
℥35 × 30 = 1050 c.c. × .55 = 577.50 c.c. sugar solution, adds,			5.50

Working formula — 420 c.c. decimal cream.
577.50 c.c. standard sugar solution.
52.50 c.c. water.
1050.00 c.c.

One feeding:

	Per cent. Fat.	Proteids.	Sugar.
Quantity, ℥5. Formula desired,	4.	1.50	7.
℥5 × 30 = 150 c.c. × .4 = 60. c.c. decimal cream, adds,	4.	1.	1.
Leaves,	0	.50	6.
℥5 × 30 = 150 c.c. × ⅛ = 18.75 c.c. skimmed milk, adds,		.50	1.25
Leaves,	0	0	4.75
℥5 × 30 = 100 c.c. × .475 = 71.75 c.c. sugar solution, adds,			4.75

Working formula — 60 c.c. decimal cream.
18.75 c.c. saccharated skimmed milk.
71.25 c.c. standard sugar solution.
150.00 c.c.

Other methods have been introduced by Holt and by Westcott, but require the memorizing of algebraic formulæ or of certain combinations, while this method of Coit is the simplest of all, really requiring no memorizing, as the whole process can be deduced from the formulæ for milk-constituents.

In cities where milk with certain *definite fat-percentages* is sold a less exact method of home modification and one not admitting of gradual changes of the constituents, but still of practical use where precision is not specially requisite, is very easy to adopt.

The so-called **8 per cent. milk** analyzes: proteids, 3.9 per cent.; fat, 8 per cent.; sugar, 4.3 per cent.

The so-called **12 per cent.** milk analyzes: proteids, 3.8 per cent.; fat, 12 per cent.; sugar, 4.2 per cent.

The changes in the sugar and proteids are so small that they can be almost overlooked, especially as we are not aiming at marked exactness.

If we wish a milk of *medium proteid strength*, we choose the *12 per cent.* milk, and dilute it with twice its volume of

sterile water. This mixture analyzes: proteids, 1.26 per cent.; fat, 4 per cent.; sugar, 1.4 per cent. If to this we add 5 per cent. of milk-sugar, we have a modified milk of fair average proportions.

If we wish a milk of *higher proteid strength*, we choose the *8 per cent.* milk, and dilute it with an equal quantity of sterile water. This mixture analyzes: proteids, 1.95 per cent.; fat, 4 per cent.; sugar, 2.15 per cent. Add to this 4 per cent. of milk-sugar, and we get a stronger milk than before.

By making slight changes in the proportions of water and these milks we can modify the constituents still further. Here again the addition of sodium bicarbonate, one grain to the ounce, or of lime-water, 1 : 20, is used to alkalinize the milk.

Cows' milk—methods of examination: The reaction and specific gravity should be taken and the amount of fat calculated. Holt's cream-tube may be used; but it is not so accurate as in analyzing breast-milk. If used, the cream should be made to rise rapidly by standing the fresh milk on ice for about eight hours. The fat is now about one-fourth of the cream.

The best *optical test* is the Feser lactoscope. It depends on the obscuring of dark lines seen through the milk diluted with more or less water. This test is only approximate, and depends too much on individual experience.

The most accurate and quickest method is by means of Babcock's centrifugal machine, in which the milk is mixed with sulphuric acid and then revolved rapidly in the machine, the fat coming to the top of the narrow tube in five minutes and being read off.

So far there is no clinical method for estimating the proteids, this requiring the work of a skilled chemist. The sugar is fairly uniform at 4.5 per cent.

CONDENSED MILK.

Condensed milk is made by evaporating fresh milk, which has been sterilized by heat, to about one-quarter its volume.

After this it is preserved by adding about one-third its weight of cane-sugar before being sealed in the cans. In many cities fresh condensed milk, without the addition of sugar, is sold. The following table from Holt gives the composition of condensed milk, and of dilutions of it six, twelve, and eighteen times:

	Condensed Milk.	Diluted 6 times.	Diluted 12 times.	Diluted 18 times.
Fat	6.94	.99	.53	.36
Proteids	8.43	1.20	.65	.44
Sugar { Cane-, 40.44 / Milk-, 10.25	50.69	7.23	3.90	2.67
Salts	1.39	.17	.10	.07
Water	31.30	90.49	94.82	96.46

Condensed milk as a food: The dilution of twelve parts is nearest that ordinarily used for infant-feeding. A study of its composition as diluted thus shows that the infant is getting almost no fat, a quite low proteid percentage, about the right quantity of sugar, and altogether too little salts. The total solids lack 7 per cent. of what they should be. Such a food is easy of digestion, but decidedly lacking in nutrition. Infants thus fed have as a rule little trouble, if any, with their digestion, and are fat and plump from the sugar, but invariably show signs of more or less rachitis, depending on the length of time the condensed milk is continued. This latter fact seems to depend on the lack of fat and salts, and possibly the proteids also. Further, these children although appearing healthy and well nourished, have very little resisting power and readily succumb to acute diseases.

The addition of cream to condensed milk, or the coincident use of cod-liver oil, will prevent these bad symptoms from its prolonged use.

Condensed milk has some marked *advantages* as a food: it is sterile, it is very easy and simple to prepare, and it is very cheap. As a temporary food to bridge over sickness, or to use in travelling, it is very valuable; but it ought never to be used for any length of time unless other food is given with it.

INFANT FOODS.

Of recent years a large number of **proprietary** or patent or manufactured **foods** have been put on the market and widely advertised and largely used for feeding infants. They are mostly dried powders, intended to be dissolved in water for use. It is quite practical to divide them into two distinct classes at once: those free from, and those containing, unchanged starch.

In the first class are malted milk and Mellin's food; in the latter are all the rest, which, having raw starch as a constituent, are manifestly unfit for infant feeding during the first nine or ten months of life. No further attention will be paid to these.

Malted milk and **Mellin's food** contain no ingredient that is objectionable for feeding, but are lacking in one of the important ingredients of milk—the fat, and contain a very large excess of sugar. Mellin's food is recommended to be dissolved in fresh milk to overcome partially this defect.

Analyses of these when dissolved in about the amount of water in milk are as follows:

	Malted Milk.	*Mellin's Food.*
Fat, ...	.39 per cent.	.04 per cent.
Proteids, . . .	2.28 "	1.50 "
Sugar, ...	10.18 "	11.56 "
Salts, ...	.5 "	.45 "
Water, ...	86.65 "	86.45 "

Looking over these analyses we find about a normal per cent. of proteids, but of vegetable and not animal origin, virtually no fat, and a great excess of carbohydrates. As food they often produce, like condensed milk, fat, plump babies; but these infants again are not resistant and succumb readily to acute diseases. These foods are often well digested, and are useful at times as temporary foods when it is desirable for any reason not to use cows' milk; but so many cases of both rickets and scurvy have been traced to dried foods as a cause that it is well never to use any of them for any great length of

time without using simultaneously some fresh food. The addition of cream to either does away with the lack of fat, and adds the element of freshness to the food, but what value the addition of the food to fresh cows' milk has, is as yet unknown.

During the *second year* of life they form a more useful addition to an infant's dietary.

Peptonized milk: This is milk in which the proteids are changed to peptones, or, in other words, digested, by the addition and action of pancreatic ferment. The process may be stopped when partially performed, giving a product of which the taste is not objectionable; or may be carried on to complete peptonization, when the product has a very bitter, disagreeable taste.

Method: To peptonize milk *partially*, add to a pint of fresh cows' milk and four ounces of water five grains of pancreatic extract and fifteen grains of bicarbonate of soda. Allow this to stand at a temperature of 105° to 115° F. for five to twenty minutes, then bring to a boil to kill the ferment, or stand on ice to prevent its further action. If the milk is to be used at once, neither of these latter is necessary.

To peptonize the milk *completely*, allow the process to continue for one to two hours. After this time the addition of acid produces no coagulation.

In infant feeding it is better to peptonize a modified than a whole milk. Peptonized milk is frequently very useful in feeding an infant with feeble digestive powers; but it is unwise to continue its use over too long a period, as then the infant's stomach, being called on to do no work, becomes enfeebled from disuse and gradually unable to perform its proper function.

Whey: By coagulating one pint of fresh milk by adding a teaspoonful of essence of pepsin, and allowing this to stand, a solid curd is formed swimming in a liquid—whey. This has the following composition: proteids, .86 per cent.; fat, .32 per cent.; sugar, 4.79 per cent.; salts, .65 per cent.; water, 93.38 per cent.

This at times makes a very valuable food for infants in cases of gastric or intestinal disorder, where the use of milk

must for a time be interdicted. Babies like it, it is very easy of digestion, and does not irritate the stomach. A little wine may be added if desired.

Egg-water: This is made by mixing thoroughly the white of one egg with six ounces of water and adding a little salt. The addition of a few grains of sugar will make the child take it better, and adds also a food-element.

Such a mixture is one of the best foods we have for temporarily feeding an infant with digestive disturbances when we wish for a time to stop temporarily all milk food.

Beef-juice: Expressed beef-juice is obtained by slightly broiling a piece of lean beef, and then squeezing the juice from it by a lemon-squeezer. One pound of steak yields two or three ounces of juice. This is flavored with salt and given cold or warm. Do not heat enough to coagulate the albumin. This is very nutritious and usually well taken. It may be given at the rate of a tablespoonful three times a day.

Scraped beef: This is another valuable and easily digested food. It is prepared by scraping with a dull knife some raw or rarely done lean beef. A tablespoonful of this salted is the amount usually given at a feeding.

Broths: These are made by first soaking and then boiling one pound of lean beef, mutton, veal, or chicken, in one pint of water. They do not contain a large quantity of nourishment, but do have in them many extractives, and hence are stimulating rather than nutritious.

Barley-, oatmeal-, or rice-water: These are made by boiling an ounce of barley, oatmeal, or rice in a quart of water to a pint, and straining before use.

Feeding in the second year: During the second year a child should have five meals a day, about 7 and 10 A. M. and 1, 4, and 7 P. M., and nothing between meals. A *sample diet* is as follows: 7 A. M., a tablespoonful of some well-boiled cereal—wheat, rice, oatmeal, barley, or hominy, with cream and a little sugar if necessary; 10 A. M., a half pint of milk; 1 P. M., tablespoonful of scraped beef, or soft-boiled egg, piece of dry bread, a half pint of milk; 4 P. M., a half pint of milk; 7 P. M., a tablespoonful of cereal with cream

and sugar; a little orange juice may be given from time to time.

Feeding in the third and fourth years: During this time four daily meals are sufficient, at 7 and 10.30 A. M. and 1.30 and 6 P. M., and again nothing should be given between meals. A sample diet is as follows: 7 A. M., orange, cereal with cream and sugar, glass of milk; 10.30 A. M., glass of milk or cup of broth and slice of stale bread or toast or zwiebach; 1.30 P. M., piece of meat—steak, chop, or chicken—two vegetables, potatoes and spinach, rice or bread pudding, or prunes, or apple sauce; 6 P. M., bread and milk, or milk toast.

CHAPTER VI.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

CATARRHAL STOMATITIS.

Definition: This is a simple catarrhal inflammation of the mucous membrane of the mouth, unaccompanied by ulceration.

Etiology: Irritants taken into the mouth, as too hot fluids, dirty teething-rings, sugar-teats, or substances the child may pick up from the floor, are active causes. It is at times started by excessive or rough efforts at cleanliness. It complicates the infectious diseases, as measles, scarlet fever, diphtheria, or influenza. The eruption of the teeth may at times produce it.

Pathology: There is congestion, accompanied by desquamation of the buccal epithelium. This is followed by increased secretion of the mucous and salivary glands.

Catarrhal stomatitis—symptoms: The mucous membrane of the mouth is red, swollen; at first hot and dry, later bathed in a profuse secretion. The temperature is slightly elevated, the child is restless and fretful. The mouth is tender, as shown by marked evidences of pain when anything is put into the mouth or on examination. In severe cases the child refuses food. At times the inflammation may be severe enough to produce a slightly blood-stained secretion. The tongue is coated on the surface and reddened on the edges. The neighboring lymphatic glands may be enlarged and tender.

Prognosis: This is good. The duration depends on the cause.

Catarrhal stomatitis—treatment: Remove the cause, if it

can be found. Attend to the general hygiene and diet. Use frequently gentle antiseptic cleansing of the mouth with cold washes of saturated solution of boric acid, or borax 2 per cent. In the severer cases a daily application of a 1 per cent. nitrate of silver solution will hasten the cure.

FOLLICULAR STOMATITIS.

Synonyms: This is also called *aphthous* and *herpetic stomatitis* from the formation of small vesicles, which later form superficial ulcers. These ulcers at first are discrete, but may coalesce into larger ones. They, however, always remain superficial.

Etiology: There seems to be a reflex nervous origin of this form of stomatitis, similar to that seen in connection with herpes elsewhere. Gastro-intestinal disorders and dentition seem to be factors of this sort. The cause is more frequently general than local.

Pathology: On the mucous membrane of the cheeks or lips, or the edges of the tongue, but not in the gums, there are present *pearl-colored vesicles*, about the size of a pin's head, and later small ulcers formed by abrasion of the epithelial covering of these vesicles. More or less catarrhal stomatitis is always associated.

Follicular stomatitis—symptoms: The symptoms resemble those of catarrhal stomatitis, but are more marked. There are fever, furred tongue, heat, redness, swelling, pain, and increased secretion. The mouth presents the appearance described of vesicles and superficial ulcers, and in addition general redness. The lymphatic glands of the neck are swollen and tender.

Prognosis: This is good. It is a self-limited disease; but if improperly managed, may go on to ulcerative stomatitis.

Follicular stomatitis—treatment: Regulate the diet and general condition of the patient. Bathe the mouth frequently with solution of potassium permanganate, grains three to the ounce. Chlorate of potassium given internally, grains two every three hours, well diluted, has repute with some. Solid silver nitrate may be applied to the ulcers.

ULCERATIVE STOMATITIS.

Definition: This form of stomatitis is only seen with the presence of teeth, and consists in an ulcerative process beginning in the mucous membrane of the gums around the teeth, and spreading from this point to the rest of the mouth. It is accompanied by a peculiar fetor of the breath.

Etiology: Overuse of mercury, decayed teeth, improper food, bad hygiene, exhausting diseases, and scurvy are frequent causes. It is often a sequel of the infectious diseases.

Pathology: The process begins around a tooth and involves the gum and the contiguous surfaces of the lips, cheek, and edges of the tongue. The mucous membrane is much swollen and of a deep livid hue. The ulceration may extend deeply to the periosteum and cause necrosis of the maxilla. The ulcerative process never extends beyond the mouth.

Ulcerative stomatitis—symptoms: These are pain, fretfulness, change in disposition, crying, and wakefulness. There is an increase in the buccal secretions, to which there is a fœtid odor. The mouth and gums bleed frequently. On inspection the gums are swollen, spongy, livid, and bleed easily. A line of ulceration, with a white necrotic appearance, will be found around one or more of the teeth. In severe cases the teeth may be loosened and sequestra of bone found. The submaxillary lymphatic glands are badly swollen and painful. The tongue is swollen, thickly coated, and shows the indentation of the teeth on the edges.

Prognosis: If left to itself, the disease progressively increases, the ulceration extending further and further until a frightful condition of the mouth results. *Gangrenous stomatitis* may supervene. If properly treated in the early stages, a rapid cure results; and even when further advanced treatment is very efficacious.

Ulcerative stomatitis—treatment: First remove the cause by stopping the use of mercury, improving the hygienic surroundings, and treating the scorbutus if present. Keep the mouth scrupulously clean by frequent washings with peroxide of hydrogen or permanganate of potassium. If there is much bleeding, a solution of alum is useful. Internally, chlorate of

potassium is almost a specific, being excreted after absorption by the buccal mucous membrane. It is best given at the rate of two grains, largely diluted, every two hours. If under this form of treatment the ulcers do not heal, some of the teeth may need extracting, as a sequestrum is probably present. The ulcers may heal faster under daily pencillings with silver nitrate in stick-form.

THRUSH.

Etiology: This form of stomatitis is caused by the presence and growth in the mouth of a fungus called the *saccharomyces albicans*. It is a parasite of the class of yeast fungus.

The fungus is the only cause, but it never grows except on a previously unhealthy mucous membrane. It develops specially in feeble, badly nourished, and marasmic infants, and in those suffering from gastro-intestinal diseases. The use of improper food, uncleanliness of the mouth, fermentation of particles of food, or a previous catarrhal stomatitis favors its growth. Under favorable conditions the fungus will grow on any of the mucous membranes of the body. The spores are conveyed to the mouth usually by dirty nipples, sugar-teats, and such like, but may spread through the atmosphere.

Pathology: The parasite consists, microscopically, of long threads (the mycelium) interwoven together, and, in their "meshes," oval bodies (the spores). The fungus lodges on the mucous membrane of the mouth and grows in little clumps of white between the epithelial cells, thence spreading to the surface. These white tufts may be scattered uniformly all through the mouth, and many may coalesce into larger lumps. They seldom produce pus. The mucous membrane of the mouth is the seat of catarrhal stomatitis.

Thrush—symptoms: The subjective symptoms are very slight. The appearance of the mouth is very characteristic. The whole mouth, or only parts, is studded with little white feathery spots, seeming to rise above the surface of the mucous membrane, and which do not rub off with ease, but leave a bleeding spot behind. They appear first on the tongue and cheeks, but may spread to the lips, palate, tonsil, and pharynx, and at times to the œsophagus and stomach.

Each spot has the appearance of a little lump of coagulated milk, but is differentiated from this by the difficulty of removal. These cases usually have acrid, irritating stools with erythema of the buttocks.

Prognosis: It is not in itself a serious disease, but so often appears in a much debilitated child that it is associated in the lay mind with severe cases. If properly treated in a child strong enough to withstand the original disease, thrush is always recovered from.

Treatment: Thrush may almost always be prevented by thorough cleanliness of nipples, bottles, and mouth. If present, the disease is treated by the use of an antiseptic mouth-wash, the best of which is a solution of boric acid in glycerin, grains ten to the ounce, applied four times a day with a soft rag or camel's-hair brush. Special attention should be paid to remedy any underlying condition.

GANGRENOUS STOMATITIS.

Definition: This disease is also called noma and cancrum oris, and consists essentially of a gangrene beginning in the mucous membrane of the gums, cheeks, or lips, spreading rapidly, and destroying all the tissue it attacks.

Etiology: It is usually a secondary disease following measles, whooping-cough, chronic intestinal catarrhs, or general sepsis. It seems never to develop in previously healthy children. At times it supervenes on ulcerative stomatitis. Streptococci are found in most of the cases, but no specific germ has as yet been isolated.

Pathology: The mucous membrane first presents a brawny induration, followed by a sloughing ulcer. This induration extending to the skin gives rise to a livid, glazed appearance in the integument, which later becomes black, and perforation of the cheek follows. The sloughing process may extend and involve the whole side of the face and the bones of the jaw. The vessels become thrombosed and hemorrhage is rare. A line of demarcation rarely forms, but the gangrene steadily spreads till death ensues.

Symptoms: The constitutional symptoms of gangrenous

stomatitis are those of great prostration and sepsis, being more or less marked at the beginning, but rapidly increasing in severity as the disease progresses. The temperature runs from 102° to 105° F. The pulse is rapid and feeble, the appetite is lost, and a severe diarrhœa is frequent. Septic pneumonia frequently supervenes.

Locally, in the early stages there is moderate pain, but it is never very marked. The typical appearance of the ulceration spreads in all directions from its starting-point, and a gangrenous odor is present in the breath. The secretions of the mouth are increased in quantity, and soon become thick and sanious. The gangrene may involve the whole cheek, the eyelids, and even the eye. The appearance is one of the most repulsive ever seen. The duration of the disease is one to two weeks.

Prognosis: This is very bad, fully three-fourths of the cases dying.

Gangrenous stomatitis—treatment: Support the patient's strength with nutritious food, stimulants, and tonics. *Locally*, the necrotic area, as soon as a diagnosis is made, should be freely destroyed, under the influence of an anesthetic, by the actual cautery. The cauterization should go well beyond the diseased into the healthy tissue. If new gangrenous spots appear, these should be treated in the same way. The wound should be dressed afterward with strong antiseptics and the mouth kept very clean by the use of peroxide of hydrogen.

CROUPOUS STOMATITIS.

Definition: This is the form of stomatitis in which the buccal mucous membrane undergoes an inflammatory process accompanied by the production of a *false membrane*. It is also called diphtheritic stomatitis.

Etiology: Intense chemical irritants may rarely form a false membrane; but the large majority of the cases are due to the growth on the buccal mucous membrane of the *Klebs-Löffler bacillus*. In the mouth it is almost invariably secondary to the presence of diphtheritic membranes elsewhere, as

on the tonsils or pharynx; but the mouth may possibly be the primary seat.

Pathology: There is a stomatitis affecting the lips and cheeks which is accompanied by the growth on these inflamed parts of a pseudo-membrane, which is firmly adherent to its seat.

The **symptoms** of croupous stomatitis are usually those of the primary diphtheria, with the addition of sore, tender mouth. Diphtheria of the mouth always belongs to the severe cases of the disease.

Croupous stomatitis—treatment: The primary diphtheria is to be treated as always, and the mouth by frequent and gentle cleansing with a saturated solution of boric acid. The membrane should not be forcibly removed.

ADHÆSIA LINGUÆ.

This condition, commonly known as **tongue-tie**, consists of an abnormally short frænum. It may interfere with suckling, and later may possibly affect the speech, but is not nearly so important as is commonly supposed.

Treatment: Snip the frænum near its attachment to the tongue with a pair of scissors, and then tear the cut deeper by a dull instrument, as the finger-nail.

RANULA.

Definition: This is a cyst, forming in the floor of the mouth on either side of the frænum. It varies in size, but may become large enough to interfere seriously with the uses of the mouth. It is due to occlusion of a mucous duct, or the duct of the sublingual gland. The cyst may be simple or multilocular.

Symptoms: This growth is painless, fluctuates, and is the color of the buccal mucous membrane. The fluid it contains is a glairy mucus.

Ranula—treatment—Snip off the top of the cyst-wall, evacuate the fluid, and cauterize the interior of the sac with solid nitrate of silver or iodine.

ALVEOLAR ABSCESS.

Definition: This is fairly common in children with teeth allowed to be kept in a state of decay or uncleanliness. It consists of an inflammation going on to the production of pus beginning around the roots of a tooth. The periosteum of the jaw may be involved, and, if neglected, necrosis of the jaw may result.

Alveolar abscess—symptoms: There is pain in the affected part, with fever and other constitutional symptoms. The face is always badly swollen on the outside, and in the mouth is a similar condition. After the formation of pus fluctuation can be made out within the mouth. The pus may perforate into the antrum, if in the upper jaw, or will discharge through the buccal mucous membrane or the skin, if left alone.

Alveolar abscess—treatment: The teeth should be kept clean by the use of a tooth-brush, and decayed fangs should be extracted. If the abscess begins to form, it should be hastened by the use of hot applications in the form of poultices externally and hot washes in the mouth. As soon as pus is detected the abscess should be lanced from within the mouth (to prevent cutaneous scars), the pus evacuated, and the cavity well drained and packed.

DISEASES OF THE THROAT.

ACUTE PHARYNGITIS.

Pathology: In this condition the whole pharynx and the tonsils are inflamed and red. It may be, and frequently is, a primary disease; or it may be part of one of the infections, as scarlet fever, measles, diphtheria, or influenza.

Etiology: It is most commonly caused by exposure to cold, but probably behind this is some bacterial invasion. The disease at times appears infectious. A rheumatic diathesis is frequently present. Certain individuals present a marked predisposition and have recurrent attacks.

The **symptoms** of acute pharyngitis are pain in swallowing and dryness in the throat, with later an increase in the

secretion. There is frequently an irritating purposeless cough. On examination the soft palate, uvula, tonsils, and pharynx are seen to be red and inflamed. The posterior surface of the soft palate is often attacked early and the whole naso-pharynx involved.

The constitutional symptoms may be marked, with rise of temperature at times to 103° F., and its accompanying symptoms. Vomiting may be present.

Diagnosis: This is easy from the inspection of the throat; but we should never forget the possibility of the pharyngitis being the initial lesion of one of the infectious diseases.

Acute pharyngitis—treatment: The bowels should be opened by fractional doses of calomel frequently repeated. Small doses of phenacetin given every three hours will reduce the fever, ease the pain, and give general comfort to the patient.

Locally, the throat should at short intervals be sprayed or swabbed, or the naso-pharynx washed out, according as the child is old enough to allow one or the other method of application. Some mild alkaline wash, such as Seiler's solution, seems the best.

Chronic pharyngitis: This is a rare condition in childhood, but may develop as the result of frequent attacks of the acute form. One of its common results is to produce an elongation of the uvula. With this is associated an *harassing cough*, from the uvula tickling the base of the tongue by its constant presence.

Treatment: Astringent local applications are useful in this condition, but amputation of the *tip* of the uvula may be required for cure.

RETRO-PHARYNGEAL ABSCESS.

Definition: In this disease a collection of pus makes its appearance in the posterior pharyngeal wall. In a general way there are two sources of this pus: either from a suppurative inflammation of the *connective tissue*, or of a *lymphatic gland*, of the posterior pharyngeal wall; or from *caries of the cervical vertebræ*. What is ordinarily understood by retro-

pharyngeal abscess is the former variety—*i. e.*, that without disease of the bone.

Retro-pharyngeal abscess—etiology: The cause usually lies in an infection of the lymphatics from a precedent inflammation of the nose or pharynx. The disease is regularly one of infancy, and infants are particularly prone to adenitis. The disease is most frequent in winter and spring, when diseases of the nose and throat are commonest. It may follow an attack of influenza, or more rarely scarlatina or measles. It is usually seen only in delicate children.

Pathology: The simple form is primarily a suppurative inflammation of one or more of the lymphatic glands lying just in front of the cervical vertebræ. The inflammation spreads to and involves the cellular tissue. Some few cases never go on to the formation of pus. In the form due to cervical caries a much more serious condition, with broken-down bone, is present.

Retro-pharyngeal abscess—symptoms: The three symptoms of most importance are *stiff neck*, *dysphagia*, and *dyspnœa*. This dyspnœa may come on in sudden attacks and appear quite alarming. The temperature is raised to 102° F., and the child is usually sick for five or six days before the abscess develops.

The *diagnosis* is made by a careful ocular and digital examination. A fluctuating swelling is found directly in front of the bodies of the vertebræ, but a little to one or the other side. The mucous membrane of the soft palate is usually red and swollen. There may be a tumor at the angle of the jaw on the same side.

Prognosis: The abscess, if left to itself, usually ruptures in the course of a week or two. The pus may be swallowed or expectorated. If rupture does not occur, the pus may burrow in the neck. Fatal cases may occur from stoppage of the respiration or the bursting of the abscess into the larynx. If properly treated, rapid recovery is the rule.

The cases due to spinal caries, of course, take their prognosis from this, the underlying factor.

Retro-pharyngeal abscess—treatment: The formation of pus should be hurried by hot applications to the neck. As soon

as fluctuation is made out, prompt incision should be made. The child should be upright, and the abscess freely opened at its lowest point, the child's head being bent forward quickly to allow escape of the pus. The opening may be made by a protected knife, a pair of dressing-forceps, or the finger-nail. After-treatment is usually unnecessary.

ADENOIDS OF THE NASO-PHARYNX.

Definition: This is a hypertrophy of the so-called *third* or *pharyngeal tonsil*, a mass of adenoid tissue located in the naso-pharynx, just below the basilar portion of the occipital bone.

Etiology: There seems to be a predisposition in some children to overgrowth of the lymphatic structures of the body. The diathesis is congenital, but the disease seems to be acquired. Delicate children are most frequently affected. Damp, changeable climates seem to predispose to it. It may follow attacks of the infectious diseases.

Pathology: The growths are a simple hypertrophy of the natural tissue of the third tonsil due to folding in and over of the mucous membrane covering it. They are attached to the bone above and behind.

The **symptoms** of adenoids are mainly the result of the accompanying chronic naso-pharyngeal catarrh, plus the mechanical obstruction of the growths to the breathing. *The catarrh* is evidenced mainly by a persistent discharge from the nose, growing better or worse with changes in the weather, but always persisting. There is great difficulty in blowing the nose and clearing out this discharge. Attacks of *otitis* are frequent and recurrent. Sleeping is interfered with and nervous symptoms are prominent, such as dreams, night-terrors, and somnambulism.

The *obstructive symptoms* are mouth-breathing, snoring during sleep, a nasal voice, and deafness from occlusion of the Eustachian tubes. The persistent mouth-breathing produces a typical shape of the face, with a pinched nose, and deflected septum, a prominent pointed upper jaw with misplaced teeth; a high-peaked hard palate, and a deficient

lower jaw. The whole expression of the face is characteristic and very stupid. In fact, these children are stupid, their mental condition usually being below par. The chest is apt to be deformed or pigeon-breasted.

Adenoids—prognosis: They have a tendency to increase till puberty, when a spontaneous atrophy seems to occur, the enlargement of the naso-pharyngeal space itself giving increased room for them. The deformities produced and the evils done by their presence, however, never disappear, but remain as a handicap for life. Patients with adenoids are prone to diphtheria, and have more severe attacks of it than normal children. The same is true of others of the infectious diseases.

Adenoids—treatment: The only adequate treatment is operative removal of the growths. This is best done under full anæsthesia with the use of a mouth-jag, using the adenoid forceps first, and cleaning out the ragged remnants afterward by a Gottstein's curette. The head can be held so as to prevent blood from running down the trachea. There is no especial after-treatment, and the results of the operation are very brilliant. Recurrence is rare. The syrup of the iodide of iron may be given to build up the health afterward.

ACUTE FOLLICULAR TONSILLITIS.

Definition: This is the form of acute inflammation attacking and confined to the tonsils proper. It is called, wrongly, diphtheritic sore throat.

Acute follicular tonsillitis—etiology: It is undoubtedly an infectious disease due to the presence in the tonsillar crypts of some form of streptococcus or staphylococcus. There is a very marked predisposition in some children to attacks of this disease. A rheumatic diathesis seems a predisposing factor. Those with chronic hypertrophy of the tonsils are often the victims. "Catching cold" is often the exciting cause.

Pathology: The infection begins in the mucous membrane at the bottom of the tonsillar crypts. The crypt is soon filled with a whitish plug of pus, fibrin, and epithelium, which pro-

jects from the surface of the tonsil. The separate crypts being filled the same way, give a white spotted appearance to the whole tonsil. The contents of one or more crypts may coalesce. The whole tonsil is much swelled and inflamed. The disease is bilateral.

Acute follicular tonsillitis—symptoms: The disease begins suddenly with chilly sensations, a rapid rise of temperature to 102° to 104° F., and marked general malaise with headache and backache. Pain in the throat of quite a severe type follows, made worse by swallowing. The severe symptoms last three or four days. The glands at the angle of the jaw are inflamed, swollen, and tender.

On *inspection*, at first the tonsils appear red and swollen, but the characteristic white spots soon make their appearance. In *differentiating* them from diphtheria it is to be observed that these spots can be easily rubbed off with a swab, and when removed leave no bleeding spot behind as is the case in diphtheria. Tonsillitis is also much more sudden in its onset. The exudate in tonsillitis never appears elsewhere than on the tonsils. It may be necessary to make a culture to prove the presence or absence of Klebs-Löffler bacilli.

Prognosis: This is good, as recovery occurs even without treatment.

Acute follicular tonsillitis—treatment: A combination of small doses of phenacetin and sodium salicylate, five grains of each given every four hours, has a very comfortable effect, alleviating the fever and many of the unpleasant constitutional symptoms. *Locally*, a gargle or spray or swabbing with Dobell's or Seiler's solution hurries the cure of the disease.

PERITONSILLAR ABSCESS.

Synonyms: Other names for this condition are phlegmonous, or suppurative tonsillitis and quinsy.

Peritonsillar abscess—etiology: The direct cause is infection of the connective tissue in the neighborhood of the tonsil by pus-producing micro-organisms. Predisposing causes are chronic pharyngitis and hypertrophy of the tonsils. Certain individuals have a marked tendency to recurrent attacks.

Pathology: The peritonsillar connective tissue, and not the tonsil itself, is the part in which the lesion is located. The abscess is unilateral and tends to point through the anterior faucial pillar a little above the tonsil.

Peritonsillar abscess—symptoms: The disease begins abruptly with somewhat the same general symptoms as follicular tonsillitis, but less marked: chilly sensations, fever, headache, and backache. The *local* symptoms are more pronounced, the pain in the throat being very severe and shooting into the ear. Swallowing and moving the jaw for any purpose make the pain intense. After a few days the patient presents quite a characteristic appearance with immovable jaw, slightly opened mouth, thick nasal voice, and mucus and saliva drooling from the lips. The neck on the same side is quite swollen.

On *inspection* the region around one tonsil is badly swollen and œdematous. The uvula is swollen and pushed to the opposite side. It seems impossible for the patient to open the mouth wide. On *palpation* a soft fluctuating swelling is detected just above and in front of the tonsil.

Prognosis: Recovery is the rule. Death may occur from rupture of the abscess into the larynx.

Peritonsillar abscess—treatment: The disease may possibly be aborted in the first stages by heroic dosing with sodium salicylate after the use of a saline purge, and local applications of strong solutions of nitrate of silver. There is, however, always the question of doubt as to the diagnosis in the beginning.

If we decide that the disease is going on to the formation of pus, the process may be hastened by hot applications to the neck and the frequent inhalation of steam by the mouth. These applications likewise give some relief to the pain. As soon as pus shows its presence a free incision should be made with a pointed bistoury in the fluctuating point. This will usually be above the tonsil at about the juncture of the hard and soft palates. If pus is well localized, the relief is immediate. After-treatment consists in washing out the sac and keeping the exit open.

CHRONIC TONSILLITIS.

Hypertrophy of the tonsils is the better name for this condition, as the essential lesion is a marked increase in the size of the glands.

Etiology: This condition is part of the same so-called "lymphatism" as is seen in adenoids, a constitutional tendency to hypertrophy of lymphatic structures. They begin to enlarge very early in life, and are at times even congenital. Frequent acute attacks and climatic conditions probably predispose.

Pathology: There is a hyperplasia of both the connective-tissue stroma and of the lymphoid tissue of the tonsils. Either may predominate, producing a harder or softer variety of hypertrophy.

Chronic tonsillitis—symptoms: Hypertrophied tonsils so frequently coexist with adenoids of the naso-pharynx that the symptoms of the two conditions are blended. They cause mechanical obstruction to nasal breathing, with snoring and nasal voice, and to swallowing. They predispose to attacks of acute tonsillitis. Deafness, mouth-breathing and its concomitants—change in the shape of the maxillary bones—follow. The blood being imperfectly aërated, these children suffer from disturbed sleep and night-terrors. Both the tonsils are affected, and remain in the same condition up to puberty, when they may shrink some, especially if they are of the soft variety. The enlargement of the throat at this times also gives them more room, and the local symptoms after this are less troublesome.

The tonsils appear prominent, and project toward the median line, at times almost touching each other. Their surfaces are full of deep excavations, the natural crypts.

Chronic tonsillitis—treatment: Local applications and drugs are of no value in these cases. The only satisfactory treatment is their *surgical removal*.

This is best done by one of the instruments specially constructed for this purpose, of which MacKenzie's is the least complicated. The tonsils can be removed quickly with very little pain, and the hemorrhage afterward is trifling in chil-

dren. It is, however, always well to have some means for stopping bleeding at hand, as a few rare cases are reported where such means was needed. Styptics or digital compression may be tried. If enough of the tonsil is removed, recurrence is rare.

If operation cannot be done, *local astringents* may be tried, and syrup of the iodide of iron given internally. After the operation a gray appearance is frequently seen on the stump, which may arouse suspicion of diphtheria, but is only the coating of the cut surface.

ACUTE ŒSOPHAGITIS.

Etiology: Inflammation of the mucous membrane of the œsophagus is most often caused by the passage of some hard substance, as metal or bone; or some hot or corroding chemical, as too hot food, or acid, or lye.

Symptoms: Burning pain in the œsophagus, neck, back, and pericardium, with painful swallowing, intense thirst, retching, and vomiting are the regular symptoms. Ulcerations are almost always formed, and in three to six months symptoms of œsophageal stricture, due to cicatrization and cauterization of these ulcers, follow.

Acute œsophagitis—treatment: This depends on the cause. If a *foreign body* is lodged in the gullet, attempts to remove it should be made. If a *corrosive fluid* has been swallowed, the proper antidotes should be given, followed by oils and demulcent drinks. Opium is necessary to relieve the pain. If *stricture is forming*, regular passage of œsophageal sounds should be practised. If stricture has formed, the treatment is surgical.

CONGENITAL FISTULA OF THE NECK.

In embryonic life the openings between the second and third branchial clefts may fail to close, leaving a small or large opening in the neck just above and a little outside of the sterno-clavicular joint. These openings usually communicate with the œsophagus. They are not serious, but

may be troublesome from being occasionally occluded, the secretion being dammed up behind and forming a *cystic tumor*. The discharge from the sinus may be bothersome.

Treatment: This is entirely surgical.

RETRO-ŒSOPHAGEAL ABSCESS.

Definition: This condition, as its name implies, is a collection of pus behind the œsophagus and in front of the bodies of the lower cervical or upper dorsal vertebræ, lower than the retro-pharyngeal abscess.

Etiology: There are three forms of this abscess: that due to simple suppuration of the lymph-glands in the posterior mediastinum; that due to tubercular inflammation and breaking down of these same glands; and that due to caries of the body of one or more of the dorsal vertebræ.

Retro-œsophageal abscess—symptoms: These are vague, and a diagnosis is very difficult to make. The main symptoms seem to be dependent on irritation of one or the other of the pneumogastric nerves, which lie next to the œsophagus. *Sudden attacks of dyspnœa*, or of *inhibition of the heart's action*, are the commonest reported symptoms. Dysphagia seems not to be marked.

Prognosis: This is bad, as diagnosis is next to impossible, and hence *treatment* is out of the question. If the abscess ruptures spontaneously into the œsophagus, recovery may follow.

SWALLOWING FOREIGN BODIES.

Children are frequently brought to a physician with the story of having swallowed a foreign body of some kind, as coins, buttons, jackstones, tacks, pins, pieces of bone, and various other objects. Always investigate the history carefully, to be sure of the fact. As a rule, these bodies pass into the stomach and through the intestines, and are discharged from the rectum without causing any disturbance.

At times they *lodge in the pharynx*, or *in the œsophagus*, and may cause unpleasant symptoms from their presence. These *symptoms* are pain, dysphagia, retching, and cough. Some blood-stained mucus may be brought up.

The pharynx should be carefully inspected and palpated by the finger, and if the body can be found it should be removed.

If it is lodged in the œsophagus, attempts to remove it with a probang or to push it down with a sound may be made. Œsophagotomy may be necessary.

If the foreign body has passed into the stomach, do not give a purge or emetic, but give the child a good meal of potatoes or bread, to form a protective coating for the body during its passage through the intestines.

DISEASES OF THE STOMACH.

ACUTE GASTRIC INDIGESTION.

Definition: In this condition the stomach is unable, through functional causes only, to perform temporarily its digestive duties.

Etiology: The main causes are improper food, the too early use of solid food in infants, sudden weaning, and overloading the stomach. In other words, the use of food of an indigestible character for a child of the age to whom it is given. The stomach may itself be at fault in certain cases through general causes, as fatigue, or general depression, or excessive heat.

Pathology: Two conditions may be present, a decrease in the gastric juice and a lack of muscular peristalsis. Inflammation is absent.

Acute gastric indigestion—symptoms: The food remains in the stomach longer than normal, and excites pain in the epigastrium, nausea, vomiting, and a marked malaise. The appetite is lost, the patient has attacks of faintness, and a good deal of headache. After the stomach is completely emptied the symptoms, as a rule, rapidly disappear. In some cases there is fever up to 102° F., and symptoms of toxæmia, dulness and stupor; or the opposite, restlessness and even convulsions, may develop. The pulse becomes weak, and prostration is pronounced. There is distention of the abdomen, and later usually diarrhœa. The shortness of the attack and

the termination of the symptoms after thorough vomiting, differentiate the condition from gastritis.

Acute gastric indigestion—treatment: If the stomach has not been thoroughly emptied, it should be cleaned out completely, and this is best done by means of the stomach-tube. If unable to use lavage, large quantities of lukewarm water may be given, or an emetic, as ipecac. If vomiting is persistent, cracked ice, or lime-water, or soda-water, given in small quantities, will usually check it. Fractional doses of calomel, grain $\frac{1}{10}$, every hour for ten doses, will tend to quiet the stomach and to remove by the bowels any indigestible matter left behind.

After cleaning the stomach stop completely the food which has been used, and, if possible, stop all food for some hours, giving the stomach absolute rest. If the demand for food is marked, egg-water mixture or whey given in small quantities is the best form of food for a day or two, when the original food may be gradually resumed. During the continuance of the pain hot applications to the epigastrium are very soothing.

ACUTE GASTRITIS.

Definition: Here there is an inflammatory change in the mucous membrane lining the stomach.

Etiology: The causes are the same as of gastric indigestion: indigestible foods and the swallowing of irritants, drugs, and chemicals.

Pathology: The gastric mucous membrane is in a condition of catarrhal inflammation, with congestion and swelling, and exudation of cells into the stroma, accompanied by marked increase of the mucous secretion of the membrane, and desquamation of the epithelium. The changes are fairly well distributed throughout the stomach. The organ is full of undigested food and mucus. There may be slight blood extravasations. In rare cases a false membrane may form on the mucous membrane. In cases due to swallowing of chemicals ulcers may be found scattered irregularly around the organ, but usually on the greater curvature.

Acute gastritis—symptoms: The disease begins, as does

gastric indigestion, with pain in the epigastrium, nausea, vomiting, headache, faintness, loss of appetite, coated tongue, prostration, and fever. But these symptoms, instead of disappearing after the stomach has been emptied, persist, and vomiting of mucus continues even after all the food has been ejected. The temperature continues somewhat raised and thirst is very prominent. The pulse and respiration are likewise quickened. Intestinal symptoms very commonly coexist or follow. The attacks last somewhat less than a week.

Prognosis: Simple acute gastritis in a previously strong child is usually recovered from. In delicate children, or if improperly managed, serious results may follow, or the disease may be the forerunner of a severe gastro-enteritis. We must never forget that the attack may be the beginning of one of the infectious diseases, more particularly *scarlatina*.

Acute gastritis—treatment: The stomach must be completely emptied of any irritants, and this is best done by *lavage*. Warm water or ipecac may be used as emetics if lavage is unadvisable. Afterward give calomel, grain $\frac{1}{10}$ every hour for ten doses, to remove any remaining portion of the irritant. Cracked ice, or bicarbonate of sodium, or bismuth will usually check the vomiting if it becomes persistent. Hot applications to the epigastrium are helpful.

The *dietetic* treatment is very important, with complete stoppage of all food for as long as possible, and when begun again using small amounts of something very bland and easy to digest, as egg-water or whey. Gradual return to regular food should be made after the symptoms have subsided.

If the gastritis is due to swallowing some *chemical*, the proper antidote should be given, and followed by demulcents. Opium may be needed for the pain.

GASTRO-DUODENITIS.

Definition: This is an acute inflammation of the stomach and duodenum, with an extension of the inflammatory process into the common bile-duct and resultant obstructive jaundice. Another name for it is *catarrhal jaundice*.

Etiology: The causes are not well understood, but prob-

ably they are somewhat similar to those of acute gastritis—the use of improper food. There may be an infectious element.

Pathology: There is inflammation, with congestion, swelling, increased secretion, and desquamation of the epithelium from the mucous membrane of the pyloric end of the stomach, of the duodenum, and of the common bile-duct. The swollen mucous membrane of the duct causes its occlusion and subsequent obstruction to the blow of bile.

Gastro-duodenitis—symptoms: The attack begins rather suddenly, with pain in the neighborhood of the duodenum, nausea, vomiting, constipation, fever, rapid pulse, and general malaise.

After a few days the typical symptom of the disease, *jaundice*, makes its appearance first in the conjunctivæ, then in the skin. The urine will contain bile and the fæces be clay colored. The appetite is lost and the tongue thickly coated. After the jaundice has been present a short time the skin becomes itchy, and the pulse may be slow. The patient is languid and good for nothing. The liver may show slight enlargement and some tenderness.

Prognosis: The disease lasts about two weeks, and recovery is the regular outcome.

In the **treatment** of gastro-duodenitis the *diet* should be restricted to milk or scraped meat, the fats and starches being specially excluded. Water should be given freely, and the bowels kept loose by fractional doses of calomel given on alternate days, and followed by some saline laxative, as phosphate of sodium. Hot applications to the epigastrium will relieve the pain. Alkalies or lavage may be needed to stop the vomiting in the early stages.

CHRONIC GASTRITIS.

Definition: This is a chronic inflammatory change in the gastric mucous membrane, and a consequent interference, more or less marked, with the functions of the stomach.

Etiology: The causes are the same as those producing acute gastritis, only being prolonged in their action. The use of improper, badly prepared, or indigestible food; improper

quantities at a feeding; irregularity in feeding; rapid eating or imperfect mastication, if continued long enough, will produce this condition. These causes apply equally to infants as to children on solid food. Bad teeth are a potent cause.

Frequent attacks of acute gastritis predispose to the chronic form. The presence of anæmia, rachitis, tuberculosis, syphilis, or malnutrition is also a predisposing factor. Chronic heart, liver, or kidney diseases, by producing venous engorgement, likewise are predisposing agents.

Pathology: The lesions are in the mucous membrane, consisting of degeneration of the epithelium of the gastric tubules and increased production of mucus. If the process advances further, there is round-cell infiltration with production of new connective tissue, and consequent destruction of the glandular structure. In old cases the stomach becomes dilated and the mucous membrane is covered with a large quantity of sticky mucus.

The **symptoms** of chronic gastritis are those of impaired digestion and failing nutrition. The appetite may be lessened or increased, belching of wind and nausea are regularly present, and vomiting occurs more or less frequently. The vomitus consists not only of undigested food, but also of large quantities of mucus. The younger the child the more marked is the vomiting. There are pain, uneasy sensations, or a feeling of fulness in the region of the stomach. Headache, irritability, and disturbed sleep may follow. The *bowels* are apt to be constipated, but there may be diarrhœa from the passage of undigested food into the intestines. This is most common in infants. The tongue is coated, the breath smells badly, and there is a bad taste in the mouth on awaking.

Signs of failure of nutrition follow, either a lack of gain or a loss in weight. The child grows anæmic and feeble, and loses its energy.

Prognosis: In *infants* the disease is serious by interfering with the normal growth of the child and in predisposing to attacks of intestinal disturbance. In *older children*, if the cause is removed and proper treatment instituted, many recover; but lacking these the disease tends to go on and grow

worse as time advances in the presence of a continuously acting cause. Although these older children seldom die from the disease or its complications, they become the confirmed dyspeptics of adult life.

Chronic gastritis—treatment: The *dietetic care* is most important, and all the causative factors of this class should be investigated and errors corrected. The right food, in proper quantities, at regular intervals, correctly prepared, and thoroughly masticated should be taken. If the teeth are faulty, they should be attended to. A hygienic life should be prescribed, with proper exercise, sleep, and bathing.

Daily *washing of the stomach* with plain boiled water, or with warm water to which some alkali has been added, is the very best local treatment. It removes the mucus and undigested food, and stimulates the production of gastric juice and the muscular tone of the stomach.

The *drugs* that are of value are either sodium bicarbonate, or hydrochloric acid, or nux vomica, or pepsin, alone or in various combinations; but too much reliance must not be placed on any one of them.

DILATATION OF THE STOMACH.

Definition: A more or less enlarged stomach in infants is a fairly frequent condition, especially when fed artificially.

Etiology: The most common cause is the almost universal habit of overfeeding artificially fed infants. The other causes which predispose are rickets and chronic gastritis.

Pathology: The dilatation is usually symmetrical and may become enormous. In rare instances there may be a congenital stenosis of the pylorus underlying the condition.

Dilatation of the stomach—symptoms: The main symptoms are due to the accompanying chronic indigestion. Vomiting attends the cases due to pyloric blockage.

The **diagnosis** is made by physical examination, proving the presence of a dilated stomach after it has been filled with gas or water.

Prognosis: This is good except in cases of pyloric obstruction.

Treatment: Remove the cause by regulating the meals. Improve the tone of the stomach by lavage and the use of nux vomica. If rickets is present, treat this.

ULCER OF THE STOMACH.

Gastric ulcer is found only rarely in children; but a few cases are reported from time to time.

Etiology: Ulcers may be due to follicular gastritis or tuberculosis, or belong to the same category as in adults—of unknown cause.

Pathology: The ulcer may be single or multiple, and usually involves only the mucous membrane. Its position on the stomach-wall is uncertain.

Symptoms: Gastric pain increased by the ingestion of food, nausea, vomiting, and hæmatemesis are the characteristic symptoms. Usually some gastritis is present, adding its symptoms.

Prognosis: This is rather unfavorable.

Ulcer of the stomach—treatment: If diagnosis is made, the child should be kept in bed and the stomach given absolute rest by withholding all food by mouth, the child being nourished by enemata of predigested food. The *drugs* used are silver nitrate, bismuth, and opium, but none is of much value.

DISEASES OF THE INTESTINES.

ACUTE IRRITATIVE DIARRHŒA.

Synonyms: Other names for this condition are simple diarrhœa, mechanical diarrhœa, and nervous diarrhœa. It is meant to include cases without anatomical changes in the intestines, and without involvement of, or influence from, the stomach. The absence of a bacterial cause is shown by reference to the etiology.

Etiology: The exciting causes are various, but the same underlying predispositions exist as in all diarrhœas. These are age, the first two years of life, unhygienic surroundings, malnutrition from any cause, and hot weather.

The *active causes* are *excessive feeding;* the use of *foods unsuitable* to the age of the child, and which consequently act virtually as foreign bodies, such as green corn, cabbage, radishes, partially cooked starches, fruits, and such stuff; the *swallowing of foreign bodies; ordinary drugs* in susceptible infants *used as laxatives; reflex nervous influences,* as exhaustion, chilling the surface, excessive heat, fright, and rarely dentition; eliminative *efforts* of nature to *excrete toxic substances* from the body by the intestines, of which *uræmic diarrhœa* is the best example.

Acute irritative diarrhœa—pathology: There is neither intestinal fermentation nor intestinal inflammation. Increased peristalsis seems at the bottom of this form of diarrhœa, caused by local direct irritation or reflexly. With it some hyperæmia of the intestinal mucous membrane and an increased secretion from its glands are present. If allowed to progress, an intestinal inflammation may supersede, or bacterial invasion and fermentation of the intestinal contents may follow.

Symptoms: These usually begin suddenly with abdominal pain and diarrhœa. The first stools are soft fæces; later they become thin and watery, averaging six to ten a day. The child is restless, somewhat weak and exhausted, and has a clammy perspiration. There is no vomiting and no fever. The stools are yellow or brown. Their odor is not bad. If due to irritating foreign bodies, these will appear in the stools. The abdomen is swollen and the increased peristaltic movements of the intestines are evident to the sight and touch.

Prognosis: These cases regularly recover in a few days, nature removing the irritant from the bowels. Under unfavorable conditions the attack may lead to one of the more serious forms of diarrhœa.

Acute irritative diarrhœa—treatment: Follow nature's lead and first give a cathartic, of which castor oil is the best. Calomel in divided doses may be used, but is slower. This removes the irritant completely from the bowel. Four to six hours later give the proper dose of opium and repeat as occasion requires. This had best be given uncombined, and five to ten drops of paregoric to a child a year old are the right

sized dose. Food should be withheld as far as possible for a day, and when begun should be bland and given in small quantities.

ACUTE FERMENTAL DIARRHŒA.

Synonyms: This is the common diarrhœa of summer that is the cause of the high infantile mortality at that time. The names given it are almost as many as the authors writing of it. They are summer diarrhœa, acute dyspeptic diarrhœa, gastro-intestinal catarrh, infectious diarrhœa, gastro-enteric infection, and, very erroneously, cholera infantum.

The name chosen expresses the condition as well as any—*i. e.*, that of fermentation or decomposition of the intestinal contents by bacterial invasion.

Etiology: The causes are summer heat, artificial feeding, bad habits of feeding, overfeeding, improper food, impure milk, bad hygienic surroundings, and residence in the city. The excessive heat of the summer combined with high humidity in the atmosphere seems the main predisposing factor. During long terms of hot weather this form of diarrhœa seems to sweep in epidemics through the infants in large cities.

Behind all these causes, but acting as the direct excitants of the disease, are undoubtedly various forms of *germ life*, which are introduced with the food into the child's digestive tract, and for which the milk or other food taken acts as a culture-medium. Attempts are being made to isolate and separate the forms of micro-organisms responsible for these conditions, but as yet no very definite results have been reached. The probabilities are that there are many different varieties of germ life in each case, each contributing its share to the disturbance, and that no one form of germ is alone the cause of the disease.

In a child previously unhealthy from any cause these factors, *germs*, *bad feeding*, and *heat*, are far more likely to produce this disease than in a perfectly well infant. Further, the great majority of these cases are seen in artificially fed children, breast-fed babies being rarely attacked, thus showing the necessity for the action of all three factors at once.

Pathology: Essentially this disease is non-inflammatory, but is a putrefaction of the food-contents of the intestine due to the presence of bacteria of one kind or another. In a healthy child and with a short attack of the disease, virtually no anatomical changes take place. If the child is non-resistent and the attack severe and lasting, early changes in the intestines are desquamation of the epithelium of the mucous membrane, going on if further continued to the changes seen in the inflammatory diarrhœas. The gross appearances in the intestinal mucous membrane are almost none.

Acute fermental diarrhœa—symptoms: The disease may begin gradually with slight looseness of the bowels, associated with symptoms of general malaise, some fever, restlessness, and fretfulness; or more acutely with high fever, frequent vomiting, and marked diarrhœa.

In the *gradual form* the diarrhœa is the main symptom. The stools are fairly frequent, eight to ten a day, yellow or more frequently green and thin, and contain masses of undigested food, curdled proteids, and fat. They may contain mucus and at times a little blood. At first the odor is sour only, but later becomes offensive. Anorexia is usually present. The tongue is coated white. Thrush frequently develops in the mouth. The child soon becomes pale, the muscles grow soft and flabby, and he loses flesh from week to week.

The disease terminates under favorable circumstances by a gradual change to the normal in the stools, and by a gain in strength and flesh. Under unfavorable conditions the cases go on to a chronic intestinal fermentation which remains till the cold weather comes; or it may develop suddenly, under the influence of very hot weather, into a case of genuine cholera infantum; or it may be the starting-point of an entero-colitis.

When *beginning acutely* the symptoms usually continue acute. The fever keeps at about 102° F.; there are marked restlessness, irritability, and often convulsions; or, on the contrary, stupor and great prostration. The vomiting remains frequent, at first curdled milk being ejected, and later mucus, serum, and bile may follow. Any food or drink is

immediately returned. The appetite is gone, but thirst is marked and fluid food is eagerly taken to quench it.

The *bowels* move frequently, in the beginning the stools being fæcal, and later being thin, yellowish or greenish, with a great deal of gas and an offensive odor. There may be a dozen or more stools per day. The diarrhœa is characterized especially by the large amount of gas expelled and the very putrid odor of the stools. The abdomen is distended and tender, and the infant evidently suffers from intense colicky pains. After three or four days of these symptoms the temperature falls, the stools become less frequent, the vomiting stops, and under proper management the child will go on to complete recovery.

Under less favorable circumstances—that is, a feeble child and continued bad feeding—an inflammatory entero-colitis supervenes. Others terminate in death during the acuteness of the attack.

Diagnosis: This form of diarrhœa must be differentiated from cholera infantum, entero-colitis, and the beginning of several of the acute diseases, such as tonsillitis, scarlet fever, pneumonia, and malaria. The diagnosis may be difficult at first, but a few days of careful observation will usually clear up the case.

Acute fermental diarrhœa—prognosis: In a previously healthy child, and with proper management, which means feeding, these cases usually recover. In institutions; in children suffering from marasmus, rickets, and other nutritional disorders; among unhygienic surroundings; with previous chronic indigestion from wrong feeding; and in very hot weather, the mortality from this form of diarrhœa is high.

Prophylaxis: This is of great importance, as the majority of these cases of acute fermental diarrhœa may be prevented by proper attention to a few essentials. During the hot summer such babies, as can, should be sent from the cities to the country, and those in a hot country to a cooler climate. If this is impossible, much may be done by keeping the child in the air and out of doors as much as possible both day and night. Frequent cool bathing both promotes cleanliness and assists in keeping the temperature of the surface lower. The

diapers must be kept scrupulously clean, especially where infants are congregated. In any case of diarrhœa the diapers particularly should be disinfected.

Feeding should be carefully regulated. Encourage breast-feeding in every way possible during the hot months, and postpone weaning till cool weather begins. If artificial feeding is necessary, this should be thoroughly regulated as to quality, quantity, and intervals. During hot weather special attention must be paid to the purity of the milk, and unless this can be assured some form of sterilization should be adopted. Foods unsuited to a child's digestion should be absolutely interdicted. Excessive feeding particularly should be avoided. Little and seemingly unimportant gastric or intestinal derangements should be promptly corrected, as these are often the beginnings of more serious disease.

Acute fermental diarrhœa—treatment: First and most important is attention to the food. In the rare cases occurring in *breast-fed* infants all food should be withheld for a short time, particularly until the tendency to vomiting is passed. Small quantities of water may be given instead to quench the thirst. As the breast is resumed, the quantity allowed at a nursing should be small and the return to full feeding gradual.

In *artificially fed* children, among whom the vast majority of these cases occur, all milk food should be temporarily prohibited. As food during this abstinence from milk albumin-water is usually the best. Broths, rice- or barley-water, or one of the infant foods free from starch, made with water only, may be tried. The reason for the prohibition of milk during the acuteness of the attack is that this forms the best culture-medium for the germs whose action is causing the disease. By depriving them of their food we starve them out.

After the attack is ended we must return to a milk food very slowly, at first using a very dilute form, and gradually increasing the proportions of the solid ingredients to the proper limit for the child. Small quantities at a feeding should also be adhered to. In some of these cases, especially the very young, a wet-nurse must be secured for a time.

Milk given after such an attack is over should always be carefully sterilized. In a few cases *peptonized milk* is useful for a time until the stomach regains its tone.

In *older children* on solid food this must be stopped, and only easily digested fluids used until the attack is over, and then the return to the regular diet should be gradual.

Medicinally our first purpose is to clean out thoroughly from both stomach and intestine all the fermenting food-products left behind. The stomach ordinarily will be cleaned of itself; but if vomiting is persistent, it may require our special attention. In these cases nothing is so efficacious as lavage. For an infant a No. 16, American scale, soft-rubber catheter, attached by rubber tubing to a funnel, is the best size. It is easily passed, the passage being facilitated by wrapping the child, arms and all, in a blanket, and the act of washing is quickly accomplished. Warm, boiled water is the best medium, although a little bicarbonate of sodium may be added. One washing is usually sufficient.

The *intestines* should next be thoroughly emptied by a teaspoonful or more of castor oil. This is undoubtedly the most efficient of the drugs of its class; but if the stomach is irritable, it may be omitted. If the stomach-tube has been used, before removal the oil may be given through this. Fractional doses of calomel (a tenth of a grain every half hour till ten or twelve doses are taken) have the advantage of ease of administration, do not irritate the stomach, and have some slight antifermentative action in the intestines. It is not so thorough and not so quick as castor oil, however. After the cathartic has acted, a thorough irrigation of the colon with warm saline solution through a long rectal tube cleans out the last remnants of decomposition. This may be repeated every day with advantage in most cases.

After the whole alimentary canal has been thus thoroughly emptied the use of some *antiseptic* drug given regularly is in order. These drugs are many, but the best of them all is the old subnitrate of bismuth, as it may be given in large doses without fear of poisonous effects. It is best given in doses of ten to twenty grains every three or four hours. Other drugs of this class are salol, salicylate of sodium, salicylate of

bismuth, subgallate of bismuth, calomel, bichloride of mercury, and creosote.

In the more subacute and prolonged cases the use of one of the mineral acids is at times of value.

The use of *opium* in these cases is indicated by marked pain and evident peristalsis. It should always be given by itself, and never combined with other drugs. Never use it until after the alimentary canal is thoroughly cleaned out. Paregoric may be used, five to ten drops; or Dover's powder, one-fourth to one-half a grain. These doses may be repeated in two to four hours as needed.

The *vegetable astringents* are often used, but are not very reliable, although the tannin in them does combine with the toxins to form insoluble compounds.

Stimulants may be necessary in the cases with marked prostration. Brandy, or whiskey, or champagne may be used. Blackberry brandy answers the double purpose of a mild astringent and stimulant. Hot baths, mustard applications, etc., may be used in great weakness.

In prolonged or convalescing cases there seems profit at times in the use of some of the *digestive ferments.*

The *hygienic care* of these children is likewise important. The child should be given fresh air in abundance; should be kept as cool as possible; should be frequently bathed in cool water; its clothing should be thin, and special attention should be paid to disinfection of the diapers. If possible, these children should be sent away from the hot cities to the country, especially when the disease shows any tendency to become protracted.

CHOLERA INFANTUM.

Definition: This disease is undoubtedly a specific infection of the milk used as food by the infant affected. It is also called choleriform diarrhœa, and the name has been used wrongly as a generic term for all summer diarrhœas of infancy. This name should be restricted, however, to the less common class of cases differing essentially from the form just described, and also from the inflammatory varieties.

Cholera infantum—etiology: This disease practically never occurs in an entirely breast-fed baby. It never occurs except in hot weather. Although careful researches have been made to find a specific micro-organism in cholera infantum, as yet no such germ has been isolated. Various forms of bacteria, however, have been found; but as yet it has not been proved to be caused by one special variety. The cause is invariably in the *milk*, and there may be enough of the toxic elements present in the milk as taken to produce the symptoms immediately on absorption; or they may be manufactured from the milk by the bacteria in the digestive tract. Each case is one of poisoning by toxins generated in the milk by growth of bacteria in it.

The disease is frequently grafted on a case of irritative or fermental diarrhœa, or occurs in a convalescent from some form of inflammatory diarrhœa. It may attack a previously healthy child, but this is far less common than the above.

Cholera infantum—pathology: This again is not a diarrhœa with anatomical changes in the intestinal mucous membrane, but is purely a poisoning of the system by the swallowing, or manufacture in the digestive tract, of chemical toxins. In fact, the symptoms are due far more to absorption of poisonous toxins into the blood than to the presence of the germs in the stomach and intestines.

Cholera infantum—symptoms: In a previously healthy child, or in one already showing some mild intestinal disorder, there is a quite sudden attack of violent vomiting and purging. These two symptoms are the most characteristic of the disease, and may continue uninterruptedly throughout. The vomiting is frequent, and follows every attempt to introduce food or drink into the stomach. At first curdled milk is ejected, and later mucus and serum and bile. The stools are frequent, fifteen to twenty a day, at first fæcal, of yellow, brown, or green color, and later losing all color, and consisting simply of large quantities of serous fluid. These are the typical stools of the disease. They are acid in the beginning, but when they become serous are alkaline. They are usually without typical odor, but in some cases may have the putrid smell of those in fermental diarrhœa. Under the microscope

they show epithelial and round cells and large numbers of bacteria.

The child loses flesh and color very rapidly ; the eyes sink in their sockets, a marked pallor develops in the skin, and the flesh seems to disappear almost under our very eyes. The skin is cool and clammy, but the temperature from the first is high, 102° to 104° F., and often reaches 107° F. It is somewhat in proportion to the severity of the attack. High temperature, as the disease progresses, points to a fatal termination. The pulse is weak and rapid ; the respirations shallow and fast. The tongue is coated early, but soon becomes dry and red. The abdomen, instead of being distended, is sunken. Thirst is intense, the child eagerly taking any fluid given it. The urine is almost suppressed, only very small quantities being secreted.

The *nervous* symptoms are marked, the child crying or moaning, and throwing itself about in a very restless way. Delirium and convulsions may follow. Certain cases develop the opposite condition of stupor and later coma. They may pass into a condition like the algid stage of Asiatic cholera, with pinched features, subnormal temperature, collapse, depressed fontanelle, irregular respiration, and very feeble pulse.

In some cases the gastro-intestinal symptoms subside, but the nervous symptoms become especially prominent, so much so as to suggest meningeal complications. Any actual changes in the brain or its membranes are, however, very rare.

Cases of cholera infantum either die or show marked changes for the better in two or three days. In those going on to recovery the vomiting usually stops first, then the stools become less frequent and lose their serous character, the nervous symptoms subside, the temperature falls, and the pulse and respiration regain their power. Convalescence is likely to be quite slow.

Diagnosis: If the picture of the disease is kept well in mind, it will scarcely be confounded with anything else. The frequent vomiting, large serous stools, high temperature, marked prostration and collapse, great thirst, dry mouth and tongue, combined with the nervous symptoms of great rest-

lessness or stupor and coma, and the rapid, feeble pulse, sudden loss of weight, with pinched face and sunken fontanelle, are characteristic of this disease only.

In times of an *epidemic* of Asiatic cholera there might be some difficulty in differentiating these two conditions.

Prognosis: This is distinctly bad. If the cases of real cholera infantum only are considered, the mortality is probably 60 to 70 per cent. The younger and feebler the child the less are its chances of recovery. The severity of the infection is, however, of most importance in prognosis.

Cholera infantum—treatment: Compare these cases with those of poisoning by some intense chemical irritant for purposes of treatment. Prompt and energetic action should be taken. First, not a particle of food is to be given for twenty-four hours at least. Immediately and thoroughly *wash out both stomach* and *bowels* with large quantities of boiled water, or normal saline solution. This will not exhaust the patient nearly so much as the constant vomiting and purging, and assists nature in her efforts to remove the poisons from the system. After washing, tannin may be thrown into the stomach and intestines to make insoluble compounds with any of the toxins that may be left behind. If the vomiting and purging recur, repeat the washings.

There is very little value in any medication given by mouth as it is either ejected or not absorbed. *Stimulants* will be needed, and may be given in the form of whiskey diluted with cold water by mouth, in small quantities frequently repeated; or hypodermatically. To stimulate the heart, quiet the nervous manifestations, and inhibit the enormous excretion of serum from the intestinal bloodvessels, *morphine* grain $\frac{1}{100}$, and *atropine* grain $\frac{1}{800}$, given hypodermatically, and repeated hourly to watch their effects, seem the very best combination yet suggested. This is contraindicated only in the cases with stupor.

To allay the great thirst and supply fluid to the tissues normal salt solution is to be injected slowly and in large quantities into the subcutaneous tissues. Giving large amounts of water by mouth only increases the irritability of the stomach.

To combat the high temperature *baths* gradually cooled should be used.

If the symptoms begin to abate and recovery seems probable, great care should be exercised as regards the return to food, and the strictest surveillance of the diet should be kept up for some weeks. Recurrences are fairly common after very slight dietetic errors. The same general rules should be followed as after recovery from fermental diarrhœa. In the cases of intense collapse with subnormal temperature applications of heat are decidedly indicated.

CHRONIC INTESTINAL INDIGESTION.

Synonyms: This common form of chronic functional disturbance of the intestines is also called chronic diarrhœa and chronic intestinal catarrh.

Etiology: It is especially seen in institution-children and in those massed together for any cause. It is also an accompaniment of general constitutional diseases, as rachitis, syphilis, and chronic pulmonary diseases. It is often seen in children who have been reduced by attacks of one of the acute infectious diseases.

Unhygienic surroundings of any kind also predispose. It may occur at any season of the year, but is more serious during hot weather. It attacks both breast-fed and artificially fed children, the latter, however, more commonly. In breast-fed infants the mother's milk is at fault in being indigestible for her particular child. Such mothers are usually neurotic or anæmic, or run-down, or pregnant, or some of the constituents of her milk are present in abnormal quantities. Lactation prolonged far beyond the normal time may produce this condition.

In children on the bottle too frequent feeding, or overfeeding, or too concentrated food is usually the cause. A high proteid percentage seems the commonest factor. The prepared foods containing starch may cause this condition.

In children on general diet overuse of carbohydrates seems to be the commonest cause. It occurs often in children whose feeding in early life has been faulty. Children allowed to

eat anything they wish—sweets, pastry, and fancy foods—are commonly affected.

Pathology: There are really no lesions in chronic intestinal indigestion, as the condition is one of chronic indigestion, or lack of performance of function by the intestinal juices, and consequent fermentative changes in the undigested food-products. After the disease has existed for some time the constant irritation in the bowels will produce a mild form of chronic catarrhal inflammation, evidenced mainly by a hyperplasia of the solitary and agminated follicles of the small and large gut, and by an increased production of mucus.

Chronic intestinal indigestion—symptoms: A mild form of *diarrhœa* characterizes this condition. The stools seldom exceed six or eight in the twenty-four hours, and are greenish, or yellowish, or gray, and after the diarrhœa has existed some time contain mucus and at times streaks of blood. They contain undigested food, lumps of coagulated casein, and unchanged fat. They are very dry, or semisolid in consistency, and the odor is very offensive, an evidence of albuminous decomposition.

The child is irritable, nervous, and sleeps badly. The abdomen is markedly distended and tympanitic, and the veins on the abdominal wall are marked out in their course. The stomach is not regularly involved, and consequently vomiting is the exception. The tongue is red and dry, and thrush and stomatitis are frequent complications. The skin of the buttocks is usually erythematous and excoriated. The temperature may rise slightly and irregularly, but may be found subnormal. The pulse grows rapid and feeble, and the respiration shallow. The appetite, instead of being lost, is regularly increased, the child taking its food with seeming pleasure. The patient loses flesh slowly but steadily, and, if the disease is prolonged, may waste to a mere skeleton. As this condition of emaciation develops the patients lie in a semistupor, sucking their fingers and otherwise indifferent to their surroundings.

The *duration* of the cases is very indefinite, exacerbations and remissions being common.

In *children on general diet* the skin is pale and sallow, the

muscles are flabby, the whole body is thin, but the abdomen is protuberant and distended. These children are emotional, cross, and hard to control. Their sleep is restless and disturbed, and during sleep they frequently grind their teeth. The bowels may be constipated, with light-gray lumpy stools of a foul odor, and an excessive quantity of gas; or diarrhœa may exist with four or five stools a day containing undigested food and with an offensive smell. They may at times contain mucus. Colicky pains are frequent. The appetite is variable, with a craving for indigestible articles of food. The tongue is thickly coated white and the breath is bad.

There are many *nervous* symptoms, in addition to the emotional changes, as tetany, fainting-attacks, headache, dulness, stupor, and at times convulsions. There is often slight irregular fever.

Diagnosis: The history, with examination of the child and inspection of the stools, usually quickly establishes the diagnosis. Special attention should be given to the other organs to prove the presence or absence of disease of any of them. Marasmus and tuberculosis especially must be differentiated.

Prognosis: Without intelligent care cases of chronic intestinal indigestion continue to grow worse and die from exhaustion or from some intercurrent acute diarrhœa, or pulmonary disease. If the disease is in a child with some constitutional disorder, or in an institution, the prognosis is bad. If the child is strong, and if intelligent treatment can be carried out by removing the cause, recovery should take place. The younger the child the fewer the chances for recovery. The disease is more difficult to cure in the summer time than during cold weather.

In children on general diet a fatal ending is not common, but a permanent relief of the symptoms is difficult to accomplish; and these cases grow to adult life with digestive systems that are always troublesome.

Chronic intestinal indigestion—treatment: The *preventive* treatment is most important, and consists in strict attention to the feeding on hygienic principles of all infants and young children.

If the disease has started, the *dietetic* and *hygienic* manage-

ment is far more important than any drugs. Seek hard for the cause in the *food* the child is taking. Have a chemical analysis of the milk, breast or cows', made to find what constituent is at fault. Inspect the stools to find what forms of food are most undigested. Regulate the quantity of food and the intervals of feeding, as well as the quality. If any constitutional ailments are present, treat them. Have the child properly clothed for the season of the year. If it is summer, insist on change of climate if possible. At any rate, have plenty of fresh air with sanitary surroundings.

It is often helpful to put an artificially fed child on some non-milk food for some time, and whatever food is given should be well diluted. Egg-water, whey, broths, or one of the non-starchy proprietary foods may be tried. The predigested foods, peptonized milk, or peptonoids, find a useful field in these cases. It is better to underfeed than to overfeed these children.

As regards *drugs*, none is very satisfactory. Opium and astringents are useless. The only cases where opium is indicated are those in which the bowels move immediately on the introduction of food into the mouth. Here it counteracts the reflexly increased peristalsis. The intestinal antiseptics may be helpful: bismuth subnitrate in large doses, or salol, or salicylate of sodium. Dilute hydrochloric acid and pepsin given with each feeding are theoretically indicated. Calomel in divided doses, or castor oil, from time to time, are good adjuvants. An occasional thorough washing of the colon is advisable. As improvement begins tonics, as iron and arsenic, are indicated.

In children *on general diet* results of treatment, if carefully carried out, are brilliant. The regulation of the diet is here also of primary importance. A diet of beef-juice, or scraped beef, or partially peptonized milk, with avoidance of carbohydrate food and absolute prohibition of all indigestible and fancy foods, will accomplish wonders in these children. The proprietary foods here have a useful field. The meals should be given at regular intervals. As improvement occurs, a gradual return to the diet proper for a child of its age should be substituted.

Calomel given from time to time aids our treatment, especially in the constipated cases, and colon-irrigation may be helpful, particularly in the cases with mucus. Salol or sodium salicylate may aid in lessening flatulence. Tincture of nux vomica is a useful tonic. Regular exercise and fresh air, and a general sanitary life, must be included in our management. Relapses will follow slight indiscretions.

ACUTE ENTERO-COLITIS.

Definition and synonyms: So far, all the forms of diarrhœa described have been essentially *without anatomical lesions*, but depend rather on *changes* taking place in the *food* than in the intestinal walls. This form, on the contrary, is really an inflammation of the intestinal mucous membrane. Other names by which it is known are ileo-colitis, enteritis, dysentery, and inflammatory diarrhœa.

Acute entero-colitis—etiology: The causes are virtually the same as those of fermental diarrhœa: bad food, or bad habits of feeding, being of the greatest importance. Hot weather predisposes markedly to the disease, although in the fall, with exposure to cold, many cases develop. It is frequently the result of one of the forms of functional diarrhœa which has been improperly cared for. It may complicate the infectious diseases.

Bacterial life of some kind undoubtedly plays a prominent part in its etiology. It is far most frequent in artificially fed children, and the tendency to it exists even after general diet is allowed.

Pathology: The lesions of acute entero-colitis are found involving, as a rule, both the ileum and the colon, spreading in both directions from the ileo-cæcal valve. In a few cases the ileum only, in a larger number the colon only, is involved.

The *mildest cases* show only a catarrhal inflammation of the mucous membrane, with swelling, congestion, and increased production of mucus. The veins are engorged, and large areas of the mucous membrane appear of a deep-red color. In places small hemorrhagic spots may be seen.

In the *protracted cases* the entire intestinal wall is thickened, and the solitary follicles and Peyer's patches are swollen.

If this catarrhal inflammation is very severe and long continued, *small ulcers* appear in the mucous membrane, due to desquamation of the epithelium. These are scattered irregularly through the colon. Several of these ulcers may coalesce, forming large irregular bare areas. By this process large amounts of the mucous membrane may be destroyed, and the gut present a worm-eaten appearance.

Other cases present the lesions of inflammation and hyperplasia, with subsequent breaking down of the solitary *lymph-follicles.* These changes are seen in both the ileum and the colon. Peyer's patches may also, but rarely, be involved. Seen with the naked eye the mucous membrane is studded with little rounded elevations, the enlarged follicles, and in the early stages the top of each presents a small pit. In *more advanced* cases the excavation is larger and the elevation smaller, the follicles having been entirely destroyed. The mucous membrane then presents a uniformly pitted appearance. The ulcers do not become large like those in catarrhal ulceration, but at times two or more small ulcers may run together and form an irregular figure. The mucous membrane is never so completely destroyed as in the former ulcerative condition.

Another and the most severe form of entero-colitis shows the lesions of *croupous inflammation.* In this there are patches of false membrane adhering to the surface of the intestine. To the naked eye the intestinal wall is thick and stiffer than normal, and has a greenish appearance on its inner surface. It is difficult to strip off any large-sized pieces of the membrane from its base. The portions of gut uncovered by pseudo-membrane are red and congested, with here and there hemorrhagic spots. The lesions are most marked in the *colon,* but some patches may be found above the ileo-cæcal valve. Under the microscope a distinct layer of fibrinous exudate is seen growing on the intestinal mucous membrane. There is a large round-cell infiltration of the mucosa and submucosa. Necrosis and ulceration are very rare.

There are many *complicating lesions,* as bronchitis, bronchopneumonia, atelectasis, and acute degenerative nephritis. The

mesenteric lymph-nodes are enlarged and inflamed in almost every case.

Acute entero-colitis—symptoms: Some attempt may be made to connect certain symptoms with one or other of the groups of pathological lesions just described; but the cases vary excessively in their clinical picture, and such attempts are often proved ineffectual at the autopsy.

The cases, except in the follicular form, which is more of a subacute process, begin suddenly with *vomiting*, *diarrhœa*, *abdominal pain*, and *fever*.

The *stools* at first are fæcal, but soon become mixed with blood and mucus in considerable quantities. They are very frequent, each one small in amount, and are preceded by pain and followed by rectal tenesmus. There is very little disagreeable odor to these stools, the odor occurring in the late stools of prolonged cases. In the *follicular* variety the stools are less frequent and the presence of blood less common. In the *membranous* variety mucus and blood are present in large quantities, and shreds of pseudo-membrane add a diagnostic feature to the case. After a few days, in all the cases, the stools gradually assume a dark-brown or greenish-brown color. Prolapsus ani frequently complicates the protracted cases.

The *temperature* at first averages 103° or 104° F.; but as the disease progresses it falls some, but continues above normal so long as the inflammatory process is present. In the beginning the patients are less prostrated than in the fermentative diarrhœas; but as the case progresses the prostration increases.

There is anorexia, but increased thirst. The abdomen is distended, and usually tender along the line of the colon.

The child gradually *loses flesh* and *strength*, the pulse becomes rapid and feeble, and the respirations irregular and shallow. The skin of the buttocks becomes excoriated, and bedsores may form. The tongue is coated, or red and glazed, and the mouth is frequently the seat of some variety of stomatitis.

Nervous symptoms in the early stages are of an active na-

ture: restlessness, irritability, twitchings, and convulsions. If the case lasts, stupor and coma often develop.

The *more acute cases* die in a few days to a couple of weeks. The subacute cases may last three or four weeks, gradually losing ground till death.

In cases of *recovery* the early symptoms of improvement are seen in the stools: the mucus and blood gradually disappear, the movements are less frequent, and the constitutional symptoms subside by degrees. Convalescence in all varieties is very slow and relapses are common.

Diagnosis: The two diseases with which acute entero-colitis may be confounded are *typhoid fever* and *intussusception*. The former is rare in children, but must be remembered during an epidemic. The latter should always be thought of, as the symptoms of the two are similar: abdominal pain, tenesmus, bloody discharges, and vomiting. The fever of entero-colitis is not present in intussusception, and the subsequent constipation and presence of an abdominal tumor in the latter condition are differential points.

The effort to separate the different pathological varieties of entero-colitis depends on the onset and severity of the symptoms and the characters of the stools. Many cases cannot be classified even with all the helps.

Prognosis: The prognosis of acute entero-colitis is always grave, but is worse in feeble or anæmic children, and in those already suffering from any form of constitutional or nutritional disorder, as rickets, syphilis, tuberculosis, or marasmus. The younger the child the more are the chances of an unfavorable end. Protracted cases and those occurring in hot weather have a bad prognosis. In a previously healthy child and under proper surroundiugs, with an intelligent carrying out of the physician's directions, many cases will recover. Never forget the possibility of a relapse occurring even when the child is seemingly improving rapidly.

Acute entero-colitis—treatment: In the way of *prevention*, special attention should be paid to the careful feeding of all children during their years of liability to the disease. All the hygienic and sanitary surroundings of the children should be regulated to the best of our ability in each particular case.

Prompt treatment of all the forms of functional diarrhœas will prevent the development of many cases of these graver varieties.

If a case has developed, a change of air to a cooler climate will often work marked and rapid improvement. The dietetic regulations are most important and most difficult. In breast-fed babies, if no gross changes are evident in the milk, this form of feeding should not be interfered with. If a child has been recently weaned, a wet-nurse may be needed. In bottle-fed babies the milk should be made quite dilute or be peptonized, or stopped altogether, and meat-juice, broths, scraped beef, peptonoids, or egg-, rice-, or barley-water used instead. The point is to give food that leaves very little indigestible residue. These children have little appetite, and enough food must be given to keep up their nutrition. All food should be given at regular intervals, and not too often. As improvement occurs special care should be given to the diet to prevent relapses.

In the beginning *lavage*, performed once, may be useful; it will seldom require repetition. *Irrigation of the colon* is particularly valuable in this condition. The lesions are mostly colonic, and we can make our medication through local applications direct. A normal salt solution given warm with a high rectal tube, and in considerable quantities, injected once or twice a day is the safest solution. Tannin or starch may be added. It is unsafe to use the stronger antiseptics for fear of leaving some behind. If tenesmus is marked, starch-water with the addition of five or ten drops of laudanum is very soothing. Cocaine suppositories, each containing one-fourth to one-half grain of the drug, may be needed.

At the first, a good-sized dose of *castor oil*, one or two drachms, should be given if the stomach will retain it. Later, repeated doses of the same in small quantities, ten to fifteen minims, will often be found beneficial. Opium in one form or another will usually be needed after the purge has acted. It is always best given separately, in small doses, repeated as need requires to quiet the pain and tenesmus. Bismuth in large doses regularly repeated, fifteen to twenty grains every three or four hours, soothes and rests the inflamed mucous

membrane. Stimulants are almost always necessary to rouse the feeble circulation and combat the great prostration. Blackberry brandy, whiskey, or good old brandy may be used. Pepsin and the mineral acids are frequently given by mouth for assistance in more completely digesting the food.

As *convalescence* is established, the mineral acids, nux vomica, arsenic, and iron are helpful tonics, local and general. After complete recovery in the digestive tract cod-liver oil is useful.

CHRONIC ENTERO-COLITIS.

Definition: These are prolonged uncured cases of the acute form of entero-colitis, which have lost their active character. The change from the acute to the chronic condition is slow and gradual, and the point of change is hard to set.

Etiology: Bad management of acute cases of entero-colitis is usually the cause of the assumption of a chronic character. It is seen in the more hardy infants, who have managed to escape death during the summer months.

Pathology: The main lesions found are a chronic catarrhal inflammation of the mucous membrane, with growth of new connective tissue, and destruction of the tubular glands of the intestine; or a chronic hyperplasia of the lymph-follicles with some small ulcerations over their summits and marked pigmentation in places. Ulcerative conditions are rare, as most of the patients with ulceration die during the acute stage. Chronic pulmonary complications are common—hypostatic congestion, broncho-pneumonia, or tuberculosis.

Chronic entero-colitis—symptoms: There are no fever and no signs of active inflammation. Pain and tenderness have likewise disappeared. Food is taken readily as given; but evidences of a desire for it are not common. The main symptoms are progressive emaciation and abnormal bowel-action. The child wastes from week to week, until there seems nothing to its body but the skeleton covered with loose skin. The face is thin and sharp, the eyes sunken, and the cheeks hollow. The fontanelle is much depressed. All the subcutaneous fat has disappeared, so that the skin hangs in loose folds. The abdomen is distended and tympanitic.

The lips, tongue, and mouth are usually dry, and may be covered with sordes. Various forms of stomatitis may be present. The teeth may decay rapidly, but dentition may proceed normally. Vomiting is rare.

The *stools* average four to six per day; they are thin, and contain mucus and biliary coloring-matter, being green or brown. They contain undigested food unless the diet is carefully regulated. Blood is seldom present. They have a very offensive putrid odor. Prolapsus ani is rarer than in the acute variety of the disease. Colic and flatulence are regular accompaniments. The skin around the buttocks is erythematous and excoriated.

The pulse is rapid and feeble, the circulation sluggish, and the extremities cold. These children are restless and irritable, sleep poorly, and whine a great deal. At other times they are dull and stuporous. Convulsions may occur.

The *duration* of the cases is a few months.

Diagnosis: The main point to be determined is whether the symptoms are due only to the chronic entero-colitis, or whether there is some complicating disease. Rickets, syphilis, tuberculosis, and marasmus must each be carefully examined for and excluded.

Prognosis: This is very bad in young infants, in those previously debilitated, in those in institutions; and during hot weather. Under favorable circumstances, and when intelligent treatment can be followed, results are fair. Some of the most hopeless appearing cases recover.

Chronic entero-colitis—treatment: The main reliance is on good hygiene, carefully regulated diet, and local treatment of the colon. An occasional dose of castor oil to clean out the entire intestinal tube; opium from time to time when the peristalsis is excessive; and stimulants judiciously used, are the only drugs of any special value.

The *sanitary points* to be taken advantage of are abundance of fresh air, a change of climate, regular bathing, and cleanliness.

The *diet* should be nourishing, suitable for the age of the child, and either very easy of digestion or predigested. Peptonized milk, beef-juice, scraped beef, peptonoids, and such

forms of highly nutritious food leaving little residue are specially useful. Attempt to keep up the child's nutrition without overfeeding.

Astringent applications given regularly by means of enemata seem the most useful form of medication. They should be used daily, and such solutions as tannin, alum, boric acid, or silver nitrate may be employed. Changing the drug used in the enemata from time to time seems helpful. Bismuth may be given by mouth, as also pepsin; but brilliant results need not be expected from either.

CHRONIC TUBERCULAR ENTERITIS.

Occurrence: This condition is found usually associated with tuberculosis elsewhere in the body, but in some few cases may be primary. The mesenteric lymph-glands are always coincidently involved.

Etiology: The *tubercle bacillus* in the intestinal canal is the cause of this condition. The bacilli may be swallowed in sputum from the infected lung or in milk.

Chronic tubercular enteritis—pathology: The lesions of chronic tubercular enteritis are usually found only in the small intestine, and consist of tubercular deposits in the solitary and agminated lymph-follicles, and of necrosis of these with formation of ulcers. These *ulcers* are irregularly shaped, and lie transverse to the length of the gut. They vary greatly in number and size. The lymph-glands are enlarged, and may caseate and form abscesses.

Chronic tubercular enteritis—symptoms: There may be diarrhœa or constipation, the former being the more common. Diarrhœa in general tuberculosis does not always have ulcers as its cause. If diarrhœa is present, it is very obstinate. Hemorrhages are rare, but may be serious. The stools are large, frequent, and brown. Abdominal pain may, or may not, be present. There are progressive wasting, and fever with its accompanying symptoms.

Diagnosis: This depends on finding the tubercle bacillus in the stools. In any case of tuberculosis with diarrhœa the probabilities are in favor of ulceration being present.

Prognosis: This is distinctly bad. As a complication it makes the fatal end of pulmonary tuberculosis more rapid, due to its interference with the nutrition. If primary, it will probably lead to infection elsewhere.

Chronic tubercular enteritis—treatment: The diarrhœa is best treated by combinations of bismuth and opium. Intestinal irrigation is not used, as the lesions are seldom in the colon. Creosote is useful, as in all forms of tuberculosis. Stimulants will usually be necessary.

APPENDICITIS.

"**Appendicitis**" is now used to include all varieties of inflammation occurring in the region of the cæcum, as they are all now believed to originate in the appendix vermiformis.

Etiology: Appendicitis is commoner in males than in females, and is usualy seen in young adults. After the fourth year of life it is fairly frequent, although cases are reported from time to time even under four years, and one is recorded only seven weeks old.

Predisposing causes are the anatomical peculiarities of the appendix, such as unusual length, abnormal position, and irregularities of the mesentery. These all tend to prevent the appendix from expelling its contents. Adhesions from previous inflammations act in the same way. Chronic constipation also acts as a predisposing cause.

Exciting causes are usually mechanical—the presence of a fæcal concretion or a foreign body in the appendix. Blows, falls, or strains, with the presence of such a foreign body, act as frequent causes. Undoubtedly there is a *bacterial element* of much importance in the etiology; but as yet its exact connection with each case is not well worked out. Each case is probably due primarily to some mechanical cause interfering with the circulation in the appendix, followed by a germ infection made more easy by this stasis.

Pathology: The appendix may be the seat of a *simple catarrhal inflammation* only, with congestion and swelling of the mucous membrane, and increased production of mucus. In these mild cases resolution takes place with few symptoms and a normal appendix.

If the inflammation is *more severe*, the appendix becomes distended with the inflammatory products, and the lumen of the tube closed up. In these cases the peritoneal coat is involved as well as the mucous membrane, and from this a localized or general peritonitis may arise by contact, and without perforation. The mucous membrane is likely to be ulcerated, even when no foreign body is present. In recovery from these cases, strictures of the lumen, bendings and thickenings of the walls of the appendix, and peritoneal adhesions are left behind. Recurrences are frequent after such attacks.

In still *more severe cases* the inflammation and swelling are so intense as to cause necrosis and sloughing in the wall of the appendix, with *perforation* at one or more points. These perforations lead to a localized abscess if adhesions are present, or to a general peritoneal infection if not.

There is a form of appendicitis in which the *entire appendix* becomes rapidly *gangrenous*, with general peritonitis as an immediate complication. The whole appendix, or a portion, may become completely detached.

Inflammation and ulceration caused by the *typhoid* or *tubercle bacilli* are found in the appendix at times.

Appendicitis—symptoms: The disease usually begins with general abdominal pain, which sooner or later is localized in the region of the cæcum. With the pain there are fever of a moderate degree, at times a chill, some rapidity of the pulse, and nausea or vomiting. The bowels are usually constipated, but there may be diarrhœa.

On *examination* there is tenderness in the right iliac region, and this can usually be localized in one small area, called "McBurney's point," one-third of the distance on a line drawn from the right anterior superior spine of the ileum to the navel. The right rectus muscle offers some resistance to palpation.

If more severe, and the whole appendix is inflamed, with some localized peritonitis, the symptoms are more marked, and on palpation fulness and an indistinct feeling of a mass in the right iliac fossa can be made out. If perforation with a localized abscess results, a distinct mass, dull on percussion, is found.

The *suddenly perforating* and *gangrenous cases* may give no signs in the beginning different from the milder attacks, and may suddenly show all the symptoms of a diffuse peritonitis with general septic infection. There is very great difficulty in deciding in the early stages which are to be the mild and which the severe cases. No one symptom can be relied on to warn us of this, and hence the general picture of every case must be carefully and intelligently watched.

Perforation and *gangrene* usually succeed to a few days of the milder symptoms; but they may occur in the early stages. They are evidenced by a sudden increase in the pain, by vomiting, a rapid rise in the pulse, and the symptoms of *intense shock.*

The temperature usually rises also. If the general peritoneal cavity is shut off by adhesions, the acute symptoms gradually subside, and the patient is left with the presence of a circumscribed abscess. This abscess, if left alone, may perforate the colon, the bladder, or the peritoneal cavity, or may travel behind the peritoneum backward, upward, or downward. If no adhesions are present, the symptoms of shock are rapidly replaced by those of general peritonitis, and death follows in a few days.

After *recovery* from a primary attack the patient may have more or less frequent *recurrent* attacks of exactly the same character as the first one. The symptoms and prognosis in these recurrences are the same as in the primary attack.

Other cases have no more acute attacks, but suffer from a chronic disturbance, with pain and uneasiness, in the region of the appendix. Some of these patients tend to become chronic invalids.

Diagnosis: Remember how almost impossible it is to decide what pathological condition exists in the appendix from the symptoms and physical signs in the early stages. Reliance cannot positively be placed on anything.

In *differentiating* appendicitis from other conditions in children, ordinary colic and intussusception are most likely to cause confusion. In colic there is no fever and no tenderness. On the contrary, pressure ordinarily relieves the pain. In intussusception there is no fever in the beginning, and the

resistance or tumor is in the upper portion, on the left side of the abdomen rather than the right. Further, the tenesmus and bloody discharges from the rectum are absent in appendicitis.

Prognosis: This is always grave; but many cases recover under both medical and surgical treatment. If the case can be properly treated before general peritonitis develops, the chances for cure are good. Localized abscesses are favorable for cure. General peritonitis is uniformly fatal.

Appendicitis—treatment: The child should be kept absolutely at rest in bed, put on a fluid diet, and an ice-bag applied continuously to the right iliac region. If ice is objectionable to the patient's sensations, hot poultices may be substituted. Opium may be given in small quantities, but enough to relieve the pain if the local applications fail to do so. No cathartics should be given, but enemata may be used as needed.

It is wise for a physician and a surgeon to watch these cases together from the outset, as operation may be demanded at any time. When skilled surgical help is available there is less risk in operating on a doubtful case than in delaying the operation too long. The difficulty in deciding how a case will develop has much to do with the varying opinions of physicians and surgeons as to the place of operative interference in dealing with these cases.

If an abscess is present, an operation is indicated at once.

COLIC.

"**Colic**" is a name for a symptom only; but it is so common in infancy, and so often requires treatment of itself, that a separate description is usually accorded it.

Etiology: Colic is a regular symptom of almost all the functional and inflammatory diarrhœas, of appendicitis, of intussusception, and of worms. It may be present, however, without diarrhœa, vomiting, obstruction, or foreign bodies; and in these cases is due to flatulent distention of the intestine and irregular peristalsis. The distending gases are formed by fermentative changes in the food-contents of the

alimentary canal. It may occur in both breast-fed and artificially fed babies; but is commoner in the latter class. In either case the food contains some indigestible constituent, and this is usually the proteids. Starchy foods may be the cause, and also over-feeding. Chronic constipation is frequently present. Colic is most frequent during the early months of life.

Pathology: The condition is one of painful muscular contraction of the intestinal walls in the endeavor to remove the accumulated distending gases.

Colic—symptoms: These are crying, which is sharp and persistent, drawing up of the legs, and in boys retraction of the scrotum. The abdomen is distended, and pressure or kneading usually relieves the pain and quiets the child. If the gas is expelled by the mouth or anus, the crying usually ceases at once. There is always difficulty in getting these children asleep and in keeping them so. The least noise or movement disturbs them. They will usually take food ravenously, as if it temporarily relieved them, but in a few minutes they are crying again with pain.

In *mild cases* the infant is simply wakeful and fretful. If *severe*, there may be prostration and cold extremities. Very many infants have a chronic colic, and are continually crying and very restless.

Diagnosis: This is usually easy; but we must never forget the possibility of some grave intestinal disease being the cause of the colicky pain. Appendicitis and intussusception, and earache especially, must be excluded before calling the case a simple colic.

Prognosis: This is good. Prolonged cases can be cured by proper care of the diet.

Colic—treatment: *During the attack*, by mouth a little brandy and hot water, or ginger and hot water, or hot peppermint-water, or soda and hot water, will assist in removing the gas from the stomach. To remove the intestinal gases an enema of warm water or a glycerin suppository is most efficient. Hot applications and massage to the abdomen are very soothing. Opium preparations should be avoided as much as possible.

To *prevent recurrence* of the colic the *diet* should be investigated and any errors in it corrected. If any one of the food-constituents seems at fault, reduce its quantity in the food. If all the constituents seem normal, try reducing the total quantity of food given. Cure constipation if it exists.

CONSTIPATION.

Constipation is one of the most frequent disorders of infancy, and one of great difficulty in its satisfactory management.

Etiology: The causes exist either in the child or in its food. Probably the latter causes are the more important.

Of the *former*, *feeble muscular power in the intestinal wall* is of most importance. This may be due to the various forms of malnutrition, of which rachitis is the commonest. In older children, *lack of attention to the desire to go to stool*, and of the formation of a regular habit, is a prominent cause. *Decreased secretion* of the intestinal fluids and of the bile is probably next in importance. Certain *anatomical peculiarities* are believed to play their part, as a very long sigmoid flexure, stricture of the gut—congenital or acquired—kinks and bends from adhesions, tumors inside and outside the gut, and painful fissures at the anus, producing a voluntary constipation.

Of the *latter class* a deficiency in fat is probably of greatest importance. This occurs in babies fed on the breast and in those fed on the bottle. Next most common as a cause is the lack of enough total solids in the food to leave any residue. Here there is nothing to provoke the intestinal peristalsis. Other causes are ingestion of too little water, or excessive excretion of it by the skin or kidneys, leaving the fæces dry. Prolonged use of sterilized milk will often be the cause of constipation, as a return to fresh milk will frequently produce regular movements again. Drugs given for other causes, and containing opium or astringents, are factors of importance.

In older children the lack of a proper variety to the diet, such as green vegetables and fruit, may produce constipation.

Constipation—symptoms: The local symptoms are usually all that are present. Constipation is a relative term, as the normal number of stools per day varies with different individuals and with different periods of life. During the first year less than two stools per day is abnormal. After the first year the passing of any day without a stool is abnormal. But the character of the stool, and the ease or difficulty in its discharge, should be taken into consideration as well as the frequency.

The other symptoms associated with constipation are flatulence and colic, and a tendency to piles and to hernia. The hard masses of fæces may irritate the rectum and anus, and be streaked with mucus and blood.

Absorption of intestinal toxins may produce various general symptoms, as headache, languor, disturbed sleep, and some interference with nutrition. The tongue is furred, the breath is foul, and anorexia may be present.

In some cases the hardened fæces may block up and irritate the rectum and thus set up a false diarrhœa, the fluid movements taking place around or through the solid masses, the patient presenting the signs of diarrhœa while really suffering from constipation.

Prognosis: This depends on the cause and the possibility of its removal. Some cases are very stubborn to treatment.

Constipation—treatment: More than the fact of the existence of constipation must be known. We must find the cause of the condition, and, if possible, the part of the intestine at fault. If we can do these, we can treat the cases far more intelligently. If structural or pathological conditions are present, such that we cannot remove them, we must content ourselves by treating the case symptomatically.

In a breast-fed child have a chemical examination of the mother's milk made; and if some constituent is found abnormally low, as, for instance, the fat, take measures to increase this. In bottle-fed babies modify the proportions of the milk-ingredients by adding fat, or diminishing the proteids, or increasing the total solids, until some combination is arrived at that will produce daily stools. In both give plain water or oatmeal-water freely. Cane-sugar instead of lac-

tose will at times assist, and maltose will often do better yet. Stopping the sterilization of the milk may also aid.

In older children the addition of fruit and green vegetables, as orange-juice, stewed prunes, baked or stewed apples, is of value. Massage of the colon and along its course will be a help in some cases. Muscular exercise is of value by increasing the tone of all the muscles. If rachitis or malnutrition exist, the proper treatment should be undertaken for the cure of these.

When these modifications in diet and improvements in general condition fail to cure, our next resort is to specific treatment of the constipation itself. There are three general methods for this: *suppositories*, *enemata*, and *drugs*. If the seat of the constipation is in the rectum only, *suppositories* are of value. Soap, glycerin, and gluten are all used, and each works well; but any of them, if too long continued, may produce rectal irritation. They should be greased before insertion.

If *enemata* are used, plain water, or soap-suds, or sweet oil, or glycerin, may be injected. They produce a cleansing of the colon, and reflexly excite peristalsis in the small gut. It is best to use small quantities, so as not to dilate the intestine too much. Their effect gradually wears off, particularly if too large quantities of fluid are used.

Drugs are very unsatisfactory for prolonged use. Calomel, castor oil, rhubarb, cascara, aloes, and phosphate of sodium are the best. In all cases attempt to form a regular habit of going to stool. In children who are old enough to be taught this, much good may be accomplished.

INTESTINAL OBSTRUCTION.

This means a **mechanical obstruction** to the passage of the contents of the intestinal canal. The varieties of the obstruction in children are not so many as those in adults; but almost any form may occur, and one form particularly is a disease of childhood. The forms with rare causes will be mentioned, while the important variety—intussusception—will be described in detail.

Rarer causes: *Foreign bodies* may be the cause of obstruction. These may be objects swallowed, as solid substances; or masses of caked drugs, as bismuth. A lump of lumbricoid worms rolled up together has been found as the cause. *Volvulus*, or the twisting of the gut upon itself, is a rare cause in children; more common in adults. *Strangulation*, or kinking of the gut, by abnormal bands from previous peritonitis; or from abnormal openings in the mesentery; or by the remains of Meckel's diverticulum; or by an adherent appendix, may likewise cause strangulation. *Strangulated hernia* as a cause of obstruction should never be forgotten.

INTUSSUSCEPTION.

All the above forms of obstruction are rare in childhood when compared with this condition.

Definition: Intussusception, or invagination of the intestine, consists in one portion of the bowel passing into a succeeding portion, and through its mechanical presence and subsequent swelling and bending by the attached mesentery, blocking up the lumen of the bowel and causing the obstruction.

Varieties: Intussusceptions may occur at any portion of the bowel, but are commoner in certain parts than in others.

The commonest location is for the *apex* of the intussusceptum, or entering portion, to be formed by the *ileo-cæcal valve*. This draws in after it the colon, and is called the *ileo-cæcal variety*.

A subvariety, the *ileo-colic*, is formed by the invagination of the ileum through the valve and into the colon, but without necessarily invaginating the colon.

The *enteric variety* is rarer, and consists of the invagination of some part of the small gut into another part.

The rarest variety of all, the *colic*, consists of the invagination of some part of the colon into another part.

Intussusception may occur in two places at once. It usually takes place in the direction of the peristalsis—that is, *downward*; but the reverse may occur rarely.

Except in the ileo-colic variety, the apex of the intussusceptum is fixed, the outer sheaths, or intussuscipiens, being

gradually infolded. In the ordinary variety a few inches to six or more feet of bowel may be involved.

Mild forms of intussusception occur frequently just after death. They are usually multiple and enteric, always short, and usually *upward.*

Etiology: There are no facts of any value in causation. It is most frequent from four to nine months of age, and in males. Most cases occur in previously healthy children. Previous intestinal disorder is present in a small proportion of the cases. Irregular peristaltic action of the intestinal walls seems the exciting cause.

Intussusception—pathology: There is great congestion of the invaginated bowel; and if long enough continued, this leads to gangrene and sloughing. The two peritoneal surfaces in contact are liable to form adhesions to each other, and this, combined with the swelling and the dragging of the mesentery, makes reduction often very difficult. If adhesions form and the intussusceptum sloughs, it may be discharged entire or piecemeal through the bowel, and spontaneous cure result. If adhesions do not form, the sloughing leads to perforation of the gut and infection of the peritoneum.

Intussusception—symptoms: These regularly begin suddenly, with severe *abdominal pain.* It is located in the neighborhood of the navel, and causes the child great agony. The pain is paroxysmal in character, and is almost at once accompanied by *vomiting.* The stomach first empties itself of its contents, and afterward ejects bile in large quantities; later the vomiting may be *stercoraceous.* At first there are one or two loose fæcal stools; but afterward absolutely *no fæcal matter* is passed, but only bloody mucus. With this is marked *rectal tenesmus.* Soon after the attack begins, on palpating the abdomen it is found relaxed; and usually in the neighborhood of the transverse or descending colon a tumor is found. In a fair proportion of the cases rectal examination discloses the apex of the advancing intussusceptum. In some this may protrude from the anus.

As the case advances, *tympanitic distention* of the abdomen and tenderness develop. At first there is no fever. If in-

flammatory signs appear in the intussusception, it may develop.

There are intense *prostration*, feeble pulse, cold extremities, and pallor. A marked diminution in the quantity of urine secreted, or complete suppression, is frequently present.

There are *chronic cases* with vague abdominal symptoms, but without complete obstruction. In these cases the presence of the tumor makes the diagnosis.

In the *acute cases* the disease lasts less than a week. Spontaneous reduction probably occurs at times. Spontaneous cure by adhesions and sloughing may occur. Death is usually from shock ; or, if shock is survived, from peritonitis.

In the *chronic cases* the duration may be from two weeks to a month. These usually die from exhaustion or the development of complications.

Diagnosis: With a typical history, and a careful examination showing the presence of a tumor, the diagnosis should be easy. Never neglect a *rectal examination*. In all cases of bloody and mucous discharges from the bowels think of intussusception as well as entero-colitis. They are easy to differentiate if both are in mind. In chronic cases the tumor is the diagnostic point.

Prognosis: This is always grave. The younger the child and the more advanced the case the more serious is the prognosis. With early diagnosis and proper treatment many recover. Spontaneous recovery, while it does occur, is rare. Chronic cases seldom recover.

Intussusception—treatment: As soon as the diagnosis can be made adopt energetic methods of treatment. Every hour's delay increases the dangers. Give absolutely no cathartics. These only increase the intestinal peristalsis and drive the intussusceptum farther in. On the contrary, keep the patient under the influence of opium. This fulfils two indications ; it relieves the pain and decreases the peristalsis, thus preventing further advance of the invagination.

Next, an attempt should at once be made to reduce the intussusception by means of *mechanical devices*. These are inflation by air and injection of fluids. Each has its advocates. Either is best done under an anæsthetic.

Inflation is performed with the patient on his back, and the air is pumped into the bowel through a rubber rectal tube attached to a Davidson syringe or to a small hand-bellows. This should be done quite slowly. While the air is being introduced the abdomen may be gently massaged upward over the tumor. From time to time the child may be inverted to aid reduction by the action of gravity. The amount of air introduced should be governed by the tension of the abdominal walls. Sounds or physical signs may suggest reduction; but to be sure of it the air should be let out and a careful examination for the disappearance of the tumor made. After complete reduction the symptoms should cease, and fæcal movements or passage of gas begin. If inflation fails the first time, it may be repeated; but more than two trials are useless.

Injection of fluids may also be tried to reduce the intussusception. It may be tried first, or after the failure of inflation. Again the child is anæsthetized, and a warm saline solution is introduced into the bowel, best by means of a fountain-syringe having an elevation of four to six feet. The abdomen may be massaged during the introduction of the fluid, and inversion may also be tried. A bandage rolled around the rectal tube prevents escape of the fluid. The fluid should be introduced slowly, with interruptions, and the amount will vary in individual cases. It is more difficult to decide whether reduction has been accomplished in these cases than when using air. Again the fluid must be let out and examination made for the tumor.

If *reduction is accomplished* in either way, the child must be kept quiet, given little food, and held under the influence of opium for a few days, until the danger of recurrence has passed.

Too much time must not be lost in trying to reduce by inflation or injection. If both methods fail—and they often will—resort must be had to *laparotomy*, as otherwise we leave our patient to almost certain death.

It is best to be prepared for operation while trying the mechanical devices for reduction; and if they fail, operate before the patient recovers from the anæsthetic. Of late

years many of the cases operated on during the first few days, and even in infants, recover.

In chronic cases operative results are quite brilliant.

INTESTINAL PARASITES.

Varieties: There are *three general forms* of worm that commonly inhabit the intestines of children. These are the *round-worm*, ascarides lumbricoides; the *pin-worm*, oxyuris vermicularis; and two varieties of *tapeworm*, tænia mediocanellata and tænia solium.

Worms, however, are very much less common than is ordinarily supposed by the public at large. The life-history of each form of parasite is different, and as each requires a special treatment a separate description of each is necessary.

ROUND-WORMS.

Description: These worms, the *ascarides lumbricoides*, are from five to ten inches long, the female being the longer. They are cylindrical-shaped, taper at both ends, and are of a pinkish-gray color, being not unlike the ordinary angle-worm in appearance. The worms live in the small intestine, and rarely are single. Half a dozen to a hundred may be present. They frequently roll up into large masses. They have a curious tendency to wander from their natural home in the small gut, and may be found in the colon, stomach, œsophagus, and even in the nose and larynx. At times they escape into the peritoneal cavity through an intestinal perforation. They have been known to crawl into the common bile-duct and to block it up.

The *eggs* are oval in shape, and about $\frac{1}{400}$ of an inch long. The contents appear granular, and the coat thick. They are discharged in large numbers in the stools, and have great vitality outside the body. They are probably swallowed with the food or drink, and after entering the intestine develop into mature worms there.

Round-worms—symptoms: There may be no symptoms at

all, and the finding of a worm in a stool may be the first suspicion of anything being wrong.

In other cases there may be irregular abdominal pains and tympanites, with restlessness, poor sleep, grinding of the teeth, and picking of the nose. These symptoms are all more often due to some chronic indigestion than to the presence of the worms. Various *nervous* disturbances of an emotional character are often associated with the presence of worms, as headache, dizziness, hysterical symptoms, tetany, and even convulsions. Certain *mechanical* symptoms may be troublesome, as the massing of large numbers of the worms in a ball somewhere in the intestine; or due to their tendency to travel into undesirable situations.

Diagnosis: No symptoms or set of symptoms can be relied on for a diagnosis. The only positive sign is by seeing the worms or their eggs in the stools. If one worm is found, the probability of others being present is strong.

Round-worms—treatment: The drug of most value for killing or benumbing the ascarides is *santonin*. This is best given after a few hours of fasting, and must be accompanied or followed by a cathartic, as in itself it does not remove the worms. It is well given rubbed up with calomel, and in divided doses. The dose for a child of five years is two to four grains. A dose of castor oil may be given afterward, if the calomel does not completely empty the canal.

PIN-WORMS.

Description: These are very small worms, called also thread- and seat-worms. The technical name is *oxyuris vermicularis*. They are of about the diameter of thread or a pin, and are from one-sixth to one-third of an inch long, the female again being the longer. They are white in color, and taper to a point at the tail. The *eggs* are oval and small, with a thin coat. The worms live almost entirely in the rectum and colon, and are present in enormous numbers. They are propagated by the swallowing of the ova. The worms, and also the eggs, are passed in large numbers with the stools. They are frequently found alive in the folds of

skin around the anus and genitals, and these act as a frequent source of *reinfection* to the patient.

Pin-worms—symptoms: The most important sign of the presence of the oxyuris is intense itching of the anus and genitals. This is usually worse when the child goes to bed. The scratching for relief may lead to eczema of the anus, or balanitis, or vulvitis. Masturbation and incontinence of urine are frequent results.

The child's sleep is disturbed, and he becomes restless and wakeful. The worms irritate the colon, and much mucus is usually discharged with the stools. Reflex nervous symptoms are much rarer than with the other varieties of worms.

Diagnosis: Itching of the anus and genitals in children should always make us suspect seat-worms, but the only positive diagnosis again rests on seeing the worms themselves or their ova. Examine the discharges and also the parts about the anus very carefully for either.

Pin-worms—treatment: Scrupulous cleanliness of the patient is the first thing of importance. He should be kept very clean, and his hands and the parts about the anus should be washed daily with some antiseptic solution, as the bichloride of mercury 1 : 5000. The anus should be kept anointed with a 2 per cent. carbolic salve, to prevent itching and to kill any worms that emerge from the rectum.

By *mouth*, salts should be given to produce watery movements and to wash the worms that are high up in the bowel down into the colon.

Once a day the colon should be washed out thoroughly with a large quantity of water passed through a long rectal tube. This water should contain either soapsuds, or quassia, or alum, or salt. Bichloride of mercury, in 1 : 10,000 or 1 : 20,000 solution, is very useful for killing the worms; but there is danger of poisoning from some of the solution being left behind.

The cure is slow, but, by persistent use of the above means over some length of time, can be effected. The clothing, toys, bedding, and even carpets should be thoroughly cleaned for fear of reinfection.

TAPEWORMS.

Description: Tapeworms are from twenty to fifty feet long, of a white color, and composed of many flat segments. The segments are fairly uniform in size, but taper gradually to the head, where the newly formed segments develop. The head is a modified segment, the size of a pin's head, and contains suckers by which the worm fastens itself to the gut.

Varieties: The commonest varieties of tapeworm found in this country are two, the *beef tapeworm*, or *tænia mediocanellata*, and the *pork tapeworm*, or *tænia solium*. Each segment of each variety is a sexually mature individual, and ova are cast off continually by them. Segments from the tail end are also broken off from time to time and discharged from the bowel. New segments grow from the head-end to take their place.

Cysticercus: The *eggs* from the tapeworm are swallowed by animals in their food, and passing into their stomachs the *embryos* are set free and are carried by the blood around the body and are deposited in various parts of the tissues, among others the muscles. Arrived here they form a little wall around themselves and take on a *larval condition*. They are then called *cysticerci*, and may live in this condition for some years. Each cysticercus is about the size of a pea.

When the flesh of these animals is eaten by a human being, unless the cysticercus is destroyed by the heat of cooking, it is set free in the digestive tract, and attaches itself to the mucous membrane of the small intestine and grows there into an adult tapeworm.

Habitat: The *tænia mediocanellata* lives in the bodies of cattle during its larval state, and hence is called the beef tapeworm. The *tænia solium* lives during its larval state in the bodies of hogs, and hence is called the pork tapeworm. More than one tænia mediocanellata is frequently found in a patient at a time; but the tænia solium, as its name implies, is usually single.

Tapeworms—symptoms: There may be no symptoms at all, the finding of segments in the stool being the first intimation of the presence of the worm. Irregular pains in the abdo-

men, large appetite, restlessness, and picking at the nose are observed in some cases. There may be diarrhœa. Probably these symptoms are all due more to disordered digestion than to the worm.

Tapeworms—diagnosis: This is made entirely by finding segments of the worms in the stools. The physician should always examine these segments himself.

Tapeworms—treatment: Preventive treatment consists in not eating underdone beef, or pork, or pork preparations. Thorough cooking will destroy the cysticerci.

If the presence of the worm is proven, treatment should be begun at once by first starving the patient for some time. To increase this time the night may be taken advantage of in children. Have the child go to bed with a very light meal and a laxative. In the morning, after the stool (which may be assisted by an enema) and without any breakfast, give the specific drug. Follow this in an hour by a thorough purge, the best of which is a tablespoonful of castor oil.

Practically only two drugs need to be remembered as tæniacides, *male-fern* and *pomegranate*. The first is given as oleoresina aspidii, in doses of half a drachm to one drachm, and made up in an emulsion with syrups, or in capsules, if these can be swallowed. It makes a nasty mixture, however, and in cases where expense need not be taken into account Tanret's pelletierine, made from the alkaloid of the pomegranate, is the nicest way of attacking the worm. Each bottle contains an adult dose. For a child a proportionate amount should be given. It also should be followed by a purge in an hour. To insure against recurrence the head must be removed.

DISEASES OF THE ANUS AND RECTUM.

PRURITUS ANI.

Pruritus ani is at times seen in children, and is an intense itching in the neighborhood of the anus.

Etiology: The itching may be due to pin-worms in the rectum, to pediculi, to irritating fecal discharges, to consti-

pation, or to eczema of the anus. The constant scratching tends to make it worse by setting up an artificial dermatitis.

Treatment: If possible find the cause and remove it. Keep the parts absolutely clean by bathing them after every stool. After bathing anoint the anus with some such ointment as,

℞. Acid. carbolic. . . . . ♏x.
Ung. zinc. oxid. . . . . ℥j.

PROLAPSUS ANI.

Varieties: There are two varieties of this condition: 1, Where a portion of the *mucous coat* only of the rectum is prolapsed; and 2, where the *entire rectal wall* is invaginated through the sphincter.

Etiology: This condition is quite common in children in the second and third years. It is predisposed to by the anatomical fact of the very loose attachment of the submucous connective tissue of the rectum.

Chronic intestinal disorders and constipation, by causing straining efforts at stool, are the common exciting causes. Phimosis, vesical calculus, and rectal polypus may be causative agents.

Prolapsus ani—symptoms: The characteristic symptom is the appearance during stool of a dark-red or purplish-colored tumor protruding from the anus. This is covered by mucous membrane, which may be in a condition of acute inflammation. It may bleed freely. The mass often will return spontaneously, or may be easily reduced. There is no pain connected with the protrusion. After prolapsing once, recurrence with each stool is common.

Diagnosis: This condition must be differentiated from hæmorrhoids and rectal polypi. After reduction the absence of any tumor on rectal examination excludes these conditions. More important is to diagnose it from intussusception, in which the presence of pain and obstruction are of most value.

Prolapsus ani—treatment: Lay the child on its face, and, having oiled the mass, gentle pressure will usually easily

reduce it. If difficulty is found, the application of cold or the use of an anæsthetic may be called for. After reduction keep the child on its back for an hour to prevent recurrence, and before allowing it to move about a pad to the anus held in place by a T-bandage should be applied. After this the child should not be allowed to defecate in the ordinary sitting posture. He should be made to lie down on his back or side, and to use a bedpan. The bowels should be kept open by the use of laxatives. Any of the causes of the condition that may be present should be removed. A daily enema of some astringent solution, as alum or tannin, tends to contract the mucous membrane and to prevent recurrence.

If these simple means fail, linear cauterization by nitric acid, made under an anæsthetic and followed by artificial constipation with opium for a few days will usually cure the cases permanently. The actual cautery may be used instead of nitric acid.

HÆMORRHOIDS.

Definition: These are vascular tumors growing in the lower portion of the rectum, outside or inside the sphincter, from dilatation of the bloodvessels of the part. They are quite rare in childhood, and when they do exist are generally of the *external* variety.

Etiology: Chronic constipation.

Symptoms: Presence of the vascular masses around the anus, and pain at stool. Bleeding is rare in children.

Hæmorrhoids—treatment: Regulate the bowels. Some astringent ointment, as the unguentum gallæ, may be used locally. Operative interference is rarely required; but, if necessary, *ligation* is probably the best procedure.

FISSURA ANI.

In **fissura ani** a small ulcer is present at the anal margin, and usually extends over the area that is under the action of the sphincter. It is a fairly common affection of childhood, and is seen at times even in infants.

Etiology: The passage of hardened fæces, scratching to re-

lieve pruritus, or traumatism from the nozzle of a syringe may cause the fissure.

Symptoms: Pain at and after stool is the marked symptom of this condition. On examination, an ulcer with its long diameter parallel with the long axis of the bowel, and lying over the sphincter, will be seen. It has a grayish base, and often bleeds slightly.

Fissura ani—treatment: Clean the parts and touch the base of the ulcer with silver nitrate stick. Keep the bowels open. If healing does not take place under this method, stretch the sphincter and keep the parts at rest, when cure will be rapid.

ISCHIO-RECTAL ABSCESS.

Definition: This is a collection of pus in the cellular tissue around the lower portion of the rectum.

Etiology: Traumatism is an active cause. It may arise through infection of the cellular tissue from the rectum through either the lymph- or bloodvessels.

Symptoms: A sense of fulness, intense and throbbing pain, and tenderness of the parts are the significant symptoms. Defecation causes great agony. Some fever with its constitutional symptoms is usually present.

On examination, a tense, red, tender swelling will be found on one or the other side of the anus. Fluctuation may be obtained from the skin surface or from the rectum.

Ischio-rectal abscess—treatment: Early and prompt incision through the skin, in a line radiating from the anus, should be made. The finger should be inserted in the wound, and any partitions broken down. The wound should be irrigated and packed to heal by granulation.

FISTULA IN ANO.

Fistula in ano is an unhealed ischio-rectal abscess. The sinus may lead from the rectum to the old abscess-cavity, or from the skin to the old abscess cavity, or be complete and lead from the rectum through the old abscess-cavity to the skin-surface.

Etiology: There may be a history of an acute ischio-rectal abscess which has been untreated; or the fistula may result without an attack of precedent acute and painful inflammation. These chronic cases are more likely to be of a tubercular nature.

Symptoms: There is no special pain in this condition, but the sign of suspicion is a discharge of pus or bloody fluid with the stools, through either the external or the internal opening. On examination, the opening will be found either on the skin-surface or in the rectal wall; if *complete*, a probe can be passed through the fistula, or colored fluid or peroxide of hydrogen can be syringed through to locate the internal opening.

Treatment: Any variety of incomplete fistula should first be made complete, by passing a director through from the skin-opening (an artificial one being made if necessary) into the rectum, and then bringing the point of the director out through the anus. Along this as a guide, cut through the intervening tissue, dividing the sphincter once only. If pockets exist, open into them freely from the first incision. Curette the tissue, pack with gauze, and let the wound heal by granulation.

ACUTE PROCTITIS.

"**Proctitis**" is applied to an inflammation of the rectum unaccompanied by inflammatory trouble higher up in the bowel.

Etiology: Enemata, suppositories, traumatism from the nozzle of a syringe, thread-worms, and irritation from hardened fæces are frequent causes. The inflammation may be due to infection by the germs of gonorrhœa, diphtheria, or scarlet fever.

Pathology: The rectum may be the seat of a simple catarrhal inflammation; or superficial or deep ulcers may form; or there may be an inflammation with the production of a false membrane.

Acute proctitis—symptoms: There are mild constitutional symptoms only, the local signs being the most important.

There are marked rectal tenesmus, and the frequent passage of very small stools each containing a large proportion of mucus and some blood. Prolapsus ani is a frequent complication. Pruritus ani and excoriations of the neighboring skin are common. In ulcerative cases there are pus in the stools and marked pain.

Acute proctitis—treatment: The patient should be put to bed, and on a proteid diet. The bowels should be moved by small doses of castor oil. Daily injections of some bland fluid should be employed to wash out the rectum and quiet the tenesmus. Starch-water and laudanum is a useful wash. Suppositories of opium or of cocaine may be used. If ulcers are present and can be seen, a solution of boric acid or nitrate of silver should be used as a local application. If any removable cause is present, it should be attended to.

POLYPUS RECTI.

Polypus is a much commoner condition in childhood than in adult life. No cause for the growth is known.

Pathology: The polyp is a pedunculated body about the size of a hazelnut. Histologically it is of a myxo-fibromatous or adenomatous structure. There may be only one, or many tumors. In the early stages they may be sessile, but in time they always tend to become pedunculated. They are usually located in the lower segment of the rectum.

Polypus recti—symptoms: The symptoms are bleeding from the rectum, associated with some rectal irritation and tenesmus. They may lead to a mucous discharge and to prolapsus ani. As the pedicle grows long enough the tumor is often protruded during stool.

On examination it appears the color of mucous membrane; but if protruded and pinched, it has a purplish tint. It is found to be distinct from, but attached to, the general mucous membrane, thus differentiating it from prolapsus ani.

Polypus recti—treatment: This consists in tying the pedicle off at its attachment, and cutting the tumor away beyond the ligature. If more than one polyp exist, each must be treated the same.

DISEASES OF THE LIVER.

The **liver** in new-born babies and in early childhood is, proportionately to the body-weight, a larger organ than in adults. This should be remembered in estimating its size, as it will be found normally extending below the free border of the ribs. This comparatively large size has some relation to the direct connection of the liver with the placental circulation.

Notwithstanding the large size of the gland, *diseases of the liver* in *infancy* and *childhood* are comparatively rare, and clinically the liver offers little of interest.

JAUNDICE.

Definition: Icterus neonatorum, the jaundice of early infancy, has already been described. This is a jaundice due to changes in the blood, *hæmatogenous* so called.

Jaundice due to changes in the liver, *hepatogenous*, does, however, occur in children. It is also called simple and catarrhal jaundice. Under the term gastro-duodenitis an intense and more widespread form of the disease is recognized. There may, however, be only a catarrhal inflammation of the bile-ducts present, and no involvement of the duodenum or stomach. This is the form under consideration.

Jaundice—etiology: The mucous membrane of the common or hepatic ducts is swollen, thus closing the lumen of these ducts. A plug of inspissated bile, a round-worm, or rarely a gall-stone may plug the duct. Errors in diet or exposure to cold may be the causative agent. In many cases there seems no exciting cause.

Symptoms: The main sign of simple jaundice, and often the only one, is the *yellow discoloration* of the skin and mucous membranes. It varies from a quite pale lemon tint to a decided dark-yellow color. The urine is dark brown, and contains bile-pigment in abundance. The stools are white or "clay-colored," due to the absence of bile from the intestinal tract. The pulse is often slowed, and there may be itching of the skin, particularly if the jaundice has been persistent. Urticaria may be present. The bowels are apt to be constipated.

Physical signs: On examination the liver is usually a little enlarged, and some tenderness over it is present. The gall-bladder may be found distended.

Prognosis: This is good, as most of the cases recover rather rapidly, even if left alone. The duration is one or two weeks. When the common duct is plugged by a round-worm or other solid body the prognosis is not so good.

Jaundice—treatment: The *diet* should be free from fats and sugars. The bowels should be kept open by calomel, or salines, or aloes. Phosphate of sodium is one of the best drugs to use with children, owing to its lack of taste. An excess of water should be given to drink. Large enemata of cold water have been found quite useful in the more obstinate cases.

FUNCTIONAL DISORDERS OF THE LIVER.

Functional disorders of the liver are most apt to be associated with disturbances of the functions of the stomach and intestines, but at times the liver alone may be involved.

Either the *bile-producing* function of the liver may be disturbed, or the *chemical changes* which should normally take place *in the blood* passing through the liver are imperfectly performed.

While this whole question is as yet in an unsettled state as regards exact knowledge, still much of the evidence points to disorders of the liver functions as being the basis of conditions which are variously styled, lithæmia, biliousness, uric-acid diathesis, and so on. Such conditions are fairly common in childhood, although they are undoubtedly often overlooked and ascribed to other causes.

Etiology: Heredity seems to be the main predisposing cause. The exciting cause is the habitual eating of improper food, or of too large quantities of food; and insufficient muscular exercise.

Functional disorders of the liver—symptoms: Constipation, flatulence, headaches, bad breath, coated tongue, poor nutrition, and anæmia are the most marked symptoms. The appetite is apt to be capricious. The fæces are light colored

and have an offensive odor. The urine is of high specific gravity, and contains an excess of urates or phosphates.

These children are apt to be *neurotic* and irritable, and to have frequent regular or irregular nervous "explosions" of various sorts. These frequently develop at puberty.

Functional disorders of the liver—treatment: This is fairly successful where the family compel the child to carry out the physician's directions.

In the first place, the *diet* should be regulated. Sugars and fats should be avoided. An easily digested, mixed diet, given at regular intervals, and in not excessive quantities at a time, should be prescribed. Plenty of water should be given daily to keep the various fluids of the body dilute. Regular outdoor exercise should be enforced.

As *drugs*, those that increase the production of bile are indicated, phosphate of sodium, aloes, podophyllin, and rhubarb. After a course of such treatment extended over a considerable time the tendency to outbreaks of these disorders can usually be overcome.

ACUTE CONGESTION OF THE LIVER.

Varieties: The liver may be congested either *actively* or *passively*. The *acute congestions* are much rarer than the chronic.

Etiology: It is the result of poisoning by malaria or phosphorus; may follow overeating of rich foods; and be a complication of simple jaundice or gastro-duodenitis.

Symptoms: These are very slight. Moderate jaundice may be present. The liver is moderately and uniformly enlarged, and may be slightly tender to pressure.

Acute congestion of the liver—treatment: This consists simply in removing the cause, or rather in treating the condition giving rise to the congestion.

CHRONIC CONGESTION OF THE LIVER.

Etiology: This is the commoner variety of congested liver. It is never a primary disease, but is always secondary to conditions causing stasis of the blood-current. The commonest

cause is congenital or acquired *heart-disease*, and, next to this, *chronic pulmonary conditions*, as emphysema, pleurisy, or interstitial pneumonia.

Pathology: The liver is enlarged, firm on pressure, and harder to cut than normal. The surface is dark, and on section the so-called nutmeg appearance is present, due to the dilatation of the central veins of the lobules. There is some increase in the connective tissue.

Chronic congestion of the liver—symptoms: These are due rather to the primary disease than to the congested liver. There may be some slight jaundice and other symptoms indicative of interference with the functions of the liver, such as coated tongue, poor appetite, and constipation.

On *examination* the lower edge of the liver is found to reach well below the costal border, and to be easily felt by palpation. The enlargement will be found uniform, and there will be no nodules.

Diagnosis: This should be made from hypertrophic cirrhosis and from the enlarged liver of leukæmia. The presence of the causative factor is of special value in congestion.

Prognosis: This depends entirely on the importance of the primary disease.

Chronic congestion of the liver—treatment: The removal or treatment of the cause is the rational method of caring for this disease. The occasional use of a saline laxative tends temporarily to reduce the congestion.

SUPPURATIVE HEPATITIS.

Varieties: There are *two* varieties of this condition in children, as in adults. In one there is a *single* circumscribed *abscess;* in the other there are *multiple points* of suppuration. Either form is rare in children.

Etiology: Traumatism seems to have been present in many of the cases. Dysentery occasionally precedes the abscess. A suppurative inflammation of the portal veins is more commonly found as a cause. This may arise from the umbilicus, from the appendix, or from typhoid ulcers. It may be a metastatic pyæmic abscess. In many cases no cause can be found.

Suppurative hepatitis—symptoms: In the variety with *multiple suppurative points* we usually have the preceding symptoms of the inflammation, in the area drained by the portal veins, which is the cause of the hepatitis.

The signs pointing to involvement of the liver are enlargement, pain, and tenderness over that organ. More or less jaundice usually develops coincidently. Chills and irregular fever are present. After a time the typhoid state develops, with low muttering delirium, stupor, dry, brown tongue, and sordes on the lips and teeth. Diarrhœa soon begins, with thin, offensive, light-colored stools. The urine contains bile-pigment, and later albumin and casts. Rapid emaciation is regularly seen.

In the variety with *single abscess* the symptoms are less acute. There are pain in the region of the loins, chills, sweating, irregular fever, loss of flesh and strength, and some jaundice. The typhoid state develops less rapidly.

There are cases where the symptoms are entirely latent, and the abscess is discovered by accident.

On *examination* the liver is tender and enlarged, and frequently the enlargement is irregular, the abscess being near the surface and pointing above or below.

Diagnosis: The presence of hepatic symptoms, combined with irregular chills and fever, and a uniform or irregular enlargement of the liver, are the points for diagnosis.

Withdrawal of pus through an exploring-needle, is the only positive evidence of abscess that we have. Failure to obtain pus on the first trial does not exclude its presence.

Prognosis: In multiple abscesses the prognosis is almost surely fatal. In the single variety, although still grave, it is somewhat better.

Suppurative hepatitis—treatment: This is purely surgical. If the pus is so situated that it can be reached and freely evacuated, a good many cases will recover.

FATTY LIVER.

This condition is **a fatty degeneration** of the hepatic cells, and is, as a rule, secondary to some of the wasting diseases of

children. Tuberculosis, marasmus, and chronic gastro-intestinal diseases are the usual primary conditions. It is fairly common in infants.

Pathology: The liver is large, the surface is smooth, the color is yellow, much lighter than normal, and a cut section has an oily appearance. Under the microscope fat-globules are seen in the liver-cells.

Symptoms: There are no subjective symptoms. A uniform enlargement of the liver is present. There is no pain and no tenderness. If the fatty degeneration is marked, some interference with the hepatic functions may be present, but not enough to give any marked symptoms.

Fatty liver—treatment: This is entirely that of the original disease on which the liver condition depends.

AMYLOID LIVER.

Amyloid degeneration of the liver-cells is dependent on chronic suppurative disease in other parts of the body. Chronic bone-disease is the commonest precursor. Phthisis, empyema, and hereditary syphilis are less common primary causes. This condition is fairly common in childhood. The spleen and kidneys are likely to be similarly affected.

Pathology: The liver is quite large, and symmetrically so. It has a smooth surface, with a gray waxy color. On section it is fairly firm. Iodine gives a mahogany color to the degenerated cells.

Amyloid liver—symptoms: There are no special symptoms from the liver itself. In the presence of the original disease, a markedly enlarged liver, with no pain, no tenderness, and no jaundice, will usually be waxy. Slight interference with the liver-functions may occur, but the symptoms are so merged with those of the primary disease as to give them no value. The spleen is regularly enlarged, and the urine sometimes shows the changes seen in amyloid kidneys.

Amyloid liver—treatment: This is entirely that of the primary disease. In cases where the originating focus can be eradicated cure may be hoped for, but otherwise little can be expected.

CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is quite rare in infancy and childhood, but from time to time cases in children under puberty are reported.

Etiology: Alcoholism is a cause as in adults. Some children inherit an appetite for liquor; and to others it is given medicinally in such quantities and over such periods as to produce in the liver the degenerative changes of chronic alcoholism. Syphilis, malaria, and chronic ptomain-absorption from the intestines are recognized as distinct causes.

Pathology: There are two general varieties, the atrophic and the hypertrophic cirrhosis. The latter form is the rarer in children.

In the *atrophic form* the liver is smaller than normal; its surface rough and yellowish; it is firm and hard to the touch and cuts like cartilage. The liver-structure shows a marked increase of the connective-tissue stroma. The hepatic cells are atrophied and replaced by new connective tissue. The smaller veins and bile-ducts may be obliterated.

In the *hypertrophic variety* the liver is enlarged, firm, and yellowish colored. The new growth of connective tissue begins around and follows the intralobular branches of the bile-duct, giving a more uniform distribution. The portal veins are less interfered with, while the bile-ducts are more apt to be destroyed. From this distribution of the connective tissue the term *biliary cirrhosis* is often applied to this condition.

Cirrhosis of the liver—symptoms: In the *early stages* the symptoms are mainly those of disturbance of the functions of the liver. Such symptoms as furred tongue, bad breath, bad taste in the mouth, capricious appetite, and constipation with foul stools are usually first noticed.

After some time the more definite symptoms of the disease develop, as vomiting, hematemesis, slight ascites, enlarged spleen, diarrhœa, bloody stools, and hæmorrhoids. Slight jaundice may occur. The patients lose flesh and strength, and become anæmic.

On *examination* the liver is found to be small, the spleen

large, and the presence of fluid in the abdominal cavity can be demonstrated. There may be albuminuria.

The *hypertrophic form* has somewhat the same symptoms, except for the presence of a marked and rather malignant form of jaundice, an enlarged liver, and usually not much ascites.

Toward the end both varieties present some irregular fever, with low delirium and other cerebral symptoms.

Prognosis: This is quite unfavorable. The course of the disease is slow, although seemingly less so than in adults. The hypertrophic form is more rapidly fatal.

Cirrhosis of the liver—treatment: If any causative factor is present, remove it at once. A *milk-diet* is the best for this disease. Any tendency to congestion in the portal system should be relieved by the use of *salines*. The patient should be out of doors and should *exercise freely*. *Plenty of water* should be drunk daily. If syphilis is present, mercury and iodide of potassium are to be used, and may produce good results. Chloride of ammonium also seems to have some value in this disease. The ascites is to be relieved by diuretics, purging, or aspiration.

HYDATIDS OF THE LIVER.

Hydatids are rare in the United States, and especially rare in children. A few cases are reported from time to time. At any rate, children are not immune to the disease.

Hydatids of the liver—etiology: They are produced by the development in the liver of the embryo of the tapeworm in its larval state. The eggs of a tapeworm are swallowed by the child, and the embryo, being set free in the stomach or intestines, travels through the walls of the viscus and is carried by the portal blood to the liver. Here it forms a wall about itself, and develops a so-called echinococcus-cyst. This cyst, growing gradually in size, is the *hydatid*.

Pathology: The echinococcus is enclosed by a thick wall made up of connective tissue from the organ in which it is growing. The cyst is single (unilocular), or contains smaller cysts inside the larger ones (multilocular). The contents are an opalescent fluid, slightly albuminous, containing crystals of cholesterin and *echinococcus hooklets* or *scolices*.

Hydatids of the liver—symptoms: The cyst is usually latent for some time, until it grows large enough to cause symptoms by its mechanical presence. It grows very gradually, and often a good-sized cyst will be discovered by accident on examination when no previous history has been present.

Usually the first thing noticed by the patient is an enlargement in the region of the loin, or of the whole abdomen. There is no pain and no tenderness. Jaundice is rare, only occurring when the cyst presses on the hepatic duct. Pressure on the portal vein may produce ascites. The cyst may become infected and suppurate, giving the symptoms of encapsulated abscess. The cyst may rupture, usually as the result of trauma. This may take place into the stomach, or bowel, or pleural cavity, or lung, or peritoneal cavity, or externally.

Hydatids of the liver—physical signs: The liver is found enlarged, and usually irregularly so. The mass may point upward to the lung, or downward to the pelvis, or forward to the abdominal wall. If the prominence can be palpated, it is found to fluctuate, and the so-called *hydatid fremitus*, a sort of tremor of the cyst, is felt.

By aspiration, the typical fluid showing the presence of cholesterin and hooklets is obtained.

Diagnosis: This is positively made, with the above symptoms and physical signs, by the discovery of the *scolices* in the aspirated fluid.

Prognosis: If a diagnosis is made, and the tumor is accessible to operation, the prognosis is good. If untreated, the disease is apt to be fatal.

Hydatids of the liver—treatment: This is purely surgical. The cyst should be opened and the contents evacuated. Every bit of the contents, especially all the small "daughter-cysts," should be carefully removed to prevent recurrence. The cavity should be packed and allowed to granulate.

BILIARY CALCULI.

Gall-stones, although rare in childhood, are found from time to time. Probably their presence is often overlooked on account of their extreme rarity.

Etiology: They are caused by the precipitation from the bile of its solids. Cholesterin, bile-pigment, and lime are the main constituents. Probably catarrh of the bile-ducts enter into the etiology, by causing a desquamation of the epithelium.

Pathology: The gall-stones may be single or multiple. In children the latter condition is the commoner. They are usually small and faceted from mutual pressure. They are rather friable and of a brownish color. They are found usually in the gall-bladder, but may be found in any part of the hepatic ducts. The mucous membrane of such a gall-bladder, or of the ducts around a stone, is in a state of mild catarrhal inflammation. The gall-bladder may be dilated. Suppurative inflammation of the gall-bladder or of the ducts may be present.

Biliary calculi—symptoms: So-called *biliary colic* is the most characteristic symptom. This is due to the passage of a gall-stone from its place of rest, through the duct to the duodenum. The attack begins suddenly, with sharp agonizing pain in the right hypochondrium, making the child cry vigorously and roll around in the bed. The screaming is incessant, and nothing seems to relieve the pain. The skin is pale and clammy, and vomiting soon begins. After some few hours of this suffering the temperature rises, and a chill may accompany this rise. In highly neurotic children convulsions may take place. The pain may continue for a day or more, and then usually ceases as suddenly as it began. By the second day some jaundice is usually present, with bile-pigment in the urine, and later the stools may be clay-colored.

On *examination* there is some sensitiveness over the region of the liver. The liver itself may be slightly enlarged. The gall-bladder may be found distended.

The above is the history of an attack in which the gall-stone passes completely through the common duct and into the duodenum. Instead of complete expulsion, the stone may become *impacted* in the cystic, or common, duct.

If it *remains in the cystic duct*, the gall-bladder becomes painfully distended, and eventually the stone may be pushed

out; or the bile in the bladder may be absorbed and the bladder gradually contract and atrophy. In other cases the gall-bladder becomes the seat of an infectious inflammation, with formation of an abscess.

If the stone is *impacted in the common duct*, the pain gradually ceases, but the jaundice with white stools increases. Later the duct forms a dilatation around the stone, allowing the passage of bile around it, the jaundice disappears, and the bile again reaches the intestine. This impacted stone may thus produce no trouble; but there is always danger of the ducts being infected, causing a suppurative inflammation which spreads to the liver and forms multiple abscesses in that organ.

Biliary calculi—diagnosis: Sudden severe pain in the region of the liver, associated with jaundice and slight tenderness and enlargement of that organ, are the points for diagnosis. Ordinary intestinal colic, intussusception, and perforation of some abdominal viscus, must be thought of as causing similar attacks. *Renal colic* may simulate these cases closely. The fæces should be searched daily after such an attack, to endeavor to find a calculus.

Prognosis: The attack itself is rarely fatal. One attack is likely to be followed by others. Cases with persistent jaundice, or with infectious inflammation of the bile-passages, are very serious.

Biliary calculi—treatment: The pain should be relieved by hot baths and the hypodermatic use of morphine. Large quantities of the latter are often needed, and care should be taken to consider the fact that children bear opium badly. Hot stupes sprinkled with chloroform may be applied to the liver region. At times it may be necessary to administer an anæsthetic to control the pain.

After the attack of pain is over treatment should be directed to the prevention of further formation of gall-stones. For this, the *diet* should be regulated, sugars and fats being excluded, abundance of water should be drunk, and out-door exercises should be enforced. Doses of olive oil, of phosphate of sodium, and of other salines should be used from time to time.

Of late years the surgery of the gall-bladder and ducts has made great strides, and all chronic cases should be given the benefit of surgical advice.

DISEASES OF THE PANCREAS.

PANCREATIC CYST.

Diseases of the pancreas are rare under all circumstances, and especially among children. The only disease of clinical importance is *cyst*.

Etiology: This is due to blocking of the pancreatic duct and retention of secretion behind the point of obstruction. A larger or smaller cyst may result; only the larger ones, however, being recognized.

Pancreatic cyst—symptoms: There is nothing definite in the symptomatology. The presence of a fluctuating tumor in the epigastrium, with ill-defined signs of intestinal indigestion and with malnutrition, are suggestive of this condition. In some cases the urine may contain sugar. Exploratory puncture with a clean aspirating-needle, by withdrawing fluid having the digestive qualities of the pancreatic juice, is the only positive means of diagnosis.

Treatment: This is purely surgical, viz., incision and drainage.

DISEASES OF THE SPLEEN.

The **normal spleen** of a child lies at the left extremity of the diaphragm, and extends along the ninth, tenth, and eleventh ribs. It cannot be easily percussed or felt when of normal size. If *enlarged*, the lower edge can be easily palpated, especially on deep inspiration, extending below the free border of the ribs. If much enlarged, it may be felt extending well down toward the ileum and often the notch on the anterior edge can be easily made out.

The only *pathological change* that takes place in the spleen that is evident to us is its *enlargement*. This enlargement is always secondary to some general constitutional state, except in a few very rare cases of *primary splenic tumor*.

ENLARGEMENT OF THE SPLEEN.

The commonest of the *causes of acute enlargement* of the spleen are malaria, typhoid fever, and septicæmia. Any of the infectious diseases, however, may cause it to swell. Such swelling is due to congestion, and the spleen becomes very dark-red and soft. When the infection has disappeared, the spleen returns to normal.

There are also a number of *chronic conditions* in which the spleen becomes hypertrophied. In these cases the chronic congestion of the spleen is succeeded by a growth of new connective tissue, so that the spleen becomes harder and more fibrous than normal. Such diseases are tuberculosis, syphilis, leukæmia, Hodgkin's disease, amyloid degeneration, cirrhosis of the liver, chronic endocarditis and rachitis.

The other causes of enlarged spleen are *primary new growths.* Any of these are rare, but a few cases of sarcoma and of echinococcus have been reported.

Enlargement of the spleen—treatment: This is dependent on the original cause. In some of the chronic cases extirpation by surgery is advisable.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition: This is an acute inflammation of the serous membrane covering the abdominal viscera. The inflammation may be *local*, confined to some small area of the peritoneum, such as that covering one viscus; or *general*, when it spreads over the whole peritoneum. A localized peritonitis is liable to become general.

Etiology: Acute peritonitis occurs quite commonly among new-born infants, even being found in intra-uterine life. It is rarer after this until the child reaches about its fifth year. The causes in *early infancy* are such as take their origin from the umbilical cord and navel.

In *later childhood* the common causes are abdominal traumatism, surgical operations on the abdomen, appendicitis, exposure to cold and wet, intussusception, strangulated hernia,

rupture of cysts or abscesses into the peritoneal cavity, ulcers, ruptures or perforations of the stomach or intestines, inflammations of the uterus, tubes, or ovaries, rupture or inflammation of the bladder, inflammation or abscess of the abdominal lymphatic glands, and diseases of the vertebræ or pelvic bones. It may complicate rheumatism, erysipelas, chronic nephritis, or the infectious diseases. Finally, so-called idiopathic cases occur in which no cause can be discovered.

There are always found various *micro-organisms* which are the exciting causes of the peritonitis. The peritoneum is particularly sensitive to these. The staphylococci and streptococci are commonly found. The pneumococcus and bacillus coli communis are also frequently present.

Pathology: In the beginning the peritoneum is simply congested, its normal shining surface becomes dull, and the subperitoneal vessels are visibly enlarged. The surface becomes roughened, and an exudation of serum, or of serum and fibrin, or of pus, follows. Either form of exudate may be in excess, and very large quantities of any one of them may be present. Adjacent surfaces of inflamed peritoneum become loosely adherent, and small sacculated collections of the exudate may thus be formed. The intestinal walls are paralyzed, and the intestines are distended with gas.

If the disease lasts and recovery ensues, these adhesions become organized and permanent. The formation of these adhesions may be the means of saving the patient's life, by walling off the general peritoneal cavity and circumscribing the peritonitis.

Acute peritonitis—symptoms: Abdominal pain usually begins early. At first it is localized over the inflamed area, but soon spreads and becomes general even when the inflammation may be fairly circumscribed. The abdomen is very tender, and all motion, even that of breathing, increases the pain. On this account the respiration is almost entirely costal. There is marked tympanitic distention of the abdomen, and the patient lies on his back with the knees drawn up to relax the abdominal parietes as much as possible. Vomiting begins early in the disease and is quite intractable.

The bowels are usually constipated, not even much gas being passed, but in some cases diarrhœa may be present.

The temperature is moderately high with quite an irregular curve. The pulse is rapid and feeble and wiry. Respiration is rapid and superficial, the diaphragm being nearly immobile. The tongue is coated and the mouth dry. The face is drawn and shows the evidences of pain. The urine is scanty and may contain albumin.

All the cases increase in severity as the disease advances, and the fatal cases usually end at the expiration of a week or less. The mind is usually clear throughout.

Diagnosis: This disease may be confused with intestinal, renal, or hepatic colic; acute gastritis, or entero-colitis, and with intestinal obstruction. A little care will soon clear up the differential points.

It is not only necessary to diagnose the presence of peritonitis, but a careful attempt should be made to locate the point where the peritonitis began; in other words, to find the causative factor. The diagnosis of the cause of the peritonitis is of very great importance from the therapeutical standpoint.

Prognosis: Localized peritonitis has a much less grave prognosis than the generalized form. The latter is a very fatal disease. Seemingly very sick cases, however, do recover.

Acute peritonitis—treatment: If the cause of the attack can be found, it will influence the treatment markedly (mainly from a surgical standpoint). Where the case is seen early, and the evidence points to some localized area as the region of the inflammation, surgical interference should always be considered. An example is seen in the localized peritonitis accompanying appendicitis. If the appendix can be removed before the inflammation spreads to the general peritoneal cavity, the patient can usually be saved.

In some few cases recovery has been recorded following operation even after general peritonitis had set in. Abscesses in the peritoneal cavity; perforation of any viscus; and intestinal obstruction causing peritonitis are definite indications for operation.

The *medical treatment* consists of rest in bed and continuous cold applications to the abdomen. This may be accomplished by ice-bags or the cold coil. If for any reason the cold is very objectionable to the patient, *hot applications* may be substituted. Turpentine stupes—flannel cloths wrung out of hot water and sprinkled with turpentine—are also very good local applications.

The question of *diet* is of great importance on account of the incessant vomiting. Food is very likely to be immediately rejected. If any form can be retained, it must be predigested and given in very small quantities at a time. Peptonized milk, peptonoids, and koumyss are all allowable. At times gavage will assist in preventing vomiting. If the stomach rejects these, nutrient enemata of predigested foods must be used. Cracked ice and champagne by the mouth will usually be well retained, soothing the stomach and acting as a general stimulant.

If the peritonitis is recognized in the very beginning, a *saline purge*, as Rochelle salts or citrate of magnesium, in small doses every hour until the bowels move, will frequently help. It acts by depleting the walls of the intestine, and so may cut short the attack. Any time later the saline is useless, and will only irritate the stomach. From this time on opium should be used freely. There is probably no disease in which such large quantities can be safely and profitably used as in acute peritonitis. It is best given hypodermatically, and in doses enough, and repeated frequently enough, to stop the pain absolutely, and to paralyze the intestinal peristalsis. Begin with about $\frac{1}{20}$ of a grain of morphine for a five-year-old child, and repeat it in about two hours as conditions point the way. Atropine may be well combined with it, but care must be taken not to produce belladonna-poisoning. Gas may be removed from the bowels by the introduction of a long soft-rubber rectal tube. As the disease advances heart-stimulants will be required, and had probably best be given by the rectum or hypodermatically. Alcohol, digitalis, or strychnine may be used. Special symptoms are to be treated as they arise.

CHRONIC PERITONITIS.

Definition: This is a rare condition in children when compared with the tubercular variety; but, undoubtedly, cases of simple chronic peritonitis do occur.

Etiology: It may follow an attack of the acute variety, or be chronic from the outset. It may be the result of a chronic inflammation extending to the peritoneum covering some one of the abdominal viscera, the organ itself being inflamed. Trauma is given as a cause in many cases. In the largest number, however, no adequate exciting cause can be made out.

Pathology: There is a general thickening of the peritoneum, due to the growth of new connective tissue. There are frequent adhesions between adjacent portions of the peritoneum, and threads and membranes, due to stretched adhesions, are seen in different places. There is a good deal of serum, which may be slightly purulent, scattered among the coils of intestine.

Chronic peritonitis—symptoms: The symptoms begin very gradually, with some interference with the general health, slight digestive disturbances, and irregular colicky pains in the abdomen. The abdomen is distended, but is not tender; nor is steady pain a regular symptom. The bowels are apt to be constipated, but diarrhœa may occur. As the effusion takes place, the abdomen becomes more distended and the superficial veins become prominent. The appetite is fair, and the strength keeps up tolerably well. There is little or no fever, but the pulse gradually loses in strength.

If the case continues to advance, a gradual, slow exhaustion supervenes, from which the patient may die, or some intercurrent disease carries him off. If recovery is to ensue, the fluid is gradually absorbed, the strength returns, and the various organs take on their normal functions.

Chronic peritonitis—physical signs: The *tympanitic distention* of the abdomen, together with a feeling as if the bowels might be matted together; and, if fluid is present, *dulness* in the dependent parts of the abdomen, accompanied by a *fluid wave*, are the physical signs to be expected.

Diagnosis: If fluid is present, various causes for this, such

as endocarditis, nephritis, and cirrhosis, must be eliminated. The difficulty lies in differentiating this condition from *tubercular peritonitis.* The main points are the absence of any evidences of tuberculosis elsewhere, and the more marked constitutional symptoms present in the tubercular variety.

Prognosis: Recovery is rather to be expected, although some changes of a permanent nature are usually left behind, which may interfere with the functions of the intestines. Many cases gradually grow worse, and die emaciated and exhausted.

Chronic peritonitis—treatment: The child should be kept at rest, with plenty of fresh air and sunshine, and under the best hygienic surroundings. The diet should be easily digestible and highly nutritious. Milk, eggs, and meat should be the staples. Carbohydrates had best be limited. Hot applications, or chloroform stupes, or some irritant liniment, should be applied to the abdomen for some time each day. The syrup of the iodide of iron given internally seems a good tonic treatment to be used. If fluid is present, occasional saline purges or diuretics should be used. If it shows no tendency to absorption, the abdomen should be tapped. This may be repeated if the fluid reaccumulates. If, after repeated aspirations, the fluid continually returns, laparotomy is justifiable.

TUBERCULAR PERITONITIS.

Occurrence: The peritoneum is frequently infected by tuberculosis in childhood. The disease may be primary in the peritoneum, and at autopsy no other portion of the body may be found involved; but it is more frequently secondary to tuberculosis elsewhere.

Etiology: The *tubercle bacillus* growing in the peritoneum is the only active cause of the disease. The bacilli may be carried there by the blood or lymph without involving other organs; but usually the peritoneum is secondarily infected from the lungs, intestines, mesenteric, or other lymph-glands.

Pathology: The peritoneum may contain few or numerous *small miliary tubercles* scattered irregularly over its surface,

and no other changes be present. In some cases a serous exudation accompanies this.

In other cases the tubercles may be massed together into *large nodules* or *plates*. Many of these may be *caseating*. With these are numerous *adhesions* between adjacent portions of the intestines, and a variable quantity of sero-fibrinous or purulent exudation. This effusion may be free or encapsulated, forming a cystic tumor.

In *advanced cases ulceration* of the tubercular nodules occurs, with formation of small abscesses and intestinal perforation.

All these lesions may be mixed together in any one case, but usually one or the other pathological variety predominates. In some cases the ascites is the main lesion, in others the adhesions, and in others the ulcerations.

Tubercular peritonitis—symptoms: Tubercular peritonitis may run its course acutely or chronically; or the same case at different times may show decided differences in the course of its symptoms. The tendency, however, is to be chronic.

The *ascitic cases* are usually the more rapid. There are irregular fever with moderate rises, symptoms of indigestion, diarrhœa, and usually some abdominal pain or discomfort. The abdomen enlarges and shows the presence of some fluid, free or encapsulated, and the intestines give a feeling as of being matted together. The omentum may be thickened, and give a sensation of an indefinite tumor extending across the upper part of the abdomen. Tenderness is present, but is not marked. The child feels sick, is weak and prostrated, and loses flesh regularly. Vomiting is seldom present.

In the *variety with adhesions*, all the symptoms are more chronic and gradual in their development, but otherwise do not differ much from the above.

In the *variety with caseating nodules* and *ulceration*, there are more marked and more serious constitutional symptoms. The temperature is higher, and often assumes a hectic course. Chills and evidences of septic absorption are present. Diarrhœa is marked, and blood and necrotic tissue are present in the stools. The abdomen presents irregular areas of dulness and tympanitic resonance. Tumors of irregular size can be

felt on abdominal palpation. Encapsulated fluid can be often made out. The abdominal outlines are decidedly asymmetrical. In the neighborhood of the umbilicus, the abdominal wall may be affected by contiguity, and an abscess burst through and discharge itself.

In *all* varieties the disease steadily progresses, the patient growing weaker and weaker, and the signs of the disease increasing, until death supervenes from exhaustion or tuberculosis elsewhere. The cases last from two to twelve months, the longer ones being of the adhesive variety.

Diagnosis: The ascites of tubercular peritonitis must be distinguished from that due to cirrhosis of the liver, to chronic endocarditis, to chronic nephritis, and to simple chronic peritonitis. The last is the hardest to differentiate. The points in favor of tubercular peritonitis are the family history, the presence of any evidences of tuberculosis elsewhere, and the more marked constitutional symptoms. If there is no ascites, the presence of evidences of matted intestines, or of irregular masses scattered over the abdomen, is in favor of tubercular peritonitis. In many cases, however, it will be almost impossible to do more than to suspect the presence of the disease.

Prognosis: Many cases of the ascitic and adhesive variety recover, some without, and more with, treatment. The ulcerative form is quite fatal. The disease is very serious.

Tubercular peritonitis—treatment: The constitutional treatment of the patient is that of general tuberculosis: rich, nourishing, easily digested diet, attention to the functions of the stomach and intestines, abundant fresh air, and the administration of creosote. Local applications are almost useless. Aspiration is of little value.

After many trials it has been found that *laparotomy*, with evacuation of any fluid present, and washing out of the whole peritoneal cavity with sterile water, will cure a large percentage of the cases. Any variety except the ulcerative is amenable to this treatment. Tuberculosis elsewhere in the body is not a contraindication. The reason why this operation is curative is not yet decided; in fact, no satisfactory explanation has been offered.

CHAPTER VII.

DISORDERS OF NUTRITION.

MALNUTRITION.

THIS, in one form or another, is a very common occurrence in children.

Malnutrition—etiology: The tendency to malnutrition may be inherited from delicate or unhealthy parents. Persons of highly neurotic constitution are often the parents of these children. Infants born before term, or of unusually light weight, are apt to be affected. But commoner yet, it is seen in children who in early infancy have been fed on diet entirely unsuitable to their age. Bad hygienic surroundings, impure air, overheated houses, and lack of proper cleanliness are also causes. Other cases follow and seem the result of precedent acute disease, as the infections, bronchopneumonia, or some form of intestinal disease. In many of the cases, two or more of the causative factors are active at the same time.

Pathology: Nothing further is found than an anæmia of all the tissues, together with a flabby condition of all the organs.

Malnutrition—symptoms: There is a great difference in the weight of these infants as compared with a normal child of the same age. Not only do they weigh less, but their gain in weight is decidedly slower. The child also grows much more slowly, and is much shorter than he should be. The muscles are flabby and feeble, and sitting up, creeping, and walking are very late in being attempted. Dentition may also be delayed. The child is anæmic, and the circulation poor. The various lymph-glands of the body enlarge easily under slight irritation. The digestive functions are poorly performed, and great care is necessary to prevent digestive disorders from developing. The resistance of these children to

any acute disease is very small; not only do they seem prone to these diseases, but if the disease once develops they have little power to withstand it.

If these children grow up they usually are continuously cursed by the presence of these abnormal conditions. They frequently have in addition many nervous symptoms, and may develop one or other of the functional neuroses. Some never outgrow the condition, and in adult life exhibit similar symptoms.

Diagnosis: In diagnosing such a disorder great care should be taken to rule out all organic disease of every kind. There are many latent states any one of which may produce just such a set of symptoms. Tuberculosis, syphilis, rickets, malaria, malignant disease, blood-diseases, and actual disease of any of the important organs must be excluded. Do not make the diagnosis of simple malnutrition until every one of the possible causes is eliminated.

Prognosis: This depends on the ability to find and remove the cause. The cases with a bad heredity are the least favorable for cure. When due to bad feeding, or bad hygienic surroundings, or even after some acute disease, they can usually be cured.

Malnutrition—treatment: This rests on the proper diagnosis of the causative factor of the condition. If it is decided that the food is at fault, steps should be taken to remedy this. In very young children a wet-nurse will often be necessary. If this is out of the question, artificial feeding, begun with a dilute modified milk which is gradually changed to one with higher percentages of constituents, is the best method.

In children on mixed diet great care should be taken to have them fed only on such food as can be easily digested by the particular child; and on one that is highly nutritious. We must remember that often in such children food suitable for the average child of the same age is often altogether unsuitable. They often require the regular diet of a much younger child. In every way the problem of feeding these children is a difficult one, and will require great intelligence and patience on the part of both physician and parent.

If the sanitary surroundings are bad, these must be changed.

Abundance of fresh air and the avoidance of overheated living- and sleeping-rooms should be insisted on. Daily cool baths stimulate the various functions and prevent "catching cold" by accustoming the child to changes of temperature. After the bath thorough rubbing of the surface before a warm fire should be indulged in. Regular habits of eating and sleeping should be enforced.

In children able to walk outdoor exercises are to be encouraged. Habits of reading and studying and other sedentary occupations should be supervised.

As *internal treatment*, nux vomica, iron, cod-liver oil, and wines are our main assistants. Moderation should be used in the amount of each given. Use doses only large enough to be assimilated easily by a child of the age at which it is given. This is of special importance as regards iron and cod-liver oil.

MARASMUS.

Definition: This common condition of wasting is also known as athrepsia, or infantile atrophy. Excessive emaciation is an accompaniment of many diseases of infancy, particularly of tuberculosis and of those of the digestive tract; but in this condition of malnutrition it is understood that none of these well-known causative factors is present. In other words, marasmus is wasting without recognizable organic lesion.

Marasmus—etiology: Marasmus is a disease of the first year of life, but cases are seen with some frequency even in the second year. It is seen in the large majority of cases in artificially fed infants, but at times a breast-fed baby is the victim. It occurs with special frequency among the poor and in institutions. It might almost be considered an institutional disease.

Probably three factors enter into the causation of these cases: an *inherited delicate constitution*, *improper methods of feeding*, and *unhygienic surroundings*. There is an inability to digest and assimilate the food given, which food is usually decidedly improper; but in many cases is such as properly nourishes the average child of the same age. In fact, the

two other elements, and possibly something further which is not yet recognized, enter into the case as well as the feeding.

Pathology: After death the results of the marasmus are found rather than any changes which may be looked on as causes. The muscles are atrophied, all fat has disappeared from the body, and fatty degeneration of the kidneys and liver is often present. Some hypostatic pneumonia is frequently found along the posterior borders of the lungs. The stomach shows no special changes. The solitary and agminated follicles of the intestines are usually enlarged and may be slightly pigmented. The mesenteric glands may likewise be enlarged. So many of these cases die of some intercurrent disease that other lesions due to those diseases are present, but in a pure case of marasmus nothing further is found.

Marasmus—symptoms: The disease begins gradually and progresses gradually. Loss of weight and emaciation are the characteristic features. The regular infantile plumpness gradually disappears, the muscles grow soft and atrophy, the skin becomes wrinkled and dry, the face grows thin, pinched, and pale, the anterior fontanelle becomes depressed, while the abdomen grows prominent and distended. Eventually there seems nothing left but the skeleton covered by skin. There is marked anæmia, the hæmoglobin often being only one-third of the normal. The temperature is usually subnormal, even when taken by rectum. The pulse is rapid and feeble, and the respirations inefficient. The tongue is coated, and the mouth is frequently the seat of thrush. The appetite is usually voracious, the call of the starved tissues for nutriment being strong and constant. The taking of food does not seem to satisfy this hunger, and naturally so, as the tissues do not receive it. Vomiting is rather frequent from the constant attempts made to satisfy the appetite. The stools may be fairly normal, but usually contain undigested food. They may be green, and of offensive odor, and are usually large in amount from the small absorption of the food. The buttocks are regularly excoriated and red, and bedsores develop on the occiput, sacrum, and heels.

The child lies quietly, dozing a good deal of the time, and constantly sucking the fingers and hands. Restless sleep and

fretfulness may be present, especially when the child is disturbed. Nervous symptoms, twitching, rolling of the eyeballs, and convulsions are frequently present. The neck is often retracted. The disease advances steadily to a fatal issue, which may result from exhaustion, from convulsions, or frequently from some intercurrent disease.

Marasmus—diagnosis: The diagnosis rests on the exclusion of all forms of organic disease. The main diseases under this category are tuberculosis, congenital syphilis, and chronic digestive disturbances. Tuberculosis shows the physical signs in the lungs, and is associated with fever. The hypostatic pneumonia of marasmus may, however, give physical signs that are confusing. In inherited syphilis the child has snuffles, and has a dry skin showing some form of rash. The mouth and anus show mucous patches. In digestive disorders the history points to the diagnosis.

Prognosis: The prognosis is very bad in young infants, in institutions, and among the poor and ignorant. Even under the best circumstances it is often difficult to start the nutrition on the up grade. Return to normal is complete when recovery does occur.

Marasmus—treatment: Attention should be paid to the three etiological factors. As yet we have no way of improving the heredity, but the sanitary surroundings can be changed for the better. Plenty of fresh air should be given all infants, and especially should this be so in all institutions.

The question of diet, the third factor, is of vast importance. Wet-nursing will often be the only means of safely feeding these infants. If this is impossible, modified cow's milk given in small quantities and regularly is the best food. We must begin with a dilute milk for the age, and gradually increase the ingredients with the child's ability to digest them. Peptonized milk is of special value in many of these cases.

The child should be bathed regularly, and the bath finished with a cold douche to stimulate the respiration. Thrush, intertrigo, and bedsores must be treated in the ordinary way. Cod-liver oil is much used in this condition; but its utility depends on its absorption, and in very many of the cases no absorption takes place. The children should be kept warm,

especially when the temperature is subnormal. Special care should be used to prevent them from contracting any acute disease, as such diseases are very fatal.

SCORBUTUS.

Definition: This is commonly called *scurvy*. It is a constitutional disease, which in infants, until recent years, has often been called acute rickets, its exact classification being unrecognized.

Etiology: "Infantile scurvy follows the prolonged employment of some diet unsuitable to the individual child." While no one particular diet seems to be always at fault, in a general way "the farther a food is removed from the natural food of a child, the more likely is its use to be followed by the development of scurvy." The lack of the quality of *freshness* in the food seems to be the most marked single factor. The proprietary foods, condensed milk, and sterilized milk seem to have the most cases assigned to them as a cause. A few cases fed on breast-milk, or on raw cow's milk, or on table diet, are reported.

The disease occurs usually during the first two years of life. The largest proportion of the cases are seen in the better walks of life. It is not any more common in the city than in the country.

Pathology: So few cases of scorbutus die that opportunity for pathological investigation is rare. The changes that are found are those due to *hemorrhage*. In an affected limb there will be present a large subperiosteal hæmatoma. The periosteum will be stripped from the bone, and in bad cases detachment of the epiphysis may be present. The bone, especially in the neighborhood of the epiphysis, will be quite congested.

Scorbutus—symptoms: The patients are apt to be anæmic and show signs of malnutrition; but these signs may be evident only to a skilled observer, a layman considering the child well developed. Scurvy frequently develops in a rickety child, although there is no relation between the diseases.

Usually the first symptoms that definitely point to scurvy are *pain* and *tenderness*. The pain may be present when the

child is at rest, but usually is evident only on moving or handling the child. This tenderness may be so great as to cause the child to scream if anyone approaches him, or if the least movement is made that shakes him. On searching for the situation of the pain, it will usually be located in the *limbs.* Another symptom, which is a direct result of this tenderness, is a *pseudo-* or *voluntary paralysis.* The child holds the affected limb immobile, in order to prevent the pain which results from motion; the limbs are apt to be kept in a flexed position.

If now the extremities are carefully examined, there will usually be found a more or less marked *fusiform swelling* of one or more of the limbs. These swellings, which are due to the subperiosteal hemorrhages, will usually be found involving the shafts of the long bones, more particularly the femur, tibia, or humerus, and extending into the epiphyseal area. In some cases the hemorrhages are confined to the epiphyses, and in these the case seems more like a joint-involvement, and is very likely to be mistaken for rheumatism. There may be some redness around these joints, but it is not regularly present.

In the great majority of cases the *gums* are affected at the same time. They are swollen, spongy, and bleed easily. Ulcerative stomatitis may develop from these. These signs are more marked in cases when the teeth have erupted, but at times are seen in infants with no teeth. *Purpuric* and *ecchymotic spots* are fairly frequent. They may occur anywhere on the body, but are quite regularly seen about the eyes. Hemorrhages from the mucous membranes may be present, such as those of the stomach, intestines, or nose. There is no regular fever in this disease, but slight elevations of temperature may occur. *Albuminuria* at times is present.

Cases are seen of all degrees of severity, from mild attacks of tender joints to very marked cases with hemorrhagic gingivitis and large subperiosteal hemorrhages.

Diagnosis: The three conditions most likely to be confused with scurvy are *rheumatism*, *paralysis*, and *osteo-sarcoma.* If scurvy is always kept in mind, it will be fairly easy to make the differential diagnosis. The prompt improvement on

proper treatment offers a therapeutical proof of the diagnosis in cases of doubt.

Prognosis: This is good. Recovery will be rapid under proper treatment. Scurvy occurring in a case of severe marasmus, or other condition of exhaustion, is naturally a more serious disease.

Scorbutus—treatment: The child should be kept as quiet as possible to protect it from pain. If the epiphysis is separated, the limb should be put up in splints. An antiseptic mouth-wash may be used with advantage.

A change in the diet is imperative. In a general way it may be said, stop all proprietary foods, condensed milk, and sterilized milk. Put the child on raw cow's milk and orange-juice. Beef-juice, and in older children potato, are also useful adjuncts.

The symptoms may be confidently expected to improve greatly in three or four days, and cure may be expected in three to four weeks. After improvement begins steps should be taken to correct the malnutrition from which the child is usually suffering. This can be best accomplished by fresh air, iron, and cod-liver oil.

RACHITIS.

Rickets is a constitutional disease with its main pathological lesions located in the bones. It must be remembered, though, that almost all the organs of the body take part in the nutritional changes.

Etiology: Rickets is far more frequent in the cities, among the ill-fed and badly housed. These conditions, together with the diet, are the actual causes. Prolonged feeding on a diet which does not contain all the proximate principles of milk in comparatively proper quantities is regularly a cause. The fat first and the proteids second are usually deficient. Condensed milk and the proprietary foods fulfil this condition of lack of fat and proteids, and the large proportion of cases will be found to have been fed on one or the other of these foods. Prolonged nursing at the breast, as when it is continued into the second year, and even breast-feeding

FIG. 1.

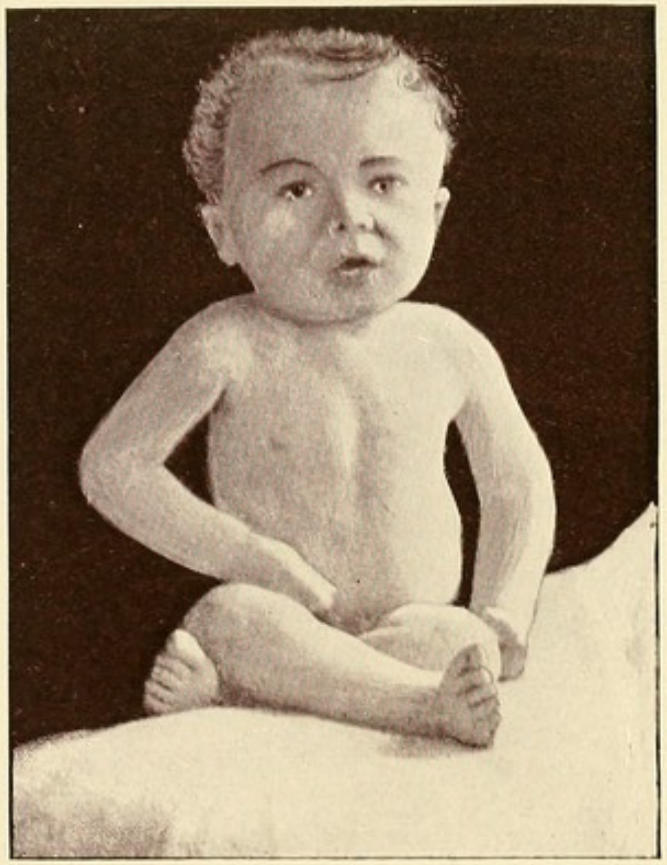


FIG. 2.

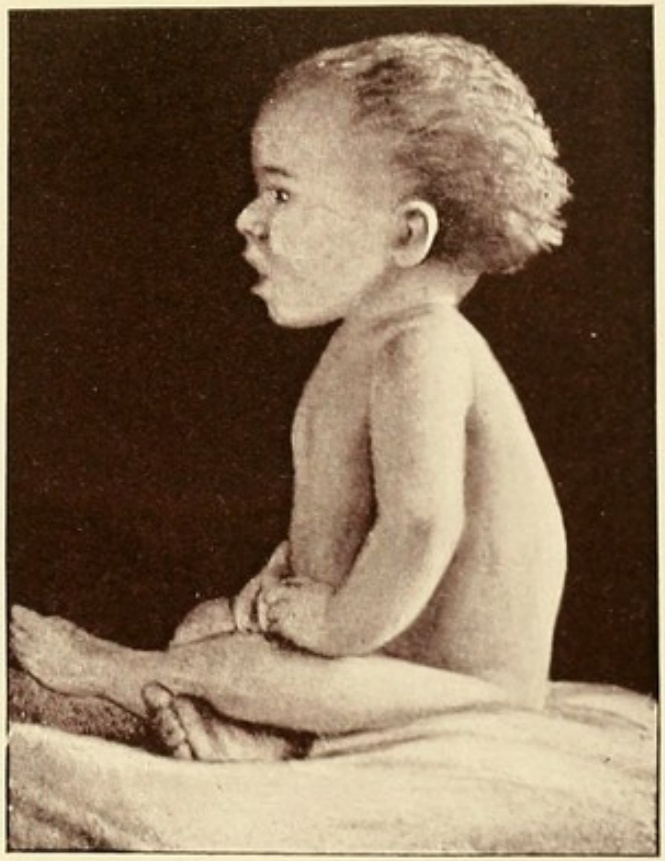


Fig. 1.—Rhachitis. Attitude in sitting; one hand raised to exhibit swelling at the wrist. (Williams.)

Fig. 2.—Rhachitis in moderate degree in a boy aged 15 months; showing backward excurvation of the spine. (Williams.)

when the mother's milk is deficient in quality, will produce rickets. Two nationalities in this country, the negroes and Italians, seem especially prone to the disease. The age of greatest susceptibility seems to be from the sixth month to the end of the second year.

Pathology: Although all the tissues of the body are involved by the nutritional changes, still the lesions evident to the naked eye are mostly in the bones. There is a general anæmia of the voluntary muscles, and of those of the heart. The lungs show the furrows from the depressed thorax. The stomach and intestines are dilated and show some evidences of chronic catarrhal inflammation. The spleen is enlarged. The liver and kidneys are negative. The lymphatic glands in different parts of the body are apt to be enlarged.

In the *bones* are the constant signs of the disease. These changes take place in the regions of the bones where ossification is in progress. These regions are the cartilage between the epiphysis and the shaft, underneath the periosteum, and in the flat bones about the centres of ossification. The bones grow by the proliferation of the cartilage-cells in these locations, which cartilage-cells have lime salts deposited in them, thus undergoing ossification. In rickets the proliferation of these cartilage-cells is stimulated to undue activity, while the deposit of lime salts in the same areas does not keep pace with the cartilaginous growth. The result of these abnormal changes is to produce a marked enlargement at the epiphyses of the long bones and at the centres of ossification of the flat bones. These bones become very soft and flexible owing to the deficiency of lime salts in them. The normal two-thirds mineral matter is reduced to one-third. This softness and flexibility explain the *rachitic deformities*, which are especially marked where the bones are subject to muscular action or to pressure, as in the femur, tibia, radius, ulna, or the ribs.

A *section* through the epiphyseal junction of a rachitic bone shows a very vascular, bluish-colored condition, which is softer than normal when cut. In the shaft next to the periosteum the bone is soft and thickened, but deeper it is hard. Section through thickened masses on the flat bones shows a spongy vascular substance which is soft enough to be indented easily.

Microscopical examination shows a marked increase of new cartilage-cells and increased vascularity of the proliferating zone. The areas which should be calcified show large quantities of cartilaginous tissue instead. The under layer of the periosteum is very vascular, and again there is a great excess of uncalcified cartilage. In the flat bones the bony trabeculæ are eroded, and their places taken by newly formed minute blood-vessels.

When the rachitic process ceases and recovery begins, this excessive proliferation stops. Calcification and ossification of these tissues take place, the enlargements due to the hyperplasia are absorbed, and the bone returns to a normal condition save for any deformities that may have resulted during the activity of the rachitic process.

Rachitis—symptoms: Rickets is a slow disease, with a very gradual onset and progression. It is difficult to say when the disease begins, as the early symptoms are not marked enough to attract much attention. A fully developed case of the disease is easily recognized; but it is of more practical value to be able to diagnose the beginning of rickets, so as to prevent its further development.

At the first, we may expect to find some *anæmia*, marked sweating around the head and neck, especially during sleep, and *slight beading of the ribs*. The baby is restless in its sleep and is usually constipated. The hair will frequently be rubbed off the occiput by the continual rolling of the head in the pillow.

As the disease advances the bony changes become more prominent, although the other symptoms still continue. The *beading of the ribs*, the so-called "rachitic rosary," increases until the little lumps are evident to the eye through the skin. In the early stages they are evident only to palpation. The "beads" are due to overgrowth of cartilage at the junction of the ribs with the costal cartilages. They are found at the end of each rib, the row thus made running downward and outward to the costal margin. The same beading is found on the inner side of the thorax, but naturally only shows in this situation post mortem. The atmospheric pressure exerted on these softened ribs causes in advanced cases

a marked *depression of the thorax* in a line parallel with and on each side of the sternum, and just along the course of these beads. A second depression is also frequently present extending in a horizontal direction around the lower portion of the thorax, and is probably due to traction of the diaphragm on the lower ribs. The sternum is apt to be protruded, or at times depressed, by this same action of the atmospheric pressure, causing the deformity known as *pigeon-breast* or *funnel-breast.*

The *vertebræ* likewise are partially softened, and the weight of the head and shoulders on these causes a posterior or lateral *bending of the spinal column.* The *kyphosis,* or *scoliosis,* so produced, forms a long, regular curve, no sharp angles being found in rachitic spines. In the early stages these curvatures will disappear when the child lies down or is suspended, but in the long-standing cases the deformity becomes permanent.

The changes in the *cranium* are well marked and characteristic. The head appears large and square, the forehead broad and projecting, the top is flattened, and the suggestion of two furrows crossing each other at the anterior fontanelle at right angles is often present. These appearances are due to the thickened masses of bone, called "*bosses,*" which exist at the frontal and parietal eminences. These bosses often grow quite thick and prominent.

In the occipito-parietal regions are frequently found soft spots in the bones. This condition is known as *craniotabes. On pressure with the finger* these small areas dent in, but spring out again when the pressure is removed. Craniotabes is seen in syphilis as well as in rickets.

The sutures and fontanelles are very late in closing; often the anterior fontanelle will be open at the end of the second or even the third year. The veins of the scalp seem large, and are plainly outlined through the thin skin.

The teeth are cut quite late, are often irregular in the order of their appearance, and are subject to early decay. The various disturbances connected with dentition are more apt to be seen in rachitic children than in normal ones.

Changes in the *long bones of the limbs* are seen early and

constantly. The earliest is an enlarged, rounded knob, found at the epiphyseal junctions. The *wrists*, *ankles*, and *knees* show this change most commonly and in the order named. Other joints may be affected in the most severe cases. Later the long bones become bent into abnormal curves. These bendings are most marked in the leg, thigh, and forearm, but the upper arm may also be affected.

In the forearm the bones are usually bowed backward, and in the upper arm outward. It is in the *legs* though that the deformities are most marked. The usual variety is bowing outward of the tibiæ, and, in marked cases, of the femora also, producing the condition known as *bow-legs* or *genu varum*. In these patients, when the feet are put together, the knees are far apart. In others the opposite condition of knock-knee, or *genu valgum*, is present. In these the inner condyles of the femur are hypertrophied, so that when the knees are put together the feet are far apart, the legs making an obtuse angle with the thighs. In very severe cases the rachitic softening is so marked that irregular and very distressing deformities in the long bones are produced.

In the *pelvic bones* rickets causes certain changes that are of importance only from an obstetrical standpoint, since the deformity may interfere with the passage of the child through the pelvis. The usual deformity of rickets is a decided shortening of the antero-posterior diameter of the pelvis, from pushing forward of the sacrum.

The *ligaments* about all the joints are relaxed and weakened, thus assisting in the production of the deformities. The *muscles* also are flabby, small, and feeble, so that sitting and standing are difficult for these children. Walking is always learned late, and at times they are first brought to the physician to know why they do not walk, the mother dreading paralysis.

Children with rickets may be either fat and seemingly well nourished, or thin and suffering from malnutrition. Almost always they are *anæmic*.

The *abdomen* is *enlarged* and *tympanitic*, for which there are two probable reasons: the pressing downward of the diaphragm from the diminished chest-capacity, and the disten-

tion of the stomach and intestines from the accompanying chronic indigestion.

The pulse and temperature are about normal. A bruit may often be heard over the anterior fontanelle, but is of no special significance. The urine is negative.

Rachitic children are quite prone to *catarrhal inflammations* of the gastro-intestinal tract or of the respiratory system. The *reflex excitability* of their nervous systems is highly exaggerated, and laryngismus stridulus, tetany, and general convulsions are frequent.

The *course* of rickets is chronic, and the disease usually lasts for one or two years. Spontaneous recovery regularly occurs, as the child is put on a mixed and nourishing diet.

Diagnosis: In a developed case the diagnosis is not difficult, and hydrocephalus is the only disease with which it may be confused. A careful examination of the head, and the presence of *rickety changes elsewhere*, are the deciding points.

In mild cases careful examination for beginning changes in the bones, at the epiphyses and costal cartilages, will usually settle the diagnosis. Any of the bony signs are sufficient to establish the presence of rachitis, when the child is supposed to be suffering only from anæmia, debility, or even from paralysis.

Prognosis: The disease is self-limited, and will usually recover spontaneously when the diet becomes such as to furnish the proper nutrition. Rachitis is seldom fatal in itself, but from its tendency to act as a predisposing cause of gastro-intestinal, respiratory, and nervous diseases, it is partially responsible for much infantile mortality.

The bony deformities which have taken place during the disease are, as a rule, permanent throughout life.

Rachitis—treatment: Proper attention to the hygienic surroundings and care with the food are the two general points to be followed both in the prevention and in the treatment of rachitis.

The children should be kept in cool, dry, well-ventilated rooms. They should be given as much out-of-door life as possible, and particularly plenty of sunshine. Each day they should be given a cool bath to stimulate the respiration and

circulation, and to accustom the system to changes in temperature, and thus do away with the tendency to "catching cold."

The *diet* should be made to conform as nearly as possible to the normal for a child of the same age. Proprietary foods and condensed milk should especially be avoided. An abundance of fats and proteids should be given, while the carbohydrates should be diminished. Cream, beef-juice, and scraped beef fulfil these conditions admirably.

As regards *drugs*, three are used quite regularly, and all rationally. They are *cod-liver oil*, *phosphorus*, and *lime*. *Cod-liver oil* is given more as a fat food than a drug, in just sufficient dosage to be absorbed easily and not to upset the digestion. *Phosphorus* is given in doses of $\frac{1}{200}$ to $\frac{1}{100}$ of a grain three times a day. Thompson's solution, containing one-twentieth of a grain to the drachm, is the easiest way of giving it. *Lime* is best given as the *hypophosphite* of *calcium*. If the child is anæmic, iron as the syrup of the iodide is useful.

During the active stage of the disease attention should be given to the prevention of bony deformities as far as possible, by keeping the child from making too much mechanical exertion. *Light supports* and *braces* may often be used with advantage. If marked deformities are present after the cure of the disease, orthopædic apparatus may be tried, but osteotomy will usually be necessary to straighten the bones.

DIABETES MELLITUS.

Diabetes mellitus: This is a rare disease in children, and is probably as well grouped under the errors of nutrition as elsewhere, in the present state of our knowledge. It is a far more serious disease in childhood than in adults, its seriousness being inversely to the age.

Etiology: It is commoner in girls than in boys, and there is an hereditary element present in many of the cases. Otherwise nothing is known as to the causes.

Pathology: There are no recognized pathological facts connected with diabetes other than the occasional association of pancreatic disease and diabetes.

Diabetes mellitus—symptoms: The symptoms are frequent urination, which is found to be due to increased secretion of urine, great thirst, increased appetite, and wasting. On examining the urine the specific gravity is high, and sugar is present in larger or smaller quantities. As the case advances albumin may also be found in the urine. Loss of weight is very rapid, the skin becomes dry, and constipation is apt to be present. Furunculosis may exist as a complication. Dermatitis about the genitals may develop from the irritation of the glycosuric urine on the skin. This is commoner in girls. The disease progresses rapidly, and a fatal termination usually comes before six months from the outset. Diabetic coma is the usual cause of death. A few die of pneumonia or tuberculosis.

Diagnosis: This is based on the *urinary analysis*, a method of examination too seldom used in dealing with children.

Prognosis: This is very bad, almost surely fatal, in childhood.

Diabetes mellitus—treatment: This differs in no wise from that in adults. The *diet* should contain as little carbohydrates as possible, milk, meat, and eggs being the staples. Good hygiene should be insisted on—fresh air, exercise, and bathing. As *drugs*, codeine given regularly seems the most valuable one. Arsenic and the salicylate of sodium have some reputation as being of value.

ACUTE RHEUMATISM.

Nature: We are still in the dark as to the true nature of rheumatism, and, as there is evidence as to its being due to *faulty tissue-metamorphosis*, it is here classified mainly as a matter of expediency. In fact, rheumatism in childhood is more of a diathesis with local evidences breaking out in various portions of the body, than distinctly a joint-disease as it is looked on in adults. Cases are seen with the joint-symptoms so very slight that they are entirely overlooked.

Etiology: Nothing is as yet positively known on this point. There may be more than one exciting cause at work at the same time. There may be some poisonous substances, due to

faulty metamorphosis of the tissues, floating in the body-fluids, which irritate and inflame the serous membranes; or there may be specific micro-organisms at work as the cause. Hereditary influences play a certain part, and exposure to cold and damp undoubtedly acts as an assistant exciting cause.

Acute rheumatism—pathology: The inflamed membrane, whether it be in a joint, the pericardium, or the endocardium, is congested, and may show slight hemorrhagic spots. There is an increased secretion of serum accompanying. The tissues in the neighborhood are swollen by inflammatory effusion, which may contain fibrin, leucocytes, and at times red cells. The articular cartilages are also swollen and inflamed. Suppuration is very rare.

Acute rheumatism—symptoms: Rheumatism in childhood is seldom of the very acute type so often seen in adults, but is more apt to be subacute in its manifestations. The *general symptoms* are those of more or less fever, which is regularly lower than 102° F., and which runs an irregular and rather short course; and its accompaniments, malaise, anorexia, at times nausea and vomiting, some delirium, and a more or less free perspiration. At the same time one or more *joints* become painful and may appear a little swollen and red. These joint-symptoms may be so slight as to give no signs save a little tenderness on use. The urine is of high specific gravity and deposits urates on standing. The child may not feel sick enough to desire to go to bed. These attacks are often called "growing pains" by the family. The attack usually lasts only one or two weeks.

In other cases *no joint* may be affected, but the rheumatic poison centres itself in some one of the *muscles*. The muscles most commonly affected are the deltoid, the trapezius, and sternomastoid, those of the lumbar region, or the intercostals. In such cases there are scarcely any constitutional symptoms, the only sign being pain on motion in the affected muscles, and at times some continuous spasm of the same.

In other children, and unfortunately fairly often, with very slight joint or muscular signs, the poison especially attacks the *pericardium* or *endocardium*. This is, of course, far more serious. In fact, the heart is more frequently involved in

children than in adults, and at times the only way of feeling sure that the child has been suffering from rheumatism is by finding a murmur or a friction-rub. No case that is even suspicious of rheumatism should be allowed to go a day without a careful examination of the heart. Heart-complications, as a rule, give no special symptoms, except a slightly rapid or irregular heart-action, or a little precordial pain if pericarditis develops.

In other cases the rheumatic diathesis is evidenced by an outbreak of *chorea*, either with or without signs in the joints; and in others by an attack of *acute tonsillitis*, or by recurrent attacks of the same; in others *subcutaneous nodules* are found developing on almost any of the fibrous structures of the body. They are mainly about the joints and in the sheaths of tendons, and vary in size from a pinhead to an almond. They seem particularly associated with cardiac disease. They are not tender and not permanent.

Cutaneous eruptions, either erythematous or even purpuric, are often developed by the rheumatic diathesis. Rheumatism, no matter in what variety it manifests itself objectively, is always accompanied by a rapidly developing *anæmia*.

Acute rheumatism—diagnosis: The slightness of the joint-affection makes the diagnosis difficult at times. Previous similar attacks, or attacks of tonsillitis, or a rheumatic family-history assist. Signs of pericarditis, or of endocarditis, or rash, or fibrous nodules, or chorea, are the best evidence. Scurvy, rachitis, tuberculosis, and pyæmia are to be differentiated. By care no confusion need exist.

Acute rheumatism—prognosis: This is good in every way except as regards the development of heart-lesions. The heart-lesions become permanent; but the changes in the joints and muscles, the chorea, the fibrous nodules, and the tonsillitis are curable. A bad point in the prognosis is the tendency for further attacks to develop after the first.

Acute rheumatism—treatment: A child with any of the rheumatic manifestations should be an object of care for the prevention of heart-lesions. He should be kept at rest in bed, and in an equable temperature, till all signs have disappeared. After an attack is over he should be constantly

under supervision to prevent subsequent attacks. He should wear flannel underclothing and should be kept from all damp surroundings, and especially from wet feet.

During the attack the inflamed joint or muscle should have hot local applications made to it, and should be rubbed gently with chloroform liniment. *Internally* the salicylate of sodium or the oil of wintergreen should be given in fairly large doses. In some cases citrate of potassium in doses large enough to keep the urine alkaline should be used. It may be profitably combined with the salicylate. During the fever the best diet is milk; later it may be more varied. Water should be drunk freely. After the attack iron is indicated to combat the anæmia. The heart-lesions and chorea are treated as usual.

CHAPTER VIII.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE HEART AND PERICARDIUM.

CONGENITAL HEART-DISEASE.

All abnomalities found in the heart at birth are classified under this head. Some form of congenital heart-lesion is found with moderate frequency.

Congenital heart-disease—etiology: The causes are well grouped under three headings: 1. Non-closure of openings existing normally in fœtal life. These are the *foramen ovale* leading from the right to the left auricle, and the *ductus arteriosus* connecting the pulmonary artery with the descending aorta. 2. Actual developmental areas. These are partial or complete *absence of the septa* between the auricles or ventricles; *transposition of the great vessels; atresia* or absence of one of the valves. 3. *Endocarditis* occurring during fœtal life. This is capable of producing any of the lesions which follow endocarditis of post-natal life.

Pathology: The commonest lesions found are absence or defects of the ventricular septum, patent foramen ovale, stenosis of the pulmonary artery, persistent ductus arteriosus, and abnormalities in the origin of the great vessels. Many lesions are found connected with the valvular openings, both stenoses and insufficiencies, but in nowise differing from the same lesions as seen in ordinary endocarditis, except that the right side of the heart is more often involved than the left. A large number of the cases have more than one lesion existing at the same time, as if when a defect occurred a partial endocarditis was engrafted on it. The heart is ordinarily hypertrophied, as the result of the extra work thrown on it by these anomalies.

Congenital heart-disease—symptoms: Cyanosis and the presence of a murmur are the characteristic signs of this condition. The cyanosis is present continuously in most of the cases, but may be developed only by exertion, as crying, coughing, or vomiting. At any rate, it is always intensified by such action. The blueness is due to imperfect oxygenation of the blood, and is present in all the tissues. The mucous membranes, as the lips and tongue, appear very purplish.

The *murmur* heard over the heart is usually rough and loud, and systolic in time. It may be heard loudest at the base or apex. Murmurs synchronous with the other heart-sounds are rare. It is, however, impossible to diagnose the form of lesion by the variety of murmur, as many different pathological changes produce the same murmur.

There are cases of undoubted congenital heart-disease with absolutely *no murmur*, the cyanosis being the only symptom present.

Hemorrhages from the nose and elsewhere are fairly common. A result of the chronic congestion of the tissues is seen in the *clubbed fingers* and *toes* which these children present. The last phalanges become enlarged and thick, and the nails somewhat deformed. Dyspnœa, increased on exertion, is a marked symptom and is always troublesome. Dropsy in the limbs and serous membranes may supervene.

Diagnosis: In the presence of cyanosis and a murmur the diagnosis is made. In the absence of a murmur other causes, which are mainly pulmonary, of cyanosis must be excluded. Nothing more than a guess as to the variety of abnormality in the heart can be made.

Prognosis: This is bad. Most of the cases die before they are twenty, and during their life they are in a state of continual danger and discomfort. Their functions are badly performed, they are delicate, and bear badly any acute disease, particularly in the respiratory system. The more marked the cyanosis the worse the prognosis.

Congenital heart-disease—treatment: There is nothing to do for the abnormalities themselves. Good hygiene and prevention of colds and exertion are necessary. Otherwise we can only treat symptoms as they arise.

ACUTE PERICARDITIS.

Etiology: This is a quite rare disease in infancy, but is as common in older children as in adults. The principal cause is rheumatism. It may complicate pneumonia, or pleurisy, or scarlet fever. General sepsis may involve the pericardium. Direct injury and extension of the inflammation from a neighboring organ are frequent causes.

Pathology: The pericardium may be the seat of a dry inflammation in which the membrane is swollen and rough and coated with fibrin; or, more commonly, there is at the same time an effusion of serum in greater or less quantity in the sac. In children the serous variety is commoner than in adults. In other cases there is a distinct purulent character to this effusion. Rarely it is hemorrhagic. After recovery the fluid is absorbed, but the fibrin becomes organized, leaving adhesions between the two layers of pericardium.

Acute pericarditis—symptoms: The subjective symptoms are few and slight. There may be a little precordial pain, and some interference with the heart's action. If effusion embarrasses the heart, there may be some dyspnœa and a weak, irregular pulse. The other symptoms are those of the primary disease. Pericarditis lasts usually two or three weeks.

Acute pericarditis—physical signs: In *dry pericarditis* there is heard a superficial to-and-fro friction-sound directly over the heart and uninfluenced by inspiration. It is loudest in the third and fourth spaces just to the left of the sternum.

In the *serous variety* the area of cardiac dulness is increased in all directions, this dulness extending further to the left than the apex-beat. There is usually a small area of dulness extending to the right of the sternum. The heart-sounds are heard feebly, and a preceding friction-sound will disappear.

Diagnosis: This depends entirely on the physical examination of the heart. The use of an exploring-needle to corroborate the diagnosis of effusion and to decide on the kind of fluid present is always allowable.

Prognosis: The younger the child the more serious the

prognosis. While pericarditis is always a serious disease, and always adds a bad element to the condition which it complicates, still it is frequently recovered from. The presence of fluid aggravates the danger as it increases in quantity. Purulent pericarditis is the most dangerous form. The adhesions left after recovery are often the source later of serious trouble.

Acute pericarditis—treatment: Absolute rest in bed should be enforced from the first. *Locally*, hot applications, or mild counter-irritation, or an ice-bag with a layer of flannel between it and the skin, seem to have some power of limiting the inflammation.

The *diet* should be easily digestible and in moderate quantity, so as not to overload the stomach. The primary disease to which the pericarditis is secondary should be treated thoroughly. Probably the best *drug* to use is opium to quiet the patient and the heart. Aconite or digitalis may be used to slow an overacting heart. The latter is especially good if the heart's action becomes feeble.

If effusion is present, counter-irritation and some diuretic, as caffeine, will usually remove it. If it is persistent and interferes with the heart by its mechanical presence, *aspiration* done in the fifth intercostal space just to the left of the sternum is advisable. If the effusion is purulent, it is best removed by incision.

After the attack is over renewal of physical exertion should be gradual, and tonic treatment is indicated.

CHRONIC PERICARDITIS.

Pathology: This condition is mainly the result of one or more attacks of acute pericardial inflammation. At times it is tubercular.

The main lesions are a thickening of the pericardium and the presence of permanent connective-tissue adhesions between the visceral and parietal layers.

Symptoms: There are usually no subjective symptoms, unless some feeling of interference about the heart and dyspnœa on exertion.

Chronic pericarditis—physical signs: The heart is usually enlarged, the apex being displaced to the left and downward. The only characteristic sign is retraction of a small spot of the chest-wall during the systole of the heart. This is present, though, in but a small percentage of the cases.

Prognosis: The lesion is permanent, but is compatible with long life.

Chronic pericarditis—treatment: There is no special treatment for the disease. Good hygiene and attention to the body-functions comprise the therapeutics.

ACUTE ENDOCARDITIS.

Inflammation of the endocardium, especially that part entering into the formation of the valves, is quite as frequent in childhood as in adult life. In fœtal life usually the right heart is affected; after birth the left heart, as in adults.

Etiology: Rheumatism is the most frequent cause of the disease. Scarlet fever, septicæmia, and, less frequently, the other infectious diseases, are complicated by endocarditis.

Pathology: The endocardium is swollen only and remains smooth, or there is an extensive growth of new connective-tissue cells in its substance. This produces warty excrescences on the surface of the endocardium involved, and on these excrescences fibrin coagulates from the blood and organizes, exaggerating the changes. The valve becomes deformed, the chordæ tendineæ shortened, and, the new connective tissue being deficiently vascularized, tends to break down and ulcerate. Portions of these "vegetations," as they are called, may be broken off and carried by the blood-current to different parts of the body, where they lodge, and are called *emboli*. The vessel in which they are caught is stopped up, and the process is called *embolism*.

Acute endocarditis—symptoms: There may be no rational symptoms at all, or the endocarditis may show itself by some fever, malaise, disturbed heart-action, and restlessness. The pulse is usually rapid and not very strong, and there may be venous congestion of the body with dyspnœa. Delirium or stupor may be present.

At times the signs of an embolism in some portion of the body is the first symptom, suggesting a heart-lesion. If the embolus goes to the brain, hemiplegia is the usual result; to the lungs, sharp dyspnœa; to the kidneys, hæmaturia; to the liver and spleen, local pain and enlargement; to the mesentery, diarrhœa; to the limbs, obstructed circulation.

An attack of acute endocarditis lasts two or three weeks, and ends in recovery or death, or often becomes the starting-point for a chronic endocarditis.

Acute endocarditis—physical signs: Regular physical examination of the heart will often show the presence of an acute endocarditis during an attack of rheumatism or scarlet fever when no subjective symptoms of any kind are present. The physical signs depend on the changes in the valves—insufficiency, or roughening, or stenosis. According to the valve involved, and the lesion of this valve, will be the form of *murmur* present.

With *mitral regurgitation* we hear a *systolic murmur*, loudest over the apex; with *mitral stenosis*, a *presystolic murmur*, heard loudest over the mitral area; with *aortic regurgitation*, a *diastolic murmur*, heard over the aortic valve and carried down the sternum; with *aortic roughening*, a *systolic murmur*, heard loudest over the aortic valve. The valves of the right side, and combinations of valves, give their distinct murmurs also.

Diagnosis: This depends on the physical signs rather than on the symptoms. Pericarditis is differentiated by its friction-sound, which is superficial and not connected with valvular closure. Functional or anæmic murmurs are at times difficult to exclude. They may be heard over the apex, but are most frequent over the pulmonary valve—the second left interspace. The second pulmonic sound will not be accentuated in anæmic murmurs. An old valvular lesion being present and not before recognized, may raise the suspicion of an acute endocarditis when none exists.

Prognosis: Recovery without some change left behind in the valves is rare, but at times all murmurs and all evident signs of heart-lesion do disappear permanently. In some cases the valves remain without undergoing further change

throughout life. In others, slow changes continue and the case becomes one of chronic endocarditis. Recurrent attacks are common. Very few die in the acute stage.

Acute endocarditis—treatment: If any suspicion of a rheumatic diathesis is present, anti-rheumatic treatment should be instituted. The salicylates, combined with the citrate of potassium, in large enough doses to render the urine alkaline, is the best form to give this. Absolute rest in bed is of primary importance. If the heart's action is exaggerated and tumultuous, aconite or opium is indicated to quiet it. If the heart's action is feeble and rapid, digitalis is the best drug.

After the acute stage has passed, rest should be insisted on for some time, and exertion should be resumed very gradually. Iron and tonics are to be used freely during this stage. Efforts should be made to prevent subsequent attacks of rheumatism in these patients.

MALIGNANT ENDOCARDITIS.

This is often called **ulcerative endocarditis**, and in childhood occurs most always after the tenth year. Hence it differs in no respect from the same condition when seen in adults.

Etiology: The disease is a *cardiac sepsis*, combined with an inflammation of the endocardium. It occurs with rheumatism, pneumonia, erysipelas, scarlet fever, gonorrhœa, and septicæmia. Streptococci, staphylococci, and pneumococci are found in the endocardium. Probably, it is never a primary condition.

Pathology: The endocardium is swollen, infiltrated with round cells, and often ulcerated. The surface is coated, in patches, with a thin or thick layer of fibrin and micrococci. Vegetations and ulcerations form in the cavities and on the valves. Portions of these vegetations being detached are carried by the blood to distant parts of the body and lodge as *emboli*, causing not only the mechanical results of embolism, but setting up an infectious inflammation at the same spot.

Malignant endocarditis—symptoms: The disease is very irregular in its symptomatology, and really presents nothing characteristic. The symptoms are those of a *general septicæ-*

mia. Fever of an irregular variety and often quite high, accompanied by an occasional chill, is regularly present. Frequent sweats, marked prostration, anorexia, vomiting, and diarrhœa, are usually found. The so-called typhoid state develops rather rapidly, with dry tongue, sordes on the lips, rapid emaciation and alternating stupor and delirium. Petechial eruptions and the signs of embolism in various parts of the body may exist. The heart and pulse may not be much disturbed, or may be rapid and feeble, or may be irregular. Dyspnœa, more or less marked, may supervene. The disease lasts from a week to ten days in the more rapid cases, to a month in the slower ones. Death is usually due to exhaustion.

The **physical signs** of malignant endocarditis depend on the development of a murmur. This *murmur* is most apt to be that of mitral insufficiency. Aortic regurgitation may develop with its characteristic murmur. In some cases *no murmur* may be detected, when after death there may be marked evidence of valvular disease. The spleen is regularly enlarged. Albuminuria may be present.

Diagnosis: The presence of symptoms of septicæmia or pyæmia, together with a heart-murmur, are the points for diagnosis. Typhoid fever and general tuberculosis are to be differentiated by their special symptoms. In many cases the diagnosis is impossible, but the possibility of malignant endocarditis in all obscure cases of a septic type must be remembered.

Prognosis: This is almost uniformly fatal.

Malignant endocarditis—treatment: Nothing more can be done than to nourish the patient properly and to use alcoholic stimulants.

CHRONIC ENDOCARDITIS.

Definition: This is a slowly developing, insidious disease, usually leading to marked deformities of the valves of the heart.

Etiology: Rheumatism is the most frequent etiological factor. Many of the cases are secondary to acute endocarditis. Some cases develop during scarlet fever or chorea. Syphilis is at times a cause.

Pathology: The inflammation involves oftenest the endocardium of the mitral or aortic valves. The endocardium of the tricuspid or pulmonary valves, or of the ventricles or auricles, is less often affected. The endocardium is thickened by the infiltration of new cells and the growth of new connective tissue in its substance. Little beaded vegetations may form on its surface, thickening and roughening the valves. In other cases the surface of these vegetations becomes ulcerated and roughened, and thrombi form on them or lime-salts are deposited in them. These changes occurring, as they do, most markedly in the neighborhood of the valves, cause them to be contracted, thickened, and deformed. After this the valves cannot be opened or closed properly, and this causes *stenosis* or *insufficiency* of the valvular opening.

In *insufficiency* the valves cannot be shut properly, and some blood is forced back by the contraction of the heart. In *stenosis* the valvular opening is so small that the blood is pumped through it with difficulty. More than one form of lesion may be present at the same time.

Complicating lesions are always present: dilatation of the ventricles, hypertrophy of their walls, and disturbances in the circulation in other organs of the body. Due to this venous obstruction, we get congestion of the lungs, liver, spleen, and brain. Dropsy of the serous cavities and of the subcutaneous tissues is also often found.

Chronic endocarditis—symptoms: The pathological changes take place so slowly, and the interference with the valvular action is so gradual, that the heart-muscle accommodates itself to the extra work required to pump the blood with the valves diseased. This accommodation of the heart is called *compensation* and lasts up to a certain point only, after which the compensation is lost and the heart becomes unable to perform its functions in full.

During the time of full compensation there are virtually no symptoms from the endocarditis, and often the discovery of a murmur is the first intimation the patient has of any trouble with his heart. This condition of compensation may last many years, or may give way in a short time. Much depends on the endocarditis becoming stationary or advanc-

ing, and on the general condition of health and the habits of life of the patient. Marked deformities of the valves, acute illness, or chronic malnutrition, or excessive muscular exertion tends to destroy compensation.

Usually the earliest symptom of chronic valvular disease is *dyspnœa* on exertion, and this may be present even during fair compensation. Palpitation of the heart may be felt at times, but is not constant. Pain around the heart is rare. As compensation begins to fail the rational symptoms of the disease appear. They are mainly due to venous congestion of the various tissues of the body. In the *brain* the congestion produces headache, vertigo, stupor, delirium, at times convulsions; in the *lungs*, dyspnœa, orthopnœa, cough, hæmoptysis, chronic bronchitis, the pneumonia of heart-disease, and œdema of the lungs; in the *pleural cavity*, hydrothorax; in the *stomach* and *intestines*, indigestion, vomiting, at times hæmatemesis; in the *liver*, enlargement and functional disturbances; in the *peritoneum*, effusion; in the *kidneys*, scanty urine and albuminuria; in the *limbs*, dropsy. The pulse is usually rapid, feeble, and often irregular. It has different characteristics in different lesions of the valves, which are described under physical signs. Emboli from the heart may be carried to any part of the body, and produce their characteristic symptoms there: in the *brain*, paralysis; in the *lungs*, dyspnœa; in the mesentery, *bloody stools;* in the *spleen* or *liver*, pain; in the *kidneys*, hæmaturia; in the *limbs*, thrombosis and œdema. The symptoms gradually increase in severity until death, or periods of greater or less improvement occur, to be followed by a return of the symptoms later. The disease may last for many years.

Chronic endocarditis—physical signs: These depend on the valve affected. In *aortic stenosis*, which is the rarest lesion of the left heart, a *systolic murmur* is heard with its greatest intensity over the second right intercostal space quite near to the sternum, and is transmitted upward into the great vessels of the neck. A similar murmur is caused by simple roughening of the valves, and also in anæmia; but in stenosis there is decided hypertrophy of the left ventricle, with dis-

placement of the apex to the left and downward. The pulse is small and usually regular.

In *aortic insufficiency* there is a *diastolic murmur* heard loudest over the sternum at about the level of the third rib, and transmitted down the sternum and to the apex. The left ventricle is greatly hypertrophied and dilated. The pulse in all the arteries is exaggerated, and distinct throbbing is present in the larger ones. This is often felt quite unpleasantly by the patient. The pulse on palpation is quite characteristic, and is called "water-hammer," or "Corrigan." The upstroke is very sudden and the collapse equally so. The arteries are almost empty in the interval. Raising the arm brings out the characteristics of this pulse.

In *mitral stenosis* a *presystolic murmur* is heard over a limited area directly over the location of the mitral valve. This murmur is not transmitted. On palpation a distinct thrill is felt with each systole of the heart. The left auricle and right ventricle became dilated and hypertrophied in this condition, displacing the apex to the left, but not markedly downward. The pulse is small, feeble, and apt to be irregular.

In *mitral regurgitation* a *systolic murmur* is heard with maximum intensity over the apex, and is transmitted around the subaxillary space into the back. The second pulmonic sound is accentuated. The left ventricle becomes dilated and hypertrophied. The pulse is small, weak, and apt to be irregular.

The *right-sided lesions* are rare and difficult to diagnose, but tricuspid insufficiency may develop as a secondary lesion to mitral valve disease. It gives a systolic murmur heard mainly over the ensiform cartilage. The great veins of the neck are apt to pulsate with the systole of the heart.

Various combinations are often present, giving two or more murmurs at the same time.

Chronic endocarditis—diagnosis: This depends on the physical signs. The only thing to be remembered is the possibility of the murmurs being functional. No functional murmurs have associated the changes of hypertrophy and dilatation, which are present with actual valvular disease.

Prognosis: On the whole, the prognosis in children is not

so good as in adults. At the time of puberty, particularly, the extra strain put on the heart is apt to destroy compensation. Aortic stenosis and mitral insufficiency are the least serious varieties of endocarditis. Mitral stenosis is the more serious, and aortic insufficiency the most serious of all. Absolute recovery from the disease is exceedingly rare, and the presence of endocarditis always increases the danger from attacks of other diseases. Long life is compatible with valvular lesions under some circumstances.

Chronic endocarditis—treatment: If *compensation is present*, all treatment directed to the heart is useless. During this period the patient's general hygiene should be carefully under regulation. The diet should be varied and easily digestible, and moderated outdoor exercise should be carried out, with avoidance of all great and sudden strain. Daily bathing, proper sleeping, and regulation of the bowels are important. Measures should be taken to prevent all forms of acute disease, especially rheumatism and the infections. The child's nutrition should be particularly watched, and iron, cod-liver oil, and tonics given if there are evidences of anæmia or malnutrition.

If *compensation fails*, then is the time for treatment directed to the heart proper. The first and most important factor in this is *absolute rest in bed* for a considerable length of time. This at once makes the work of the heart much less, and allows it to recover its strength. Combined with this, the *drug* of most value is *digitalis*. It should be given alone if the arteries are soft; and should be used with nitroglycerin, or chloral, or iodide of potassium, if the arteries are tense. At the same time the general venous congestion is reduced by diuretics and saline purges, either of which class of drugs removes much water from the system. The diet had best be reduced to milk only for a time during this stage. As the heart improves, return to solid and highly nourishing diet, and the use of strychnine and iron are indicated.

The *special* symptoms as they arise require special treatment. Dropsy of the serous cavities may be removed by diuretics and purges; but tapping may be necessary. The œdema of the limbs had best not be interfered with surgically

from the danger of infection. Intense dyspnœa and orthopnœa are relieved by nitroglycerin and opium. Sleeplessness is relieved by trional.

ACUTE MYOCARDITIS.

Definition: This is an inflammation of the muscular wall of the heart.

Etiology: It is secondary to the infectious diseases—diphtheria, scarlatina, typhoid fever, and septicæmia. It is rather commoner in children than in adults. Some cases are associated with pericarditis, and others with endocarditis.

Pathology: The inflammatory changes may be diffuse or circumscribed, and may be interstitial or parenchymatous. In the *interstitial form* the heart-wall is infiltrated with inflammatory products, round cells, sero-fibrin, and even blood-cells. At times the process may be so intense in spots as to form a small abscess. The heart is flabby and softened, and of a mottled yellowish color.

In the *parenchymatous* form there is a distinct fatty degeneration of the muscle-fibers. The two varieties are apt to be associated at the same time in the same heart. These hearts are regularly dilated.

Acute myocarditis—symptoms: There are no typical symptoms or physical signs of this disease. The heart's action may be too slow or too rapid, or feeble or irregular.

The attacks of *cardiac failure*, slow or rapid, coming on in the course of the acute infections are apt to be due to this disease. The physical signs depend entirely on the enlargement of the heart from the dilatation.

Diagnosis: It is impossible to make a sure diagnosis. The association of these symptoms with one of the infectious diseases makes the diagnosis probable.

Prognosis: Recovery is possible, but death is the rule.

Acute myocarditis—treatment: Careful nursing, absolute rest, and proper nourishment during the course of an infectious disease tend to prevent this condition from developing. Strychnine by mouth or hypodermatically is the best drug we have for use in this disease. It should be used in large

doses, and often up to the physiological limit. In imminent cases hypodermatics of some rapidly diffusible stimulant, such as ether, brandy, or camphor, are useful. Iron and tonics are needed during convalescence.

CARDIAC NEUROSES.

These are rather rare in children, as most of the causative factors are usually the developments of adult life.

Etiology: Gastro-intestinal disorders are the commonest provoking cause of these neuroses. A neurotic heredity is usually present. Sudden fright, grief, and other emotions may cause them. The early use of tobacco in excess, as in cigarettes, and the use of tea and coffee are other causes. Anæmia and malnutrition are often present.

Pathology: There is no known lesion present in these conditions. They are undoubtedly due to some *reflex influence* acting on the *nervous supply* of the heart. Possibly the absorption of toxins from the digestive tract may explain the functional derangements.

Cardiac neuroses—symptoms: These are palpitations, irregularities in rhythm, excessively rapid or slow rate, and attacks of syncope. Respiration is liable to be hurried along with the change in the heart-rate. Normally in children some irregularity of the heart's rhythm is fairly common, and occasions no disturbance. Especially is this so during sleep.

Most of these conditions are paroxysmal. They come on in attacks lasting a short time, and disappear equally quickly. The *physical examination* of the heart in these cases is negative other than to show its rate and rhythm.

Diagnosis: The only point in diagnosis is absolutely to exclude all forms of organic heart-disease, pericardial, endocardial, and myocardial. This in some cases can be done only after repeated careful examinations of the heart, and observation of the child for some time.

Prognosis: This is good. The neuroses are never fatal in the attack, and with proper treatment and removal of the cause the tendency to the attacks can be cured. Certain cases with marked hereditary predisposition are least amenable to treatment.

Cardiac neuroses—treatment: The cause should be carefully searched for and removed. Gastro-intestinal derangements should be corrected. Attention to the diet is of great importance. Tea, coffee, and tobacco should be stopped. Proper exercise, fresh air, and regular sleeping hours should be enforced. If the child is at school, supervision of its studies should be undertaken. Anæmia and malnutrition should be treated in the usual manner.

During attacks digitalis, strophanthus, aromatic spirits of ammonia, valerian, and the bromides are the most useful drugs. Between attacks no special treatment for the heart proper is indicated.

DISEASES OF THE ARTERIES AND VEINS.

CHRONIC ENDARTERITIS.

Inflammations of the arteries are rare in children, as they are regularly the result of senile changes.

Etiology: Syphilis or tuberculosis is the usual cause.

Pathology: The arterial wall undergoes hyperplastic inflammation and a subsequent degeneration of the new tissue, with weakening and giving way of the wall.

Chronic endarteritis—symptoms: The development of an *aneurism* in this weakened wall is the typical symptom of this condition in children. Aneurisms are quite rare, but they have been found in the abdominal aorta, the iliac, femoral, and cerebral arteries. The presence of a pulsating tumor in the line of an artery over which a systolic bruit is heard; and the mechanical symptoms due to its pressure, are the regular signs of the disease. Endarteritis in patches, without aneurism, can scarcely be diagnosed.

Prognosis: This is bad, as the disease is apt to appear in subjects in bad general health.

Chronic endarteritis—treatment: This is the same as in adults. Antisyphilitic treatment should be used in the presence of a specific history. Surgical intervention in external aneurism is always advisable.

ACUTE PHLEBITIS.

Definition: This is an acute inflammation of the walls of a vein.

Etiology: Injury of the vein, extension of inflammation from neighboring structures, the presence of an infective embolus, and exposure to cold act as actual causes. It also complicates the infectious diseases, particularly typhoid fever and septicæmia. In some cases anæmia seems to be the only cause.

Pathology: The wall of the vein is the seat of an acute inflammation with swelling and infiltration by inflammatory products. The internal surface becomes roughened, and the lumen of the vein diminished, thus favoring coagulation of the blood in the vein. The inflammation may be so intense as to cause minute abscesses in the wall of the vein. Thrombosis, or clotting of the blood in the vein, may be the result or the cause of the phlebitis.

Acute phlebitis—symptoms: There are pain and tenderness along the course of the affected vein, and, if it is a superficial vein, a red swollen line follows its course. Thrombosis of the blood inside occurs, and the parts below, that are drained by this vein, are swollen and œdematous. There are usually slight fever and some general malaise. In infective cases abscesses may form in the course of the vein.

The disease may last but a couple of weeks, with cessation of the inflammation, absorption of the thrombus, and return of the vein to normal; or it may run a chronic course, and in some cases the vein remain permanently blocked, all signs of acute inflammation, however, disappearing.

Diagnosis: In the veins of the limbs the diagnosis is fairly easy, and rests on the presence of signs of inflammation over the vein and thrombosis. In the trunk the symptoms are mainly those of thrombosis and blocking of the circulation, and the diagnosis is more difficult.

Prognosis: This is fairly good unless the disease occurs in the veins of some one of the vital organs, as for instance the brain, or unless it is part of a general septic process.

Acute phlebitis—treatment: Rest and the avoidance of all

unnecessary movement in the part containing the affected vein are of primary importance. This is to reduce the chance of breaking off a piece of the clot, and the formation by it of an embolus elsewhere. Hot applications, lead and opium wash, and in the chronic stage mild counter-irritation, as by iodine, assist in absorption of the clot. The bowels should be kept freely open and the patient fed on nourishing diet. No drugs are of any special value.

DISEASES OF THE CAPILLARIES.

NÆVUS.

Definition: This condition, which is also called *angioma*, is one of the commonest congenital disfigurements of childhood.

Etiology: *Nævi* are probably always congenital. At times they may be so small at birth as not to be noticed, and then grow distinctly after birth. In olden times they were erroneously ascribed to maternal impressions.

Pathology: There are two general varieties: the *capillary*, with a great increase in the number and size of the capillaries; and the *cavernous*, in which there is a form of erectile tissue, the blood circulating in irregular anastomosing spaces without distinct vascular walls.

Nævus—symptoms: When *superficial*, the so-called cutaneous nævus, it forms the well-known port-wine stain, or "mother's mark." These are purplish blotches on the skin of irregular **shape and size.** They may be situated anywhere on the surface.

In the *subcutaneous* variety there is a soft, irregular tumor of varying size under the skin. It is increased in volume by crying, coughing, or exertion. The skin over it may be normal or bluish, or be so thin that the tumor shows the blood color through it. Pressure reduces its size, but it fills again on removing the pressure.

As a rule no other symptoms than the presence of a mark or a tumor are found; but in certain localities pain, or the displacement of certain organs, may be caused by the nævus. This is seen, for instance, in the orbit. Hemorrhage, which

may be dangerous, may occur from them. They are most often found around the head and neck. They may occur in the viscera, as the liver, spleen, or kidneys, but are only diagnosed at autopsy.

Diagnosis: The cutaneous form is simple. The subcutaneous may be mistaken for other tumors, and especially for lipomata. The increase in size on exertion and decrease on pressure are the points in favor of nævus.

Prognosis: They rarely disappear spontaneously. Usually they remain stationary and are only disfigurements. They may grow, however, and become not only very unsightly, but even disabling. They are not dangerous.

Nævus—treatment: This is based on replacing the vascular tissue by cicatricial tissue, which may be brought about by the local application of caustics, the actual cautery, or electrolysis. Diffuse scarification may be tried in large port-wine stains. In the subcutaneous variety any of the above methods may be tried; or injections of coagulating fluids, or ligation by passing a thread around the tumor and strangling it.

DISEASES OF THE BLOOD.

The blood in early infancy: During the first week or two of extra-uterine life, and in premature infants, the blood differs considerably from that of adults. The specific gravity and the proportion of hæmoglobin are high, the number of red cells exceeds the normal five millions per cubic millimetre, and there are present nucleated erythrocytes or hæmatoblasts in considerable numbers, and also many red cells with the hæmoglobin dissolved out.

The *leucocytes* are also present in larger number than the normal ten thousand per cubic millimetre. They may increase to twenty or even thirty thousand. In other words, *leucocytosis* is a *normal condition* during this stage.

The five different varieties of leucocytes, small mononuclear cells, large mononuclear cells, mononuclear cells with nucleus undergoing transition, polynuclear neutrophile cells, and eosinophile cells, are found as in adult blood, and in approximately the same proportions. Within a short time all

the elements gradually take on the proportions of adult blood, except that for a year or two the hæmoglobin is lower than the normal 100 per cent. of adult life. Otherwise the blood in childhood differs very little from that of maturity.

In *health*, within certain rather definite limits, the proportion of the different constituents remains constant. In *disease*, diminished production or increased destruction of one or another element produces marked changes in their relative proportions, and gives us our only means for the classification of diseases of the blood.

SIMPLE ANÆMIA.

This is also called **primary anæmia**, and when it occurs in young girls about the time of puberty it is called **chlorosis**, from the greenish appearance produced in the skin.

Etiology: The disease is commonest in girls and during the second decade of life. Confining occupations, impure air, lack of exercise, improper food, and constipation all act as causes. The last probably acts through absorption of poisonous substances into the blood, which should be excreted with the stools.

Pathology: On examining the blood the *hæmoglobin* is found far below normal, often being down to 30 or 40 per cent. The *red cells* and *leucocytes* are not diminished. In other words, each red cell has lost a certain proportion of its hæmoglobin.

Simple anæmia—symptoms: The patient is languid, feeble, and incapable of exertion. There are headache, dyspnœa on exertion, vertigo, palpitation of the heart, attacks of syncope, constipation, anorexia or capricious appetite, and amenorrhœa, one or all in almost every case. The skin and mucous membranes are pale, and the muscles flabby, but the subcutaneous fat is not diminished. The disease comes on gradually, and without treatment lasts an indefinite length of time.

Simple anæmia—physical signs: The blood-changes described under pathology are most important. There is usually a systolic murmur heard over the heart, with its maximum intensity over the pulmonic area, although it may be loudest at

the apex. There is a loud venous hum heard over the great vessels in the neck.

Diagnosis: This, in the presence of the typical symptoms, depends on the blood-examination.

Prognosis: This is good. It is not a fatal disease, and under proper treatment recovery is rapid.

Simple anæmia—treatment: The treatment is very simple and very satisfactory. The bowels should be kept open, daily exercise in the fresh air should be prescribed, and iron should be given regularly. Blaud's pills, grains three or five, three times a day after meals, give most excellent results. Other forms, as the tincture of the chloride or the bitter wine, may be used when it is impossible to swallow pills.

SECONDARY ANÆMIA.

Definition: This disease is not due to any fault of the blood-making apparatus, but is secondary to general constitutional states which increase the normal wear and tear on the blood.

Etiology: It is a common disease of infancy and childhood, and is seen complicating and after such conditions as rheumatism, tuberculosis, syphilis, acute and chronic digestive disorders, rachitis, hemorrhages, the infectious diseases, nephritis, prolonged suppuration, malaria, marasmus, improper food and insufficient air, prolonged fevers, and malignant growths. In some cases very rapid bodily growth seems to be the only discoverable cause.

Pathology: Aside from the lesions of the primary disease, the changes in the blood are all that we expect to find. There is a moderate decrease in the quantity of hæmoglobin and in the number of red cells in the blood. The diminution in each is fairly proportionate—that is, the cells are decreased in number, and those that are left behind have less than their proportion of hæmoglobin. The decrease is seldom as great as it often becomes in simple anæmia. There is no increase in the leucocytes.

Secondary anæmia—symptoms: It is difficult to separate the symptoms due to the anæmia from those of the primary

disease. The skin and mucous membranes are pale and blanched. The muscles are flabby. The extremities are apt to be cold. There are disinclination to exertion, dyspnœa on motion, attacks of palpitation of the heart and of syncope, poor appetite, general restlessness, and disturbed sleep. The patients tire easily, and, if old enough, complain of headache. Anæmic heart-murmurs are often present, but are less marked than in the essential form of the disease.

Diagnosis: This depends on the blood-changes shown by examination in the presence of one of the primary diseases. The absence of any primary cause would point to a *simple anæmia.* The proportionate percentage of hæmoglobin and cells in the two diseases differs also.

Prognosis: This depends entirely on the primary disease. The anæmia itself is not a cause for special prognosis.

Secondary anæmia—treatment: The cure of the primary disease is of first importance. All digestive disturbances particularly must be corrected. Good hygiene, proper food, and, above all, fresh air, must be insisted on. The drugs of most value directly to increase the richness of the blood are iron and arsenic. The former may be given as the bitter wine, the syrup of the iodide, or the pepto-manganate; the latter as Fowler's solution. In children who can swallow pills, Blaud's pill, with arsenic, is the best form. Cod-liver oil seems to assist the treatment in certain cases.

PERNICIOUS ANÆMIA.

Definition: This is a primary disease of the blood which progresses regularly to a fatal end.

Etiology: No real cause for the disease is known. It is of rare occurrence in childhood. Late researches point to the fact of the disease being due to an increased destruction of the red blood-cells in the liver, which may be brought about by the presence in the portal blood, fresh from the intestinal tract, of some *toxic principles.*

Some cases are due to the presence of the *anchylostoma duodenale* in the intestines. Such are rare in this country.

Pathology: There is a fatty degeneration of the walls of

the heart, of the hepatic and renal cells, and of the walls of the arteries and capillaries. The blood shows a marked diminution in the red cells and in the hæmoglobin. The cells that are left have a normal proportion of hæmoglobin, but are of irregular shapes and sizes, and often are nucleated. The leucocytes are not absolutely increased, although they are so relatively to the number of erythrocytes present.

Pernicious anæmia—symptoms: The symptoms develop slowly and insidiously. The skin and mucous membranes become pale, and later lemon tinted. The muscles grow flabby and soft, and progressively feeble. The disinclination to exercise is most marked. There are palpitation of the heart, dyspnœa on exertion, attacks of syncope, and a gradual muscular and mental enfeeblement. The functions of the stomach and intestines are weakened, with the consequent symptoms of indigestion and malnutrition. There may be hemorrhages into the retina, or from the mucous membrane of the nose, mouth, stomach, or intestines, or even under the skin.

In the *later stages* subcutaneous œdema may develop, but without albuminuria. Irregular fever is present at some time in the disease. A fair degree of nutrition—that is, of subcutaneous fat—is usually retained. Anæmic heart-murmurs are usually present. Toward the end the feebleness becomes so marked that the patient is absolutely bedridden. The cases regularly last some time, with intervals of seeming improvement.

Diagnosis: This depends on the blood-examination showing both the great decrease in red cells and hæmoglobin, and the morphological changes in the erythrocytes. By this means the other varieties of anæmia are differentiated; and all diseases of other organs should also be excluded.

Prognosis: This is distinctly bad, although periods of improvement are to be expected.

Pernicious anæmia—treatment: Rest, fresh air, a highly nutritious and easily digestible diet, and minute attention to all the functions of the stomach and intestine are very important. No constipation should be permitted. The only drug from which any help can be expected is *arsenic*. It

should be given in Fowler's solution, in gradually increasing doses, until full tolerance is reached.

LEUKÆMIA.

Definition: This is a disease of the blood, characterized by a marked increase in the leucocytes; a diminution in the number of erythrocytes, and in the quantity of hæmoglobin; enlargement of the spleen and lymphatic glands; and increase in amount of the marrow of the long bones. With the leucocytosis, which is always present, one or more of the other organs—spleen, glands, and marrow—may be involved.

Etiology: Nothing definite on this point is known. It is seen with some frequency in children, and even in infants, and in boys more often than in girls. In some cases it seems to be secondary to malaria, or syphilis, or trauma over the spleen, or at times to starvation.

Pathology: The essential lesions are in the *blood*, *spleen*, *lymph-glands*, and *bone-marrow*. The blood is lighter colored than normal, and in advanced cases has a whitish purulent appearance. The number of red cells and the amount of hæmoglobin are moderately diminished. The leucocytes are very much increased in numbers, at times being as many as the erythrocytes. This, of course, occurs in extreme cases only. The proportions of the different varieties of leucocytes present vary with the form of leukæmia present. In the *lymphatic* variety the small mononuclear cells are mainly increased. In the *spleno-medullary* variety the large mononuclear cells are mainly in excess, while the eosinophiles are also increased. The *spleen* is usually much enlarged, the changes being mainly those of a simple hypertrophy. The *lymphatic glands* are hypertrophied in various parts of the body, single or multiple groups being involved. These again undergo simply a hyperplasia of their normal tissue. New lymphoid tissue in the form of tumors may grow in the liver, kidneys, or peritoneum. The *marrow* of the bones is hypertrophied, its color may be yellow or red, and both its cells and stroma are regularly involved.

All of the organs, blood, spleen, glands, and marrow, may

be involved at once, or only the blood with the glands, when it is called *lymphatic leukæmia;* or the blood with the spleen and marrow, when it is called *spleno-medullary leukæmia.* In any variety all the organs are usually somewhat involved, although certain ones are more markedly so.

Leukæmia—symptoms: The disease usually begins insidiously, but advances rather more rapidly in children than in adults. The child is pale, very weak and feeble; has marked dyspnœa and attacks of fainting. Hemorrhages from the various mucous membranes, as into the retina or under the skin, are often seen quite early. These bleedings at times may be large enough to be quite serious. Enlargement of the abdomen from the hypertrophied spleen or of the various superficial or deep lymphatic glands soon begins, and at times is the earliest symptom. The heart's action is rapid and feeble, and there may often be irregular rises of temperature lasting over some days. There may be pains and tenderness in the bones from the changes in the marrow.

As the disease progresses extreme feebleness, subcutaneous dropsy, headaches, failing sight, diarrhœa, and hemorrhages gradually bring on the fatal issue. The cases last from a few months to a year.

Diagnosis: This is based on the blood-examination—that is, the increase in the number of leucocytes and the proportions of the different varieties.

Prognosis: This is almost absolutely fatal, although a few cases of recovery have been reported.

Leukæmia—treatment: Rest, a highly nourishing diet, and fresh air are of importance. *Arsenic* in gradually increasing doses is the best drug. Iron and phosphorus are often used as adjuvants.

PSEUDOLEUKÆMIA.

Definition: This is often called Hodgkin's disease, malignant lymphoma, lymphatic anæmia, and splenic anæmia. It is *characterized* by anæmia and enlargement of the lymphatic glands of the body. Often the spleen is enlarged at the same time, and in some cases the spleen is involved without the

glands. To this variety the term *splenic anæmia* is especially applicable.

Etiology: The disease is more frequent in boys than in girls. Syphilis, tuberculosis, and malaria are possible predisposing factors. Local traumatism may be assigned as the beginning. Very little is known, however, of the causes.

Pathology: In the blood the red cells are decreased in number and the hæmoglobin correspondingly in amount, but the white cells are unaltered.

One or more groups of lymphatic glands are enlarged. The glands are simply hypertrophied, both the stroma and the cells being involved. They have no tendency to suppurate nor caseate. The glands in the neck or axillæ are usually first involved.

In the spleen new growths of lymphoid tissue are often found. These may cause a uniform or irregular enlargement of that viscus. Similar growths of lymphoid tissue may be found in the liver, kidneys, and other organs, but much less commonly than in the spleen. The marrow of the bones in rare cases may be involved in this hyperplastic growth.

Pseudoleukæmia—symptoms: The first symptom is regularly an enlargement, without known cause, of some set of superficial lymph-glands. Those in the neck are usually earliest involved. The axillary or inguinal glands may soon be affected, or even some of the deeper sets, as the thoracic or retroperitoneal.

After some little time the *constitutional symptoms* of the disease appear. These are mainly the result of the anæmia, such as pallor, weakness, palpitation of the heart, dyspnœa on exertion, fainting attacks, vertigo, and disordered digestion. Later, hemorrhages from the mucous membranes and into the skin, subcutaneous œdema, fever, and marked loss of flesh and strength develop. Toward the end nervous symptoms, delirium, coma, or general convulsions may occur.

As the glands grow in size *pressure-symptoms* become prominent. In the *neck* they produce dyspnœa, dysphagia, and interference with the blood-vessels or pneumogastric nerves; in the *thorax* they may press on the trachea, œsoph-

agus, or descending vena cava; in the *abdomen* they may produce jaundice by pressure on the bile-duct, or ascites by pressure on the portal vein, or œdema of the lower extremities by pressure on the ascending vena cava; in the *spleen* they produce enlargement; in any position they may produce *pain* from pressure on sensory nerves. The glands are not tender and are freely movable.

The disease progresses rapidly, with periods of improvement, and regularly lasts a number of years.

Diagnosis: The disease must be differentiated from leukæmia, which is done by the blood-examination, and from tubercular adenitis. In the latter condition the glands are prone to fuse together, soften, and suppurate, all of which phenomena are rare in pseudoleukæmia. In the splenic form splenic leukæmia is distinguished by the blood-examination; and other causes of splenic enlargement must be excluded before diagnosing splenic pseudoleukæmia.

Prognosis: Absolute recovery is rare, but temporary improvements extending over many years are often seen. Where the deeper glands are involved the cases seem more serious.

Pseudoleukæmia—treatment: The patient must be kept under strict hygienic surroundings, with good food, good air, and moderate exercise. Of drugs, arsenic seems the only one of much value; it is given in gradually increasing doses. The iodides at times seem useful.

The question of the *operative removal* of the enlarged glands has much to be said on both sides. At times, after operation, there seems to be a fresh outburst of the disease elsewhere. At other times good results follow. Operation is indicated when pressure-symptoms are causing trouble.

ADDISON'S DISEASE.

Definition: This is a disease characterized by anæmia, general languor and debility, feeble heart-action, irritability of the stomach, bronzing of the skin, and disease of the suprarenal capsules.

Etiology: It is a rare disease under any circumstances, but especially so in children. A few cases are recorded under

fifteen years of age, some being in mere babies. The causal conditions are not known.

Pathology: The *supra-renal capsules* are usually the seat of a tubercular inflammation, with conversion of the glands into cheesy and fibrous tissue. At other times they are atrophied or absent. There is a deposit of pigment in the cutis vera. The blood shows a decrease in red cells and hæmoglobin, but no change in the white cells.

Addison's disease—symptoms: There is a slow, gradual loss of flesh and strength. The mind becomes dull and apathetic. The temper is often highly irritable. The action of the heart grows very rapid and feeble. Dyspnœa is present, increased by exertion. The stomach is upset; there are pains, nausea, and vomiting.

The typical *bronzing of the skin* is most evident on the face and hands, and in the regions where the skin is naturally most pigmented.

Patches are also found on the various mucous membranes, as that of the mouth.

Toward the end asthenia becomes most marked, and stupor, delirium, coma, or convulsions may occur. The disease is chronic, but progressive in its course, and with its periods of remission and exacerbation may last for years.

Diagnosis: This is difficult except in cases where all the typical symptoms are present at the same time.

Prognosis: This is bad, as almost all the cases die in a few years. General tuberculosis may develop.

Addison's disease—treatment: Absolute rest and nourishing and digestible diet are of prime importance. Iron, arsenic, strychnine, and phosphorus are all recommended as useful drugs.

HÆMOPHILIA.

Definition: This is a condition in which even after the slightest injury *hemorrhage*, which is very difficult to control, occurs. Such a person is called a "bleeder."

Etiology: There is a marked *hereditary tendency* to the disease. It is *transmitted* through the *female side* of a hæmo-

philic family, but regularly *appears* in the *male members.* No real cause for the disease is known.

Pathology: No marked changes either in the blood or in the bloodvessels are found. In a few cases the walls of the vessels seem to be very thin. In the other tissues nothing abnormal is found.

Hæmophilia—symptoms: As a rule, there is nothing in the appearance of the child to suggest that he is a "bleeder." The first sign is apt to be a prolonged hemorrhage from some trifling wound, or from some mucous membrane. Epistaxis is the commonest form of bleeding. The bleedings usually do not make their appearance before the second year. For instance, such hemorrhages from the umbilical cord are rare. The first hemorrhage is apt to be recovered from. The fatal ones, as a rule, are subsequent. Petechiæ in the skin, or hæmatoma in the deeper parts, are apt to take place. In some cases there are swelling and inflammatory signs in the joints.

Diagnosis: No way exists to tell that these children are "bleeders," except by experience with a wound in each individual case.

Prognosis: A large proportion of these cases never reach adult life. They are in constant danger of death from hemorrhage from a small or large traumatism.

In the few women who are "bleeders," neither in menstruation nor in parturition does the hæmophilia seem to add to the dangers.

Hæmophilia—treatment: In case of bleeding the usual surgical measures—pressure, position, plugging, and ligaturing—should be followed. If a coagulum is formed, great care should be taken not to disturb it.

After the bleeding is stopped measures should be taken to prevent further accidents. All minor operations and all traumatisms should be avoided, vaccination and pulling of the teeth being among these. The child should be kept in the open air and given plenty of exercise and iron. There seems some value in giving lime salts over a considerable length of time. Girls in these families should not marry.

PURPURA.

Definition: This term is used to include *spontaneous hemorrhages* into the skin, mucous membranes, and internal organs. The different varieties have received different names; but they all seem related except in degree, and hence will be grouped together. Such varieties are *purpura simplex*, in which purpuric spots are seen in the skin; *purpura hæmorrhagica*, or Werlhof's disease, in which in addition free hemorrhages appear from one or another of the mucous membranes; and *rheumatic purpura*, or *peliosis rheumatica*, occurring in cases at the same time suffering from inflamed joints.

Etiology: This is quite a common disease of children, as in them the bloodvessels are still immature. In very many cases no etiological factor can be made out. In others, such causes as cachexia from scurvy; tuberculosis; chronic pulmonary or intestinal disorders; from malignant disease or infections from septicæmia, malignant endocarditis; or the exanthemata, are active. Certain *drugs*, as quinine, the iodides, chlorate of potassium, and phosphorus, will produce it. A few cases are mechanical from venous stasis, as in endocarditis, pertussis, and epilepsy. In others the rheumatic diathesis seems in some way to act as a cause. In others there is reason for believing that some form of micro-organism is the actual cause. This is especially true in Werlhof's disease.

Pathology: In the blood itself no changes have as yet been demonstrated other than those of slight anæmia. In some cases the walls of the small arteries and capillaries, from which the hemorrhages regularly take place, are found in an abnormal condition, permitting free escape of the blood through them. In the variety with an infectious origin an infectious embolus followed by thrombosis and inflammation of the wall of the vessel is found. In the very acute hemorrhagic cases different forms of germs have been found, but no specific one.

Hemorrhagic areas are found in the skin, the serous and mucous membranes, the joints, and the viscera. The spleen is usually enlarged.

Purpura—symptoms: These vary a good deal with the form of purpura present.

In *purpura simplex* there may be no constitutional symptoms at all, or slight fever with its accompanying malaise, anorexia, and headache, may be present. The local symptoms are the occurrence of hemorrhagic spots or blotches situated in almost any part of the skin. These spots are a dark red at first, later fading to a brown, and then to a yellow. They are not tender and do not disappear on pressure. After fading some pigmentation is left for a considerable time. In the course of two or three weeks the disease is over.

In *purpura hæmorrhagica* there are regularly present more or less marked constitutional symptoms, as fever, headache, prostration, and digestive disturbances. With these occur extravasations into the skin and free bleedings from the mucous membranes. These hemorrhages are from the nose, mouth, throat, stomach, lungs, intestines, or genito-urinary tract. In many cases the bleeding is severe enough to endanger life. In other cases the hemorrhage may take place *en masse* in some internal portion of the body where the tissues are loose, as the orbit, producing exophthalmos.

Some of the *more severe cases* of this variety run a very rapid fatal course, presenting the symptoms of an infectious disease with high fever and signs of general toxæmia. All of these hemorrhagic cases last several weeks, and, if recovery occurs, the patient is left anæmic and feeble.

In *purpura rheumatica* the symptoms are those of acute rheumatism of one or more joints with the lesions of simple purpura added. They are probably not cases of actual rheumatism, however. The joints are inflamed, red, hot, swollen, and tender. There are fever, with its accompaniments, and on the extremities purpuric spots scattered irregularly about. This form lasts several weeks and relapses are quite common.

Diagnosis: Purpura being more of a symptom than a disease, diagnosis in any variety is fairly easy. Scurvy is differentiated by its signs in the gums, the different locality of the hemorrhages, the etiology, and, if necessary, by the effects of treatment. Hemophilia is distinguished by the family history and the absence of constitutional symptoms. If possible, the cause of the purpura must be diagnosed as well as the presence of the disease.

Prognosis: The large proportion of cases end in recovery. The fatal cases are the very severe ones of the hemorrhagic variety, with marked symptoms of infection, the so-called purpura fulminans. Relapses are common.

Purpura—treatment: The patient should be put to bed and on a nourishing diet. The causal factor should be searched for and treated, if possible. Tonics, as iron and arsenic, are indicated. Stimulants should be used in the presence of fever and toxæmia. Fresh fruit-juice and vegetables should be tried on account of their value in the similar condition of scurvy.

Of *drugs*, aromatic sulphuric acid, turpentine, ergot, tannic or gallic acid and calcium chloride have been used. In the rheumatic form the salicylates should be given.

CHAPTER IX.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE.

EPISTAXIS.

Hemorrhage from the nose is quite a common occurrence in children.

Etiology: Boys are more apt to suffer from nose-bleed than girls. It occurs especially in delicate children who are not accustomed to outdoor life. It is often a symptom of adenoids and of rhinitis. It may begin without any exciting cause, but more often follows picking the nose, a slight blow on the nose, or other local injury. Severe exertion may bring on an attack. It occurs in cases of endocarditis and other conditions in which there is venous stasis of the blood. It may be an early symptom of typhoid fever, malaria, or almost any of the infectious diseases. It may be part of the hemophilic diathesis or a complication of almost any of the anæmias.

Pathology: The source of the blood may be situated almost anywhere in the nasal fossæ, but usually is in the anterior nares. As a rule there is a small erosion of the mucous membrane, which may be situated over a fair-sized vessel, in which case the bleeding is more marked than when there is capillary oozing only.

Epistaxis—symptoms: Bleeding from the nose is the only sign of any moment. The blood may come from one or both nostrils, or, if the bleeding point is posterior, it frequently will come through the pharynx, the blood being either spit up or swallowed. In some cases the bleedings may be large enough to produce feelings of faintness and to leave the child

anæmic. The hemorrhages as a rule last only a few minutes, but recurrences are common.

Diagnosis: It is, of course, easy to make the diagnosis of epistaxis, but it is at the same time important to find the cause of the bleeding and its exact source.

Prognosis: Ordinarily epistaxis is a simple, harmless trouble. In the various constitutional diatheses of which it is a symptom, as hemophilia and the anæmias, it is more serious. In conditions of venous stasis from any cause it is ordinarily helpful. Occurring in the late stages of the infectious diseases it indicates a serious form of the disease.

Epistaxis—treatment: A child prone to attacks of epistaxis should be put under the best hygienic conditions, and by the use of daily cold baths and being kept in the fresh air should have his mucous membranes toughened so as to resist changes in temperature. If the epistaxis is due to any of the predisposing causes, as rhinitis, adenoids, venous congestion, or any of the hemorrhagic diatheses, these should be treated.

In an attack the local application of cold to the nose externally, or to the back of the neck, or in the nostril, will usually stop the bleeding. If these are not effective, astringent solutions may be applied to the nostrils, such as tannic acid, antipyrin, or alum. In other cases the anterior nares may be plugged with cotton. In more severe cases the posterior nares had better be plugged at the same time. This may easily be done by passing a soft-rubber catheter, in which a piece of silk is threaded, through the nostril into the pharynx. The catheter is withdrawn, the silk being left in place by catching the end with a pair of forceps. With this silk a piece of cotton can be drawn into the naso-pharynx and tied there.

After the hemorrhage has ceased it is wise to examine the nares carefully for the bleeding spot, and to touch this with silver nitrate.

ACUTE RHINITIS.

This is commonly called "**cold in the head**," and technically *coryza*.

Etiology: This very common disease of adult life is equally

often seen in babies and children. It is seen oftenest in children who are coddled too much, being dressed too warmly, and kept in overheated, unventilated rooms, and never bathed in cool water.

It follows exposure to draughts, sudden chilling of the surface while warm, wet feet, and cold to the abdomen. Probably some form of micro-organism is also present as a co-existing cause. The disease certainly seems somewhat infectious. Coryza is an early symptom of measles.

Pathology: The nasal mucous membrane is congested and swollen, and at first has a very scanty secretion, or at best only a watery one. Later, as the inflammation passes off, the mucous glands begin to secrete, and mucus and muco-pus are discharged in large quantities.

Acute rhinitis—symptoms: The nose is stuffed up, necessitating mouth-breathing, and in infants causing difficulty in nursing. There is a feeling of fulness in the nose and frontal sinuses, and there may be decided aching in the same localities. There are slight fever and general malaise. Sneezing begins early. At first there is no discharge; later there is a profuse acrid watery one, and later yet mucus and muco-pus in large amounts may be expelled by blowing. If the naso-pharynx is involved, the openings of the Eustachian tubes may be swollen, and ringing in the ears and deafness follow.

The attack lasts a little less than a week, but the mucous discharge continues for some time longer. Complications, as excoriation of the upper lip, ear-ache from catarrhal otitis media, conjunctivitis from extension through the lachrymal duct, and cervical adenitis from absorption into the lymph-glands may be present.

Diagnosis: The diagnosis of the rhinitis is easy enough; but we must exclude the beginnings of measles, influenza, or diphtheria, and the presence of congenital syphilis.

Prognosis: This is good. Recovery regularly follows in about one week. Recurrences are frequent.

Acute rhinitis—treatment: To cure the tendency to catching cold, the children should be accustomed to sleeping in cool, well-ventilated rooms, and to being out of doors almost every day. They should be warmly and properly dressed,

but not bundled up. Every day they should take a cool-water sponge or plunge after the cleansing bath. This tones up the vascular system, and keeps it more resistant to sudden temperature-changes. The nose and naso-pharynx should be examined for any chronic conditions, such as chronic rhinitis or adenoids, that may be present.

To treat the coryza itself, the child should be kept indoors and in a uniform temperature. A purge should be given early, which will remove the congestion and hasten the inflammatory process in the nose. After this there may be given internally some combination of quinine, belladonna, and camphor, as in "pill rhinitis;" or of quinine, ammonium chloride, camphor, belladonna, opium, and aconite, as in "pill coryza," in small doses suitable for the age of the child, and frequently repeated. The nostrils may be washed out two or three times a day by a cleansing solution, such as Seiler's, and then a little melted vaseline poured into them. If reduction of the congestion is necessary, a weak cocaine solution may be used cautiously. A blunt-pointed piston syringe, or a medicine-dropper, or a Bermingham douche, are better for these local applications than sprays or atomizers.

CHRONIC RHINITIS.

This is a **chronic inflammation of the nasal mucous membrane** in which there is neither hypertrophy nor atrophy.

Etiology: Frequent attacks of the acute form are the commonest cause. Adnoids in the naso-pharynx, foreign bodies in the nose, and nasal polyps are often found in these cases.

Chronic rhinitis—symptoms: A mucous or muco-purulent discharge from the nostrils, of greater or less amount, is the early symptom of this disease. The edges of the nostrils and the upper lip may be excoriated by the discharge. The discharge can usually be easily removed by blowing.

Diagnosis: A thorough examination of the anterior and posterior nares should be made when possible, to discover the condition of the nasal mucous membrane and the presence of any of the local causes.

Prognosis: The disease may be cured by proper treatment;

but if left alone, it is apt to terminate in one of the more serious forms of chronic rhinitis, the hypertrophic, or more often the atrophic.

Chronic rhinitis—treatment: The prophylactic treatment recommended for acute rhinitis must be followed. Local causes, if they exist, must be removed, such as adenoids, polypi, or foreign bodies. The nostrils should be thoroughly cleaned out once or twice a day by spraying or syringing with some mild alkaline solution, such as Seiler's. A good home wash is a teaspoonful each of soda, salt, and borax to a quart of water. Always use nasal solutions warm. A useful and simple little instrument for this purpose is the Bermingham douche. After cleaning, some astringent such as sulphocarbolate of zinc in 1 per cent. solution, or nitrate of silver in 1 or 2 per cent. solution, should be applied with cotton.

HYPERTROPHIC RHINITIS.

Definition: This is a form of chronic rhinitis in which the nasal mucous membrane is very much hypertrophied, blocking up the nasal fossæ and interfering with the passage of air through them.

Etiology: It is most commonly secondary to repeated attacks of acute rhinitis.

Pathology: All the parts of the mucous membrane are hyperplastic. The vascular portion is particularly affected, the number of vessels being increased and their size enlarged. The membrane over the turbinated bones takes on the characteristics of erectile tissue.

Hypertrophic rhinitis—symptoms: The two symptoms commonly present are nasal discharge and obstructed nasal breathing. The discharge is more difficult of expulsion than that due to simple rhinitis, but is not so irritating to the upper lip. The nasal obstruction leads to mouth-breathing and to the so-called nasal tone of voice. This is not due to talking "through the nose," but to the inability to ventilate the naso-pharynx through the nose.

Diagnosis: This is made by examining the anterior and posterior nares when possible. The turbinated bodies are seen

to be swollen, and red both in front and behind, and the space of the nostrils blocked by them so much that a probe is passed with difficulty.

Prognosis: Much good can be accomplished by removing this hypertrophic condition by treatment. It is questionable if the inflammation can be completely cured. If untreated, the cases usually go on to atrophy.

Hypertrophic rhinitis—treatment: The same prophylactic and cleansing measures should be used as in simple chronic rhinitis. The hypertrophied turbinates can be reduced by forming a linear horizontal scar along them by the application of nitric, glacial acetic, or chromic acid, or by the use of an electro-cautery. In some cases the mucous membrane is so hypertrophied that portions of it may be removed by a snare.

ATROPHIC RHINITIS.

Atrophic rhinitis is a somewhat rare condition in children. It is often called ozæna or fœtid rhinitis. These terms are better reserved for cases with necrosis of the bones of the nose.

Etiology: It is sometimes secondary to repeated attacks of acute rhinitis and to hypertrophic rhinitis. Other cases come without known cause.

Pathology: There is a gradual atrophy of all the elements of the nasal mucous membrane. The submucous tissue with its glands and vessels disappears in time.

Atrophic rhinitis—symptoms: The liquid discharge from the nostrils is small. On the contrary the secretions dry in the fossæ, and form crusts and scales which adhere more or less firmly to the mucous membrane. The nostrils are roomy and the passage of air through them free. If the crusts are allowed to accumulate, they begin to decompose and give a disagreeable odor to the air passing through the nostrils. From this arises the name fœtid rhinitis. This fœtor depends almost entirely on the lack of proper cleansing of the nostrils. The naso-pharynx and the pharynx are usually the seat of an atrophic inflammation at the same time.

Diagnosis: Careful inspection of the anterior and posterior

nares, showing the free nasal cavities, the pale appearance of the mucous membrane, and the presence of crusts, usually settles the diagnosis.

Prognosis: Cure is impossible, but amelioration of the symptoms can easily be brought about, and a little continuous care afterward will prevent their recurrence.

Atrophic rhinitis—treatment: This consists in cleanliness, and a stimulating application to the nose to keep the mucous glands secreting as much as possible. For purposes of cleanliness a nasal douche of warm water containing some alkali, as soda, salt, or borax, or Seiler's solution, may be used daily. This may be given by the ordinary fountain-syringe to which a nasal nozzle is attached, or by the Bermingham douche. After washing, some stimulating oily solution should be applied, as menthol or thymol, about ten grains to the ounce of sweet oil. If the fœtor is bad, listerine may be added to the alkaline douche. Any crusts that cannot be washed out should be removed mechanically with a cotton-wrapped probe.

MEMBRANOUS RHINITIS.

This is nothing more nor less than **diphtheria of the nose**, and on account of its importance as a source of infection in others it should be so considered.

Etiology: The *Klebs-Löffler bacillus* is the cause of almost all the cases of this disease.

Pathology: The nasal mucous membrane is highly inflamed, and its surface coated with a layer of false membrane. If the inflammation extends backward into the naso-pharynx, the cervical lymph-glands will be enlarged and tender.

Membranous rhinitis—symptoms: The *local symptoms* in the nose are the main ones, as, unless the inflammation spreads to the naso-pharynx, absorption is slight, and hence constitutional symptoms few.

There are obstruction of the nares, and serous, or mucous, or bloody discharge from the nostrils, and usually excoriation of the upper lip. On examination the nostrils are choked up, and the gray or dirty white membrane is easily seen covering the whole of the inside of the nose. It can be

removed only with difficulty, and leaves a bleeding place behind. The membrane may remain localized or may spread to the pharynx or larynx. In case it does not spread the membrane will come away piecemeal, or as a whole, in the course of a couple of weeks, and recovery will rapidly follow.

Diagnosis: This depends on the presence of a *false membrane* in the nose, and should be accompanied by a *bacteriological report* as to the presence or absence of the Klebs-Löffler bacillus.

Prognosis: As long as the disease remains localized in the nose, the prognosis is fairly good. If it spreads, the case takes on the characters of a general diphtheria.

Membranous rhinitis—treatment: The patient should be isolated, and the nose washed out two or three times a day with a warm weak solution of bichloride of mercury, about 1 : 50,000 or 1 : 30,000. The question of giving *antitoxin* will depend on the same rules as in general diphtheria. Constitutional treatment of other kinds will seldom be needed.

SYPHILITIC RHINITIS.

This is seen with some frequency in infants the victims of **inherited syphilis.**

Etiology: The disease is the result of a syphilis contracted in intra-uterine life, and manifesting itself during the first three or four months of extra-uterine life.

Pathology: The lesions are those of a catarrhal rhinitis. In places, superficial ulcers, the so-called *mucous patches*, form in the nostrils.

In other children the lesions are those of the tertiary stage of the disease, when *gummata* form and break down, with destruction of mucous membrane, cartilage, and bone. The septum may be perforated, the various bones may necrose, and marked deformities of the nose may follow. The so-called "saddle-back" nose is the most typical, where the bridge is sunken and the nose becomes very broad. These tertiary lesions are seldom confined to the nose, but the hard and soft palates are usually affected coincidently. After syphilitic

ulceration, cicatricial healing regularly takes place, with contraction of the scar-tissue subsequently and increase of the deformity.

Syphilitic rhinitis—symptoms: In the secondary form a coryza, or the so-called "snuffles," is the only symptom. The discharge from the nose is serous, or mucous, or bloody, and excoriates the upper lip.

In the tertiary form the physical signs, as described under pathology, together with a very offensive odor and some discharge, are the symptoms.

Diagnosis: The secondary form is differentiated from simple coryza mainly by its longer course and greater discharge. Other evidences of congenital syphilis should be looked for, and will usually be found. The family history will help at times.

In the tertiary variety the signs are more characteristic, ulceration and scars being often seen side by side.

Prognosis: This is that of syphilis in general, in children.

Syphilitic rhinitis—treatment: Constitutional treatment for syphilis should be at once instituted. Locally, the nose should be kept clean by an alkaline wash, and afterward an application of bichloride of mercury in weak solution or insufflations of calomel powder should be made.

NASAL POLYPI.

Nasal polypi occur only in older children, and are comparatively rare even in them.

Etiology: The causes are unknown; but polyps seem to be always secondary to, or at least associated with, chronic inflammatory conditions in the nose.

Pathology: The polyps are usually composed of a mixture of mucous and fibrous tissue, one or the other predominating. They are covered with ciliated epithelium. They are regularly pedunculated, and of a round form and pink color. They originate usually from the middle meatus.

Nasal polypi—symptoms: These are due mainly to the accompanying rhinitis, and are discharge and interference with the passage of air through the affected nostril. There

may be headache and sneezing, and frequent attacks of acute coryza.

Diagnosis: This depends on a careful examination of the nostrils and the discovery of the pale, soft pediculated growth. Frequently a number of polypi will be found at the same time.

Prognosis: Polyps are not serious, but are troublesome; and unfortunately, after removal, have a tendency to recur.

Nasal polypi—treatment: The polyps are best removed by the cold-wire snare passed around the pedicle and drawn tight. After removal the base, if it can be found, should be cauterized. The use of cocaine makes the operation easier. The accompanying chronic rhinitis should be carefully treated on the regular plan.

HAY FEVER.

Synonyms: This is also called *rose cold*, *autumnal catarrh*, and *hay asthma*. It is a condition of intense coryza, with which conjunctivitis is regularly, and asthma often, associated.

Etiology: It is not common except in older children. The cause is not known, but it is believed to be a neurotic predisposition, and the presence in the atmosphere of some irritant arising from vegetation. The cases regularly begin in the fall months, and usually at a very definite time. Certain localities seem perfectly free from the disease, and a patient going to one of these will be cured almost at once.

Pathology: The disease is functional; there are no lesions, but the irritated mucous membranes are temporarily in a state of acute inflammation with increased secretion.

Hay fever—symptoms: The disease begins suddenly, at a rather definite time of year, with sneezing, obstructed nasal breathing, a watery irritating discharge from the nostrils, lachrymation, and injected conjunctivæ. The nose itches excessively and the sense of smell is much impaired. These symptoms continue throughout the attack.

In certain cases, in addition to the involvement of the nasal and ocular mucous membranes, the bronchial walls and lining are invaded and the patient suffers from attacks of *coughing*, with scanty expectoration, and from attacks of asthma. The

difficult breathing is continuous day and night, but is often exacerbated and may become very distressing. The attacks last for a month or six weeks, and usually disappear with the first frost.

Diagnosis: The first attack may be a little difficult to recognize; but, as a rule, even in this the character of the disease is shown. Subsequent attacks should be quickly diagnosed.

Prognosis: It is not a serious disease; but it is difficult to cure an attack, and equally so to prevent subsequent ones. Many cases of undoubted cures are, however, reported.

Hay fever—treatment: During the attack much relief may be afforded by applications to the nose of atropine in 1 per cent. solution, or of cocaine in 2 per cent. solution. Some ointment, or even vaseline, applied to the nostrils prevents excoriations from the discharges.

In cases with asthma combinations of belladonna and iodide of potassium given internally produce relief. The inhalation of smoke from stramonium cigarettes is often helpful.

Between attacks the nostrils and naso-pharynx should be carefully searched for any abnormalities, and these, if found, should be thoroughly treated.

If no amelioration can be effected, the child should be sent away regularly to a region free from the disease during the time of its prevalence.

DISEASES OF THE LARYNX.

SPASMODIC LARYNGITIS.

This disease is also called **laryngismus stridulus** and *spasmodic croup.*

Etiology: This disease is a *neurosis* of the internal laryngeal muscles. It is seen most often in rachitic children or in those suffering from any form of malnutrition. In these conditions all the reflexes are markedly increased. Children of a neurotic heredity are often affected. *Local causes* are frequently found, such as adenoids, hypertrophied tonsils, elongated uvula, or acute inflammations of the throat, nose,

or bronchi. Indigestion, exposure to draughts, excessive exertion, and great emotion will often excite attacks.

Pathology: There are no lesions of this disease. The condition is a reflex spasm of the laryngeal muscles. The pathological changes of the predisposing diseases are found.

Spasmodic laryngitis—symptoms: The symptoms consist in sudden, unexpected attacks of interference with the passage of air through the larynx. The spasm seems at first to close the glottis completely, and during this time the child changes from a pale to a cyanotic color, throws back his head, and makes violent efforts to breathe. In about half a minute the spasm gradually relaxes, and this is shown by a noisy crowing or stridulous inspiration. Expiration is, however, more difficult. This may be the end, but recurrences in a few minutes or hours are to be expected. There may be a great many paroxysms during the day.

General convulsions and tetany are apt to complicate the attacks. There are all grades of severity to the paroxysm, from the mildest form which may be scarcely noticeable to a spasm so severe and prolonged as to endanger life. The tendency to recurring attacks lasts usually from a few weeks to a month or two.

Diagnosis: The disease must be differentiated from catarrhal laryngitis and from diphtheria of the larynx. The suddenness of the seizure and the intermissions in the attacks, together with the absence of fever and other signs of inflammatory stenosis of the larynx, suffice for a diagnosis. The general condition of the child also assists.

With a little care in following the history there should be no occasion to confuse the disease with whooping-cough.

Prognosis: The prognosis is good, although fatal cases do occur. The presence of general convulsions, the general condition of the child, and the severity of the paroxysm must be taken into consideration in estimating the chances for recovery.

Spasmodic laryngitis—treatment: To relieve the spasm, strong counter-irritation to the skin is quickly efficacious. This may be brought about by dashing cold water on the head and face or by putting the patient in a hot bath. In

more severe cases chloroform-inhalations may be used, and at times intubation may be necessary.

In the *intervals* between attacks antispasmodics should be given to prevent recurrences. The best drugs of this class are bromide of potassium, chloral, and antipyrin. They may be used separately or in combination, and should be given so as to keep up a continuous impression on the nervous system.

To remove the tendency to attacks, all measures directed to improving the child's general health, particularly its nutrition and digestion, should be carefully followed out. Fresh air, cool bathing, and proper food should be insisted on. Rickets must be treated, if present. The nose, naso-pharynx, and throat should be carefully examined, and any abnormalities that are found should be properly treated.

ACUTE CATARRHAL LARYNGITIS.

This disease is also called **catarrhal croup**, and, in its milder or severer form, is the *ordinary croup* from which children so frequently suffer.

Etiology: The disease commonly results from exposure to cold, or damp, or draught, in a child that is ordinarily too much protected from these influences. It occurs most commonly before the third year, but is seen even in much older children. Certain children have a marked predisposition to the disease, attacks recurring with great persistency. It often *complicates* influenza, measles, scarlet fever, or others of the infectious diseases.

Pathology: The mucous membrane of the larynx is hyperæmic and swollen, with—in the beginning—a diminished secretion, so that the membrane is dry. As the disease advances the secretion increases until it eventually becomes abnormally large in quantity. The swollen mucous membrane over the cords decreases the space of the glottis, and, as more or less muscular spasm usually accompanies the inflammation, the stenosis becomes more marked. In the severe and prolonged form, the trachea takes part in the inflammatory process.

Acute catarrhal laryngitis—symptoms: In the *milder cases*,

which are the ones ordinarily seen, about the only symptoms are a change in the *character of the voice.* This becomes hoarse and at times metallic. In young infants it can only be noticed when crying, but in older children the spoken voice also shows it. At times aphonia may be present. A cough of a dry, brassy character usually accompanies this. If the chink of the glottis is much narrowed, particularly if some adductor spasm is present, as so often is seen in these cases, there is more or less *interference with the breathing,* which is the alarming symptom of the disease.

The constitutional symptoms are slight, the temperature rising only a degree or two, and the child not feeling at all sick. These cases regularly have an exacerbation of the symptoms during the night, with remissions during the day.

In the *severe cases* the symptoms due to the larynx are the same, but intensified ; a croupy voice and cough, no expectoration at first, and marked interference with respiration. The inspirations are stridulous and the child seems to be suffocating. The skin becomes somewhat cyanotic and dyspnœa is very marked. The suprasternal and infracostal spaces sink somewhat in inspiration, and all the signs of laryngeal stenosis are present.

The constitutional symptoms are likewise marked. The temperature may be from 101° to 104° F., the pulse and respiration are rapid, and the child seems sick and prostrated. The symptoms increase in severity for two or three days, and then gradually diminish as the mucous glands begin to secrete and the inflammation to be resolved. The cases usually last about a week or ten days, unless bronchial or pulmonary complications set in, when they are much prolonged.

Diagnosis : Spasmodic laryngitis is distinguished by the absence of fever and constitutional symptoms, by its occurring usually in rachitis, and by its paroxysmal character.

The differentiation from membranous laryngitis is more difficult and more important. The dyspnœa in the membranous form is more constant, and does not show the daytime remissions seen in catarrhal croup. The presence of visible membranes elsewhere in the throat is almost certain proof of the membranous variety. Cultures can be made from the

pharynx and larynx for the Klebs-Löffler bacilli, even where no membrane can be seen. In doubtful cases where other children are exposed it is better to isolate the patient until the diagnosis can be made positive.

Prognosis: This improves with the age of the child. Although most of the cases recover, still caution must be used in prognosing, as very severe symptoms may develop, and any case may turn out to be of the membranous variety.

Acute catarrhal laryngitis—treatment: The child should be put to bed in a room with a uniform temperature, in which the air is artificially moistened by steam, and put on a lighter diet than it normally takes. The clothing should be sufficiently warm, and the neck should be rubbed with camphorated oil or turpentine, and then bound up with flannel. A hot mustard foot-bath may also be given. The bowels should be opened by fractional doses of calomel given hourly. If the case is in any way severe, the child should be under a tent and breathe steam plain, or medicated with turpentine, creosote, or compound tincture of benzoin. *Internally*, the so-called expectorants are indicated, of which the best are ipecac and antimony. They are best given in doses just short of producing emesis, frequently repeated so as to keep up a continuous effect. A convenient method is by use of the tablet containing $\frac{1}{100}$ grain each of ipecac and antimony, or combinations of the wines of ipecac and antimony with ammonium chloride may be used. At times there seems value in giving repeated doses of pilocarpine until the laryngeal mucous membrane begins to secrete. If the spasmodic element is marked, chloral, or the bromides, or antipyrin, are useful additions and are indicated. In very severe cases where medicinal measures do not relieve the stenosis, recourse must be had to intubation.

To prevent future attacks these children should have the nose and naso-pharynx and pharynx carefully examined and any abnormalities found treated. Such lesions as adenoids or enlarged tonsils are often found, and act as predisposing causes. Each day the child should be given a cool bath, and it should be taken out of doors daily and accustomed to cool, fresh air. The clothing should be comfortably warm, but

not too heavy. The tendency to croup can be completely removed in these children by attention to these little details.

MEMBRANOUS LARYNGITIS.

Synonyms for this are true croup, or membranous croup, or laryngeal diphtheria. The latter particularly must be remembered, as in the vast majority of the cases the whole disease is diphtheria with its lesion located in the larynx.

Etiology: While infection by the Klebs-Löffler bacillus is, in the great mass of the cases, the cause of this disease, still pseudomembranes may rarely form in the larynx from irritation produced by the streptococci, and at times after intense chemical irritants, as lye.

Pathology: There is an inflammation of the laryngeal mucous membrane so intense that a new false membrane is formed on its surface and attached quite intimately to it. The false membrane by its mechanical presence blocks up the opening between the cords, which is already diminished in size by the swelling. In this false membrane the Klebs-Löffler bacillus is easily demonstrated, together with colonies of streptococci.

Membranous laryngitis — symptoms: The constitutional symptoms, particularly early in the disease, are quite secondary to the local symptoms from the larynx. The disease begins gradually with a steadily increasing diminution in the calibre of the glottis, but with very slight or no remissions, thus distinguishing the catarrhal variety. They have the croupy voice and cough, and the stridulous, noisy respiration. Later on aphonia may develop and the stenosis become marked enough to produce general cyanosis of the patient.

In the meantime the fever is slight and the pulse only a little fast. The respiration is more rapid. As the dyspnœa increases restlessness and excitement and a struggle for air are prominent symptoms. There is no enlargement of the lymph-glands in the neck. All these signs presuppose the presence of pseudomembrane only in the larynx. If the result is to be favorable, after four or five days the cough grows looser, the voice returns, the breathing is easier, and

often fair-sized pieces of false membrane are spit up. If the end is fatal, all the dyspnœic symptoms increase in severity, and cerebral symptoms become especially prominent, as restlessness, delirium, and convulsions; or stupor and coma. The cases are rapid in their course, recovery or death usually being certain by the end of a week.

The spreading of the membrane to the lung, setting up bronchitis or pneumonia, adds an important element to the symptoms of the disease.

Diagnosis: This is often quite difficult to make in the beginning. The presence of membrane elsewhere in the throat and the lack of general symptoms are of most importance. Bacteriological examinations should be made in any doubtful case.

Prognosis: This is very serious, but since the advent of antitoxin the mortality has been very considerably reduced.

Membranous laryngitis—treatment: The child should be put to bed and isolated from other children and properly fed and stimulated. In preantitoxin days, emetics, steam inhalations, and calomel fumigations were used as the medical treatment of this disease. At the present time they may be used as aids to the specific antitoxin treatment, but are never to be relied on alone. Emetics that are of value are ipecac, or antimony, or turpeth mineral given in small doses repeated frequently until vomiting is produced. Plain or medicated steam inhalations are used in the same way as in catarrhal laryngitis, under a tent. Calomel fumigations are given by subliming ten to twenty grains of pure calomel over an alcohol lamp, the calomel being placed in a porcelain receptacle. The child and the fumigating apparatus should be under a tent, and the only caution necessary is to prevent accidents by fire. Various forms of apparatus are sold for this purpose, but by a little ingenuity a simple and efficient home-made one may be devised. The fumigations are usually repeated every three hours, watching results. The *antitoxin* should be given early, in good-sized dose, and repeated in twelve hours, according to results. In cases of doubtful diagnosis it should be given without waiting for the case to develop to a certainty, as, should the case turn out to be some other form, no harm has been done.

Notwithstanding antitoxin and medical treatment, very many of these cases require mechanical help to overcome the laryngeal stenosis, and it is due to these only in many cases that the child is saved from absolute suffocation. Mechanical measures or operative procedures are indicated by a steadily increasing dyspnœa, restlessness, cyanosis, and retraction of the suprasternal and infracostal regions. It is always a matter of some anxiety to know when the time for operation is necessary, and there is no good rule to follow. It is, however, better to perform it too early than too late, after the child is exhausted by its struggles for air and the tissues are poisoned by the venous blood. Of the two operative measures employed, *intubation* and *tracheotomy*, the evidence in favor of the former is steadily increasing, and tracheotomy is now very seldom used as a first recourse, but only in certain cases where intubation cannot be performed, or where it may have failed. Intubation has many advantages over tracheotomy. It is simple, it is more easy to obtain the parents' consent to an early operation ; it has no prejudicial effects on the throat in forming a wound for fresh infection ; if it fails, tracheotomy can be done as easily afterward as before, and there is no need of an anæsthetic. The only objections are the difficulty in feeding some children who are wearing tubes, and the rare accident of crowding false membrane into the trachea ahead of the tube and spreading the infection, and the possibility of not having the apparatus.

Intubation consists in the passing into the larynx through the mouth, and leaving there, a tube through which the air passes in entering and leaving the lungs.

The **O'Dwyer apparatus** is the best one for use. It consists of various sized tubes, an introducer, an extractor, a mouth-gag, a gauge for deciding the size of the tube required, and some strong silk.

To insert the tube the child should be wrapped, arms and all, in a blanket or sheet, and then held upright by the nurse with his head resting against her chest. The gag is inserted in the left side of the mouth. The operator holds the tube on the introducer in his right hand, the silk being first passed through an eye in the tube and the ends held by the same

hand. He then passes his left index finger over the epiglottis, and thus locates the upper part of the larynx. With this finger as a guide he passes the end of the tube through the space between the cords and pushes the tube easily into place. The introducer is then removed, leaving the tube with its attached silk *in situ*. If the intubation has been successful, the relief to the dyspnœa is immediate, and some attempts at coughing are usually made. If unsuccessful, the tube is again affixed to the introducer and again introduced. After the child quiets down from the intubation and becomes accustomed to the tube, the silk may be removed. If it is left, it should be fastened to the cheek by adhesive plaster. After a successful intubation usually very few troubles arise. One is the coughing up of the tube. This is remedied by passing the next larger size.

Feeding with the tube in place should usually be done with the head held lower than the body, or at least lying down; and, if possible, semi-solid food rather than liquids should be used. If the tube becomes blocked, it is usually coughed up by the patient; but if not, it must be quickly extracted. During the wearing of a tube the child should be carefully watched to prevent accidents, and the physician should be within easy call to replace a coughed-up tube or to remove an offending one.

In **removing a tube** the child is held in the same way as in introduction. With the left index finger the head of the tube is located, and on this as a guide the extractor is passed into the lumen of the tube and the tube removed.

The **time for removal** is difficult often to decide. Await the subsidence of the disease before trying it. Often after removal it will have to be reintroduced, and worn for a few days longer before it can be safely left out.

Tracheotomy is sometimes necessary, and is performed in the way regularly described in all the text-books on surgery.

CHRONIC LARYNGITIS.

Chronic inflammations of the larynx are found with some frequency in infants and children.

Etiology: Most of the cases follow attacks of acute laryngitis, and are apt to be associated with chronic catarrhal conditions elsewhere in the throat. Other cases are due to syphilis and others to tuberculosis.

Pathology: In the *simple catarrhal cases* and in the early syphilitic ones, the mucous membrane is in a state of chronic inflammation, with at first an increased and later a decreased secretion, and hence abnormal dryness. The *later syphilitic form* attacks the deeper tissues of the larynx, producing ulceration and deformity.

The *tubercular cases* show thickening of parts of the larynx by tubercular infiltration, and later necrosis of this new tissue with ulceration.

Chronic laryngitis—symptoms: These are hoarseness, a dry cough, more or less aphonia, and a thick, sticky, but not profuse, expectoration. In the *tubercular* variety, tubercle bacilli may be found in the sputum, and there is severe pain on swallowing.

Diagnosis: This depends on the above symptoms, in addition to the physical signs seen on examination. Ulceration, with involvement of the arytenoid areas, usually means tuberculosis. General ulceration with scar-tissue present at the same time usually means syphilis. Evidence of either disease elsewhere assists the diagnosis. The simple variety simply shows a general thickening of the cords, with often dried secretion on them.

Prognosis: The simple form can usually be cured by removing the cause. The tubercular form usually complicates tuberculosis elsewhere, and the prognosis is that of tuberculosis in general.

The syphilitic form can be regularly cured; but in the tertiary variety, after extensive ulceration, the larynx is apt to be left badly deformed.

Chronic laryngitis—treatment: In the *simple form*, attention to the whole pharynx is important. Any chronic conditions there must be treated. The larynx should be touched daily with astringent applications, or by weak solutions of nitrate of silver. Inhalations of turpentine or of tar are

valuable. Internally, expectorants such as ammonia and ipecac may be given.

In the *syphilitic variety*, the main reliance is to be placed on antisyphilitic treatment. Local applications of a cleansing nature often aid. With marked cicatricial contraction intubation is often necessary.

In the *tubercular variety*, cleansing applications should be made, and the ulcers touched with nitrate of silver, or lactic acid, or dusted with iodoform. If pain on swallowing is marked, applications of cocaine give temporary relief. Special means should be taken to build up the general health, and to overnourish the child.

PAPILLOMA OF THE LARYNX.

Papilloma of the larynx is found with some frequency in children, and at times even in infancy. Nothing is known as to its causation.

It consists of one or more warty growths from the neighborhood of the cords. The tumor is either sessile or pedunculated, and presents a pinkish appearance.

Papilloma of the larynx—symptoms: The symptoms are hoarseness, going on to aphonia, an irritating cough, and a gradually increasing dyspnœa as the growth of the tumor blocks up the larynx. The symptoms progress slowly, as the tumor grows slowly.

Diagnosis: This depends on direct examination with the laryngoscope if possible. If not, much can be learned by palpation of the larynx with the index finger.

Prognosis: This is not good, as the tumor is apt to recur after removal, and seems at times to take on almost a malignant character.

Papilloma of the larynx—treatment: In some older children they can be removed by endolaryngeal operations through the mouth. Most cases require *thyrotomy* and the excision of the growths. In some cases tracheotomy and leaving the growth entirely alone is all that can be done.

ŒDEMA GLOTTIDIS.

Definition: By this is meant an effusion of serum into the submucous tissue of the larynx.

Etiology: It is secondary to disease elsewhere, as nephritis, and occurs often as a complication of the exanthemata. It also results from intense local irritation, as the inhalation of hot vapors, the swallowing of corrosive liquids, and from ulceration and foreign bodies.

Pathology: The cellular submucous tissue is infiltrated with serum and with new cells. The larynx, especially in the aryepiglottic folds, is intensely swollen.

Œdema glottidis—symptoms: The characteristic symptom is inspiratory dyspnœa, expiration being comparatively easy. If any local inflammation is present, there is intense pain. The symptoms develop rapidly, and in a few hours the patient may be in danger of death from suffocation.

Diagnosis: A laryngoscopic, or digital, or at times a mere visual, examination will settle the diagnosis.

Prognosis: With proper treatment this is good. Untreated, the mortality is high.

Œdema glottidis—treatment: The œdematous tissues must be freely scarified, and *ice* must be given to swallow, and also packed externally around the throat. In some cases tracheotomy is necessary. Intubation is not, as a rule, very successful.

FOREIGN BODY IN THE LARYNX.

This **occurs** from time to time, usually as a result of a child laughing, or crying, or coughing, while holding some foreign body in its mouth. It may lodge in the larynx, or trachea, or drop down into a bronchus, usually the right one.

Symptoms: Violent paroxysmal cough and dyspnœa, with often localized pain and hæmoptysis, are the regular results of this accident.

Diagnosis: This depends on the history and symptoms. In a few cases the object may be seen by the laryngoscope.

Prognosis: In many cases the body is eventually expelled. It may remain and be encapsulated, causing no trouble; but

in no case can a positive prognosis, good or bad, be given. There is always danger from its presence.

Foreign body in the larynx—treatment: The patient should be inverted and shaken, and in some cases the body will be expelled. If impacted in the larynx or trachea, it may be removed by properly constructed forceps either through the mouth or after tracheotomy.

DISEASES OF THE BRONCHI, LUNGS, AND PLEURA.

ACUTE BRONCHITIS.

Definition: This very common condition in infants and children is an inflammation of the mucous membrane lining the smaller and larger bronchi.

Etiology: It commonly follows exposure to cold and dampness. It occurs most often during the winter months, especially when the changes of temperature are sudden and marked. Certain children seem predisposed to the disease, especially those who have frequent attacks of coryza and sore throat. It is often secondary to the infectious diseases, particularly to influenza, measles, and whooping-cough. It is common in children suffering from any form of malnutrition, especially rickets. *Micro-organisms* undoubtedly play a prominent part in the causation of this disease.

Pathology: The small or the large, or both kinds of *tubes* may be involved, and regularly those of both sides. The mucous membrane is congested and swollen, the superficial epithelium is shed, and the glands secrete an excessive quantity of mucus and muco-pus, due to the inflammatory exudate.

Acute bronchitis—symptoms: The disease begins gradually, and frequently after a preceding coryza, with fever, cough, and malaise. The respiration is rapid, and the child is restless and fretful. In the milder cases these symptoms last about a week and recovery occurs.

In *more severe cases* all the above symptoms are intensified, the temperature runs higher, the cough becomes distressing, and the respiration quite rapid and difficult, but its normal

rhythm is retained. The pulse is rapid and the child decidedly prostrated. In the early stages the secretion from the bronchi is scanty ; and later, when it increases, the child swallows what it coughs up into the throat. Expectoration is a habit formed later in life, the young child always swallowing its sputa. The appetite is commonly lost ; there may be vomiting and diarrhœa. In these severe cases the disease lasts for a couple of weeks, and may be protracted even over a longer time.

Acute bronchitis—physical signs : In the very beginning there are often no physical signs, save the very rapid respiration and the movement of the alæ nasi. After a day or two both chests are full of sibilant and sonorous breathing. Later, as the secretion becomes loose, this breathing is replaced by very numerous fine or coarse râles. The kind of râle depends on the size of the bronchi in which it is formed. As the disease advances the râles grow looser and fewer, and gradually disappear.

Diagnosis : This depends on the history and the physical signs. The only difficulty is in excluding broncho-pneumonia, and in many cases this may be impossible.

Prognosis :—In children of good physique this is good. Delicate children, those the subjects of rickets or of the infectious diseases, and very young infants bear the disease badly. In young infants the mucus is very apt to block up the bronchi and to impede respiration seriously.

Acute bronchitis—treatment : The child should be kept in the house in an equable temperature, and put on a light, easily digestible diet.

It should be given first fractional doses of calomel repeated till the bowels move. The chest should be rubbed with some mild counterirritant, as camphorated oil, or mustard paste may be applied. *Internally* a prescription containing small doses of an expectorant, such as ammonium chloride and ipecac, may be given and repeated frequently.

Inhalations of steam, containing turpentine or creosote, are often useful. *Stimulants* are frequently necessary, and brandy or strychnine may be used. Opium should be given sparingly in this disease.

In cases of sudden collapse, or of respiratory feebleness, nothing acts so quickly as a stimulant as a hot mustard bath.

CHRONIC BRONCHITIS.

Etiology: This condition is not so common in children as in adults. It regularly follows an attack of acute bronchitis in a patient who has a poor constitution, or is weak from some disease which the acute bronchitis complicated.

Pathology: The bronchial mucous membrane is in a condition of chronic catarrhal inflammation, with an excessive production of mucus.

Chronic bronchitis—symptoms: A troublesome, persistent cough, with the raising of a great deal of mucus, are the regular symptoms of this disease. The cough is apt to be worse in the morning and at night. There are no fever and no malaise, and very little interference with the general health.

Chronic bronchitis—physical signs: These are simply the presence of coarse mucous râles scattered over both lungs.

Diagnosis: This depends on the cough and the signs in the lungs. Tuberculosis must be excluded by the difference in the physical signs and the absence of tubercle bacilli from the sputum.

Prognosis: This is good, as most of these children by intelligent treatment can be cured.

Chronic bronchitis—treatment: General strengthening, constitutional treatment is of most importance. All measures to improve the child's general health should be carefully followed out. These children should not be confined to the house, but should have the freedom of the fresh air and sunshine in good weather. A change of climate for a short time will often be very beneficial. As *drugs*, creosote, tar, or terebene seem of most value. Iron and cod-liver oil are especially helpful.

FIBRINOUS BRONCHITIS.

Definition: This rather rare disease consists in the formation in the bronchi of false membranes, which form casts

of the tubes in which they occur. Some cases are undoubtedly diphtheritic, but others are not so, and their etiology is unknown.

Pathology: The trachea and large bronchi are usually involved, but the inflammation may reach down into the bronchioles. The mucous membrane is swollen and congested, and coated with a layer of coagulated fibrin, which may be free in the lumen, or attached to the surface of the bronchi.

Fibrinous bronchitis—symptoms: The symptoms are those of a severe catarrhal bronchitis, acute or chronic, as the case may be. They differ mainly in the severity of the dyspnœa, which in the croupous variety is excessive.

The *physical signs* are likewise the same.

Diagnosis: This is made by the expulsion and recognition of the fibrinous casts of the tubes. Otherwise the diagnosis from ordinary bronchitis is impossible.

Prognosis: This is bad, as considerably over half the cases die.

Fibrinous bronchitis—treatment: This is the same as for the catarrhal variety. Inhalations of oxygen may be of service in the excessive dyspnœa. If the diagnosis of the fibrinous form is made, an emetic may assist in the expulsion of the membrane.

With the possibility of the disease being diphtheritic *antitoxin* might be used to aid in loosening the cast.

BRONCHO-PNEUMONIA.

Synonyms and definition: Other names for this common children's disease are catarrhal pneumonia, lobular pneumonia, and capillary bronchitis.

It is an inflammation involving both the terminal bronchioles and the air-vesicles. There is consequently a lobular distribution to this inflammation.

Etiology: This is the usual "pneumonia" of childhood. It may be primary, as after exposure to cold, or damp, or draughts; but is more commonly secondary to bronchitis, or measles, or whooping-cough, or diphtheria, or influenza. It

attacks previously healthy children, but more often those of feeble constitution. *Micro-organisms* of one form or another are the actual exciting cause.

Pathology: The inflammation involves the entire thickness of the bronchial wall, and not the mucous membrane only. The terminal bronchi are those usually attacked, and the air-vesicles connected with them and those surrounding them are involved in the inflammatory process. The walls are thickened and infiltrated by new cells; the air-vesicles are filled with exuded fibrin, pus, and epithelium, and in places this is changed into new connective tissue. Parts of both lungs are regularly involved. Catarrhal bronchitis, pleurisy, atelectasis, and diffuse consolidation of portions of the lungs are ordinary complications. This inflammation is such that resolution is slow, and often it may become chronic.

Broncho-pneumonia—symptoms: The disease, whether it be primary or follow a preceding inflammation elsewhere, begins gradually with a slowly rising temperature, an increasing rapidity of the pulse and respiration, and a cough. The *temperature* runs irregularly, with remissions and exacerbations, the highest point reached being ordinarily 103° or 104° F. The fever subsides gradually, by lysis.

The *pulse* goes to 130 to 150 beats to the minute. The *respiration* is excessively rapid and shallow, and may reach 50 to 75 per minute. The respiratory rhythm is changed, so that the breath is held after inspiration, giving the so-called grunting respiration. The pulse-respiration ratio is entirely lost. The *cough* is short and dry, and quite incessant. Later it is looser and easier, but expectoration is uncommon, the sputa being swallowed.

The *face* and *skin* are suffused and cyanotic, the alæ nasi are playing freely. The *tongue* is coated and dry. There may be *vomiting* and *diarrhœa*. All the cases are restless, and in some the *nervous symptoms* are especially marked, with delirium, or stupor and coma, and often convulsions. A good many cases are ushered in by general spasms. *Prostration* is marked in all the severe cases.

If the case is to go on to recovery, a gradual improvement in all the symptoms occurs, but convalescence is extended over

a considerable time, and the duration of the attack is quite indefinite.

In other cases the acute symptoms gradually subside, and the disease protracts itself into a chronic state.

The bad cases increase in severity, and die exhausted, or from respiratory failure, or from convulsions, before the second week.

Broncho-pneumonia—physical signs: If only the walls of the bronchi and of the air-spaces are involved, there will be no physical signs at all. If enough neighboring air-spaces are inflamed to produce patches of consolidation, we have areas of *dulness* on percussion, *increased fremitus*, and *bronchial voice* and *breathing*.

If a catarrhal bronchitis is present as a complication, which is very common, we find the fine or *coarse mucous râles* of this disease. The smaller and the finer the râles, the nearer to the air-vesicles they are made, and the more the probability, in the absence of signs of consolidation, of the presence of broncho-pneumonia.

If any pleurisy is present, we find *crepitant râles* or a *friction-rub*.

Diagnosis: In some cases the diagnosis must be made by the presence of fever, rapid breathing, cough, and prostration.

Catarrhal bronchitis often gives exactly the same signs, but broncho-pneumonia has more fever and more prostration. Without signs of areas of consolidation the differentiation cannot be positive.

From lobar pneumonia the diagnosis is fairly easy, the history and physical signs of the two being quite different.

The only important difficulty is in excluding tubercular broncho-pneumonia in the protracted cases. This should always be borne in mind, but often only time will decide the question.

Prognosis: A serious prognosis must always be given. Certain conditions add decidedly to the mortality-rate. These are age and previous condition of health. The younger the infant the worse the prognosis. Previous disease, as measles, pertussis, diphtheria, or influenza, or bad nutritional states,

as rachitis, add to the dangers. The amount of lung involved and the presence of a large amount of bronchitis are elements to be considered in estimating any particular case.

Broncho-pneumonia—treatment: As *prophylaxis*, measures should be taken in all cases of bronchitis and the infectious diseases to prevent the development of broncho-pneumonia. Proper attention to the air of the sick-room, and to the mouth and throat, and to the treatment of these diseases will prevent many cases of broncho-pneumonia.

If the disease has developed, the child must be put to bed in a room with an equable temperature of about 70° F., and kept on easily digestible but nutritious diet. Special attention should be given not to overload the stomach. The child should not be allowed to lie in one position for any length of time, as this favors hypostatic congestion of the lungs. There seems to be some value in keeping the air in the room moist by allowing water to boil over a fire.

The child's chest should be rubbed twice a day with camphorated oil or diluted turpentine, and then kept covered with a cotton jacket. If stronger counter-irritation is needed, repeated applications of mustard paste made with flour, in strength of 1 to 6 up to 1 to 2, may be used.

In the beginning of the disease fractional doses of calomel, frequently repeated, should be given. Following this, doses of ammonium chloride and ipecac, in syrup of tolu, or simple elixir, should be given frequently enough to keep up a continuous action on the bronchial mucous membrane but not to nauseate, throughout the disease.

Inhalations of tar or of creosote seem to have some value. Stimulants will usually be needed after the disease has run a few days. Alcohol and strychnine are the best. Atropine may be used to stimulate the respiration.

The fever will seldom need any treatment; but if it should, *cool sponging* should be our resource, rather than antipyretics internally. If the child is very restless and the nervous symptoms are marked, small doses of opium, or of bromide, may be used. As a rule, the less opium given in this disease the better. In sudden attacks of collapse, hypodermics of caffeine, or of camphor, or of nitroglycerin, may be used.

Immersion in hot water is, however, a quicker and more reliable stimulant than drugs.

If the disease becomes protracted, attention to the general health and a *change of climate* are our main resources. Iron, quinine, and cod-liver oil, as general tonics, are of most value. Creosote should also be given in slowly increasing doses.

LOBAR PNEUMONIA.

Definition: This disease is also called croupous or fibrinous pneumonia, and while broncho-pneumonia is the ordinary variety as seen in children, still the *lobar form* occurs with more frequency in them than is usually supposed.

Etiology: The older the child the more frequent is this form of pneumonia. It is most frequent during the cold months of the year. It is due in the large number of cases to the presence of the *diplococcus pneumoniæ*. The disease at times seems to be somewhat epidemic.

Pathology: The lower lobe of one or the other lung is most often involved, although the upper lobe may be attacked. At times more than one lobe in the same lung is affected, or even parts of both lungs.

The disease goes through the same stages as in adults, first that of *congestion*, in which in the affected lobe the air-cells contain the products of inflammation, and their walls are swollen and thickened.

The second stage—of *red hepatization*—follows, in which the above process is simply so intensified as to cause a complete consolidation of the affected part. This is red, and resembles liver on cut section. During this stage the pleura over the affected lobe becomes inflamed and is coated with fibrin.

This is followed by the third stage—that of *gray hepatization*—in which the inflammatory exudate begins to soften and break down. The red color is changed to gray, but the consolidation remains.

Lastly comes the *stage of resolution*, in which the exudates become still softer and more liquid, and are gradually removed by absorption and expectoration. After this stage is passed the lung returns to its original condition.

Lobar pneumonia—symptoms: The disease begins quite suddenly with chilly sensations, a rapid rise in temperature, pain in the chest, and cough. In many cases the onset is announced by convulsions or vomiting.

The *temperature* soon reaches about 105° F., and remains there with very slight remissions until the disease is over, when it falls quickly, by crisis. The *respiration* is accelerated to 40 or 50 per minute, and is usually painful. The *pulse* becomes rapid, 120 to 140 per minute, but should be strong and full. The appetite is lost, and the *bowels* may be loose or constipated. There is apt to be rather a *lively delirium* throughout the fever. The *cough* is persistent and frequently painful. In the beginning the *expectoration*, if not swallowed, is a stringy, sticky, blood-stained mucus; later it grows looser, and consists of little yellowish lumps. During the whole course of the disease the child sleeps very little.

The disease lasts with very little change in its character for a week or ten days, when the temperature rapidly drops to normal by the so-called crisis. With the fall in temperature is a coincident drop in the rate of the pulse and respiration to nearly normal. In some cases is a *pseudo-crisis* with some fall of temperature and remission in the symptoms a day or so preceding the real crisis. It is just before the crisis that the sudden attacks of heart-failure, that are such a serious feature of lobar pneumonia, develop. Some cases, instead of defervescing at the regular time, go on with the same symptoms, this indicating usually a spreading of the disease to new portions of the lungs.

Lobar pneumonia—physical signs: During the *congestive stage* there is slight *dulness* on percussion, while the breath- and voice-sounds are somewhat exaggerated. Over the same area a few *crepitant râles* are usually heard.

During the *stages of hepatization* there is distinct *dulness* on percussion over the consolidated area. The *vocal fremitus* is markedly *increased*, and both the breathing and the voice are *bronchial*. *Râles* are usually absent.

As the stage of resolution appears the *dulness diminishes*, the breathing and voice gradually take on their normal quality, and the *crepitant râle*, the *râle redux, returns*. Later,

coarser râles take their place, and are present until the lungs return to normal.

Signs of dry pleurisy, or of fluid in the chest, or of pericarditis, are often found as complications.

Diagnosis: *Broncho-pneumonia* and *pleurisy with effusion* are the two diseases most likely to be confused with lobar pneumonia. The history and physical signs, if carefully followed, will usually prevent any error being made.

Prognosis: The prognosis in children is much better than in adults. The great danger depends on failure of the heart, and in children this does not occur so readily as in adults. The height of the temperature and the extent of lung involved are of great importance in estimating the prognosis. The danger of post-pneumonic complications, particularly pleurisy with effusion and empyema, must be remembered.

Lobar pneumonia—treatment: The child should be kept absolutely quiet in bed from the first. The food should be liquid, and given in small quantities frequently repeated.

If there is *pain in the chest*, the application of mustard paste and a cotton jacket are of value. For the *restlessness* and *cough*, small doses of *opium* are allowable. For the *temperature* it is best to do nothing. If its height seems detrimental to the child, *cool sponging* is the best way of bringing it down. If the *heart's action* is too excessive, small doses of *aconite* may be given. The *main reliance* must be put on *heart-stimulants*. They will always be necessary just before the crisis, and had better be used in moderation throughout the disease. The best of these are *alcohol* and *strychnine*. Digitalis is often used, but is not so reliable as the above. They should be used in full dose. The *inhalation of oxygen* is useful in certain cases of *excessive dyspnœa*. During convalescence, general tonic treatment is indicated.

INTERSTITIAL PNEUMONIA.

Definition: This condition is also called chronic broncho-pneumonia and fibroid phthisis. Clinically it is a rather indefinite disease and difficult of diagnosis.

Etiology: It follows attacks of broncho-pneumonia, of

chronic pleurisy, and of bronchitis. Its connection with a precedent cured tuberculosis is in doubt. It is a comparatively rare disease of childhood.

Pathology: The disease is regularly unilateral, and consists in a substitution of connective tissue for the normal pulmonary tissue throughout the affected lung. The pleura on the lung is thickened and adherent, and the lung is filled with bands of dense fibrous tissue. In fact, there is a growth of new connective tissue all through the lung. The walls of the bronchi are thickened or thinned, and in places the bronchi are much dilated, forming *bronchiectases.*

Interstitial pneumonia—symptoms: Following the history of the acute disease is the persistence of the symptoms of *cough*, *expectoration*, *dyspnœa* on exertion, and at times *pain in the chest.* The patient does not gain in flesh or strength, but remains thin and feeble. The cough is troublesome and the expectoration usually profuse and muco-purulent. Hæmoptysis may be present. Fever is ordinarily not present. The disease lasts for months and years. If only a small area of the lung is involved, the inflammation may stop, and the patient recover. If the whole lung is involved, the patient will probably die, exhausted, after a long time. Often a tubercular inflammation is engrafted on the interstitial change.

Interstitial pneumonia—physical signs: These are not distinctive. The *chest is retracted.* There is *dulness* on percussion, or *flatness.* The *vocal fremitus* is *increased.* The breathing-sounds are feeble, or bronchial, or cavernous in cases where bronchiectatic cavities are found. Various forms of *râles* are usually present.

Diagnosis: The diagnosis of this disease from *tuberculosis* is of special importance, and is extremely difficult to make. The history and physical signs of the two diseases are almost identical. The diagnosis usually rests on frequent examinations of the sputa for tubercle bacilli.

Prognosis: This is bad. Some cases with an involvement of only a small area of lung recover. Life may be prolonged over very many years.

Interstitial pneumonia—treatment: Climatic and hygienic

treatment are of special value, and the only ones on which we can place much reliance. The child should live where he can be out of doors most of the time. Cod-liver oil and creosote seem to have some value.

PULMONARY EMPHYSEMA.

Definition: The variety occurring regularly in children is the so-called *vesicular emphysema*, in which the air-spaces are abnormally dilated, but without any of the chronic inflammatory changes seen in adult life.

Etiology: The cause is either *compensatory*, in which portions of the lung are overdistended, because other parts are disabled; or *obstructive*, where something mechanically prevents the lung from emptying itself properly. In other words, emphysema in children rarely exists unless complicated by some other form of disease in the lung.

Pathology: The causative disease produces its lesions, and in the parts affected by emphysema the air-spaces are found dilated and their walls thinned. In some the walls are ruptured.

Pulmonary emphysema—symptoms: There are no distinctive symptoms of this disease, as those of the primary condition completely conceal any slight ones that might be produced by the emphysema. *Dyspnœa*, persistent or in attacks, belongs to this disease, but is regularly present before the emphysema develops.

Pulmonary emphysema—physical signs: These are likewise liable to be concealed by the primary disease. The emphysematous areas give a tympanitic percussion-note, with feeble breathing-sounds and prolonged, low-pitched expiration.

Diagnosis: This is rarely made in dealing with children.

Prognosis: If the original disease is cured, the emphysema is regularly recovered from also.

Treatment: This is that of the original disease. There are no special measures for the emphysema itself.

ASTHMA.

Definition: This is also called *bronchial* or *spasmodic asthma*. It is a paroxysmal dyspnœa in which, during the

intervals between the paroxysms, no evidences of disease of the lungs are present.

Etiology: The disease occurs with some frequency among older children, and seems specially to attack those of a gouty or neurotic heredity. The disease is looked on as a *vaso-motor neurosis* without lesion, but in certain cases the presence of some abnormality in some part of the respiratory tract seems to act as the exciting cause. Hypertrophied turbinates, adenoids, enlarged tonsils, elongated uvula, bronchitis, or enlarged bronchial glands are such causes. The inhalation of irritants as in hay-fever, and gastro-intestinal disturbances often provoke an attack.

Asthma—symptoms: The attack regularly comes on suddenly without warning, and often in the night. It consists of intense dyspnœa and air-hunger. The child sits up or stands bracing its arms, to bring into play the accessory muscles of respiration. The skin becomes bluish, the eyes prominent, and the alæ nasi widely dilated. The skin may be covered with perspiration. The respiration is noisy and wheezing, and the rate is rather under than above normal. The pulse is rapid and small, and the temperature ordinarily normal.

After a few hours the attack gradually subsides and the respiration becomes regular and easy, and the patient drops to sleep exhausted. At times the attack lasts days instead of hours. Recurrences are to be expected.

Asthma—physical signs: The chest is found fairly fixed. On percussion there is a rather *hyperresonant note*. The inspiration is short and jerky; the expiration is very prolonged and wheezing. The whole chest is full of sibilant and sonorous breathing. If bronchitis exists, moist râles are heard.

Diagnosis: This is not difficult. After the attack is over a searching examination of the heart, kidneys, lungs, and upper air-passages should be made to determine the cause if possible.

Prognosis: The paroxysm is almost invariably recovered from. The prognosis for complete recovery from the attacks is fairly good, especially if climatic treatment can be taken advantage of.

Asthma—treatment: *During the paroxysms* the best treat-

ment is the *inhalation of the fumes of saltpetre paper*, or of the smoke of *stramonium cigarettes*. A hypodermic of a small dose of morphine and atropine will cut short an attack, but had better not be used except under exceptional circumstances.

Between attacks the child should be put in the best hygienic condition and kept in a good climate. Any abnormalities in the respiratory tract should be carefully treated. As *drugs*, belladonna, iodide of potassium, and cod-liver oil are of most value. Antipyrine and chloral in small doses regularly seem to have a good effect in some cases.

PULMONARY GANGRENE.

This is a **localized necrosis** of the pulmonary tissue, and is rare under all circumstances, being rather commoner in children than in adults.

Etiology: It is almost invariably secondary to pneumonia, septic infections, or some equally intense inflammation in the lung. It is seen usually in children of feeble constitution.

Pathology: The process may be circumscribed or diffuse. The gangrenous patches are greenish-looking and emit an intensely foul odor. There is regularly found a thrombosis of the vessels leading to the necrotic mass, and this is consequently wedge-shaped. Putrefactive bacteria are present in large numbers. If the gangrene is on the surface, as it usually is, there is regularly present a complicating pleurisy.

Pulmonary gangrene—symptoms: Those of the primary disease are always present and mask the symptoms of gangrene. The distinctive features of this condition are the *gangrenous odor of the breath* and the *expectoration of necrotic masses* of tissue from the lungs. Irregular fever, sweating, cough, and emaciation are present, but do not point especially to gangrene.

Physical signs: There may be only the signs of bronchitis, or of consolidated lung, or of a cavity—none of them distinctive.

Diagnosis: This is very difficult, and can only be surmised unless in the presence of the two characteristic features of the

disease, the odor of the breath and the peculiar expectoration.

Prognosis: This is bad. Death is the usual result.

Pulmonary gangrene—treatment: Proper nourishment and stimulants of the alcoholic variety are the methods of value. The inhalation of the fumes of turpentine, or creosote, disguises the foul odor of the breath.

PULMONARY ATELECTASIS.

Definition: This is a condition in which from different causes *the alveoli of the lung collapse* and return to their fœtal condition of non-aëration.

Etiology: It occurs especially in feeble children, and in those suffering from the various congenital and acquired forms of malnutrition. The actual causes are *compression* or *obstruction*. Such conditions as effusions in the pleural or pericardial sacs, deformities of the spine or thorax, and tumors in the cavity of the chest may act to cause atelectasis from *compression*. The *obstructive causes* are those which occlude a small or large bronchus. The swollen mucous membrane, the increased mucous secretion, and the feeble breathing are all to be taken into account in this.

Pathology: The collapsed areas may be scattered through the lung, but are usually situated in the posterior portion and near the spine. The area involved is darker than the rest of the lung, and is somewhat depressed below its surface. If put in water, it sinks. The surrounding alveoli are apt to take on a condition of compensatory emphysema.

Symptoms: Dyspnœa and cyanosis are the ordinary symptoms of pulmonary atelectasis, but as it usually occurs complicating some other condition, the symptoms of the two blend, and are not characteristic. The circulation is poor, the extremities are cold, and the child is usually in a condition of collapse.

Pulmonary atelectasis—physical signs: The respiratory murmur over the affected area is feeble, and fine râles are usually heard in the neighborhood. On percussion, dulness is regularly present over the collapsed area if it is of any

size. The physical signs of the complicating condition must always be taken into consideration.

Diagnosis: Except when a large continuous area of lung is involved, the condition is seldom recognized. Even if its presence be suspected, a positive diagnosis can seldom be made.

Prognosis: This is serious, as it regularly occurs in feeble infants who are already suffering from another disease.

Pulmonary atelectasis—treatment: In addition to the care of the primary disease, stimulants, fresh air, oxygen, and very hot baths seem of most service.

DRY PLEURISY.

This form of pleurisy is the simplest, and is accompanied by the **exudation of fibrin** only.

Etiology: It follows exposure to cold and wet and injuries to the chest, and is secondary to various inflammations in the lung.

Pathology: The inflammation may begin in the pulmonary or costal pleura, but regularly spreads to the opposite portion. The inflamed area, be it a small or large one, is swollen, red, and coated with fresh fibrin. Bands of this fibrin form adhesions between the opposite pleural surfaces.

After the inflammation subsides the fibrin may be completely absorbed, but permanent adhesions are usually left.

Dry pleurisy—symptoms: There is a sharp, cutting pain over the affected area, made worse by pressure or by inspiration. There are also a dry, useless cough, and general malaise. The symptoms usually last about a week, when they completely disappear.

Dry pleurisy—physical signs: Over the affected area is heard a *friction-sound*, or *fine crepitant râles*. These sounds are developed by full inspiration, and are unchanged by coughing.

Diagnosis: This is easy with the history and physical signs. The presence of complications in the lungs must always be thought of.

Prognosis: In uncomplicated cases this is good, although a damaged pleura is regularly left behind.

Dry pleurisy—treatment: *Counter-irritation* in the form of mustard paste, iodine, or even a blister, hurries on the process. Rest in the form of a firm bandage around the chest will often ease the pain. Opium may be needed. Some simple febrile mixture, as phenacetin, together with confinement to the house, is useful.

PLEURISY WITH EFFUSION.

Definition: In this form of pleurisy the side of the thorax involved contains more or less *serum* in addition to the fibrin.

Etiology: It follows exposure to cold and damp. It is often secondary to pneumonia, rheumatism, or nephritis. It is frequently tubercular.

Pathology: Only one pleural cavity is regularly involved, but cases are occasionally seen on both sides at once. The pleura is coated with fibrin, and bands of this material form fresh adhesions between the two surfaces. The cavity contains more or less serum, which may be quite clear, or slightly turbid, or at times sanguinolent. In the largest effusions the lung is compressed in the upper and posterior part of the chest. After the inflammation subsides the serum is regularly absorbed, but adhesions are apt to be left behind.

Pleurisy with effusion—symptoms: If the disease is secondary, the symptoms of the primary disease gradually merge into those of the pleurisy. If the pleurisy is *primary*, it at times begins acutely with high fever, pain in the chest, cough, and prostration. The regular onset, though, is gradual, with slight fever, general malaise, and often no symptoms at all pointing definitely to the chest.

After any form of onset the disease regularly takes on a subacute course, the temperature only a little above normal, with some pain in the chest, and cough and dyspnœa on exertion. The patient feels sick, but often not enough to go to bed, and there are anorexia and loss of flesh and strength.

The disease if left alone lasts weeks or months, and has a tendency to spontaneous recovery. Some few die from pressure of a very large effusion. In some the effusion becomes

purulent, and the patient has empyema. In others, the pleurisy being tubercular is not recovered from, and later the child shows evidences of tuberculosis elsewhere.

Pleurisy with effusion—physical signs: Before the effusion is marked, and as it is absorbed, *friction-sounds* are heard over the inflamed pleura. After the fluid is present, we find *below its level flatness* on percussion, *diminished* or absent *breath-* and *voice-sounds*, and *loss* of *vocal fremitus*. *At the level of the fluid* the voice has the tremulous quality known as *ægophony*. Above the fluid the resonance is exaggerated and the breathing is broncho-vesicular, due to the compressed lung. The chest containing the fluid does not move in respiration, the intercostal spaces bulge, the diaphragm is depressed, and when the fluid is on the left side the heart is displaced

Irregular physical signs are frequently present; and in many cases where the fluid is sacculated by adhesions they become very perplexing.

Diagnosis: The physical signs in a typical case are very positive, but so often irregular signs are found that various forms of inflammation in the lungs may be confused with an effusion. From empyema the diagnosis can only be made by the use of an exploring-needle. Under any circumstances, the exploring-needle had better be used, in order to establish the diagnosis positively.

Prognosis: Recovery is the rule. The two dangers are the possibility of the disease changing to an empyema, and of its being a tubercular pleurisy.

Pleurisy with effusion—treatment: The patient should be put to bed, on a diet in which liquids are restricted, and some form of counterirritation should be applied to the chest. Dry cups or blisters are often very useful here. The bowels should be kept freely open, and by means of diuretics the kidneys should be kept secreting in abundance. The citrate of potassium and digitalis are good drugs for this purpose. If pain is excessive, opium may be used. The salicylate of sodium affects some cases favorably.

If the effusion is so large as to embarrass the breathing, or if it does not decrease under the above treatment, aspiration of at least half the fluid in the chest should be performed

under aseptic precautions. A second and a third aspiration are sometimes necessary.

After recovery, general tonic treatment, with a change of climate for a time, should be undertaken.

EMPYEMA.

Definition: This is a disease of some frequency in childhood, and consists in an inflammation of the pleura with a purulent effusion.

Etiology: Most of the cases are secondary to pneumonia. Some follow serous pleurisy. Others complicate the infectious diseases, or are part of a general sepsis. Trauma of the chest, or direct infection of the pleura from a necrotic rib or carious vertebra or suppurating gland, are other causes.

One of three general varieties of bacteria are regularly found in the pus—the *pneumococcus*, the *streptococcus*, or the *tubercle bacillus*. Mixed infections are also found.

Pathology: The whole of one pleura may be involved, or only a circumscribed portion. Rarely both pleural cavities may be simultaneously attacked. The pleura is inflamed and its surface coated with fibrin and pus, and the cavity is more or less filled with a *purulent exudate*. The pus settles to the bottom of the pleural sac, and floats the lung upward and toward the spine. Adhesions between opposing surfaces of the pleura are frequent, causing the pus to be *sacculated*.

There is very little tendency to absorption; and if untreated, the pus tends to rupture into the lung, or externally through an intercostal space.

Empyema—symptoms: Following the symptoms of the precedent pneumonia or infectious disease, the temperature continues high; there may be sweats, the child has pain in the chest, dyspnœa, and cough. As the disease advances, the symptoms are mainly those of infection—irregular high fever, sweating, loss of flesh and strength, anorexia, diarrhœa, prostration, and a rapidly growing anæmia. The pulse grows rapid and feeble, and a typhoid condition is developed if the case is left alone.

The disease may run a slow course, with the above symptoms more or less intensified; and if untreated, the pus may rupture externally or through the lung, or the patient may die exhausted by the disease.

Empyema—physical signs: These are identical with those of pleurisy with effusion, except that sacculation of the pus is more common, and therefore irregular physical signs more to be expected.

Diagnosis: This is a very easy disease to overlook in children. Pleurisy with effusion, pneumonia, and phthisis are likely to be confounded with it. It is always advisable in suspected cases to make use of the exploring-needle.

Prognosis: In the cases following pneumonia, under proper treatment, the prognosis is good. In the cases due to streptococci the prognosis is much worse. In those due to tubercle bacilli the prognosis is that of tuberculosis in general. The younger the child and the longer the disease has been untreated, the worse the prognosis. Some few cases recover spontaneously. The prognosis depends very decidedly on the treatment.

Empyema—treatment: The child should be put on tonic stimulating treatment.

The empyema requires surgical care and removal of the pus. Aspiration is unsatisfactory, and is best reserved for use as a temporary measure only.

Simple incision through one of the lower intercostal spaces, with evacuation of the pus and drainage by a large-sized tube, will cure most of the cases. General or local anæsthesia may be used, and the dressings changed frequently. Irrigation had better not be employed.

In cases where the ribs are close together, or where the pus is thick and fibrinous, a small piece of one rib had better be resected, and the subsequent treatment be carried out as after simple incision. Although this operation is more severe, success is more certain after it than after any other method of treatment.

In the few cases in which the lung will not expand after evacuation of the pus, and after lapse of some weeks, extensive operations with resection of many ribs may be necessary.

Pulmonary gymnastics should be advised to encourage the expansion of the lungs.

ACUTE PULMONARY TUBERCULOSIS.

This **form of tubercular inflammation** in the lung is also called *acute* or *galloping consumption*, or *acute tubercular broncho-pneumonia*. It is seen with some frequency in young children.

Etiology: The causes are predisposing and exciting. The predisposition is often inherited from a tuberculous family. Bad hygienic surroundings, poor food, bad air, and wasting diseases produce a predisposition. The infectious diseases, particularly whooping-cough, measles, and la grippe, and simple inflammations in the lung, are marked *predisposing causes*. The *exciting cause* is infection by the *tubercle bacilli of Koch*, which in most cases gain access to the victim by way of the respiratory tract. In rarer cases they are taken in with the food or drink, and at times by direct inoculation. In only a very few cases has evidence of actual intra-uterine infection or of congenital tuberculosis been proved. In any pulmonary case the bronchial lymph-glands are regularly the seat of bacilli, and from them the pulmonary infection comes.

Pathology: The lungs are more or less filled with miliary tubercles. These are scattered irregularly through the pulmonary tissue, and are also found on the surface. They are apt to have the distribution of an ordinary broncho-pneumonia, but in places may be massed together in lumps. Some of the nodules, if the case is not too recent, caseate and break down with the formation of cavities.

Catarrhal bronchitis, simple broncho-pneumonia, or even diffuse consolidation of the lung may exist as *complications*. The pleura is regularly involved if the tubercular process is near the surface. The bronchial glands will be found enlarged and caseous.

Acute pulmonary tuberculosis—symptoms: The onset and symptoms of this disease are almost identical with those of ordinary broncho-pneumonia. There is *fever* of a somewhat

irregular character, and varying from 101° F. to 104° F. Sweats are not frequent. The child has a *cough*, with *rapid respiration* and *pulse*, appears quite sick, and shows a loss of flesh and strength somewhat more marked than in simple broncho-pneumonia. Expectoration, as always in children, is lacking.

Some of the cases go on acutely with these symptoms and die within a month. Others take on a more subacute character and last for two or three months, but the tendency in them all is to exhaustion and death.

Acute pulmonary tuberculosis—physical signs: Here again the similarity with the signs of a simple bronchitis or broncho-pneumonia is most marked. If only scattered miliary tubercles are present, with the accompanying catarrhal bronchitis, we find scattered *subcrepitant* or *coarse râles*. If areas of consolidation are present, we find *dulness*, *increased fremitus*, and *broncho-vesicular* or *bronchial breathing* and *voice*. In other words, there is nothing distinctive in the signs.

Diagnosis: The history and physical signs are so nearly identical with those of simple broncho-pneumonia that a positive diagnosis is almost impossible. Unfortunately we have not the aid of finding tubercle bacilli in the sputum as in adults, as sputum is so hard to obtain. The family history, the preceding condition, and the surroundings of the child should all be carefully taken into consideration. *Enlarged glands*, or history of disease of the *bones*, are especially important.

The loss of flesh in tubercular pneumonia is more marked than in the simple form. The signs in the tubercular variety are more often in the upper lobes or anterior portions of the lungs, while in the simple form they are more often posterior.

Prognosis: This is bad. All the cases die in a short time, except the few which go on to the chronic condition.

Acute pulmonary tuberculosis—treatment: To prevent the disease all children, and especially those with a hereditary predisposition, should be well fed, well clothed, and kept most of the time in the fresh air. If consumptives are in the family, all their sputa must be carefully destroyed by chemicals or fire. These children should not sleep with nor kiss

tubercular people. All milk and meat should be carefully inspected before use. Tuberculous mothers should not nurse their children. All infectious diseases or pulmonary inflammations in these children should be most carefully treated with this end in view.

If the disease has begun, the treatment consists in keeping the stomach and bowels in good condition, so that we will be able to over-nourish the child; to allow an abundance of fresh air; and to give creosote in full doses to tolerance. Special symptoms are to be treated as they arise.

CHRONIC PULMONARY TUBERCULOSIS.

Etiology: This form is seen mostly in older children, and differs very little from the same condition as seen in adults. The predisposing and actual causes are the same as in the acute variety. A good many of the cases follow an attack which began acutely, or are secondary to protracted simple inflammations of the lungs, or develop gradually from a latent tuberculosis of the bronchial glands.

Pathology: The growth of miliary tubercles and of tubercular masses scattered through the lung, with the accompanying chronic bronchitis or broncho-pneumonia, are the beginning lesions of this disease. The same tendency to necrosis of this new-formed tissue, and subsequent formation of small cavities exists, as in adults. The apex is not so regularly the location of the *early lesions* as in older patients, this being more often in the neighborhood of the bronchial glands. In different parts of the lung are found bands of fibrous connective tissue, and the pleura is regularly thickened and adherent. The whole lung is irregularly consolidated.

Chronic pulmonary tuberculosis—symptoms: The symptoms ordinarily follow those of some previous disease during its convalescence. They are cough, irregular fever, loss of flesh and strength, increasing pallor, and general prostration. Hæmoptysis and expectoration are rare. The appetite is lost; there may be vomiting and diarrhœa. The pulse is rapid and feeble, and the respiration is accelerated. As the disease progresses sweating is common and the temperature may

take on a hectic course. The wasting becomes excessive and the strength very feeble. Tubercular diarrhœa and laryngitis may appear as complications, and toward the end a general œdema may develop, and the child dies after many months of excessive emaciation and exhaustion.

Chronic pulmonary tuberculosis—physical signs: The expansion of the affected chest is limited. The *vocal fremitus* is apt to be *increased*. On percussion, irregularly placed *areas of dulness* or *flatness* may be found. On auscultation, *exaggerated voice-* and *breathing-sounds* are found over these same areas, or there may be no change in the character of the breathing. There are always present, however, various kinds of *adventitious sounds* scattered irregularly over the lungs—fine and coarse râles, bubbling and whistling sounds—and if small cavities are present, *cavernous breathing* with moist gurgles may be heard.

The signs are not nearly so distinctive as in adults, and are much more difficult to bring out.

Diagnosis: The irregularity in the physical signs and the history are the main points of value in diagnosis. At times a simple chronic pneumonia will give precisely the same signs and symptoms, and differentiation will be almost impossible. If possible, a specimen of the sputum should be obtained and examination made for the tubercle bacilli. This is the only positive method of diagnosis.

Prognosis: Recovery is possible when the cases are seen early and put under proper treatment. As more of the lung is involved and cavities form, and especially if the stomach fails, the prognosis becomes bad.

Chronic pulmonary tuberculosis—treatment: The preventive treatment should be followed as is outlined for the acute variety.

For the treatment of the disease itself, life in the fresh air, if possible in a climate that is dry and high, is of most importance. Special attention should be paid to the condition of the stomach and bowels, in order that the child may take and digest and assimilate a large quantity of nourishing food. Food rich in fat should be taken especially, and for this reason cream and cod-liver oil are particularly desirable. Creo-

sote in increasing doses has a decidedly beneficial effect. Symptoms should be treated as they arise, the cough by soothing cough-mixtures, the fever by sponging or antipyretics, and the sweats by drugs of the atropine group. It should always be remembered, however, that harm is done by over-drugging these patients, upsetting their digestion, more often than by giving too few drugs.

CHAPTER X.

DISEASES OF THE GENITO-URINARY SYSTEM.

ENURESIS.

Definition: By this is meant an involuntary emptying of the bladder at inopportune times.

Etiology: The *organic causes* are inflammations of the bladder or other parts of the genital tract, and diseases of the brain or spinal cord. These cases, due to organic causes, are not the ones ordinarily understood by enuresis.

The *functional causes* are lack of proper inhibitory control from the cerebral centres, and increase of the reflexes of the cord. Such causes are anæmia, malnutrition, the functional neuroses, a highly acid urine, or one containing some abnormal constituent, irritation from a foreign body in the bladder, and reflex irritations from the genital tract, as phimosis, balanitis, and vulvitis, or from the rectum and anus, as polyps, worms, and fissure. Children of a neurotic heredity are particularly liable to the disease.

Enuresis—symptoms: Involuntary emptying of the bladder is the only symptom. This most commonly occurs at night only, in some cases by day only, and in others both by day and by night. It may occur every night once or even more times, or only irregularly. The condition usually lasts from infancy for some years—at times until puberty.

Diagnosis: The point of diagnosis is to discover, if possible, any causative factor or factors that may be present.

Prognosis: The organic cases cannot be cured. The functional cases eventually outgrow the trouble. In some the response to treatment is very satisfactory; in others, no means that can be taken seem to have any influence over the condition.

Enuresis—treatment: Hunt for any possible cause and re-

move it, going systematically through the whole list. All measures directed to improving the child's general health should be strictly carried out. The diet and the quantity of fluid allowed should be carefully regulated. The child should not be punished for wetting himself, but on the contrary should be rewarded when he does not do so. He should sleep in a warm room, with warm coverings, and with the foot of the bed raised higher than the head. He should empty his bladder the last thing before going to bed, and should be taken up again before the parents retire. During the daytime he should be trained to hold his urine as long as possible.

As *drugs*, the most valuable are belladonna, cantharides, and strychnine. The first should be given in maximum doses in the evening. It is well to give cantharides at the same time to add an irritating effect to the vesical neck, and hence tonicity to the sphincter. These act best in cases of neurotic, irritable children. In children whose muscular tone is below par, strychnine, given regularly, has some value.

Passage of cold sounds, galvanism to the neck of the bladder, and distention of the bladder daily until its capacity is increased, may be tried if the above measures fail.

VESICAL CALCULUS.

Stone in the bladder occurs with some frequency in children after the second or third year of life. Most of the stones are of the uric-acid variety. Rarer forms are the oxalate of lime or the phosphatic stones. They vary in size and may be multiple. They may be free in the bladder or attached to its wall.

Etiology: The stone is usually formed around a small concretion which has passed from the kidney to the bladder. Excessively concentrated urine from improper diet or the use of too little liquid, seems to favor the causation. Otherwise little is known.

Foreign bodies passed into the bladder from the urethra are rare causes.

Vesical calculus—symptoms: These are like the symptoms

seen in adults—pain at the end of urination, excessive irritability of the bladder, and *sudden stoppage* of the stream during the act of micturition. The urine may contain mucus and blood in small quantities, and signs of cystitis may develop. Enuresis and rectal tenesmus with a tendency to prolapse are often seen.

Diagnosis: The symptoms are fairly typical, but the diagnosis should always be confirmed by the passage of a searcher into the bladder.

Prognosis: Without treatment the prognosis is bad, but under intelligent surgical care these cases should be cured.

Vesical calculus—treatment: The stone, after discovery, should be removed either by lithotrity or by perineal or suprapubic cystotomy. The latter operation is most in favor during recent years.

After removal of the stone, steps should be taken to prevent formation of others, by attention to the diet and mode of life.

DIABETES INSIPIDUS.

Definition: This is also called *polyuria*, and is a neurosis characterized by the secretion of large quantities of pale urine of low specific gravity and containing no abnormal constituents.

Etiology: This disease occurs with some frequency in grown children. It is usually in some way connected with nervous causes, as shock, fright, or a neurotic predisposition. Otherwise the causative factors are unknown.

Pathology: No lesions are to be found in the kidneys. Various forms of irritation have been found over the floor of the fourth ventricle.

Diabetes insipidus—symptoms: Very large quantities of urine are passed per day. The specific gravity is low and the ingredients normal. The child is thirsty, loses flesh, and becomes anæmic, and presents irregular nervous and hysterical symptoms. The disease lasts for years, with intervals of improvement.

Diagnosis: Frequent urinary analyses must be made to exclude chronic nephritis.

Prognosis: This varies a good deal. Many of the cases recover in time, but others seem incurable.

Diabetes insipidus—treatment: Great attention should be paid to all hygienic measures to put these children in the best health possible. The *diet* should be nutritious and largely *albuminous*. As *drugs*, ergot, gallic acid, belladonna, and the mineral acids are used. The treatment should be continued over long periods of time.

FUNCTIONAL ALBUMINURIA.

Etiology: This condition is quite a frequent one in adolescents, although seldom seen in children. In some cases it is paroxysmal in character, coming at certain times with no special cause and then disappearing to return again later. In these cases it is more often present by day than by night. In others it is dietetic, always following certain articles of food, which are usually of a *proteid* nature. In others it is due to excessive exercise or to fatigue. In some cases no cause can be assigned.

Pathology: No known lesions exist. There may be irritation from some abnormal chemical ingredients in the urine, or it may be simply an *exudation* of serum through the kidneys, due to some vaso-motor disturbance.

Functional albuminuria—symptoms: The presence of albumin in the urine, without subjective symptoms, is what we expect in this condition. Casts are regularly absent. Some of the subjects are persons not in the best of health.

Diagnosis: This can be made usually only after long observation of the patient and frequent examinations of the urine, so as to exclude actual renal disease.

Prognosis: If the disease is purely functional, the prognosis is good. The point is as to whether these children may not be the persons who in adult life will develop nephritis.

Functional albuminuria—treatment: This consists in improving the patient's general health, regulating his diet and exercise, and giving iron. Intelligent care, followed for a sufficient length of time, usually effects a cure.

ACUTE DEGENERATION OF THE KIDNEYS.

Definition: This is the *simplest form* of what may be called "acute Bright's disease."

Etiology: It is seen in almost all cases of infectious disease in children, being secondary to all forms of toxæmia—chemical or bacterial.

Pathology: There are simply degeneration and death of the renal epithelium. The cells are swollen, opaque, and contain fat. Later they disintegrate and desquamate. The kidneys are usually a little swollen and pale.

Acute degeneration of the kidneys—symptoms: There are no subjective symptoms, although the presence of the nephritis adds to the dangers of the original disease. The urine is diminished in quantity, and may be suppressed. There may be small quantities of albumin and a few casts in the urine, but more often neither can be found.

Prognosis: This is good; as after recovery from the original disease the kidney returns completely to normal.

Acute degeneration of the kidneys—treatment: During the continuance of the causative infectious fever the diet should be fluid and largely milk. Otherwise no special treatment is needed, except in case of *suppression*, when the bowels and skin should be called on to act freely.

ACUTE EXUDATIVE NEPHRITIS.

Synonyms: Other names for this variety are *parenchymatous* and *desquamative nephritis*.

Etiology: It may be primary, with no discoverable cause; but is most often secondary to the infectious diseases, particularly scarlatina and diphtheria.

Pathology: The kidneys are large, soft, and congested, and their markings indistinct. The *renal epithelium* is swollen, opaque, degenerated, and detached. The *struma* is infiltrated with serum, white blood-cells, and coagulated matter. The cells of the glomeruli are swollen and increased in number. The tubules may be dilated. In places minute abscesses may be found.

Acute exudative nephritis—symptoms: The disease usually begins acutely with fever, vomiting, and rapid pulse and respiration. The child is restless, sleeps badly, and complains of headache. The bowels are apt to be loose. The urine is decreased in quantity, and in severe cases may be suppressed. Albumin, casts, and at times blood are found in it.

As the disease progresses the *fever* runs an irregular but rather low course. *Anæmia* develops rather rapidly, and the symptoms pertaining to the *nervous system* are especially prominent: restlessness, twitchings, and even convulsions being regularly present. Other patients become dull, and pass into a condition of stupor and coma. Dropsy is not a marked symptom, but is usually present.

Other cases run a much less acute course, with all the symptoms milder. The regular duration of the disease varies from two to four weeks.

Diagnosis: This combination of symptoms should always lead to a careful examination of the urine. The different forms of nephritis are often difficult to differentiate, except after observation extending over some time.

Prognosis: In very young children this is a rather fatal disease; but on the whole the majority of cases recover, and the kidneys return to normal. Some few go on to have chronic Bright's disease.

Acute exudative nephritis—treatment: The child is to be kept in bed, in a warm room, and put on a milk-diet. Large quantities of water should be given, and the bowels kept freely open by the use of salines daily. Counterirritation should be applied to the lumbar region by means of dry cups. The skin should be made to act freely by hot packs, or hot-air baths. If the *pulse* is full and strong, such drugs as nitroglycerin, or opium, or chloral hydrate should be given. If the *heart* is acting feebly, caffeine is valuable. If the *cerebral irritation* is marked, and convulsions threaten notwithstanding the above steps, venesection will act quickly and satisfactorily. There is no place where it has the value it does in these conditions of suppression of urine.

After the acuteness of the disease has passed away great care should be exercised in returning to the regular diet, and

in preventing chilling of the skin. Iron and tonics will then be needed to cure the anæmia and build up the strength.

ACUTE DIFFUSE NEPHRITIS.

This form of nephritis differs from the preceding mainly in leaving a damaged kidney behind.

Etiology: This condition may be due to exposure to cold or wet; but is most often secondary to one of the infectious diseases, and particularly to scarlatina or diphtheria.

Pathology: All the changes seen in the exudative form are present, and *in addition* we find a *growth of new connective tissue* scattered irregularly through the stroma of the kidney, and a growth of the glomerular capsule cells such that they compress the tufts of vessels. Both of these changes are permanent, and leave a kidney damaged by the replacement of some of its secreting structure by fibrous tissue.

Acute diffuse nephritis—symptoms: The disease may rarely begin acutely and behave like the exudative form; but is more commonly less active, with lower fever, progressive anæmia, anorexia, nausea and vomiting, and loss of flesh and strength. Dropsy is a regular symptom of this variety, and is quite marked. Nervous symptoms—headaches, restlessness, and delirium, or stupor and coma—are regularly present. Convulsions are fairly frequent. The urine is diminished in quantity, and contains albumin and casts, and at times blood.

The symptoms last from two to four weeks, disappearing gradually; but slight changes in the urine remain for a long time afterward.

Diagnosis: This must be made from the exudative variety. The main points are the less acute symptoms and the presence of more dropsy. Often time only will differentiate them.

Prognosis: A good many cases die during the acute stage. Others recover except for a damaged kidney, while others go on to a chronic nephritis.

Acute diffuse nephritis—treatment: During the acute stage this is the same as in the exudative form.

Afterward the child should be kept warmly dressed, and

live out of doors in a warm climate. The diet should be largely *carbohydrate*, with milk and abundance of water. Iron should be given over a considerable period of time. The continued administration of small doses of the bichloride of mercury seems to have a beneficial influence over the kidney lesion.

CHRONIC DIFFUSE NEPHRITIS.

In young children forms of **chronic Bright's disease** are rare, but probably less so than imagined on account of the frequent neglect in examining the urine of children.

Etiology: Most of the cases follow the acute forms. Others are due to chronic endocarditis, or tuberculosis, or hereditary syphilis.

Pathology: There are two general varieties, that *with* and that *without exudation*.

The *former* is evidenced by a large whitish kidney with indistinct markings. The renal epithelium is swollen, granular, fatty, and degenerated. The capillaries of the glomeruli are dilated and the tuft-cells swollen. Casts, coagulated matter, and necrotic epithelium are seen in the tubes and stroma.

In the form *without exudation*, which is commonly known as interstitial or granular nephritis, the kidney is small and rough and the capsule adherent. The cortex is thin and irregular, and the color a mottled red. The same changes in the renal epithelium and in the glomeruli are present, as in the preceding variety; but in addition there is a diffuse growth of new connective tissue in the stroma; and of the cells of the glomeruli. In places the tubules are considerably dilated.

Chronic diffuse nephritis—symptoms: The symptoms develop slowly and insidiously, whether they follow preceding acute attacks or come on primarily. There are anæmia, loss of appetite, attacks of vomiting and diarrhœa, and loss of flesh and strength. *Nervous symptoms* come and go, headache, sleeplessness, restlessness, and paroxysmal dyspnœa. *Dropsy* of the subcutaneous tissues and of the serous cavities is regularly present in more or less marked form.

The *urine*, in the variety with exudation, does not differ

much from normal in amount, but contains albumin and casts in varying quantities. The specific gravity is below normal. In the variety without exudation the urine is increased in quantity and is of low specific gravity. Albumin and casts are present only in small quantities, and may be absent altogether over considerable periods of time. This latter variety is rare in children.

The immediate danger in chronic nephritis comes from the so-called attacks of *uræmia*, in which the urinary excretion is diminished and the nervous symptoms become very marked, frequently going on to severe convulsions. With this we find a laboring heart-action with tense arteries, and very severe headaches.

The *course* of this disease is very chronic, with exacerbations and remissions.

Diagnosis: This depends on frequent, careful examinations of the urine.

Prognosis: This is bad, as recovery is virtually impossible; but under proper direction life may be prolonged comfortably over a period of many years.

Chronic diffuse nephritis—treatment: The child should live in a warm, dry climate if possible, and special attention should be paid to warm clothing and to the prevention of exposure to changes in temperature. The diet should consist largely of milk and carbohydrates, with fruit and fresh vegetables. The bowels should be kept open freely. Exercise should be taken regularly, but not to fatigue. The prolonged use of iron or of bichloride of mercury is of much value. If the dropsy is marked, we use diaphoretics, diuretics, or cathartics, and if necessary tap the serous cavities. If uræmia threatens, we give drugs to dilate the arteries; we sweat the patient by hot packs or the hot-air bath; or, if necessary, perform venesection. If convulsions develop, chloroform is our main reliance. Other symptoms are to be treated as they arise.

TUMORS OF THE KIDNEY.

Benign tumors are rare. **Malignant growths** are fairly common in children. A very few are *congenital*.

Tumors of the kidney—pathology: The tumor is usually *primary* to the kidney, and in the vast majority of the cases is a *sarcoma* of the round- or spindle-cell variety. *Myo-sarcoma* is often found. The whole kidney may be replaced by sarcomatous tissue, or the growth may develop on the surface of the kidney. The tumor may press on the ureter or inferior vena cava. Adhesions may form to neighboring organs.

Tumors of the kidney—symptoms: The symptoms are a rather fast-growing tumor, cachexia, and hæmaturia. The tumor usually begins posteriorly; but soon it grows to the front, and often becomes very large. Its surface may be smooth or lobulated. Cachexia develops much later than in malignant growths elsewhere. *Hæmaturia* is usually present at one time or another, and often early. Pain and symptoms due to pressure may be present.

The ordinary *duration of life* is less than a year from the time the tumor is discovered.

Diagnosis: The above set of symptoms is quite characteristic and usually enables a positive diagnosis to be made.

Prognosis is absolutely bad.

Treatment is purely surgical. Enucleation of the tumor and the kidney should be performed as soon as the diagnosis is made.

PYELITIS.

Definition: This term is used to describe an *inflammation* of the *pelvis* of the kidney and of the neighboring portion of the ureter. If the kidney is involved coincidently, it is called *pyelo-nephritis*. If pus is accumulated in the sac, *pyo-nephrosis*.

Etiology: This condition develops from renal calculi, tuberculosis of the kidney, abscesses in the neighborhood, extension upward of inflammation from the bladder; and as a consequence of the infectious diseases, particularly sepsis.

Pathology: It may be unilateral or bilateral. The mucous membrane is inflamed, swollen, and often shows minute hemorrhages. There is an exudation of mucus and pus from the inflamed membrane. The kidney may or may not be involved, but is regularly so in the older cases. Small collections of pus are frequently found in the pelvis of the kidney.

Pyelitis—symptoms: In an acute attack there is a sudden rise of fever, accompanied by a chill, with intense pain in the back. The temperature remains high, and the chill may or may not be repeated. Chills occur especially in the tubercular cases and in those with the formation of an abscess. The *urine* is found decreased in quantity, acid in reaction, and containing pus, a little albumin, and some red cells. The cases last a few weeks, the fever and other symptoms disappearing very gradually, unless an abscess is formed or tuberculosis exists.

In the *chronic cases* pus in the urine is often the only symptom, unless a renal tumor is formed from an accumulation of pus. Acute exacerbations in these cases are frequent.

Diagnosis: This depends on the examination of the urine. *Pyuria*, with an acid urine, and epithelial cells from the pelvis of the kidney, are usually sufficient, in the presence of the history, to establish a diagnosis. A search in the pus for tubercle bacilli should be made in the prolonged cases.

Prognosis: This is fairly good in the primary cases. There is danger of nephritis developing, however. In cases due to stone or tuberculosis the prognosis is less favorable.

Pyelitis—treatment: The child should be kept in bed, on a plain diet. Large quantities of water, containing lithia, citrate of potassium, or other saline diuretic, should be given. Employ strong counter-irritation over the loins during the acute stage, by means of mustard or cups.

The question of *surgical interference* comes up in the chronic cases, as in those which are distinctly due to calculi.

RENAL CALCULI.

Small calculi, or gravel, are common in children.

Larger calculi are found but rarely. They are usually formed of *uric acid*, and are found in the calyces or pelvis of the kidney. The small ones are usually bilateral.

Renal calculi—symptoms: The continual secretion of the urine tends to wash the calculi through the ureters into the bladder. Their passage is accompanied by a sharp pain radi-

ating down the ureter and to the pelvis. In boys retraction of the testicle accompanies it. These pains come on suddenly, and cease as soon as the stone reaches the bladder. They are very agonizing and sharp in character. There is regularly tenderness over the affected side. The urine passed just after the attack will contain a little blood and some albumin, and often some uric acid crystals.

At times the calculus will be impacted in the ureter, with a resulting dilatation of the pelvis with urine. This may be infected and become purulent.

Diagnosis: The finding of the stone in the urine is proof positive of the diagnosis, the other symptoms being corroborative.

Prognosis: This is fairly good. There is danger, however, of the development of nephritis, or pyo-nephrosis, and of a subsequent vesical calculus.

Renal calculus—treatment: The child should be put on a fluid diet, and should drink very large quantities of water to which an alkaline diuretic has been added.

If after thorough use of this method cure is not effected, and if the diagnosis of the presence of a stone is positive, the case should be put under surgical care. The results of operation are fairly good.

PERINEPHRITIS.

Definition: This is an inflammation of the cellular and fatty tissue surrounding the kidney. The inflammation frequently goes on to suppuration.

Etiology: It may be secondary to disease of the vertebræ, or to suppuration in the kidney proper. It may be due to exposure to cold, or to local traumatism, or follow some of the infectious diseases. In many cases no cause can be found.

Pathology: The perinephritic tissue is in a condition of acute exudative inflammation, in which there is often such an excessive exudation of leucocytes as to form an abscess. This abscess may extend backward and burst externally. It may burrow upward or downward, and point in most unexpected places.

Perinephritis—symptoms: The disease is acute, beginning with a chill, rise of temperature, and *pain in the lumbar region.* The pain is made worse by moving the limb of the affected side, and so lameness is produced, and there is tenderness on pressure over the kidney region.

The fever continues, chills may be repeated, the pain is constant, and the child appears quite sick and willingly stays in bed. Vomiting is a rather frequent symptom. As the disease progresses an indefinite tumor may be made out in the loin, the skin of the back over the abscess gradually reddens, and evacuation may take place through the skin. In other cases the abscess may burrow and burst through the groin, or into the bowel or peritoneum. In some cases resolution takes place without the formation of abscess.

The duration of the disease is from one to three months.

Diagnosis: This is usually not difficult if the case is studied carefully. It differs from pyelitis in being accompanied by *no changes in the urine;* and from hip-disease, with which it is sometimes confounded, by the slower course of the latter.

Prognosis: The patient usually recovers. The danger lies in rupture of the abscess into the peritoneum.

Perinephritis—treatment: The patient should be put to bed on a light diet, the bowels moved by fractional doses of calomel, and ice-bags kept applied to the inflamed side of the body. If suppuration is deemed inevitable, hot poultices should be used, and at the first signs of the abscess approaching the surface an incision should be made through the lumbar region to evacuate the pus.

PHIMOSIS.

Definition: In this condition the opening through the prepuce is so contracted that the glans cannot, or can only with great difficulty, be pushed through it. It is a congenital condition which is very common, and is regularly associated with adhesions between the prepuce and glans. At times there is enough narrowing actually to interfere with the passage of urine. If the prepuce is forcibly retracted, there is found in the sulcus about the corona a whitish secretion called the *smegma.*

Phimosis—symptoms: This condition may be unaccompanied by any symptoms, but various *local* and *reflex* results of it are frequently found. Among these are attacks of *balanitis* from the impossibility of cleanliness; hernia or prolapsus ani from straining; enuresis, masturbation, insomnia, night-terrors, and general convulsions from reflex irritation. Rarer effects are chorea, epilepsy, and gastralgia.

Phimosis—treatment: Where the prepuce is not too long, it should be forcibly retracted, the adhesions broken up, any smegma removed, and the parts anointed with a little vaseline before the foreskin is returned to its proper place. This should be repeated every day until return becomes easy.

In case of long foreskin or where retraction is impossible *circumcision* should be done.

BALANITIS.

Definition: This is an inflammation of the mucous membrane covering the glans and lining the prepuce.

Etiology: Uncleanliness from adherent prepuce and irritation or injury of the parts from infection, or masturbation, or urethritis, are the main causes.

Balanitis—symptoms: The parts are swollen, red, inflamed, and tender. After a few days there is an increased secretion; and then a rapid amelioration of the symptoms. In tightly adherent prepuce the inflammation may be so intense as to cause sloughing.

Balanitis—treatment: If the prepuce is adherent, it is often necessary to slit up the dorsal segment of it, or to perform circumcision first. In any case the prepuce must be retracted, and the parts kept scrupulously clean with soap and water. Afterward a saturated boric acid solution should be applied.

VULVO-VAGINITIS.

Definition: This is a catarrhal inflammation of the vulva and lower vagina. It is fairly common in young children.

Etiology: There are *two* general varieties, the *simple* and the *specific* or *gonorrhœal*. The *former* is due to dirty diapers

or drawers, or to wearing none, or to other lack of proper cleanliness. In some cases it is due to pinworms, or to infection from the rectum from other causes.

The *specific form* is due to infection by the gonococcus, either from attempts at rape, or more often from use of towels, rags, and utensils that have become infected.

Vulvo-vaginitis—symptoms: There are itching, burning, and irritation of the parts, with pain on urination. There is a marked thin, or thick, or creamy discharge present, which dries and forms crusts over the vulva. The mucous membrane is red and inflamed, and the skin is excoriated. In the most severe cases there are mild constitutional symptoms of fever and malaise.

The simple cases usually heal rather readily under treatment; the specific variety is very obstinate.

Complications are likely to arise—urethritis, cystitis, pelvic inflammations, and conjunctivitis.

Diagnosis: The specific form is distinguished from the simple by its greater severity, and the discovery of the gonococci in the discharge.

Vulvo-vaginitis—treatment: Absolute cleanliness is the keynote of treatment. Dried secretions should be removed with soap and warm water, and the parts—vulva and vagina—irrigated twice daily with weak bichloride or saturated boric acid solution. Vaseline should then be applied to the skin around the vulva. Pads of cotton or of gauze, damped with boric acid solution, should be worn continuously. In some cases strong solutions of silver nitrate must be used. Measures should be taken to protect the eyes from infection.

CHAPTER XI.

DISEASES OF THE NERVOUS SYSTEM.

PERIPHERAL NEURITIS.

Definition: By this term is understood an inflammation of the peripheral nerves. This may involve only one or more nerves, but often many in different regions of the body are affected.

Etiology: Local *traumatism*, *exposure to cold*, the *toxins* from the infectious diseases, as diphtheria, la grippe, and typhoid, and certain *chemical poisons*, as alcohol, arsenic, and lead, are all well-recognized causes.

Pathology: The affected nerve shows some signs of an exudative inflammation, but the characteristic changes are a degeneration of the nerve-fibres.

Peripheral neuritis—symptoms: The disease develops gradually. There is more or less neuralgic pain in the affected nerves, with distinct tenderness to pressure over them. Hyperæsthesia, with numbness and tingling, is frequently present. The muscles supplied by the affected nerves gradually lose their power, and eventually become paralyzed. The extensors are oftenest affected, giving wrist-drop and toe-drop. Atrophy is quite rapid in these same muscles. The reflexes are lost. Electrical reactions are changed, until in the extreme cases there is no response to any form of current. In old cases contractures and *deformities* occur.

In some of the more acute cases constitutional symptoms with fever and malaise are present for a time. The disease lasts for one or two months, and recovery occurs very slowly. Where nerves supplying vital organs are involved death is a frequent result, as in involvement of the vagus after diphtheria.

Diagnosis: It is necessary to distinguish paralysis due to

cerebral and spinal lesions from this form. *Cerebral lesions* cause no atrophy, and are usually accompanied by increased reflexes. *Anterior poliomyelitis* is accompanied by atrophy and loss of reflexes; but is usually confined to one set of muscles in one limb, while neuritis is apt to be symmetrical. In poliomyelitis there is no pain over the nerve-trunks.

Neuritis of the facial nerve is usually due to cold or trauma, or extension of the inflammation from the mastoid cells. Neuritis of the nerves supplying the forearm extensors is usually due to lead; and of those supplying the extensors of the feet to arsenic. Neuritis in the nerves of the throat or eyes, and in the pneumogastric, is ordinarily due to diphtheria.

Prognosis: Recovery is the rule after a long, slow convalescence. A few cases leave a permanent paralysis. If the pneumogastric is involved, death is likely to result.

Peripheral neuritis—treatment: If the causative factor can be discovered and removed, this should be the first step in the treatment. The child should be kept in bed and as quiet as possible. In the early stages the main point is to relieve the pain. This is done by dry, hot applications, and by the use of phenacetin. In some cases opium will be necessary. Strychnine is to be given regularly, and acts as a good tonic.

After the acute stage has passed regeneration is hurried by the use of electricity daily or on alternate days. Massage and general tonics, of which iron and cod-liver oil are the best, are valuable adjuncts. In severe dysphagia, due to paralysis of the throat after diphtheria, feeding by the stomach-tube may be required. In involvement of the pneumogastric, combinations of morphine and strychnine, the latter pushed to its limit, are of most value. Faradism to the diaphragm may also be tried.

ACUTE ANTERIOR POLIOMYELITIS.

Synonyms: This disease is very common in childhood. It is also called *infantile paralysis* or *infantile spinal paralysis*.

Etiology: The disease occurs oftenest before the fifth year of life, and it has been suggested that the efforts of learning

to walk have an etiological relationship to it. Trauma and changes in temperature are also ascribed as causes. Some cases develop subsequently to the infectious diseases. There is some evidence that the disease itself may be of germ origin.

Pathology: The lesion is an acute inflammation in the anterior horns of the gray matter of the spinal cord. The cells in this area, which is usually a limited one, are destroyed in the inflammatory process, and after recovery the cord in this region shrinks in size. So few cases die in the acute stage that the lesions seen are mainly those due to the subsequent atrophy of that portion of the cord. The disease most often attacks the cervical or lumbar enlargements of the cord, and usually on one side only.

Acute anterior poliomyelitis—symptoms: The disease regularly begins acutely, with fever of 101° to 104° F., vomiting, marked prostration, and pain in the limbs. Many cases also have one or more general convulsions. These acute symptoms, similar to those of an infectious disease, last for a few days without any definite local signs developing, when one or more of the limbs are found to be paralyzed. Gradually the constitutional symptoms disappear, but the paralysis remains. The affected limb is regularly entirely involved at first, but as the weeks pass by some of the muscles regain their power, and eventually only one group, as for instance the anterior tibial, is left permanently paralyzed. In some cases more than one group, or different groups in different limbs, are permanently affected.

After about three months whatever parts were to recover have done so, the paralysis in the other groups being permanent. After years this paralysis may lead to contractions and relaxations of joints, causing bad deformities, the limb being decidedly atrophied and feeble, the reflexes gone, and the reaction of degeneration to electricity being present.

Diagnosis: In the early stages it may be mistaken for one of the infectious diseases or for meningitis. A few days' observation will settle this point.

Confusion may exist between it and cerebral paralysis, or peripheral neuritis. Cerebral paralysis may begin similarly,

but the paralyzed limb is well nourished, the reflexes are increased, and its general condition is spastic rather than flaccid. Neuritis begins more gradually, and has pain along the affected nerves. In doubtful cases the diagnosis may be very difficult until some length of time elapses.

Prognosis: As far as life is concerned, this is good. Death during the acute stage is rare. Until three months have elapsed it is difficult to tell what the permanent damage will be. So long as the reaction of degeneration does not develop the prognosis for recovery in such muscles is good.

Acute anterior poliomyelitis—treatment: During the acute stage, if the diagnosis is made, the child should be kept quiet in bed, and lying on its face or side rather than its back. The diet should be liquid, and the bowels emptied by repeated fractional doses of calomel. Counter-irritation to the spine by means of mustard applications, or by cups, or the use of continuous cold, is advisable. Mild antipyretics answer for drugs. Ergot and bromides are theoretically indicated.

After the acute stage is over, and only a paralyzed limb is left, the muscles in this limb should be regularly exercised by the use of electricity in the form that will produce contractions. Massage is a very useful adjunct.

To prevent and to cure subsequent deformities various forms of orthopædic apparatus are advisable.

TRANSVERSE MYELITIS.

Various forms of general myelitis are occasionally seen in children; but this one, in which the lesion involves a complete cross-section of the cord of greater or less extent, is the only one of sufficient frequency to demand separate consideration.

Etiology: This condition is regularly due to compression of the cord from the sharp angle formed in the spine in (tuberculous) vertebral disease, or from a chronic thickening of the meninges due to the same trouble. Tumors of the cord, and fractures and dislocations of the spine produce the same lesions.

Pathology: The carious bodies of the vertebræ collapse and

produce bends in the spine. In addition, the peritoneum and meninges undergo a chronic inflammatory process, with thickening of their tissues. The mechanical bending and the inflammatory thickening together compress the cord and set up an *interstitial myelitis*, with gradual destruction of the cells and fibres, and with abolition of their function.

Secondary degenerations, ascending and descending, from the involved region, are also frequent. The nerve-roots are usually affected coincidently.

Transverse myelitis—symptoms: The essential symptom is a gradually increasing paralysis of the parts below the lesion, which paralysis is of the spastic variety and bilateral. The reflexes, superficial and deep, are increased, and there may be some anæsthesia. In old cases some atrophy may follow. If the myelitis is low down, bedsores and bladder and rectal difficulties are present. Pains radiating along the pinched nerves are an early and persistent symptom in many cases. The *course* of the disease is slow and chronic.

Diagnosis: With a careful examination of the spine as to its flexibility and deformity, the diagnosis of this disease is easy.

Prognosis: If the case is diagnosed early, and if under proper treatment of the spine the vertebral disease can be stopped, the cord will often return to normal. In the long-standing cases the prognosis is not so good, and many remain permanently paralyzed.

Transverse myelitis—treatment: This is almost entirely that of the vertebral disease causing the myelitis. The child should be put at rest on its back, and kept on a highly nourishing diet. Extension of some sort should be applied to the spine. Special efforts should be made to prevent the formation of bedsores, and the bladder must be watched. Iodide of potassium may be given internally with possible benefit.

FRIEDREICH'S ATAXIA.

Synonym: This disease of the spinal cord is also called *hereditary ataxia*.

Etiology: It is distinctly a family disease, with often more

than one child in the same family affected, and with an hereditary history. Neurotic and alcoholic families are most often affected.

Pathology: The lesion is a sclerosis of the posterior and lateral columns, and of the crossed pyramidal and direct cerebellar tracts of the cord.

Friedreich's ataxia—symptoms: There is an ataxic gait developing very early in life, and later on ataxia of the upper extremities. The motions, however, are stiffer and more rigid than those of locomotor ataxia. All movements are accompanied by a very coarse tremor. Actual paralysis is rare. Sensation is only slightly impaired. The reflexes are usually lost, or at any rate diminished. Bladder, rectal, and trophic symptoms occasionally occur. Contractures and deformities are frequent in the old cases.

The disease progresses slowly upward, and finally the medulla is involved, by which time the child becomes helpless.

Diagnosis: The two diseases most likely to be confused are locomotor ataxia and multiple sclerosis. Both are diseases of adult life, and both have many characteristic signs of difference from Friedreich's disease.

Prognosis: This is distinctly bad, although life is prolonged over many years, but the stage of complete helplessness surely approaches.

Treatment: Care for the general health is all that can be done.

PROGRESSIVE MUSCULAR ATROPHY.

Definition: By this condition is understood a slowly progressing atrophy of certain muscles in the body from disease of their "trophic centre" in the spinal cord. The causes are unknown.

Pathology: The ganglion-cells in the anterior horns of the spinal cord in some cases have been found atrophied.

Progressive muscular atrophy—symptoms: The disease begins slowly, and with no subjective symptoms save a gradual wasting and enfeeblement of certain muscles. It begins oftenest

in the muscles of the ball of the thumb, or in those of the little finger. It involves the interossei, and often later the deltoid. In other cases the anterior tibial or peroneal groups are affected. The extreme wasting is accompanied by proportionate weakness and by persistent fibrillary contractions. The electrical reactions are unchanged in kind, but are very feeble. The course of the disease is slow and chronic.

Diagnosis: This is rather easy, as no other disease simulates it.

Prognosis: This is bad. The disease is incurable, but is consistent with a fairly long life.

Treatment: General tonic treatment, with especial attention to the nutrition is of most importance.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

Synonym: This disease is also called *muscular dystrophy*, and is quite well recognized.

Etiology: It is a disease of early childhood, and shows a distinct tendency to run in families. The parents are apt to be neurotic.

Pathology: There is at first a true hypertrophy of the muscular fibres, but these soon undergo a fatty and then a fibrous degeneration, until finally all the muscle-fibres are replaced by connective tissue. The muscles on section appear yellowish. There are no lesions in the nervous system.

Pseudo-hypertrophic muscular paralysis—symptoms: The beginning of the disease is very gradual and difficult to date. A gradually progressive weakness in the legs is first noticed. The child is uncertain on its feet, and has great difficulty in climbing steps, or in rising from a sitting posture. In rising upright from the floor the child goes through a characteristic series of motions which were first described by Gowers. He first rolls over on his face, then raises himself on all fours, and then bringing his hands to his feet uses them in climbing up his legs and thighs, thus lifting up the weight of his body. Similar weakness develops in other muscles—in the *loins*, producing a marked lordosis; in the *shoulder* and *neck*, allowing the head to drop forward. These also produce a very protuberant abdomen.

Succeeding this weakness is a symmetrical *increase in bulk* of the affected muscles, evident especially in the calves, glutei, deltoids, and infraspinati. As the disease progresses this hypertrophy is succeeded by a gradually increasing atrophy, until in the end all the affected muscles are wasted and flabby.

The *gait* is swaggering, somewhat similar to the rolling gait of a sailor. There are no subjective symptoms, and no bladder or rectal trouble. The reflexes are diminished or lost, and the electrical reactions are decreased.

The *course* of the disease is gradually progressive to the stage of complete helplessness.

There are *three recognized types* of the disease—that involving the *legs* mainly; that involving the *shoulder-girdle*, or Erb's type; and that involving *the face and shoulder-girdle*, or the Landouzy-Déjérine type.

Diagnosis: There is no other disease with which this may be easily confounded, if a careful history is taken and examination is made.

Prognosis: Recovery is impossible. Death is frequently due to intercurrent disease.

Treatment: General constitutional treatment, with the use of strychnine, is all that can be done for these cases.

ACUTE MENINGITIS.

Acute meningitis, which is often called *brain fever*, may occur *sporadically* without known cause; is often *secondary* to inflammations about the head; and at times appears *epidemically*, when it is called *epidemic cerebro-spinal meningitis*. In these latter cases the membranes of the spinal cord are also regularly involved; in the others, they may or may not be.

Etiology: Injuries to the head, sunstroke, otitis, ethmoiditis, and other inflammations about the head are apt to cause meningitis.

In the sporadic and epidemic cases the cause is probably the action of some form of *coccus* in the presence of bad air and bad hygienic surroundings. The pneumococcus is most frequently found. Other cases complicate the various infectious diseases.

Pathology: The pia mater of the brain and of the cord is swollen and infiltrated with varying amounts of serum, fibrin, and pus. A distinct layer of these is usually found in the sulci and often over the convolutions. The ventricles are moderately dilated with purulent serum. The convolutions of the brain are somewhat flattened and their surface hyperæmic.

Acute meningitis—symptoms: The disease usually begins rather abruptly with a *rise of temperature;* at times a chill and intense headache. These are followed by *vomiting* of the *cerebral type*—that is, without nausea or reference to food—*hyperæsthesia* all over the body, *photophobia*, and *general pains. Convulsions* are quite frequent in children.

The fever runs an irregular course, tenderness and contraction of the muscles of the neck develop, and the headache becomes extreme. Restlessness, sleeplessness, and wild delirium gradually succeed one another. Later stupor and coma replace the active brain-symptoms. The intelligence is soon lost, and muscular contractions, with inequality of the pupils and various forms of strabismus, are seen. The bowels are usually constipated and the abdomen retracted. In the epidemic cases erythematous and petechial eruptions develop. The pulse at first is apt to be somewhat slow, but later becomes rapid and irregular.

The cases regularly progress from bad to worse, and die in the course of one to three weeks. Some few recover with a gradual disappearance of the symptoms and probable absorption of the exudate. In the cases which recover, a chronic thickening of the pia is frequently left, with a later development of hydrocephalus.

Many of the cases run a much more subacute course than the above, even beginning more slowly.

Diagnosis: In young children so many diseases have such marked cerebral symptoms suggesting meningitis that in the early stages we may frequently be in doubt over the diagnosis. As the disease progresses, however, the diagnosis becomes more positive.

The tubercular variety is differentiated by its slower course, and by the family and personal history. If a local cause

can be found for the meningitis, this also assists the diagnosis.

Prognosis: The mortality-rate is very high; but some cases do recover permanently and completely; in others various defects of the nervous system are left, as deafness, blindness, paralysis, and epilepsy; in others hydrocephalus subsequently develops.

Acute meningitis—treatment: The child should be put to bed, and kept on a fluid diet, in a quiet, darkened room. The hair should be cut short, and continuous cold applied to the head by a cold coil or by ice-bags. Counterirritation to the nape of the neck by repeated applications of iodine, or by a blister, may be used. The bowels should be kept freely open by the use of saline laxatives.

Internally the bromides, ergot, and iodide of potassium are indicated; the former to quiet the patient, the latter to relieve the cerebral congestion. If pain is severe, opium must be used. As the disease progresses alcoholic stimulation becomes necessary. Attention must be paid to the bladder to prevent retention. Restraint is often needed in the wild delirium. The sequelæ are to be treated on general principles.

TUBERCULAR MENINGITIS.

Synonym: This is also called *basilar meningitis*, and is quite a common disease of childhood.

Etiology: It is due to infection of the meninges by the tubercle bacillus. It is most apt to occur in children of a tuberculous heredity, or in those who have tuberculosis elsewhere in the body, as in the glands, the bones, or the lungs. It may develop a long time after the cure of glandular or bone-disease.

Pathology: There are miliary tubercles, and inflammatory exudate of serum, fibrin, and pus in the meshes of the pia mater. They may be found anywhere in the brain, but are most frequent over the base. The ventricles are moderately dilated with serum, the ependyma contains small tubercles, and the surface of the brain is flattened. Tubercle bacilli

are found in moderate numbers. Tubercular lesions elsewhere are apt to be found.

Tubercular meningitis—symptoms: In most of the cases there is a prodromic period lasting for a few days or weeks. The child during this time loses flesh and strength, and is less inclined to play. He is moody and irritable, sleeps badly, and may have a little evening fever with headache and a coated tongue. Sudden attacks of vomiting without apparent cause are rather characteristic of this stage. These symptoms vary a good deal, being more or less marked in the same case at different times.

Gradually these indefinite symptoms change into undoubted evidences of cerebral disease. There are now present irregular fever, intense headache, often convulsions, or alternating conditions of stupor and delirium. The child is restless, and sleeps badly, often crying out suddenly in his sleep. The bowels are constipated, and attacks of vomiting are frequent.

Photophobia, general hyperæsthesia, rigidity of different muscles, and transitory or permanent paralyses all develop. The pupils are dilated, or contracted, or unequal. There are stiffness of the neck, with some retraction, and at times opisthotonos. The pulse is slow and irregular; the respiration shallow, and also irregular. There may be retention of urine.

As the disease progresses the delirious and irritative symptoms gradually give place to stupor and coma and loss of all subjective sensation. The tongue and lips become dry and covered with sordes, and all intelligence and volition are finally destroyed. Toward the end there is inability to take food. The child dies in convulsions, or more often exhausted in coma. The temperature rises at the end.

The disease lasts from a week to a month, and almost invariably shows periods of distinct improvement, with amelioration of all the symptoms.

Diagnosis: In the prodromal stages the diagnosis is very difficult, and is seldom made. As the real meningitic symptoms develop, the disease becomes more definite, and a probable diagnosis is fairly easy. In certain cases, however, it is

almost impossible positively to differentiate the simple and tubercular forms.

From the cerebral types of non-meningitic diseases, after a few days' observation, the diagnosis is usually easily made.

Prognosis: Probably the disease is universally fatal. In the few cases of reported recovery there may have been an error in diagnosis, the condition having been the simple form.

Tubercular meningitis—treatment: This is precisely the same as for simple meningitis, and should be carried out with the possibility of an error in diagnosis having been made.

HYDROCEPHALUS.

This condition, called **water on the brain,** is of two varieties: *external hydrocephalus*, in which the exuded serum lies between the dura mater and pia; and *internal hydrocephalus*, in which the serum fills and distends the ventricles. The former condition is very rare. The latter, internal hydrocephalus, is the ordinary clinical form.

Etiology: Most of the cases begin during intra-uterine life, and possibly congenital syphilis may be an etiological factor. More than one child in the same family may be affected. In some cases different causes are found which press on the opening to the fourth ventricle and cause a mechanical obstruction to the outlet of the ventricular fluid. Such cases are tumors of the brain, or chronic thickening of the meninges. In most cases there is no satisfactory cause found.

Pathology: The ventricles are dilated, at times enormously, with thin serum having the characteristics of cerebro-spinal fluid. The fluid may vary in quantity from a few ounces to over a quart. The brain-matter is markedly thinned, undergoing pressure-absorption. The skull-bones are widely separated at the sutures and the fontanelles much enlarged. The ependyma may be normal or be somewhat thickened. Spina bifida is a rather frequent complication.

Hydrocephalus—symptoms: In some cases the hydrocephalus is so developed at birth as to be a serious obstacle to delivery, at times requiring puncture.

In other cases the symptoms develop soon after birth. The head is symmetrically enlarged, and increases in size often to extreme dimensions. The forehead overhangs the eyes and the face appears very small. The upper lid hangs so that the sclerotic shows between it and the iris. The sutures and fontanelles are wide open, and bulge and pulsate. Fluctuation may be obtained. The infant has difficulty in supporting its head, and its whole muscular system is flabby and feeble.

In the severe cases the intelligence is more or less interfered with. Strabismus, changes in the pupil, nystagmus, blindness, deafness, and various forms of paralysis are present. Convulsions often occur. In the milder cases nothing except the enlarged head is to be noted.

In some cases the exudation of fluid takes place after ossification of the cranial bones is accomplished, and then no enlargement of the head follows.

The *course* of the disease is slowly or rapidly progressive, and death regularly takes place before the development of puberty.

Diagnosis: The only disease likely to cause confusion is rachitis. In this the other signs of rickets are of assistance, and the head does not increase in size so fast, nor do the sutures or fontanelles gape so widely.

Prognosis: A few cases recover, but usually with a somewhat impaired intelligence. The large majority, however, are incurable, and continue to grow worse.

Hydrocephalus—treatment: There is nothing satisfactory to do. Attention to the general health should be strict. Mercurial inunctions may be tried. Surgical measures, such as aspiration or incision, are not satisfactory.

CEREBRAL ABSCESS.

Notwithstanding the frequency in children of the main causes of **abscess of the brain**, it is in them a comparatively rare disease.

Etiology: Suppurative otitis, with or without involvement of the mastoid cells, and traumatism to the head are the com-

monest causes of this disease. General sepsis and various inflammations of the scalp cause some cases. In a great many no cause is discoverable.

Pathology: The abscess is usually single, varies in size, and may be situated almost anywhere in the brain, but is most common in the temporo-sphenoidal lobe or the cerebellum. There may be an encapsulated collection of pus, or only an irregular cavity containing a greenish pus with broken-down brain-tissue in it, and surrounded by inflamed brain-tissue.

The abscess may rupture into the ventricles, or set up a meningitis, or cause a thrombosis of the lateral sinus.

Cerebral abscess—symptoms: There are usually described three periods in abscess of the brain—an initial, a latent, and the terminal period.

The *initial symptoms* may be mild or severe, and resemble those of acute meningitis, being fever, prostration, headache, general convulsions, vomiting, and delirium or stupor. The headache is apt to be localized to the affected area. After a variable time the symptoms gradually subside and the latent stage begins.

During the *latent period*, which may be short or long, all the symptoms may disappear, or only the headache remain. If any local symptoms have developed, they also persist.

After a variable time the *terminal stage* begins rapidly or gradually, the fever, headache, vomiting, optic neuritis, general convulsions, and delirium or stupor return. Various forms of paralysis and other local symptoms may now be present, and all the symptoms go from bad to worse.

The first stage marks the development of the abscess, and death may take place during this. The latent period means the encapsulation of the abscess. The final stage means its rupture into the ventricles, or its spread to fresh portions of the brain, or the development of a complicating meningitis. Death takes place from convulsions or from exhaustion in coma.

The disease may last for a few weeks only, or, with a long latent period, may be prolonged over many months.

Diagnosis: Tumors of the brain are distinguished by the lack of any signs of inflammation. From acute meningitis

the diagnosis is intensely difficult. Abscess is a slower disease; and, as a rule, shows more of the local and less of the general cerebral symptoms than meningitis.

Prognosis: Without proper treatment all the cases die. A few are saved by surgical interference.

Cerebral abscess—treatment: During the beginning stage it is that of acute meningitis—cold to the head, free purgation, and the use of bromides. As soon as the diagnosis of abscess can be made and its situation localized, it should be treated surgically.

CEREBRAL TUMORS.

Tumors of the brain of different kinds are fairly common in children.

Etiology: No definite cause is known for any of them. Trauma is often assigned. Tubercular, sarcomatous, and carcinomatous tumors are frequently secondary to similar growths elsewhere in the body.

Pathology: The commonest varieties are the tubercular tumor, the glioma, the sarcoma, and the cyst. Carcinoma, gumma, and mixed growths are occasionally found.

Tubercular tumors are often multiple, and are located most often in the substance of the cerebellum or cerebrum.

The gliomata resemble the structure of neuroglia, are usually single, and also found most often in the cerebellum.

Sarcomata may originate in the brain-tissue, the meninges, or the bones of the skull.

Cysts are found anywhere in the brain; they may be parasitic or degenerative.

Cerebral tumor—symptoms: These are best considered under two heads—the general symptoms, which are common to all intracranial growths; and the local symptoms, which are due to the situation of the tumor.

Under the *general symptoms* the first in importance is headache, which is regularly excessive and constant. It may be localized to one part of the head or be general. Repeated vomiting without cause and unaccompanied by nausea is quite a frequent symptom of brain tumor. Vertigo, either constant or paroxysmal, is often present. Optic neuritis, with

more or less interference with vision, is an early symptom. General convulsions occur early, and are repeated as the tumor grows. Mental changes—irritability, loss of memory, and emotional excitement—are to be expected. Insomnia begins early; later, delirium may be present; and toward the end we find stupor and coma. The disease may last for many years before death.

The *local symptoms* depend on where the tumor grows. In the frontal lobes, the emotional and intellectual functions are especially involved, but these local signs are vague. In the third left frontal there is motor aphasia.

In the *motor area*—that is, around the fissure of Rolando—there are early seen convulsive twitchings, or spasms, of the leg, arm, or face of the opposite side, according to the region involved. Later there may develop complete paralysis of the same. This is the so-called Jacksonian epilepsy, and it is important to notice where the spasm starts and how it spreads, as this enables us to localize the site of the tumor more accurately. In the parietal lobes there may be some indefinite sensory symptoms on the opposite side of the body. In the occipital lobe there is developed a homonymous hemianopsia. In the temporo-sphenoidal lobe there is sensory aphasia: the patient can speak, but cannot understand what is spoken. On the base, in the anterior fossa we have interference with smell. In the middle fossa there is atrophy of the optic nerves, or interference with the function of any of the first six pair. In the posterior fossa there will be interference with the function of one or more of the last six pair of cranial nerves. In the cerebellum there is produced cerebellar ataxia. In the substance of the cerebrum, and in the crus there are interference with the functions of the motor tract and hemiplegia of the opposite side.

Diagnosis: Tumor must be diagnosed from abscess by the presence of fever and the absence of optic neuritis in the latter condition. From tubercular meningitis of slow type the local symptoms of tumor are of main value in diagnosis.

It is also necessary to locate, if possible, the site of the tumor, and to decide its variety. The focal symptoms decide the former point, and the latter is helped by the presence

of tuberculosis or sarcoma elsewhere, or by a history of syphilis.

Prognosis: It is a fatal disease. The symptoms progress steadily till death. If the tumor is a gumma, which is very rare in children, the prognosis is better.

Cerebral tumor—treatment: If syphilis is suspected especially, but under any circumstances in every case, give mercury and iodide of potassium a thorough trial. Otherwise surgery is our only recourse, and even with successful localization of the tumor the surgical results are not brilliant. Pain will require opium.

INFANTILE CEREBRAL PALSIES.

Classification: Clinically there are *three general classes* of patients to be described under this head: those suffering from *hemiplegia*, or paralysis of one side of the body; those suffering from *diplegia*, or paralysis of both sides; and those suffering from *paraplegia*, or paralysis of both lower limbs.

Etiology and pathology: 1. Some of the cases are due to *intra-uterine disease of the brain*, which may be a congenital defect or a hemorrhage. Porencephalus and cysts are sometimes found. In others there is an arrested development of the cortical cells.

2. More often the case results from some *trauma* inflicted on the brain *during parturition*. The lesion is regularly a meningeal hemorrhage, which is more often due to prolonged tedious labor than to the use of forceps. Asphyxia at birth is usually found in these cases. Secondary changes in the brain follow the hemorrhage, as a diffuse meningo-encephalitis with atrophy and sclerosis of the cortex. Cysts are found in some cases. Secondary degenerations of the motor tract in the internal capsule and cord regularly follow in the older cases.

3. Other cases develop *after birth*, and may follow an injury to the head, or be subsequent to one of the infectious diseases. Severe whooping-cough is often assigned as a cause. General convulsions often leave behind some form of paralysis. There is found hemorrhage, thrombosis, or embo-

lism in the brain. Subsequent meningo-encephalitis, with atrophy and sclerosis, and descending degenerations in the motor tract are found.

Infantile cerebral palsy—symptoms: The symptoms are a greater or less degree of spastic paralysis in different muscular groups of the body. The distribution may be to one side, hemiplegia; to two sides, diplegia; or to the lower extremities, paraplegia.

The child is usually brought to the physician with the story that it cannot walk. Convulsions of an epileptic nature are fairly frequent. The child remains small and poorly developed. The affected limbs are rigid, and resist quick efforts at moving them. The reflexes are markedly increased both in the affected limbs and all over the body. The muscles atrophy slowly from disease, and after some time contractures of the affected parts take place with the joints in flexion. Athetoid movements are also regularly present. Efforts at walking are usually associated with such a tendency to adductor contraction as to cross the legs over each other. The mental faculties are more or less impaired, sometimes to complete idiocy. Speech is usually imperfect, and hearing and sight may be defective.

The *acquired variety* is apt to begin with a convulsion, followed by fever and symptoms like meningitis, but with hemiplegia left behind. In these cases the mental condition is more nearly normal than in the congenital or birth palsies.

Many of the congenital and birth cases give a history leading us to believe them acquired, as the mother thinks the child was normal for some months after birth, but more careful observation would decide otherwise.

Diagnosis: Cerebral palsies differ from spinal paralysis by having rigid muscles, and not flaccid, atrophied ones. The reaction of degeneration is not present. The acquired form may suggest meningitis in its onset; but the quick recovery from the acute symptoms, with paralysis left, soon decides the diagnosis.

It is well to discover, if possible, for the sake of prognosis, whether the case is congenital, due to birth-trauma, or acquired.

Prognosis: This is better in the acquired hemiplegia than in the diplegias or paraplegias of congenital or birth-origin. None, however, recover completely without some mental impairment in addition to the physical deformity, and all are subject to epilepsy.

Infantile cerebral palsy—treatment: During an acute attack treat as meningitis, by a purge, ice to the head, and bromides internally.

After this, attend to the general health, and prevent deformities by the use of proper apparatus. If deformity is present, perform tenotomies and apply braces. These children are fit subjects for education in institutions for the feeble-minded. In cases of epilepsy bromides and surgery are our recourse.

IDIOCY AND IMBECILITY.

The **difference** between an **idiot** and an **imbecile** is one of degree only. Both are permanent conditions due to changes in the cerebrum, which may be an arrest of development of a congenital nature; or the result of inflammatory or traumatic injury, or premature ossification of the skull.

An **idiot** has his intellectual faculties completely impaired, and is really little more than an animal in human form. An *imbecile* is often called simply a feeble-minded person, and is really a high-grade idiot. His mental development is often fair, but his self-control and emotional faculties are very deficient.

Idiocy and imbecility—symptoms: The main sign is the inability of the brain to receive, to utilize, and to produce mental conceptions. All varieties, from absolute lack of any mental action to simply an unbalanced mental deficiency, are seen. The least marked cases are the "backward" children in a family or in school.

In many of them the moral nature seems absolutely lacking, although they may be bright and quick at most things. This is the class that furnishes a large number of the habitual criminals to society. In other words, they are degenerates.

Deficient mental development may appear evident in chil-

dren at a very early age, but the milder degrees are usually not discovered until much later.

Physical signs are frequently seen which should attract our attention to the condition. Among these are a microcephalic or a misshapen skull, with a markedly receding forehead. The so-called stigmata of degeneration may be present, high arched palate, prognathism, irregularities in the teeth, malformations in the ears, anomalies in the fingers, and left-handedness. These children are apt to be restless, continually walking about, or keeping some limb in motion, and they go through various purposeless performances repeatedly.

Diagnosis: A little observation of the mental and physical condition rapidly makes the diagnosis sure. It is well to attempt to find the origin of the condition, whether it is congenital or acquired.

Prognosis: This is absolutely bad as regards the mental defect. It has, however, no bearing on the prolongation of life.

Idiocy and imbecility—treatment: Much can be accomplished in developing any dormant intellect in these children by *education* in a thoroughly equipped institution.

CRETINISM.

Definition: This disease consists in the arrested physical and mental development of a child. It occurs endemically in certain portions of the world, and sporadically in our country, being fairly common now that it is more often looked for.

Etiology: The condition is due to the loss of function of the thyroid gland. The gland may be congenitally absent, or its glandular structure may be replaced by other tissue, or the organ may have been removed by surgical means.

Cretinism—symptoms: The characteristic signs of the disease may appear during the first year of life, or not until later. They develop rather slowly, but steadily become more marked. When well developed the appearance is unmistakable. The child is short in stature, and light in weight for its age. The limbs and fingers and toes are short and

thick. The fontanelle is very late in closing, the nose is flat, broad and upturned, the alæ being thick, and the nostrils wide open. The lips are much thickened, and the tongue is large and constantly protruded. The teeth are cut late, and are badly formed and irregularly placed. The hair is sparse, but coarse and straight. The skin of the entire body is thick and dry, but does not pit on pressure. In the supraclavicular regions there are regularly formed pads of fatty tissue, which give the neck a shortened, thickened appearance. The thyroid gland can usually not be felt unless it contains a tumor. The abdomen is large and prominent, and an umbilical hernia is frequently present.

Walking and talking are learned late, and are very imperfectly performed even then. The sexual functions are developed very late in life; in fact, the infantile condition persists over many years. Constipation is often found, which seems to be directly due to the cretinoid condition, as it usually disappears quickly under treatment.

The temperature is apt to be subnormal, and the mental condition is one of extreme apathy and dulness.

Diagnosis: It is very important to make an early diagnosis, as when treatment is begun then the child may be brought back to virtually a normal state. By a little care, after one or two well-developed cases have been seen and their typical appearance well impressed on the mind, the condition should not be overlooked even in its incipiency.

Prognosis: If untreated, the cases grow worse and worse. Treatment begun early seems to be able to eradicate the effects of the disease. When begun late great improvement occurs, but probably the child will never become normal. The physical improvement is more marked than the mental. Under any circumstances "thyroids" have to be given indefinitely to prevent recurrence.

Cretinism—treatment: Thyroid extract daily by mouth in doses of one to five grains replaces in the system the active principle of the normal thyroid gland. At the same time attention to the diet, exercise, fresh air, and the moral and mental education of the child are of the utmost importance.

FIG. 1.



FIG. 2.



FIG. 3.



Fig. 1. Characteristic appearance of a Sporadic Cretin, aged 23 months; length, 23 inches; circumference of abdomen, 19 inches.

Fig. 2. Shows change after three months treatment with thyroid; abdomen measured 16 inches.

Fig. 3. Illustrates the condition after five and a half months' treatment; height, 30 inches; abdomen measured 15 inches. (Osler.)

CONVULSIONS.

Spasmodic contractions of the muscles are very common in infancy. They are often called *eclampsia*. They are to be looked on as a symptom of disturbance in the motor area of the brain due to various causes.

Convulsions—etiology: The great predisposing cause is the markedly increased excitability of the lower reflex centers of the nervous system, and the poor development of the higher inhibitory centres in infancy.

Nutritive disorders increase this tendency by interfering with the proper nutrition of the nervous centres. Rickets, anæmia, malnutrition, and intestinal diseases are of greatest importance as predisposing causes. An hereditary neurotic taint is also a great predisposer to convulsions.

Exciting causes are various, and often seemingly unimportant. All inflammations, injuries, and pathological lesions of the brain are apt to be causes of convulsions. Irritation from trauma elsewhere on the body; from renal, hepatic, or intestinal colic; from undigested food; from phimosis; from foreign body in the ear, and probably from dentition and worms, may start a general convulsion. Finally, fever from any cause, as heat-stroke, or the beginning of an infectious disease, is a very frequent exciting cause.

While in most cases some cause, direct or indirect, may be found; still there are a good many that must be called idiopathic.

Pathology: The probable lesion is a hyperæmic condition of the motor region of the brain.

Convulsions—symptoms: There may be prodromal symptoms of restlessness, twitchings of the facial muscles, grinding of the teeth, or rolling of the eyes.

Usually, however, the general spasm comes on suddenly and unexpectedly. The eyes become fixed, the jaw clenched, the skin pale, the limbs rigid with a tendency to flexion in all the joints, and the neck retracted. Consciousness is abolished.

This tonic contraction of the muscles may persist; but it is usually followed by clonic contractions, with jerking move-

ments of all the limbs. There are then frothing at the mouth, working of the jaw, irregular rolling of the eyeballs, twitching of the facial muscles, and spasmodic action of the muscles of the trunk and extremities. Respiration is spasmodic, due to involvement of the diaphragm. The pulse is feeble and irregular. The skin and mucous membrane become cyanotic. Emptying of the bladder and rectum is common.

These convulsions last from a few minutes to an hour, and leave the child in a condition of stupor. They may be repeated after a short or long interval of quiet. Multiple recurrences extending over many days are fairly common, and even then may be followed by complete recovery. Death may take place during the first spasm or in the subsequent ones.

Localized spasms of certain regions may occur at times without meaning permanent or organic focal lesion of the brain.

Diagnosis: The only necessity in diagnosis is to discover as quickly as possible the *cause* of the spasm. All the etiological factors should be taken up in order and each one excluded. In new-born babies the probabilities are in favor of meningeal hemorrhage or of tetanus. In older children gastro-intestinal irritations, or the beginning of one of the infectious fevers, are the commonest causes. The pulse and temperature should be taken, the fontanelles examined, the urine analyzed, and the history carefully investigated in every case.

Prognosis: In fairly strong children, and except when actual brain-lesion is present, convulsions are not commonly fatal. Those from reflex irritation, and due to fever, are ordinarily not dangerous. In very feeble children, and when they mark the beginning of intracranial disease, they are much more serious. The possibility of the convulsions being epileptic must be remembered, as this means recurrences during an indefinite number of years.

Convulsions—treatment: The child should be kept as quiet as possible, and all rubbing and unnecessary manipulation forbidden. A warm mustard bath or pack may be given

until the skin is reddened. If there is fever, a cool bath should be substituted. Ice applications to the head are always helpful.

During the convulsion the child should be immediately put under the influence of chloroform, and kept so until the convulsive tendency has disappeared. While anæsthetized it should be given by rectum suitable doses, for its age, of chloral and bromide, which may be repeated hourly. If these drugs fail to prevent recurrences, it is quite justifiable to use morphine hypodermatically in proper dose.

In cases where the *digestive tract* is believed to be the offender the stomach and bowels should be emptied by washing or drugs. In many cases a high rectal douche of warm water should be given. After the spasm is controlled hunt for the cause, and treat that condition to prevent recurrence.

TETANY.

Definition: This is a functional nervous disease characterized by tonic spasms in certain groups of muscles, especially those of the hands and feet, and occurring in paroxysms.

Etiology: It occurs almost always in infants who are suffering from rachitis, marasmus, or other forms of malnutrition, and from gastro-intestinal disorders.

The irritation of cold and wet, or of indigestible food, regularly excites an attack in those predisposed.

Pathology: There are no lesions connected with the disease. Probably malnutrition of the nerve-centres increases their reflex excitability.

Tetany—symptoms: The attack consists of tonic spasms in the extremities, and especially the hands and feet. It begins rather rapidly, and is continuous over some time. There are no loss of consciousness and no marked subjective symptoms. Pain is caused by efforts at overcoming the spasm, but is ordinarily not spontaneous. The typical position assumed in the affected limbs is for the forearm to be pronated, the wrist flexed, the thumb turned in, and the fingers flexed at the metacarpo-phalangeal joints. In the feet the position of equino-varus is assumed, with the plantar surface arched and

the toes bent. In the more marked cases the arms and legs are adducted, the spasm extending upward to the upper arms and thighs.

The duration of the attack is from a few days to several weeks, and recurrences are quite common.

Diagnosis: The lack of unconsciousness, the typical position taken by the extremities, and the presence of the predisposing cause usually decide the diagnosis. Trousseau's symptom, the production or the augmentation of the spasm by pressure on the nerve or vessels of the affected limb, may be used as a diagnostic sign.

Prognosis: It is not a serious disease, and recovery from the tendency to it, as well as from the attack, is to be expected.

Tetany—treatment: Find and remove the cause, which is usually intestinal. During the spasm use chloral, bromides, and antipyrin internally. Attend to the patient's nutrition and digestion in order to prevent recurrences.

EPILEPSY.

Definition: This is a functional neurosis characterized by convulsions, which are accompanied by loss of consciousness.

Etiology: The disease most commonly develops in children approaching puberty, although cases are seen much younger. A neurotic or alcoholic heredity predisposes to epilepsy. It develops often after convulsions occurring in infancy, and in children suffering from cerebral palsies.

In a predisposed individual sudden fright, traumatism to the head, great emotional excitement, or excessive heat will develop the disease. Adenoids, phimosis, foreign bodies in the ear, menstrual disorders, masturbation, and intestinal toxæmia are often causative factors.

Pathology: There are no discoverable lesions of the disease. It must be looked upon as an explosion of energy in the cerebral cortex.

Epilepsy—symptoms: There are two distinct types of the disease, the *grand mal* and the *petit mal*.

The *grand mal* variety consists of a sudden attack of unconsciousness, the patient falling to the floor with a sharp cry,

the eyeballs rolling up, the jaw set, and all the muscles of the body in *tonic spasm*. The skin becomes cyanotic; the bladder and rectum may be evacuated.

This condition is succeeded after a few seconds by the second stage, that of *clonic spasms*. During this stage the muscles alternately contract and relax violently. The jaw is moved up and down, the tongue is apt to be bitten, there is frothing at the mouth, and the head is twisted to one side. The extremities relax and then become rigid, the muscles of respiration again begin action, and the cyanosis passes off.

This clonic stage gradually disappears, the patient regains consciousness for a moment, and then passes into a deep sleep of exhaustion lasting some hours. After awaking he has no recollection of what has happened.

The *petit mal* variety consists essentially in a sudden, short loss of consciousness, coming on while the child is engaged in any action, lasting a few seconds; and after it is over, immediate resumption of what was being done before, without recollection of the interruption. The child does not fall, but usually remains fixed in whatever position it occupied before the onset, while the eyes have a vacant stare.

In either variety there is often preceding the attack a warning or premonition of its advent. This is called the *aura*, and when present it is usually the same before each convulsion, and may be motor or referred to any one of the senses.

The seizures are repeated at irregular intervals, growing usually more frequent as time passes, and often recurring many times in the same day.

They occur both by night and by day, and both types may be seen in the same individual.

Diagnosis: Hysterical convulsions are to be excluded by differences in the character of the movements, and by the presence of consciousness and recollection of the event. Uræmic convulsions are proved by examination of the urine. Diseases of the brain produce more localized convulsions, and leave some evidence of their occurrence after they pass off.

In cases suffering from petit mal only, observation must be

continued over some time before a diagnosis may be positively made.

Prognosis: The danger to life consists in a serious accident happening to the child during the unconsciousness of the convulsion, as falling in the fire, or in front of a car, or from a height.

If the cases are recognized early and a cause found that is removable, the chances of cure are fair. In the old cases treatment seems only to lessen the number of convulsions. Surgery has helped some of the cases where a local lesion was suspected in the skull.

Epilepsy—treatment: During the attack simply prevent the child from injuring itself or biting its tongue. Epileptics should always have a companion. Discover, if possible, any cause, direct or reflex, and remove it, going systematically through the whole list of etiological factors. Regulate all the functions, particularly the digestive, in the most careful way. Insist on the proper amount of exercise, sleep, fresh air, proper clothing, proper food, regulation of the school- and play-hours, and on suitable domestic surroundings.

As *drugs*, the bromides given in full doses over a considerable length of time are the most valuable therapeutic help we have. In petit mal belladonna combined with the bromides seems helpful.

In *traumatic epilepsy*, where a localized depression is found in the skull, cerebral surgery may be tried with some hopes of success.

CHOREA.

Definition: The ordinary name for this disease is *St. Vitus' dance*. It is a functional neurosis characterized by irregular rapid twitchings of a few or all of the muscles of the body.

Etiology: It occurs most commonly from the seventh to the fourteenth year, and more often in girls than in boys. Children born of neurotic or alcoholic parents are predisposed to the disease. It often follows scarlet fever or other of the infectious diseases. Many cases develop in anæmic children. Of all causes, however, the *rheumatic diathesis* is most often present in the subjects of chorea.

The exciting cause of an attack is often a great fright, or overwork at school, or the presence of adenoids, worms, or phimosis. The early evidences of beginning menstruation are often exciting factors.

Pathology: The pathological condition present is probably some disturbance in the nutrition of the nerve-centres in the motor areas in the brain.

Chorea—symptoms: The attack usually develops gradually, with clumsiness on the part of the child in making voluntary movements with the extremities to be affected. Twitching of the facial or trunk muscles may be first noted. Soon the typical, irregular, spasmodic movements of these muscles develop, until the child can only with difficulty use the limbs, the chronic jerkings interfering with the normal, intentional motions. The less observed and quieter the child keeps the less marked are the movements. When noticed, or when desiring to make some muscular movement, the chorea becomes worse. The same is true under efforts to control them. The movements usually cease during sleep.

The affected muscles are weak, and the mental condition of the child one of extreme irritability. The speech is regularly involved and at times very difficult to understand.

On examining the *heart*, in a large majority of the cases, a soft systolic murmur is heard over the mitral or pulmonic area. In many children this disappears after recovery, in which case it was probably anæmic; but in others it remains permanent, when it was probably a rheumatic endocarditis. These children are regularly anæmic, with poor appetites and disturbed sleep, and other evidences of disturbed nutrition.

The attacks last ordinarily from two to three months. Some become chronic and may last for years. Recurrences are rather to be expected even after complete recovery.

Diagnosis: This is usually easy. Tic convulsif, due to irritation of the fifth nerve, may be mistaken for chorea, but is never found outside the distribution of this nerve.

Prognosis: Chorea may be so severe as to endanger life in itself, or from endocarditis, but this is rare in childhood. Recovery is usually complete in the ordinary cases, the heart-lesion of the rheumatic diathesis being the only serious re-

mainder. Recurrences after some months or about an even year may be looked for.

Chorea—treatment: The child should be taken from school, and kept comparatively quiet in one room, and should not be allowed to see too many visitors. The diet should be simple and easy to digest, and the room kept properly ventilated.

If there are evidences of rheumatism present, salicylate of sodium should be used. For the chorea itself, *arsenic*, given in the form of Fowler's solution in gradually increasing doses to tolerance, is almost a specific. Iron in some easily assimilable form is advantageously combined with it. In cases where the spasmodic movements are very excessive bromides, or chloral, or both, may be given at the same time to dull the reflex irritability of the nervous system.

HYSTERIA.

Hysteria, while rare in childhood, is still seen at times in certain of its phases.

Etiology: The disease is seldom seen before the tenth year, and occurs as often in boys as in girls. A neurotic or alcoholic heredity is usually present. Malnutrition and improper school and home education and surroundings are most important etiological factors. The modern forcing forward of children is responsible for much of it. Some sudden external irritation to the nervous system awakes the attack.

Pathology: There are no known pathological changes in this disease.

Hysteria—symptoms: Almost any of the varied symptoms of disease may be simulated by hysteria.

Sensory disturbances are quite common, hyperæsthesia of almost any of the superficial or deep structures of the body being often present, such as headaches, joint-pains, and abdominal tenderness. Blindness and deafness may also occur. Anæsthesia is less frequent, but is also found.

In the *motor* sphere various forms of paralysis are common. Aphonia, monoplegia, or even paraplegia may develop. Various contractures in the extremities are quite common. The most frequent motor symptom, however, is the hysterical

convulsion. The child screams, or laughs, or cries, and rolls around, throwing the limbs in irregular purposeless movements. Opisthotonos is frequent, but the patient never hurts himself as in epilepsy. Consciousness is retained, and there is present afterward a remembrance of the fit.

Psychical symptoms are often seen—morbid appetite, refusal of food, craving for sympathy, increased self-consciousness, and depressed states bordering on melancholia. In others there are great excitability and fits of ungovernable passion. Hallucinations, night-terrors, and disturbed sleep are frequent. Symptoms of more than one variety are often seen in the same patient.

Diagnosis: Observation over a prolonged period of time is often necessary before organic disease can be excluded. The possibility of the two conditions being present at the same time must not be forgotten. Eventually the diagnosis becomes quite easy and positive.

Prognosis: This is not very good. Periods of improvement are seen, but relapses are common, and later in life these patients usually become highly neurotic.

Hysteria—treatment: Everything possible should be done to improve the general health and nutrition of these children. Fresh air, proper exercise, regulation of the bowels, easily digested and simple food; regulation of the work and the recreation; avoidance of novels, theatres, and unsuitable associates, are all points for medical supervision. Often a great deal is accomplished by removing them from family and friendly influences, and putting them under the charge of a suitable individual who will treat them with firmness and tact. In other words, the treatment is entirely hygienic and moral.

Hysterical symptoms as they arise may call for treatment, and some rather unpleasant variety should be tried for each symptom, as counter-irritation, electricity, cold douches, and other similar therapeutical means, any one of which will appeal to the child from the standpoint of suggestion.

HEADACHES.

Headaches are fairly common in children, especially in girls approaching puberty. In infants they are rare, except as a symptom of organic disease of the brain.

Etiology: The commonest causes are anæmia and other forms of malnutrition; constant breathing of impure air charged with carbonic acid gas and other impurities; chronic indigestion and constipation, with absorption of products of intestinal decomposition; uræmia, malaria, the rheumatic diathesis; and, reflexly, diseases and anomalies of the eyes, nose, and ears. A large class includes the so-called *nervous headaches*, in which there seems simply a tendency for the brain to ache under some unknown circumstances. These are usually children of a neurotic heredity. Another class are the *sick headaches*, in which nausea and vomiting accompany the pain. These are probably due to a toxæmia from an excess of the end-products of proteid metamorphosis in the blood.

Pathology: The changes in the brain that cause pain are probably entirely circulatory.

Headaches—symptoms: Pain in the head is *the* symptom. It may be localized or diffuse. It may be one-sided or on both sides. It may be accompanied by symptoms elsewhere, as nausea and vomiting, or rheumatism, or other sign pointing to the etiology.

Diagnosis: The only point in diagnosis is to discover the cause. Go carefully through all the possibilities, and observe the child closely, excluding one cause after another, and eventually it can be decided in most cases to what the headaches are due.

Headaches—treatment: To cure the attack, phenacetin and caffeine are the main drugs of value. They may be combined with cold applications to the head and hot mustard foot-baths.

After the attack is over treat the cause if it can be found. Enrich the blood, improve the nutrition, regulate the diet and the bowels, remove malarial, rheumatic, or lithæmic tendencies, see that the kidneys secrete properly, correct errors of refraction in the eyes, and cure nasal, naso-pharyngeal, and ear affections.

SPEECH-DISORDERS.

We are frequently consulted for advice about the various **functional difficulties of speech** which are so common in children.

Varieties: The main forms are late development of speech, lisping, stuttering, and aphasia.

Late development of speech: Most children should learn to talk during the second year of life. Much depends, however, on efforts made to teach them. In children with no training, or in those who have been seriously debilitated by sickness, the function of speech may be much delayed even when there is no brain-defect. Time and a little attention will quickly remedy this. Very late speech is suspicious of cerebral anomaly.

Lisping: This consists in an inability to articulate clearly the hissing sounds. It may be simply a habit; but at times it seems almost impossible to place the tongue and teeth and lips in such a position as perfectly to form these sounds. It is never a serious defect, and can usually be overcome by proper training begun early before the habit becomes too fixed.

Stuttering: *Stammering* is a term also used almost interchangeably with this. It is rare before the second dentition, and consists in an inability to connect consonants and vowels together into a continuous word. Certain consonants seem especially hard to enunciate. Singing is often done with no hesitation.

Stuttering is likely to develop in children of neurotic heredity, in those who are overworked at school, and in the badly nourished. In some it is an *imitative habit*. Removal of the cause and improvement of the general health will often cure the habit. In the older cases a process of careful training in enunciation and use of the voice, by a person skilled in such work, should be undertaken.

Aphasia: By this is meant a temporary functional loss of speech, not the form due to disease of the third left frontal convolution. It is seen at times after severe attacks of illness, as typhoid fever, and after marked emotional excite-

ment. It usually recovers spontaneously in a comparatively short time.

SLEEP-DISORDERS.

There are frequently found in children **disturbances of sleep.** The two of most importance are *marked restlessness*, amounting almost to insomnia, and *night-terrors.* The two conditions may be treated together, as they seem to depend on similar causes and to differ in degree only.

Etiology: The commonest causes are derangements of the digestive tract, due to improper feeding in some way, either over-feeding or under-feeding, or the giving of unsuitable forms of food. This leads to colicky pains, to chronic indigestion, and to absorption into the blood and circulation through the brain of intestinal toxins. Earache, obstructed respiration (the result of adenoids or other causes), and general nervous irritability from excessive fatigue, reading or hearing exciting stories, and improper home-surroundings, are the common causes of many cases.

Sleep-disorders—symptoms: There may be only a restless, disturbed sleep, with frequent waking; or an actual insomnia. This latter is comparatively rare in children.

In *night-terrors* the child suddenly awakes from his sleep in great fright, half remembering some dreadful dream. At first he is bewildered; but gradually his mind becomes clear, and after a time he goes off to sleep again. Recurrences are frequent.

Diagnosis: This is fairly easy. The night-terrors might be confused with nocturnal epilepsy; but a little careful observation will usually settle the diagnosis.

Prognosis: This is good, as by proper treatment the condition can be cured.

Sleep-disorders—treatment: Find the cause, if possible, and remove it. Regulate the diet, the bowels, the exercise, the reading of books, and listening to stories. Improve the general health in every way. Give no drugs, if possible to dispense with them. If necessary, use trional or bromides, given at bedtime.

BAD HABITS.

The two **bad habits** that are most often seen in children are *sucking* and *masturbation*. The former may lead to the latter.

Sucking: This is very common. It consists in sucking of the fingers, or toes, or some foreign substance, such as a rubber-nipple. Often the fingers will be sucked so constantly as to produce distinct maceration of the skin. In addition to its being a bad habit, probably the baby sucks some wind into its stomach by its persistent efforts. Great pains should be taken in the beginning to prevent the formation of the habit before it becomes so fixed as to be hard to cure. Often the fingers will have to be bandaged and tied.

Masturbation: Masturbation, it must not be forgotten, is quite common among small children of both sexes, and is even seen in babies of a year old. It may result from local irritation of the genitals, as from phimosis, worms, vulvitis, itching skin-diseases, or irritating urine. Other cases are taught by forms of play or exercise in which friction is made on the genitals; and still others by playmates or nurses. It may be performed by use of the hands, or by rubbing the thighs together, or by friction against some external object. Children the subject of this habit are apt to be anæmic and poorly nourished and irritable. Locally the genitals are relaxed, and there may be slight redness of the prepuce or vulva. Careful observation extended over some time is necessary to detect these cases. The younger the child, and the sooner the habit is discovered, the more apt will we be to break the indulgence.

Masturbation—treatment: Intelligent moral care from the parents is of most value. Any local cause of irritation should be removed. The general health particularly should be improved in every way. Mechanical restraints may help in very young children, but are useless in older ones. The child's companions should be carefully selected. Hypnotism has been of value in some cases. Corporal punishment will not often be of much use, except in individual cases where the child's temperament is such as to be much influenced by this kind of treatment. Many of the cases are very difficult to handle.

CHAPTER XII.

DISEASES OF THE LYMPH-NODES.

ACUTE ADENITIS.

Definition: This is an acute exudative inflammation of the lymphatic glands. It is exceedingly common in children.

Etiology: This is probably always secondary to an abrasion or inflammation, of one kind or another, of the skin, or mucous membrane of the area drained into the inflamed glands. The original lesion may be so inconspicuous as to be overlooked. The commonest causes are rhinitis, naso-pharyngitis, stomatitis, dentition, otitis, eczema, diphtheria, scarlet fever, measles, and influenza. From these the cervical glands are affected. The axillary glands are involved from vaccination, paronychia, trauma to the hand or arm, or skin-eruptions on the same. The inguinal glands inflame from lesions about the genitals, anus, or feet.

Adenitis is most frequent during the first two years of life, the absorptive power of the lymphatics seeming most active then. It occurs in both healthy and delicate children, but seems most frequent among those in institutions.

Pathology: The glands are swollen, infiltrated with the products of exudation, and the surrounding tissues are usually involved. If the infection is severe enough, there is such an excessive emigration of white cells as to cause the glands to break down and form abscesses. The milder cases go on to resolution with absorption of the inflammatory products.

Acute adenitis—symptoms: Most of the cases have some fever, which may reach 102° F. or more, with its accompanying malaise. The inflamed glands are swollen, a little painful, and usually quite tender. When under the sterno-mastoid muscle a voluntary torticollis is often caused. When only the gland is involved the skin retains its normal color; but if the surrounding tissue becomes inflamed, the skin

reddens. These are the cases apt to suppurate. Only one, or a number of neighboring glands, may be affected, and in some cases *groups* in separate locations are involved simultaneously. The tumor may reach the size of an egg. It remains hard and regular, unless it suppurates, when soft spots may be felt on its surface.

In cases complicating infectious fevers the local swelling and tenderness are all that can be recognized during the symptoms of the original disease. In cases in which there is suppuration softening usually occurs by the second week, followed by bursting externally. After evacuation, natural or artificial, healing is usually rapid and complete. In non-suppurative cases the acute stage passes away, and the swelling gradually is absorbed, requiring a month or two.

Diagnosis: Knowledge of the location of the superficial lymphatic glands, and the fact that the swelling is only of short duration, cover every point in diagnosis.

Prognosis: This is good except in seriously debilitated children. Recovery is usually rapid. Some few become chronic and are later infected by the tubercle bacillus

Acute adenitis—treatment: In all infectious diseases careful and frequent cleansing of the mouth, and nose, and naso-pharynx will prevent infection of the glands.

If adenitis has begun, search the drained area, and treat any inflammation or abrasion there. Wash the mouth, nose, and naso-pharynx with some alkaline solution; syringe the ear, care for skin-diseases, and so on. Apply cold to the inflamed gland to retard the inflammatory process, or heat if suppuration seems inevitable. As soon as softening occurs lance the abscess, as thus a smaller scar is left than when it is allowed to burst itself. In cases going on to resolution daily applications of a 10 to 20 per cent. ichthyol ointment seem to assist the process.

CHRONIC ADENITIS.

Definition: In this condition it is understood that the glands are in a state of chronic inflammation of a *simple* character, that is, neither tubercular nor syphilitic.

Etiology: This is also fairly common in children. It often remains after an attack of acute adenitis; but usually is the result of chronic inflammations of the skin or mucous membranes in the region drained. Some children seem especially prone to such conditions.

Pathology: The glands undergo a true hyperplasia, with an increase of both their cellular and connective-tissue elements. The latter, however, are usually in excess.

Chronic adenitis—symptoms: The only symptom is the enlargement of the glands. This is usually moderate in amount, and tenderness and other signs of acute inflammation are absent. There is very little tendency for the glands to increase in size, and softening and suppuration are almost unknown. More than one group is usually involved, and hypertrophied tonsils and adenoids are apt to be present in the same case.

Diagnosis: The important point is to be able to exclude tubercular and syphilitic enlargements of the glands. The finding of a local cause of irritation is of main value. Simple enlargement occurs in younger children, and shows no tendency to involve contiguous tissues nor to suppurate. A gland enlarged from a simple inflammation may later become tubercular. The diagnosis in many cases is very difficult.

Prognosis: This is very good, as the curing of the cause usually hastens absorption of the inflammation.

Chronic adenitis—treatment: Hunt carefully over the drained area for the cause of the inflammation, and treat it. If diseases of the scalp, or discharges from the ear, or adenoids, or naso-pharyngitis are present, treat them as usual. Improve the child's health in every way by iron, cod-liver oil, nourishing diet, and fresh air. Locally, applications of iodine or of ichthyol may hasten absorption, and can do no harm.

TUBERCULAR ADENITIS.

This common condition, before the knowledge of the tubercle bacillus, was called *scrofula*.

Etiology: The lymphatic system seems particularly prone to tuberculosis, and this condition is seen oftenest in children

over three years old. Those with a tubercular family history are specially subject to it. At the same time, with this predisposition there is usually present some local irritant, as chronic inflammation of the skin or mucous membranes in the drained area. Other cases follow attacks of one or other of the infectious diseases. In addition to these causes there is always present infection by the tubercle bacillus. The pharynx seems the usual place of infection, as the cervical glands are those oftenest involved.

Pathology: The glands are swollen and the seat of a simple and tubercular inflammation at the same time. The cellular and connective-tissue elements are both increased, and the gland studded more or less thickly with miliary tubercles and tubercular masses. In the older cases these tubercular masses undergo a caseous degeneration, and break down into tubercular abscesses. The surrounding tissues first become adherent to the inflamed glands, and later are involved in the same inflammatory process. Neighboring glands become likewise fused together into one mass. Tubercular lesions may or may not be found in other organs of the body. Tubercle bacilli are present in the glands in moderate numbers.

Tubercular adenitis—symptoms: There is a slowly growing enlargement of one or more groups of glands. Those of the neck are most often affected; those in the axilla or groin less often. Groups in two or more places may be involved coincidently. The increase in size continues, and fusing of the separate glands occurs until a large mass is present in which the original outlines of the glands cannot be made out. Then the surrounding tissues become adherent to the mass and likewise involved. By this time areas of softening occur which approach the surface, the skin becomes discolored, and an abscess bursts externally, discharging thick, curdy pus with bacilli. Throughout there are very seldom any pain or tenderness of the inflamed glands, or any signs other than those of their presence.

The process usually extends over many months or years from the first appearance of enlargement to the time of suppuration. The general health may be somewhat depreciated,

the child becoming anæmic and poorly nourished. After bursting, the discharge may continue for an indefinite length of time with a permanent sinus, or may stop and healing occur. Under any circumstance, irregular, large disfiguring scars are left.

Diagnosis: The diagnostic points are the tendency to conglomeration of the glands, their caseation, and the character of the discharged pus in which tubercle bacilli may be found. The child's family history is also an aid. The enlarged glands of simple chronic adenitis, or of Hodgkin's disease, do not suppurate. Syphilitic enlargement usually has the history to help us. The difficulty of diagnosis is great in the early stages.

Prognosis: Most of these cases get well; some after many years of discharging sinuses. A very few develop tuberculosis elsewhere and die of this condition.

Tubercular adenitis—treatment: The main points in treatment are the cure of local conditions in the skin, nose, throat, or elsewhere that originally caused the glands to enlarge. After this everything should be directed to the general health of the child—fresh air, exercise, nourishing diet, and change of climate if possible. Internally, iron, arsenic, and cod-liver oil are very helpful. The former is best given as the syrup of the iodide.

If, after some months of this treatment, no improvement is seen, and particularly if suppuration threatens, the case should be put under surgical care. Operation, if undertaken, should be thorough, and the scars left by this are far less disfiguring than those after spontaneous evacuation.

CHAPTER XIII.

DISEASES OF THE SKIN.

LENTIGO.

THE common name for this affection is **freckles.** It consists of small pigmented spots, occurring in groups on surfaces that are ordinarily left uncovered.

Etiology: It occurs oftenest in children over five years old, and in blondes with a tendency to red hair. There seems some connection between it and exposure to sunlight.

Lentigo—treatment: Apply a 1 per cent. solution of corrosive sublimate on a piece of lint to the affected region for three or four hours. This raises a blister, which should be pricked and dressed with dry powder. When the raised skin desquamates the new epidermis is free from pigment. The treatment is somewhat painful.

ICHTHYOSIS.

This is a congenital **deformity of the skin** characterized by the formation all over the surface of *dry scales*, and with a lack of the normal cutaneous secretions.

Etiology: The only point known is that the disease occurs in families.

Pathology: It consists in an excessive growth of the epidermal cells.

Ichthyosis—symptoms: The dry, scaly condition of the skin is the only symptom. These scales desquamate freely, and long cracks form through the skin at flexures, which may be painful. Perspiration is absent. The sense of touch is much interfered with. Except in the very worst cases the disease is not dangerous. These children are frequently exhibited as freaks, being called fish- or alligator-boys.

Prognosis: The condition is incurable, but is ordinarily compatible with long life.

Ichthyosis—treatment: Internally, the use of the fatty foods is to be recommended.

Locally, daily warm baths should be taken, with plenty of soap to remove the loose scales, and then the whole skin should be thoroughly rubbed with lanoline or vaseline, to keep it as soft and pliable as possible.

SEBORRHŒA.

This condition of **thick, dry crust-formation**, is very common on the heads of infants.

Etiology: It is caused by an excessive production of the secretions of the sebaceous glands, which are allowed to dry on the scalp and become mixed with dirt.

Seborrhœa—symptoms: The vertex of the head presents a large patch of dirty, yellowish, greasy secretion. On examination it is found to consist of epithelial cells, granular matter, fat, and dirt. The skin under it is ordinarily normal and shows no signs of inflammation.

Seborrhœa—treatment: Keep a vaseline poultice on the scalp over night, held in place by a nightcap or a bandage. Next morning the free use of warm water and soap will remove most of the crusts easily. Repeat this successive nights until the condition is gone. Afterward apply daily an ointment containing 10 grains of resorcin to the ounce of vaseline.

MILIARIA.

This is commonly called **prickly heat**, and is a very common condition in childhood.

Etiology: It is regularly caused by intense heat, with excessive production of irritating perspiration.

Pathology: This is an acute inflammation of the sweat-glands, resulting in damming up of the minute ducts and the formation of small papules with minute vesicles on their summits.

When the inflammation is very slight it is called sudamina, and consists of the minute vesicles only.

Miliaria—symptoms: The symptoms are this widely spread red rash, consisting of the minute vesiculated papules, which may become infected and turn into pustules, associated with intense smarting and burning. It is usually most marked on the trunk and head, but may be universal.

Diagnosis: This is easy. A careful inspection of the rash, the history of the case, and the state of the atmospheric temperature are the points for consideration.

Miliaria—treatment: During hot weather the child should be lightly dressed, with cotton next the skin, should be frequently bathed in cold water, and the skin kept dusted with some dry non-irritating powder, such as the stearate of zinc, which acts very nicely.

If the eruption is present, these measures should be continued; and, in addition, the bowels should be opened, the diet should be light, and internally some mild diuretic, such as sweet spirits of nitre, given. The zinc and calamine lotion—zinc oxide, ℥ss; pulv. calamine, ʒij; glycerin, ℥j; liquor calcis, *ad* ℥viij—should be applied freely. One per cent. of carbolic acid may be added, if there is any tendency to infection. Under this treatment the case should recover in a few days.

FURUNCULOSIS.

A **boil** is an intense localized inflammation occurring about a hair-follicle or a gland of the skin.

Etiology: Boils are probably due to a direct infection of the follicle by micro-organisms. Pus from one boil will infect another follicle, and in this way successive crops are formed. Local injury of a slight nature often opens the way for infection.

Boils are fairly common in children, but never seem so deep-seated, indurated, and painful as they become in adults.

Furunculosis—symptoms: A boil begins as a small red papule, but the surrounding skin rapidly becomes indurated and tender. A small whitish top soon forms over it, but on removing this no pus escapes. The pain is severe and throbbing, and the neighboring lymphatics may become inflamed

and tender. There may be some constitutional symptoms in the worst cases.

After a week or ten days pus collects in considerable quantity in the boil, and on evacuating it the centre of the boil shows the presence of a large white necrotic mass—the core. This core is the remnant of the dead follicle or gland. After its removal a little cavity is left to fill by granulation, and with this the parts soften and the pain and tenderness disappear.

Diagnosis: There is no reason for confusing this condition with any disease.

Furunculosis—treatment: Improve the general health by the use of arsenic and the hypophosphites. When a boil is beginning put over it a wet dressing of carbolic acid in 2 per cent. solution. As soon as it begins to point incise freely and continue this wet carbolic dressing. By this means we rapidly heal up any boil present and prevent the formation of others. Very careful cleaning of the skin, and subsequent applications of 1 : 1000 bichloride of mercury, or of 1 : 40 carbolic acid, will prevent the formation of additional boils.

IMPETIGO CONTAGIOSA.

This is an infectious disease of the skin characterized by the **formation of vesicles and pustules.**

Etiology: The disease occurs almost always in children, is contagious, and hence is seen oftenest in a number of children in the same family, or among the poor, living in the same tenements. Probably some as yet unknown form of bacterial infection is the actual cause.

Impetigo contagiosa—symptoms: There are often a little fever and malaise with the outbreak of the eruption. This begins as isolated vesicles, the contents of which soon become pustular, and then a dry, yellowish scab forms on the surface. After this crust drops the surface is red, but no depression is left. The eruption is usually confined to the hands and face. The lesions may remain discrete or may coalesce, and usually heal in a couple of weeks. Fresh crops from auto-inoculation may prolong the attack indefinitely.

Diagnosis: Pemphigus and chickenpox are most apt to be confused. The points are the distribution of the lesions, their contagious character, and the isolated vesico-pustules.

Prognosis is very good.

Treatment: Remove the crusts, wash the parts carefully, and apply an antiseptic ointment, as unguentum hydrargyri ammoniati.

ECZEMA.

Definition: This very common skin disease of infancy and childhood is a dermatitis, characterized by itching, redness, infiltration, moisture, and crusting. It may be acute or chronic, and presents a boundless variety of lesions.

Etiology: While the disease is especially frequent in childhood, it presents no essential differences from the eczema of adult life. The skin in most children is very susceptible to irritation both from outside and inside, but in certain ones there seems to exist an especial predisposition to inflammatory action. This tendency is at times inherited. The parents are often subjects of a gouty or rheumatic diathesis. Children brought up amid unhygienic surroundings, being improperly nourished and unaccustomed to good air, are often the subjects of eczema. On the other hand, very frequent examples of the disease are seen in children seemingly in the best of health, with their nutrition above the normal, and their skin pink and healthy. These latter seem especially prone to facial eczema.

The *exciting causes* acting from within the organism are oftenest connected with *digestion* and *elimination*. It is seen in both breast- and bottle-fed babies, but more often in the latter because they are oftenest the subjects of digestive derangements. Indigestion from over-feeding, from excessively high percentages of fat, or proteids, or sugar in the milk, or from too early use of starch, is a frequent cause. On the other hand, mother's milk or artificial food in which the nutritive value is far below normal may be responsible. Improper articles of diet for the age of the child are often causative; as are also constipation and deficient action of the liver or kidneys.

Reflex irritation, acting through the nervous system, undoubtedly may be a partial cause at least of certain cases, as in dentition.

External irritants which may cause an outbreak are cold, heat, irritating soap, powders, excessive bathing, and clothing of a rough texture. Discharges from the nose, ear, eyes, genitals, or rectum, and wet diapers will frequently start the disease. Parasites, as pediculi, acari, and trichophyta, will produce it. In most cases there is more than one cause acting. The disease is not contagious.

Eczema—symptoms: In infants eczema is more often seen about the head and face than elsewhere. Any of the various lesions—erythema, papules, vesicles, or pustules—may be present singly or combined in the individual case. There is always intense itching, and the child's scratching adds mechanical lesions to those of the original inflammation. The inflamed surface discharges a serous secretion which dries on the skin and forms yellow crusts. Scratching these causes bleeding, so that many of them become dark brown. Many of the lesions become infected, and the eczema then takes on the pustular form. This pustular variety is most frequent in the scalp. Each variety has its own particular name, but there is no special advantage in these subdivisions.

The lesions spread usually from one neighborhood to another, until finally a large portion of the skin becomes involved. It is quite common for the lymphatic glands to be inflamed, and to swell, and even to form abscesses.

In the groin, and other regions where two surfaces of skin come into close contact, intertrigo or erythematous eczema develops quite regularly.

All varieties of eczema are subject to frequent relapse, and all require great care and perseverance in treatment.

The reader is referred to special books on the subject for more detailed description as to *lesions*.

Diagnosis: *Scabies* and *syphilis* cause lesions most likely to be confused with eczema. Remember the characteristics of eczema: multiform lesions, serous discharge or "weeping," crusting, and itching. Scabies itches badly, but is usually found in other members of the family, and on special locali-

ties, as the webs of the fingers, the flexures of the wrists and elbows, and around the genitals. With care the *burrows* of the acarus may usually be found. Syphilis gives multiform lesions, but does not itch. The eruption is dark colored and the child usually shows general cachexia.

Prognosis: It is a slow, tedious disease. With proper care most cases can be cured; but relapses are common, and the patience of the physician and of the family will be tried to the utmost in the meantime.

Eczema—treatment: Search into every possible external, internal, and reflex cause of the condition; if anything is found wrong, correct it, and regulate the child's life according to the most approved hygienic rules. This means special attention to the condition of the stomach, intestines, liver, and kidneys. The most painstaking care should be taken in these lines, and no little thing must be neglected. Intelligent co-operation on the part of the mother and nurse is very important. All local irritants must be removed, and irritating discharges cured. Water should not be applied to eczematous surfaces. It has a specially irritating effect on them. A bland oil may be used for purposes of cleanliness, and the parts mopped dry afterward. Prevent scratching, if necessary, by confining the child's hands.

During the *acute stage* some soothing ointment should be applied and kept in place by a bandage or mask. The plain oxide of zinc ointment, or this with 1 per cent. of carbolic acid added to allay the itching, answers the purpose well. In the more subacute cases a stimulating ointment, as one containing tar in some form, is of most value. Another useful formula is Lassar's paste, with ten grains of salicylic acid to the ounce of ointment.

In the various text-books on dermatology will be found innumerable formulæ for the different stages. Here it is necessary to emphasize principles only: remove causes, prevent washing and scratching, apply soothing remedies to acute cases, and stimulating ones to chronic cases. All applications should be kept persistently in contact with the inflamed skin.

URTICARIA.

This is also called **hives** and **nettle-rash**. It consists of suddenly appearing, elevated, irregularly shaped blotches in the skin, called *wheals*. They are usually paler than the surrounding skin, but may be pink. They disappear equally quickly, and leave no trace behind.

Etiology: Local irritants, such as coarse underclothing, bites of insects, and some vegetable poisons, will produce the wheals. A large number are due to gastro-intestinal irritation, as indigestion from any source; but certain special articles of diet always produce attacks in certain individuals. Strawberries, fish-food, and oatmeal are among such articles. Certain drugs, as quinine, will sometimes cause it.

Urticaria—symptoms: The wheals are quite characteristic in their onset, appearance, and manner of disappearing. They itch intensely, and scratching will often develop new crops. As the attack begins some fever may accompany it, with general malaise. No other skin disease has any special resemblance to it.

Prognosis: This is perfectly good, but individuals who once have the disease are very apt to have subsequent attacks.

Urticaria—treatment: Find the cause, if possible, and remove it. All local irritants should be taken away. As the digestive system is so frequently at fault, it is always well to clean it out thoroughly at once. This is best done by a dose of a saline cathartic. After this regulate the diet.

Locally, an alkaline bath is always good to allay the itching, or a solution of menthol, gr. x to the ounce. Nothing further is necessary except so to regulate the food and clothing as to prevent subsequent attacks.

SCABIES.

This disease is called commonly the **itch**. It is not very frequent in this country.

Etiology: It is due to the boring under the skin of the female *acarus scabiei* for the purpose of laying her eggs. The male parasite remains on the surface. The little furrow

made by the acarus is a pigmented, irregular line, which is the characteristic diagnostic point of the disease. At the extremity of the burrow the minute insect will usually be found.

Pathology: Around the itch-mite in the burrow will be a zone of inflammation, which is either a papule, a vesicle, or a pustule. In fact, the lesions of scabies are quite multiform.

Scabies—symptoms: The acarus chooses places to burrow where the skin is thin, and hence the lesions are found most frequently in the webs of the fingers, on the folds of the wrists, about the breasts and genitals. The burrows, together with the multiform lesions and the intense itching, are the main symptoms. To relieve the itching the scratching is incessant, and the mechanical lesions of this are added. This probably explains the frequent presence of lesions about the genitals. In infants the face may be involved from infection from its mother's breast.

Diagnosis: From simple eczema the finding of the burrow and the acarus decides the diagnosis.

Prognosis: This is good. Cure is comparatively easy.

Scabies—treatment: The child should first be given a soap and hot-water bath, the soap being very thoroughly applied in regions where lesions are found. After this a sulphur ointment should be well rubbed in over all the affected parts. This should be repeated two or three times, and until the acari are killed. All clothes and night-dresses should be sterilized by boiling.

TINEA TRICHOPHYTINA.

This is commonly called **ring-worm**, and is frequently seen in children on the skin, general surface, or scalp.

Etiology: This disease is due to the growth in the skin of the *trichophyton fungus*. It is a vegetable parasite consisting of mycelial threads and spores.

Tinea trichophytina—symptoms: The disease begins as a slightly red scaling spot. If in the scalp, the hairs in this spot become broken off and point in all directions. The

infection spreads from this point in a circle, the centre begins to heal, and eventually by this process of peripheral spreading and central healing, a *ring* is produced, which gives the name to the disease. Two or more points may be infected at the same time, and the rings formed from each may cut into one another, forming irregular figures.

In the scalp, a common location in children, the hairs become brittle, lose their lustre, and give quite a "moth-eaten" appearance to the affected area. Often ring-worm in the scalp gets infected, and pustules form in the hair-follicles. If such a case becomes chronic, we get the condition known as kerion, where the whole area becomes boggy from the infection. The disease does not produce much itching nor any other subjective symptoms.

Diagnosis: Syphilis, eczema, and psoriasis all may form circinate figures, but by taking the other lesions into consideration usually no error need be made. By microscopical examination of scrapings taken from the lesions the fungus may be easily found.

Prognosis: In the skin it is easy to cure. In hairy areas it is more difficult, but perseverance brings it to an end here also.

Tinea trichophytina—treatment: On the skin daily applications of tincture of iodine will cure the case rapidly.

In the scalp the hair should be clipped close, and the lesions thoroughly cleaned with soap and hot water. After this an ointment of white precipitate should be rubbed thoroughly into the affected regions. This treatment, cleansing and anointing daily, should be continued till all signs of the disease are gone. There are many valuable ointments, for which reference should be made to the special text-books. In the very persistent chronic cases *epilation* may be necessary.

CHAPTER XIV.

DISEASES OF THE EAR.

ACUTE OTITIS.

Inflammations of the middle ear are excessively common in infancy and childhood.

Etiology: It is usually secondary to inflammation in the naso-pharynx, such as cold in the head, adenoids, enlarged tonsils, and pharyngitis complicating the infectious diseases. The commonest of these are scarlet fever and influenza. It may occur after measles, diphtheria, or typhoid. Picking at the ears, foreign bodies in the ears, and boxing the ears are also causes.

Pathology: The inflammation in the pharynx spreads through the Eustachian tube, swelling the mucous membrane and causing an increased secretion of mucus, which may become purulent. This inflammation may be confined to the mucous membrane of the Eustachian tube, or may spread to that lining the middle ear. In the middle ear the inflammation may be the milder *catarrhal* variety, or the more severe *purulent* form with the presence of the germs of suppuration.

Acute otitis—symptoms: Following the symptoms of the primary disease, the child has the two symptoms of otitis: fever and pain. The fever may be only 100° F.; or may be much higher, reaching often 104° F. The pain, or earache, is quite a prominent sign, and the one that leads us to suspect the ear as the cause of the fever. The pain is acute and severe, and in young infants who cannot talk is evidenced by restlessness, crying, and tenderness on pressure about the ear. The crying from earache is apt to be incessant and continuous. In infants cases do occur, however, in which the pain and tenderness seem very slight. With the fever there

are general malaise, anorexia, headache, and constipation. The symptoms last a day or two to a week, and gradually cease with the subsidence of the inflammation; or more quickly in case the otitis leads to rupture of the drumhead, with free exit to the inflammatory exudate. In the latter case the discharge from the ear is rather profuse at first, but gradually becomes less in quantity, and finally ceases with healing of the rent in the membrane.

In the *purulent variety* mastoid abscess, thrombosis of the lateral sinus, meningitis, or cerebral abscess may develop from extension of the inflammation to these parts.

Diagnosis: Fever of unknown origin, combined with pain or evidences of tenderness about the ear, is usually sufficient for diagnosis. Infants who have long crying attacks are very apt to be suffering from earache. If possible, an examination of the ear should be made through a speculum. The membrane will be found red and congested, and bulging if secretions are behind it. A discharge and a perforation may be found.

Prognosis: In the catarrhal form the prognosis for recovery without impairment of hearing is good. In the purulent form, after recovery there is apt to be either partial or complete deafness in that ear, while there are many cases of serious disease, and even of death, following the complications.

Acute otitis—treatment: In the early stages the local application of heat by a hot-water bag, a Japanese stove, or by prolonged douching with hot water by a fountain-syringe, will relieve the pain and hurry the inflammation through. At the same time touching the naso-pharynx with some astringent, as silver nitrate, is good. The bowels should be opened by broken doses of calomel, and the child kept in a warm room with an equable temperature. If the pain continues, it is justifiable to put a few drops of a solution of atropine, 1 per cent., and cocaine, 4 per cent., in the external meatus. If the membrane is found to be bulging and tense, it is better to perform paracentesis than to wait for the membrane to rupture spontaneously. After spontaneous or artificial perforation of the drum-membrane, the ear must be kept scrupulously clean and dry.

CHRONIC OTITIS.

This means really a **chronic otorrhœa.**

Etiology: It usually follows repeated attacks of the acute disease in delicate, poorly nourished children. It often is due to neglect of proper treatment.

Pathology: There is a chronic purulent inflammation in the middle ear, with rupture or destruction of the drum-membrane, and a constant purulent discharge from the external meatus.

Chronic otitis—symptoms: The main symptom is the discharge, which is often quite free and creamy, and is apt to have a very characteristic, disagreeable odor. This discharge may cause eczematous eruption of the external ear, and with it may be associated a cervical adenitis.

Diagnosis: This is made by the presence of the chronic discharge, and the examination of the drum-membrane, if it can be performed.

Prognosis: While these cases are often obstinate, they can usually be cured by perseverance.

Chronic otitis—treatment: Excessive cleanliness of the meatus, syringing twice a day with a boracic acid solution, and then drying the parts carefully, will usually heal them. Peroxide of hydrogen has a useful field here. At the same time any inflammatory condition in the naso-pharynx should be attended to, and the child's general health brought as near normal as possible.

CHAPTER XV.

DISEASES OF THE BONES.

ACUTE ARTHRITIS.

Definition: This is a suppurative disease of the extremities of the bones, occurring with some frequency in infancy. It is also called **acute epiphysitis.**

Etiology: It is an infection of the extremity of the bone by pyogenic organisms, which may gain entrance through almost any lesion of the skin or mucous membrane. Traumatism of the joint is probably a partial cause.

Pathology: The inflammation usually begins at the epiphyseal junction, and spreads into the joint and to the shaft. It is really an osteomyelitis, modified by the anatomical condition of the epiphysis in infancy. It is very apt to go on to the production of an abscess either subperiosteally or in the joint. Secondary abscesses elsewhere are common, when the true pyæmic nature of the disease becomes apparent.

Acute arthritis—symptoms: The disease begins suddenly with a chill, and fever of a remittent type. General prostration and other symptoms of fever accompany this. The affected joint becomes swollen, red, painful, and very tender, so much so as to produce a voluntary paresis. If pus forms, signs of deep fluctuation are present, and burrowing may be extensive or rupture externally may take place. Death may occur from the intensity of the infection in a few days; or resolution may take place with a gradual disappearance of the symptoms; or, after rupture of the abscess, recovery with a damaged joint may occur.

Diagnosis: The disease is most likely to be confused with *acute rheumatism*, but in infancy rheumatism seldom assumes such marked local symptoms.

Prognosis: Death takes place in about 10 per cent. of the cases. Very few recover without some disability of the joint.

Acute arthritis—treatment: The child's nutrition should be well kept up, and alcoholic stimulants will usually be necessary. Pain should be relieved by drugs and by local applications. As soon as signs of suppuration occur, or even earlier, free incision with drainage should be used and the case treated on surgical principles.

POTT'S DISEASE.

Definition: This is a tubercular inflammation of the bodies of two or more of the vertebræ.

Etiology: The *tubercle bacillus* seems particularly prone to invade the bones in children during the period after they learn to walk. Previously healthy children, with no tubercular family history, may be attacked; but it is much more common in delicate children with a bad heredity. It may follow attacks of one of the infectious diseases, but is usually the primary tubercular focus. Traumatism is often ascribed as a cause, but simply offers a *locus minoris resistentiæ* for attack by the bacillus.

Pathology: The bodies of the vertebræ are gradually invaded by the tuberculous tissue, which, having poor vitality, breaks down, producing caries of the bone. With this comes formation of tubercular abscesses, which may remain localized at the spot, or may burrow and appear at the surface at considerable distances. The intervertebral disks are destroyed by the same process. The weight of the body above the disease causes the carious vertebræ to yield, with the production of the characteristic deformities of the disease. If only one or two vertebræ are diseased, the bend is quite an acute angle; if more, the curve is more general.

The meninges, the roots of the spinal nerves, and even the cord itself, may be involved by the inflammatory process.

Pott's disease—symptoms: The disease begins very gradually. The child seems out-of-sorts, tires easily, is stiff in his movements, and has restless nights. The first definite symptom is pain, which is more often referred to the abdomen

than to the back. This is probably due to irritation of the nerve-roots, and the case is often treated for indigestion. Later, there are noticed a rigidity of the spine and the voluntary assumption of certain postures which relieve the pain. About this time a beginning deformity of the spine at the seat of the lesion may be noted, or in others paralysis from pressure on the cord. Later yet, abscesses form and remain localized at the seat of trouble, or burrow and burst at remote points.

The general health may remain fairly good ; but usually the child becomes anæmic, emaciated, and feeble.

The disease may attack the cervical, dorsal, or lumbar spine, giving localized symptoms according to the region involved. The dorsal vertebræ, however, are attacked much oftener than the others. The deformity produced is an antero-posterior bending, or *kyphosis*. Lateral curves are rare. Abscesses in the cervical region point in the pharynx ; those in the lower dorsal and lumbar travel down the sheath of the psoas muscle and point in the groin.

The *course* of the disease is essentially chronic, lasting over a number of years, and death may take place from exhaustion, or from the development of tuberculosis elsewhere. In the cases going on to recovery the inflammatory process ceases, new bone is deposited in the carious vertebræ, and ankylosis with permanent deformity takes place.

Diagnosis : This is made by rigidity of the spine, which is shown by having the child go through various movements in which the spine is necessarily bent ; by pain and tenderness in the diseased area ; by the attitude assumed, which is often characteristic ; by the presence of the typical deformity of the spine ; by paraplegia, and by abscesses. Rachitic deformities are more gradual in their curve, and, instead of mere rigidity of the spine, there may be greater mobility.

Prognosis : In the lumbar and cervical regions the prognosis is better than in the dorsal. About one-fourth of the cases die from the disease sooner or later. Death occurs most often from one of the many complications that are likely to arise during the slow course of the disease. In the cases which recover some deformity is almost certain ; but its

extent depends mainly on the kind of treatment and the stage at which it was begun.

Pott's disease—treatment: The general treatment of the patient is of great importance, as in all varieties of tuberculosis. The nutrition should be carefully attended to, fresh air, good food, cod-liver oil, and iron being the main points of value.

Locally, rest to the diseased parts, with proper position, is the keynote of treatment. This may be gotten by long confinement to bed, or by the use of proper mechanical supports which allow the child to be up and about. For details of mechanical treatment special works on orthopædics are to be consulted.

Recently these cases are being treated by *forcible reduction* of the deformity, and then the application of a proper brace to maintain the parts in position. As yet the value of this heroic method is undecided.

The earlier any treatment is begun the better the results. Abscesses and sinuses are to be treated on general surgical principles.

HIP DISEASE.

Definition: This is a chronic tubercular inflammation of the structures of the hip-joint, and is very common in childhood.

Etiology: Infection by the tubercle bacillus is the actual cause of this disease. A slight injury to the joint usually is the exciting factor. Delicate children and those with a tubercular family history are most often the victims. Why the hip is more often involved in children than other joints is unknown. The greatest liability to the disease is after the third year of life.

Pathology: The disease may begin in the head of the femur, in the acetabulum, or in the synovial sac. In any case it rapidly spreads to the other structures of the joint. There is an invasion of the neighboring parts by tubercular tissue, which destroys the bones and cartilages, and then becomes carious and breaks down, as all tubercular tissue does, with the formation of cold abscesses.

The destruction of the joint in the old cases becomes very considerable, the head of the femur at times being completely destroyed and the acetabular cavity perforated.

Hip disease—symptoms: For descriptive purposes the symptoms are usually divided into three stages.

In the *first stage* there is a very gradual onset of the symptoms, and ordinarily a slight limp is the first thing noticed. This is due to tenderness in the joint and to muscular spasm. The lameness slowly increases in severity until it becomes very evident to even the most casual observer. Pain also develops, and is usually referred to the knee, and ordinarily is worse at night. On careful examination during this stage there will be found limitation of motion, due to muscular spasm, in almost every direction, as compared with the opposite joint. After the disease has lasted a short time there will be found a certain amount of deformity, the muscles holding the joint in such a position as will prevent pressure on the inflamed parts. The deformity present is usually flexion, abduction, and outward rotation, to be followed, later, by adduction and inward rotation. There is usually seen quite early some atrophy of the muscles on the affected side. This stage may last two or three years.

In the *second stage* all the symptoms of the first stage are aggravated. The deformity, atrophy, and limitation of motion, especially, are increased. Pain is more severe, and sudden crying out during the night is common. During this stage breaking down of tissue and formation of abscesses occur, and fever makes its appearance. The abscesses may appear in any location around the joint, but are most frequent in front. Some rupture into the pelvis. After rupture, sinuses discharging carious tissue and pus remain indefinitely. This stage lasts for some months to a year.

In the *third stage* the deformity is very marked. In addition to the flexion and adduction there is real shortening from destruction of the head of the femur. The atrophy becomes more marked, the suppuration continues, the sinuses persist, and the general health fails. Tuberculosis occurs elsewhere, or the viscera undergo amyloid degeneration. Even after this stage many cases recover with ankylosis of

the joint, but most of them die. This stage may be prolonged over months or even years.

Diagnosis: The diagnosis is easy, if, with lameness and pain, we find atrophy and limitation of motion. Rheumatism is the disease most apt to be confused.

Prognosis: While the tendency in this disease is to recovery, still death takes place in a considerable proportion of the cases from exhaustion, amyloid disease, or general tuberculosis. If recovery occurs in the first stage, there is scarcely any deformity left; but in the second or third stage recovery is accompanied by marked deformity, with lameness and atrophy of the limb.

Hip disease—treatment: Constitutional treatment, with highly nutritious foods, cod-liver oil, iron, and fresh air, is very important.

Locally, the hip is to be put at rest by immobilization of the joint by extension in bed, or by special forms of orthopædic apparatus. In the third stage the question of surgical treatment of the carious joint must be considered. Abscesses and sinuses are to be cared for on general surgical principles.

KNEE-JOINT DISEASE.

This is often called **white swelling**, and is a tuberculosis of the knee-joint.

Etiology: It is due to infection by the tubercle bacillus, often being combined with slight traumatism.

Pathology: It begins in the condyles of the femur or the head of the tibia, or the synovial membrane, and spreads to the joint-structures proper, the normal tissues being replaced by new tubercular tissue. This undergoes the usual necrotic process, with formation of abscesses.

Knee-joint disease—symptoms: Stiffness of the joint, pain and tenderness, and a limp are usually first noticed. Later, swelling of the ends of the bone and atrophy of the thigh- and leg-muscles follow. There is limitation of motion, with deformity, caused by muscular contraction. This is usually flexion, with later a partial backward dislocation of the tibia. As the case advances, motion may be gotten laterally, due to

destruction of the lateral ligaments; and abscesses form and break externally. The general health does not suffer as much as in hip disease. The course of the disease is very chronic.

Diagnosis: Rheumatism is most apt to cause confusion; but the chronic nature of the disease, and the bony enlargement, and, later, the spots of softening and formation of abscesses should decide the diagnosis.

Prognosis: It is a less fatal disease than tuberculosis of the spine or hip; but death may occur from tuberculosis elsewhere, from amyloid degeneration, or from exhaustion. As regards the joint, recovery takes place usually only with very marked deformity, unless under proper treatment begun early.

Knee-joint disease—treatment: The same constitutional treatment is necessary as in hip and spine disease. *Locally*, the joint must be immobilized in good position, and by some apparatus that allows the child to be up and about. In late cases surgical measures may be required.

TUBERCULAR DACTYLITIS.

Definition: This is a chronic tubercular osteomyelitis of the phalanges of the fingers. It is seen most often during the second and third years of life, and usually in children with a tubercular heredity.

Symptoms: It is a chronic condition seemingly without much pain or tenderness. The phalanx swells and the whole finger gradually enlarges until it takes on a *fusiform appearance*. Later the soft parts are involved, including the skin, and abscesses form and burst, discharging curdy pus and bony detritus. Inside the bone shell will usually be found a sequestrum. The disease lasts for years until the dead bone is all discharged, when recovery occurs with marked deformity and shortening, and a useless finger.

Diagnosis: The only difficulty comes from separating this condition from syphilitic dactylitis, which produces almost the same lesions. The history, other signs elsewhere, and the fact that syphilitic disease is rare, are the points for guidance.

Prognosis: This is good for life, unless tuberculosis should

be present elsewhere, but a badly deformed finger is usually left.

Tubercular dactylitis—treatment: Constitutional treatment to build up the general health is of most importance. Put the finger on a splint which will keep it at rest. Open abscesses, scrape out sinuses, and treat the part on general surgical principles.

CHAPTER XVI.

THE INFECTIOUS DISEASES.

VACCINATION.

Definition: This consists in inoculating a child with the dried serum from a vesicle of a calf which has *vaccinia* or *cow-pox*. Vaccinia in a calf is undoubtedly variola modified by its passage through a number of generations in the bovine species. When a child has been successfully inoculated with vaccine it is protected almost absolutely from infection by smallpox; but in the few cases where the immunity is not perfect the child undergoes a mild attack of modified smallpox, called *varioloid*.

Vaccination—time for performance: All babies had best be vaccinated before they begin cutting teeth. Revaccination should be performed about the tenth year, and again at about the twentieth. During local epidemics of smallpox every one who has not been vaccinated successfully within five years should submit to the operation.

Vaccination—method of performance: Fresh, dried virus from the calf on an ivory point should be used. The left arm, near the deltoid insertion, preferably; or, in girls, the left leg, on the outer side of the calf, should be washed with a little alcohol. The operator's hands being scrupulously clean, with a new fine cambric needle a small square should be scratched crosswise until the blood just runs. The vaccine point is moistened in sterile water, and the lymph then rubbed thoroughly into the wound. The blood and vaccine are now allowed to dry completely and to form a crust over the wound. No dressing is necessary afterward, but the part should not be washed nor rubbed. After a few days, if there is a tendency on the part of the child to disturb the crust, a light sterile dressing may be bandaged on the arm for pro-

tection. On no account, from beginning to end, must the crust be removed, as it forms the best protection to the wound. The only objection to using the leg for vaccination is the greater difficulty of preventing infection of the wound.

Vaccination—symptoms: Four or five days after vaccination a red areola is seen around the wound, and the formation of a vesicle around the circumference begins. From the seventh to the tenth day this vesicle becomes fully developed, showing a depressed centre. The serum in the vesicle by this time begins to change into pus, and the areola around the umbilicated pustule becomes redder and more marked. The neighboring lymphatic glands become swollen and tender, and for a time the region around the vaccination may take on a very angry look. The pustule very gradually dries up with the formation of a thick crust, the inflammatory action decreases, and after two or three weeks the crust drops off, leaving a depressed, pitted scar, which remains permanently.

Constitutional symptoms, more or less marked, are apt to accompany most cases. There is slight or rather high fever, with its accompanying symptoms—headache, anorexia, and malaise. These begin usually with the formation of the vesicle, and last for a few days, when they gradually disappear.

Vaccination—abnormalities: Revaccinations seldom follow so typical a course as the first vaccination.

The pustule may ulcerate deeply into the tissues and a large slough form, leaving a deep pit.

Secondary vesicles may form around the original one, or even inoculation on to distant parts of the body, through the mucous membranes or abrasions of the skin, may take place.

Vaccination—complications: Various urticarial and erythematous eruptions are fairly frequent after vaccination. Erysipelas or cellulitis may develop from the wound, either from infection at the time of vaccination or subsequently. Boils and small abscesses follow in some cases.

Inoculation of syphilis or tuberculosis is much dreaded and talked about, but is extremely rare.

Vaccination—treatment: Prevent the wound from infection, subsequent to the vaccination, by protecting it with a sterile dressing after the formation of the vesicle. A dry powder,

such as aristol, may be dusted on if the suppuration is excessive. A wet dressing of lead and opium is useful in the later stages if the inflammation is severe.

If fever needs controlling, small doses of phenacetin may be used. Complications are to be treated as usual.

VARICELLA.

Chickenpox is an acute contagious disease, consisting of a vesicular eruption with very few constitutional symptoms.

Etiology: It is very much commoner in children than in adults. The poison is given off from the surface of the body, and is probably contained in the vesicles. The disease is markedly contagious. No specific micro-organism has as yet been isolated. One attack regularly produces a permanent immunity.

Incubation-period: This is regularly from thirteen to seventeen days.

Varicella—symptoms: Ordinarily the eruption is the first symptom, although there may be some fever and malaise during the preceding day.

The *eruption* comes in crops beginning as papules, which soon turn into vesicles, and a few of these may become pustules. The eruption is found on the scalp regularly in the beginning, but spreads and may involve any part of the surface of the body. It is also found on the mucous membrane of the mouth and pharynx. The eruption runs its course in about five days, and after recovery the skin is left unchanged, except where a pustule was present, when a pit will be left behind.

Varicella—complications: Infections of the vesicles are the only complications to be dreaded. Erysipelas, sloughing of the skin, and acute adenitis may be present.

Diagnosis: The presence of vesicles in the scalp and throat, and the absence of any marked general symptoms are the points for diagnosis.

Prognosis: This is good, as only complications in *debilitated* children seem dangerous.

Varicella—treatment: The child should be kept in the

house, and away from school and other children. The constitutional symptoms are usually so slight as to need no treatment. If the fever is high and the child uncomfortable from it, a little phenacetin may be given.

Locally, carbolized vaseline may be used to allay the itching and prevent the child from scratching.

MEASLES.

Measles is more technically called **rubeola**, and also **morbilli**. It is an acute contagious disease, characterized by fever, catarrhal inflammation of the respiratory tract, and a diffuse maculo-papular eruption on the skin and mucous membranes.

Etiology: The disease is undoubtedly due to some microorganism as yet unknown. It is, next to smallpox, the most contagious of the exanthemata, and very few of the human family escape it. The contagium is given off from the mucous membranes and surface of the body, and floats in the atmosphere. One attack regularly protects from subsequent ones.

Incubation-period: This is from ten to fourteen days.

Measles—symptoms: The invasion is marked by chilly sensations, a rather rapid rise of temperature, headache, vomiting, and prostration. General convulsions frequently usher in the disease. With these symptoms catarrhal inflammations of the conjunctivæ are regularly seen. There may be some hoarseness and sore throat at the same time. The fever remains fairly high, and with it are headache, backache, anorexia, and restlessness or drowsiness. If the throat is inspected during this time, isolated red spots are often seen on the hard palate, and pearl-colored isolated spots on the inside of the cheeks.

After three to four days this period of invasion is succeeded by the period of eruption. The typical rash of measles consists of macules and papules, which appear first around the scalp and on the face, and spread downward until they usually cover the entire body. By the time the lower extremities are involved the rash has about faded from the head. The papules are isolated, but occur in patches, which

often become crescentic. They have very little elevation above the surface of the skin. The eruption lasts about four days, and during its outbreak all the constitutional and catarrhal symptoms are exaggerated; but as it fades these also gradually disappear. The throat is quite painful, and swallowing is difficult. The eyes are sensitive to light, and there is regularly a laryngeal or bronchial cough. After the fading of the eruption the skin begins to desquamate in fine bran-like scales. This continues for a week or ten days.

Measles—complications: Laryngitis, bronchitis, broncho-pneumonia, conjunctivitis, adenitis, otitis, and noma are the most frequent sequelæ of measles.

Abnormal cases: *Black*, or *malignant*, *measles* is a form with intense infection, a hemorrhagic eruption in the skin, and free bleeding from the mucous membranes.

Diagnosis: This is made from the presence of coryza and conjunctivitis, combined with the typical eruption on the skin and hard palate.

Prognosis: Ordinarily, in healthy children, this is good. In institutions, and among people where the disease prevails for the first time, the mortality is high. Complicating broncho-pneumonia is the usual cause of death. Most of the hemorrhagic cases die.

Measles—treatment: The child should be kept in bed in a room with a steady temperature of about 70° F., and which is kept darkened. The diet should be almost entirely liquid, and large quantities of water should be given. The bowels should be opened by fractional doses of calomel, and this should be followed by some mild febrifuge, such as a small dose of phenacetin every four hours, or of tincture of aconite every two hours. If the temperature is excessive, cool sponging is to be used. If the eruption seems delayed, a hot bath may be given. For the conjunctivitis boric acid in saturated solution should be dropped in the eyes, and the edges of the lids smeared daily with vaseline. During desquamation frequent warm baths and the application of very small quantities of vaseline to the skin are useful. Some cases with intense infection will require alcoholic stimulants.

Complications are to be treated as usual, and special pains

are to be taken to prevent the occurrence of bronchitis or broncho-pneumonia.

ROTHELN.

This is also called **rubella** or **German measles.** It is a contagious disease of mild type, characterized by a rash of rather atypical appearance, and by slight malaise.

Etiology: German measles is probably due to some unrecognized germ; is contagious, and is seen in epidemic form. Adults seem equally often attacked as children. One attack confers immunity, but never against measles or scarlet fever.

Incubation-period: This is from one to three weeks. It averages two weeks.

Rotheln—symptoms: There may be a very slight fever, 100° to 101° F., with malaise, a little headache, and sore throat. The rash appears very early, and may resemble, although it is not exactly like, that of measles on the one hand, or that of scarlet fever on the other. Most cases are accompanied by a moderate swelling of the glands along the posterior border of the sterno-mastoid muscles. The eruption lasts two or three days only, and then rapidly disappears. Some slight desquamation follows.

Diagnosis: This is the most important part of the disease, and except in epidemics is very difficult to make positively. The points of value are the almost *complete absence* of any *constitutional symptoms*, thus differentiating measles and scarlet fever; the *enlarged posterior cervical glands*, and the *anomalous rash.*

Prognosis seems to be always good.

Treatment: No real treatment for this disease is necessary. Any complications may be cared for as they arise.

SCARLET FEVER.

Definition: This disease, also called **scarlatina**, is an acute contagious fever characterized by sudden onset, the early symptoms being fever, sore throat, vomiting, or general convulsions; and later, a typical eruption on the skin and mucous membranes.

Etiology: It is undoubtedly due to some form of micro-organism, but so far no specific germ has been isolated. The disease is distinctly contagious during its whole course, the poison being given off from the skin and by the breath, and being carried by clothing, bedding, and other articles from one place to another. The vitality of the organism is very marked, even after years seeming to retain its contagiousness. Children are much more susceptible to scarlatina than are adults, but still a considerable number of individuals never take the disease. Suckling babies seem rather immune to scarlet fever, as they do to others of the exanthemata. One attack regularly protects from subsequent attacks of this disease.

Pathology: The characteristic lesion is the eruption on the skin and in the throat. There is intense hyperæmia, with dilatation of all the capillaries. There is some exudation of inflammatory products into the tissue of the true skin, with blocking of the sweat-ducts. In reality there is present an acute dermatitis.

Complicating lesions are often found—inflammation of the Eustachian tube, suppurative otitis, cervical adenitis, and, most important, various forms of *nephritis*, either acute degeneration of the kidney, or acute exudative or acute diffuse nephritis.

Incubation-period: This is short, being from two to seven days.

Scarlet fever—symptoms: The period of invasion begins acutely with rather high fever—101° to 104° F.—sore throat, often vomiting, and frequently general convulsions. The child appears decidedly sick, has headache, anorexia, and often diarrhœa, and complains of the sore throat; all these symptoms being somewhat in proportion to the height of the fever.

On examining the throat there will be found a diffuse bright redness covering the tonsils, pharynx, and hard palate. On looking carefully this will be seen to be made up of fine red points massed closely together. At times false membranes may be seen on the tonsils even this early.

After one to three days the period of *eruption* succeeds

this period of invasion. The characteristic rash consists of minute red points so closely packed together as to give the appearance of a diffuse scarlet color. This rash may be continuous or appear in scattered patches. The eruption begins on the neck and upper part of the chest, and usually spreads over the whole body. The face is most likely to escape. The rash from beginning to end lasts from three to ten days. In some cases the rash completely disappears in a few hours.

During the development of the eruption the temperature rises and all the constitutional symptoms increase in severity; but with subsidence of the rash the general symptoms and fever gradually disappear.

After the rash is gone a general *desquamation* of the whole skin begins, and continues for three to six weeks. This desquamation is not in fine scales, as after measles, but in large flakes, and in some places long strips of epidermis peel off. Where the skin is thick, as on the hands and feet, the flakes are largest and the process lasts the longest.

The *tongue* in scarlet fever is rather characteristic. During the period of invasion it is coated white; but later, during the rash, the white covering disappears, and leaves a reddish surface, with raised papillæ, giving the so-called "strawberry-tongue" of this disease.

Until *desquamation is complete* the disease is not over, and danger from infection is present even if the child seems perfectly well. The disease in different cases varies in severity from very mild attacks, through the typical cases as above described, to the very malignant ones with evidences of overwhelming infection, as marked cerebral symptoms, and hemorrhages into the skin and from the mucous membranes.

Scarlet fever—complications: The complications and sequelæ of scarlet fever are of great importance, and add enormously to its seriousness.

The commonest complication is a *catarrhal inflammation* of the *pharynx* and *tonsils*. This does not add much to the disease, but paves the way for another and much more serious condition—a *croupous inflammation* of the same parts. A false membrane is formed over the same area, and may spread to the naso-pharynx, nose, middle ear, or larynx. This is but

very rarely due to the Klebs-Löffler bacillus, ordinarily only streptococci being present. With this pseudo-membranous angina there are great swelling of the lymphatic glands of the neck, general symptoms of marked sepsis, and often complications in the ears, lungs, and kidneys. At times the pharyngitis may be so severe as to produce gangrene, with sloughing of large areas in the throat.

The *lymphatic glands* of the neck are always in a state of more or less inflammation, it often being severe enough to cause suppuration with abscess-formation.

The *middle ears* are very frequently inflamed during the course of scarlet fever, from extension of the inflammatory process through the Eustachian tubes into the tympanum. Ordinarily both ears are involved, and the inflammation may be catarrhal or suppurative. With otitis there are some rise of temperature, earache, and, usually, later a discharge from the meatus, with more or less *deafness*. Many cases are left permanently deaf, or become *deaf-mutes* after this serious complication.

Meningitis, pleurisy, pericarditis, endocarditis, arthritis, and broncho-pneumonia are infrequent complications.

The most important complication in scarlatina is *nephritis* of some variety. It accompanies almost every case that is at all severe, the poison of the fever being evidently very irritating to the renal epithelium. It may take the form of acute degeneration of the kidney only; or, when more severe, may be acute exudative or diffuse nephritis. The two former conditions may be completely recovered from, but the latter leaves a permanently damaged kidney behind. With the onset of either variety the urine becomes diminished in volume, and subcutaneous œdema and uræmic symptoms may be present in some of the severe cases. It is often the cause of death.

Diagnosis: This can only be made positively after the development of the *rash*. This, combined with the fairly characteristic symptoms of invasion, usually marks the disease positively. In some cases with very slight rash the diagnosis is very difficult. The occurrence of the typical desquamation afterward is always of assistance. Urticaria, erythema, and

rötheln may produce similar eruptions, but are excluded by the absence of signs in the throat and the general symptoms.

Prognosis: The cases vary a great deal in severity and mortality in different epidemics. The younger the child, as a rule, the more fatal the disease. The more marked the symptoms during the active stage of the attack the greater is the mortality-rate. Complications add greatly to the danger from the disease. The mortality-rate in a large series of cases averaged from 12 to 15 per cent., but is often much lower and again much higher than this in local epidemics.

Scarlet fever—treatment: The child should be *isolated* in one room, which should be kept well ventilated and at a uniform temperature of 70° F. Unnecessary furnishings in this room should be removed, and the nurse and others who of necessity must go into the sick-room should frequently use a gargle of some mild antiseptic. Those going into and out of the room had better keep hanging at the door a long cotton gown to wear over the clothing when in the room.

The *hands* should be carefully washed on coming out, and a short exposure to fresh air is advisable before coming in contact with other people. The patient should be given every day a complete bath of warm water and soap, and during the period of desquamation this should be followed by a slight greasing of the whole body with vaseline.

For the care of the disease itself, the child should be kept in bed during the whole course of the fever and constitutional symptoms. The diet should be milk only, this being the best preventive of a complicating nephritis. If the child cannot take milk, only fluid foods of other kinds should be allowed. Under any circumstances an abundance of water should be used. This diet should be continued for some little time after the fever has subsided, its change being indicated by the results of careful watching of the condition of the urine.

Another important part of the treatment is the care of the *mouth* and *throat*, to prevent complicating adenitis and otitis.

The mouth should be washed frequently, the pharynx sprayed, and the nose douched or sprayed with some mild antiseptic like Dobell's solution.

Otherwise the treatment is mainly *symptomatic*. If the

temperature is excessive, it may be controlled by cool sponging or small doses of the coal-tar products. The cerebral symptoms usually accompany the high temperature, and are best treated by the same means; but the bromides may be given in addition, if they become too excessive. If the heart-action becomes feeble, stimulants are to be used; the best of which are alcohol, digitalis, and strychnine.

Complications are to be treated on the usual plan—adenitis by local applications of ice, and incision in case of suppuration; otitis by heat locally, and cleanliness of the meatus; and nephritis as laid down in the description of this disease.

During *convalescence* care with the diet and the use of iron are of most importance.

ERYSIPELAS.

Definition: This is an acute contagious disease characterized by fever and its accompanying symptoms, and the presence of a diffuse red rash having a great tendency to spread.

Etiology: The disease is due to infection by a specific micro-organism—the streptococcus of Fehleisen. It is communicated by inoculation through an abrasion of the skin or mucous membrane, but the point of inoculation cannot always be found. One attack does not confer immunity against subsequent infection. It often develops in wounds received accidentally, or at the surgeon's hands, or after vaccination; but many cases of the so-called idiopathic form develop on the face or elsewhere from a wound so small and unimportant as to be overlooked. In new-born infants it often develops from the navel during the detachment of the cord.

Pathology: There is a marked inflammation of the true skin and of the subcutaneous connective tissue. The lymphatics are found to be filled with streptococci. The skin becomes congested, swollen, and infiltrated with the inflammatory products, with at times an excessive emigration of white cells and the formation of abscesses.

Complicating lesions in the veins, meninges, pericardium, peritoneum, kidneys, and lungs are often found.

Erysipelas—symptoms: After an incubation-period of from three to seven days the child is, as a rule, rather suddenly attacked with a chill followed by fever, prostration, headache, and loss of appetite. Often a general convulsion marks the onset of the disease. The fever remains rather uniform, averaging 100° to 104° F., and all the constitutional symptoms continue. The characteristic rash appears as a diffuse redness, with some thickening of the skin and consequently a well-defined raised edge. From day to day it is seen to spread, and as new areas are involved a new accession of fever and often a slight chill appear. The eruption ordinarily spreads by direct continuity of tissue, and the whole body may be involved; but at times patches are seen with uninflamed skin between. Natural lines on the skin, as the hair-line, bony ridges, or creases, often seem to limit the spread of the rash.

The *duration* of the disease is very indefinite, depending on the severity of the infection, the limitation of the spreading of the rash, and the resistance of the patient; but the fever and constitutional symptoms continue unabated so long as the disease has a tendency to spread.

Complications: Albuminuria, pneumonia, and abscesses are the commonest complications of the disease.

Diagnosis: The sharp constitutional symptoms, combined with the typical spreading rash with its raised edge, are quite characteristic.

Prognosis: The younger the child the more fatal the disease. In the new-born death is to be expected. In children over five it is a serious, but rarely a fatal disease. Complications, naturally, add to the mortality-rate.

Erysipelas—treatment: The child is to be kept in bed, well nourished according to its age, and alcoholic stimulants used as needed. There seems to be some value in the use internally of the tincture of the chloride of iron. Special symptoms are to be treated as they arise—excessive fever by the external use of cold water or by phenacetin internally, and great restlessness and sleeplessness by the use of bromides.

Locally, the parts should be kept continuously in a wet dressing of carbolic acid, 1 per cent., or in bichloride of mer-

cury, 1 : 10,000. These may be alternated with a 10 per cent. ichthyol ointment, or other similar application.

DIPHTHERIA.

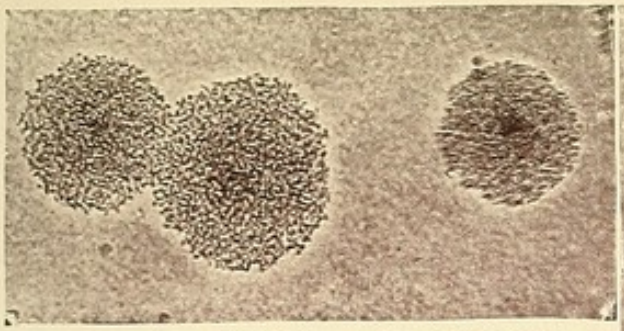
Definition: This is an acute contagious disease due to a specific bacillus, and characterized by the growth of a false membrane on some one of the mucous membranes, though usually in the respiratory tract. It may or may not be accompanied by constitutional symptoms of varying severity.

Etiology: Diphtheria is due to infection by the *Klebs-Löffler bacillus.* It exists in large cities as an endemic disease, and epidemics occur from time to time. Children are more prone to the disease than are adults. One attack does not protect from future attacks, although a short-lived immunity is produced. The bacilli exist in the pseudo-membrane, and are given off in the discharges from the nose and throat of the patient. They may live on clothing, toys, and in the fur of animals, and by these the disease may be spread to other children. The bacilli frequently remain virulent in the throat of the child after all local and general evidences of the disease have disappeared. They may also be found in the throats of attendants and other individuals who have no clinical signs or symptoms of the disease.

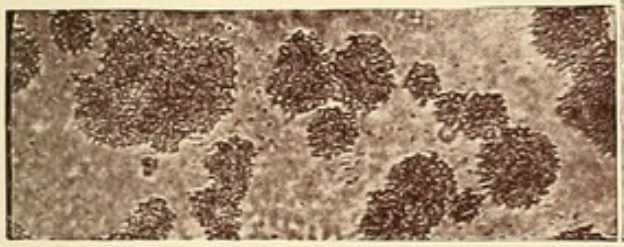
Children who have any abnormalities, or acute or chronic catarrhs of the throat or naso-pharynx, are much more liable to be attacked by diphtheria than others.

Pathology: The local lesion is a croupous inflammation of the mucous membrane involved in the diphtheritic process. The commonest locations are the tonsils, pharynx, nose, larynx, trachea, or bronchi. Other mucous membranes, or even the skin when abraded, may be involved. The mucous membrane is congested, swollen, and infiltrated, and its surface coated with a false membrane composed of fibrin, pus, and necrotic epithelium. This false membrane is tightly adherent to the mucous membrane, and if forcibly removed leaves a raw, bleeding ulcer.

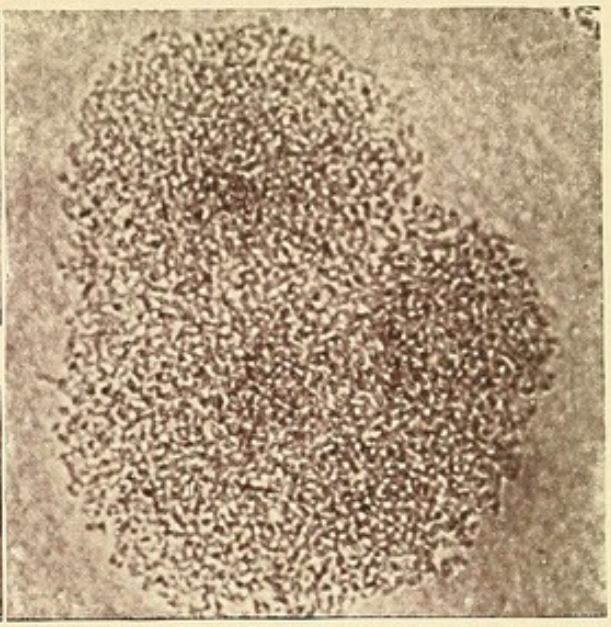
Complicating lesions, due to the presence in the blood of the toxins produced by the diphtheria bacilli, are seen in the



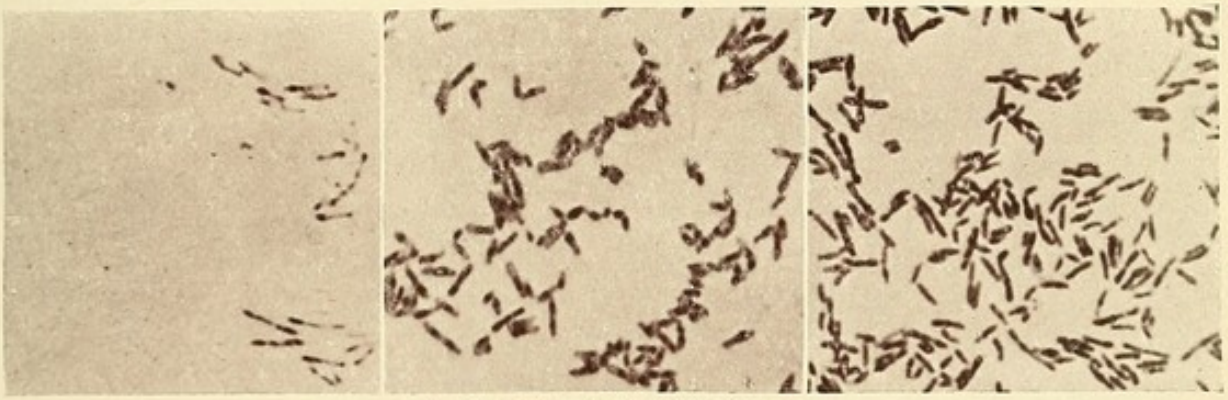
a. Colonies of diphtheria bacilli, × 160.



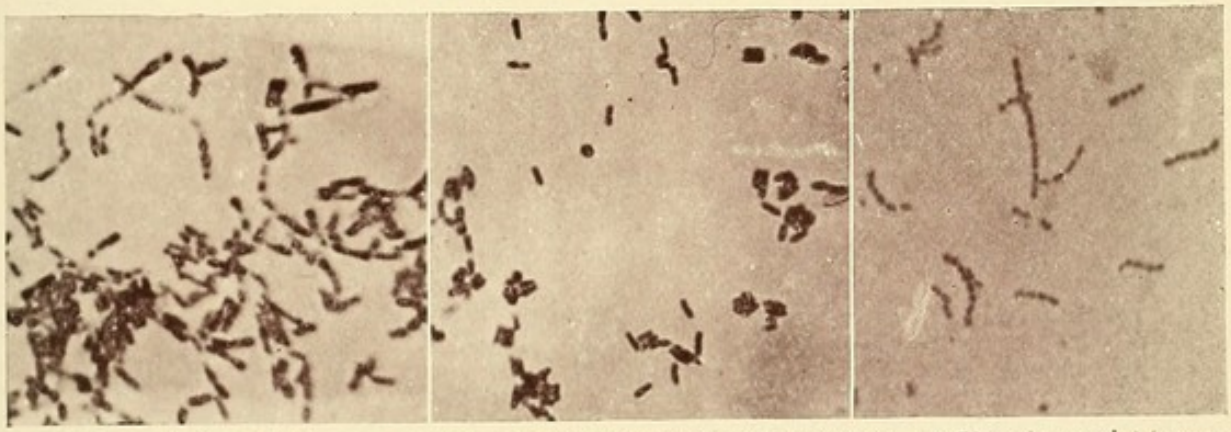
b. Colonies of pseudo-diphtheria bacilli, × 160.



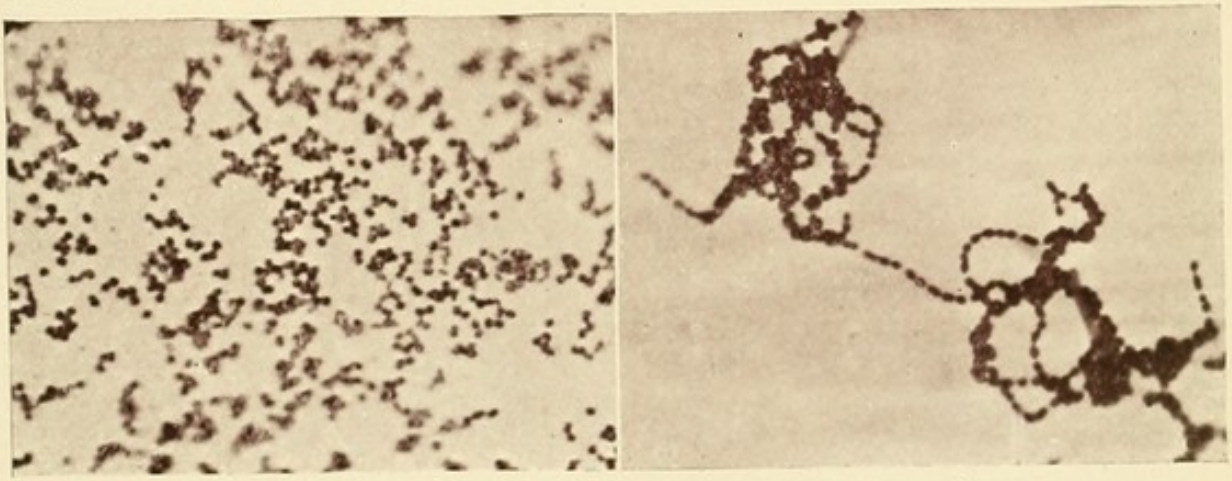
c. Colonies of diphtheria bacilli, × 240.



d. Diphtheria bacilli, × 1000.



e. Diphtheria bacilli, × 1000. *f*. Pseudo-diphtheria bacilli, × 1000. *g*. Streptococci, × 1000.



h. Streptococci, × 1000. *i*. Streptococci, × 1000.

Diphtheria Bacilli and Streptococci.

degeneration of the essential cells of various organs of the body. These are commonest in the kidneys, heart-muscles, peripheral nerves, liver, and spleen.

Other *complications* are due to coincident infection by various streptococci, among which are adenitis and broncho-pneumonia.

Incubation-period: This is from two to ten days.

Diphtheria—symptoms: The symptoms are local, due to the inflammation of the mucous membrane; and general, due to absorption of the toxins of the Klebs-Löffler bacilli into the system. The cases vary greatly both in severity of the constitutional symptoms and in the amount and location of false membrane formed.

The *local* symptoms are the presence on ordinarily some part of the *respiratory mucous membrane* of the characteristic *whitish patches* of this disease. These patches may be seen on the tonsils only, or on other parts of the pharynx, and may be continuous or with intervals between them. The pseudo-membrane is white, or gray, or black; it cannot be removed without leaving a raw, bleeding ulcer behind, and the surrounding mucous membrane is swollen, red, and inflamed. The parts are painful and tender to the touch, and dysphagia is present. The glands under the jaw are coincidently inflamed, being swollen and tender. From day to day the pseudo-membrane is often seen to spread and involve neighboring parts.

The first membrane may appear in the nose, or naso-pharynx, or larynx and out of sight. In the two former locations its presence is shown by a severe rhinitis, with irritating discharges from the nose, and in the last by the symptoms of croup. These false membranes may be thin or thick; and, since the use of bacteriological diagnosis in this disease, we know that there may be true diphtheria, as shown by the presence of the Klebs-Löffler bacilli, without any pseudo-membrane, the throat being simply in the state of catarrhal inflammation. The membrane in any one location is gradually loosened from its base, and is discharged on an average in about seven days.

The *constitutional* symptoms in some cases are absolutely absent, or so slight as to be overlooked.

The disease, however, ordinarily begins with a chill or chilly sensations, accompanied by a rise of temperature. The fever continues throughout the disease, but is usually of only moderate severity—101° to 102° F. Temperatures over 104° F. are very rare. With this are prostration, headache, vomiting, and diarrhœa. The heart's action is ordinarily rapid and feeble, and diphtheria is pre-eminently the disease in which attacks of serious heart-failure are to be dreaded. These attacks may arise suddenly when the child is in his ordinary condition, or may develop more slowly, venous congestion, dyspnœa, and rapid feeble pulse being the signs of the failure. Probably in most cases these attacks are due to degeneration of the vagus nerve by the diphtheria toxins.

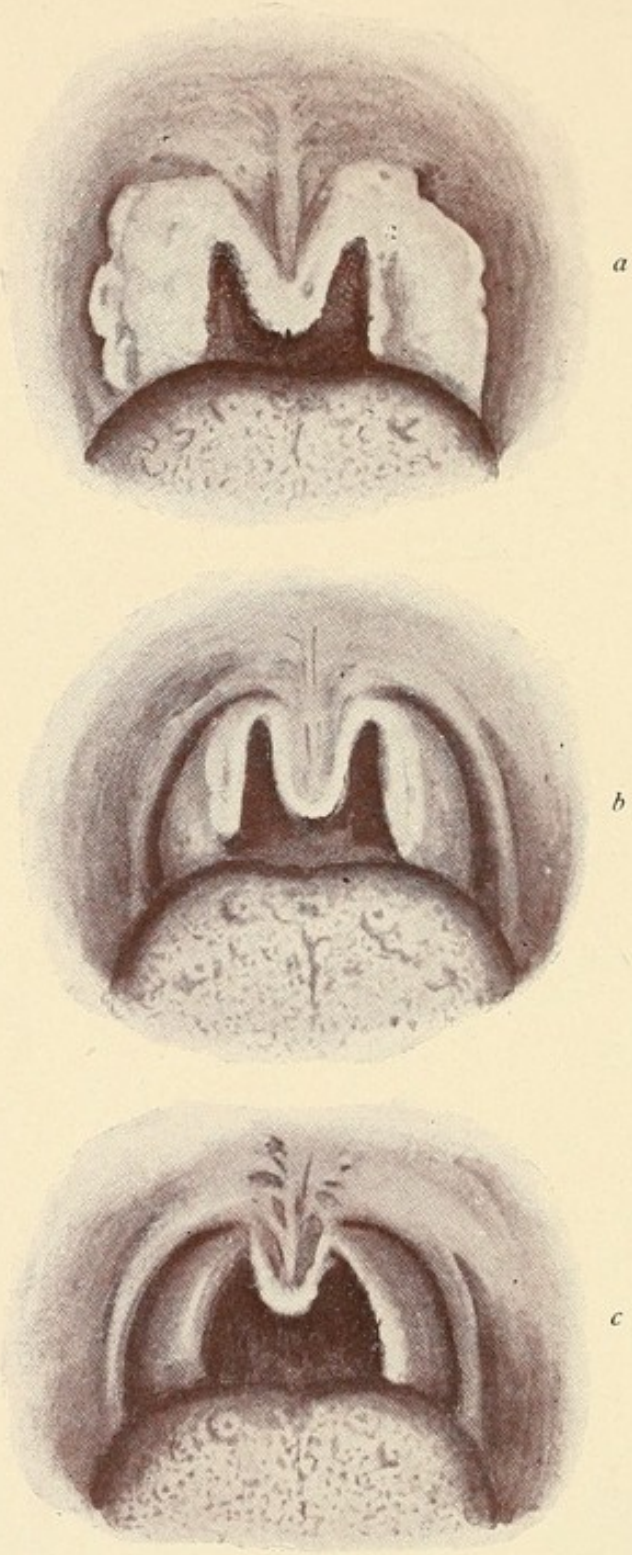
Diphtheria—abnormal cases: There are cases without very high fever and with no large quantity of membrane, in which the child seems to suffer, and usually dies from an intense septic infection or *toxæmia*. The main symptoms here are great prostration and feeble heart-action.

There are others where the only symptoms are those of a laryngitis, the membrane being confined to the larynx. These are described under Membranous Laryngitis.

Complicating lesions add their symptoms to those of the disease proper, as obstructed respiration in laryngeal cases; rapid breathing, cough, and increased prostration in bronchopneumonia; diminished urine and œdema in nephritis.

Pseudo-diphtheria: Since the introduction of the systematic bacteriological examination of cultures from most cases of diphtheria, it has been proved that there is such a thing as clinical diphtheria, differing scarcely from the true form, except that there are no Klebs-Löffler bacilli in the pseudomembrane, but instead there are *streptococci* only as its cause. This variety has been called *pseudo-* or *streptococcus-diphtheria*. Clinically it is well to consider the two forms alike, although the Klebs-Löffler diphtheria is the more serious disease of the two.

Diphtheria—sequelæ: There occur frequently after diphtheria *paralyses* of groups of muscles. These are due to de-



Diphtherial infection of uvula and anterior pillars of fauces, showing the disappearance of the membrane after injection of antitoxic serum. (*a*, 18 hours; *b*, 24 hours, and *c*, 36 hours after injection.) (Williams.)

generation or inflammation of the peripheral nerves, due to the toxins of the disease. The muscles most commonly involved are those of the soft palate, of the pharynx, of the larynx, of the eyes, of the extremities, and of respiration. Except when involving the last set of muscles, they are not dangerous to life. After considerable time the power gradually returns to the paralyzed muscles as the nerves are regenerated. If the respiratory muscles are affected, death is quite likely to occur.

Diagnosis: Clinically, the presence of general symptoms of an infectious disease, combined with the characteristic false membrane, are the points for diagnosis. This membrane is not easily wiped away, as are the spots in follicular tonsillitis. A sanious nasal discharge points to membrane in the nose, and laryngeal symptoms to its presence in the larynx. It is practically impossible to separate the cases of true from those of false diphtheria positively by the appearance alone.

Of recent years our diagnosis is made more certain by the aid of *bacteriological methods*. A swab culture is made from the throat, and this allowed to incubate for twelve to eighteen hours. At the end of this time the little colonies are stained and examined under the microscope, and the presence or absence of the Klebs-Löffler bacilli proved. This is of special assistance in laryngeal and nasal cases where no membrane is visible.

Prognosis: This is very uncertain, as the age of the patient, the virulence of the infection, the location and extent of the membrane, and the presence of complications all enter into the prognosis. The younger the child the more dangerous the disease. When the membrane remains confined to the tonsil only, the prognosis is quite good. Rapidly spreading membrane is serious. Laryngitis and broncho-pneumonia are very dangerous complications. The cases with marked evidences of toxæmia are always severe. The mortality of pseudodiphtheria is far less than that of the real form.

Diphtheria—treatment: The same preventive measures should be taken as in all infectious diseases. The patient should be closely isolated; the attendants should gargle their throats frequently; the outside clothing worn in the room

should be changed before going elsewhere; discharges from the child's mouth and nose should be received in a strong solution of mercuric chloride; the clothing and bedding of the patient should be soaked in the same solution or burned; the hangings of the room should be removed before putting the child in it. After the disease is over the walls of the room should be washed with bichloride, and a public funeral should not be allowed.

Nowadays, in addition to the old practice of swabbing out with an antiseptic the throats of children who have been exposed to the disease, a small immunizing dose of *antitoxin* should be given, averaging from 100 to 500 units, according to the age of the child. For this purpose there seems as much value in administering the serum by mouth as subcutaneously.

For the actual care of the disease, the child should be kept absolutely quiet in bed, and on a highly nourishing diet, mainly of milk, and other easily digested soft foods. Gavage is often very helpful in feeding these cases when they will not take sufficient nourishment. The room should be well ventilated and kept at an equable temperature. Internally there seems some value in giving small doses of bichloride of mercury every four hours. As stimulants, whiskey is to be given in rather large doses frequently repeated. It not only stimulates the heart, but also seems to neutralize the toxæmia. In cases of failing heart strychnine is our most useful remedy.

Locally, the inflamed parts should be kept clean. If the membrane is only on the tonsils and pharynx, frequent gentle swabbing with weak bichloride solution is helpful. If the naso-pharynx or nose is involved, the nostrils should be washed out frequently each day with Seiler's solution. This is best done by a simple piston- or a fountain-syringe. The solution should go in one nostril and out the other. Laryngitis is treated locally and mechanically as described under *membranous laryngitis.*

In addition to these measures, all cases should be treated, as soon as seen, by *injecting antitoxin.* The initial dose should be 1000 to 3000 units, or even more, according to the age of the child, the dose being repeated in about eighteen hours if no local or general improvement is manifest. It

may be given a third or a fourth time at the same interval if needed. Use the most concentrated serum, as the size of the dose is smaller, and the earlier it is given the better the result. It is given by a large-sized hypodermic syringe, under aseptic precautions, and may be inserted under the skin of the back or the side. Signs of improvement after antitoxin are seen in the diminution of the fever and constitutional symptoms, and in the stopping of the spread and the loosening of the membrane. These must not be expected before eighteen hours. Some days after the injection in certain cases erythema, urticaria, and joint-pains are noticed, but they usually quickly subside.

The results of the use of antitoxin have been most gratifying, particularly in the laryngeal cases, *intubation* and *tracheotomy* being less often required, and when used, saving more lives.

Complications are treated as always; post-diphtheritic neuritis by massage, electricity, and strychnine. Convalescence requires the use of iron, combined with good diet and plenty of fresh air.

WHOOPING-COUGH.

Definition: This disease, also called **pertussis**, is an infectious neurosis, characterized by inflammation of the respiratory tract and a peculiar paroxysmal cough.

Etiology: While whooping-cough is probably due to some germ, as yet efforts to isolate it have not been successful. The poison seems to be given off in the breath, to float in the air, and to be inhaled. Children are quite susceptible to the disease, and more so than are adults. One attack regularly produces immunity. The disease exists endemically in most cities, but epidemics occur from time to time.

Pathology: Catarrhal inflammations of the larynx, trachea, and bronchi are regularly found. Broncho-pneumonia exists as a frequent complicating lesion.

Incubation-period: This is probably about two weeks, but cannot be stated positively.

Whooping-cough—symptoms: The invasion begins with a catarrh of the larynx, trachea, or bronchi. This lasts for

from ten days to two weeks; but instead of the cough improving, toward the end of this period it grows worse. This catarrh is of varying degrees of severity, but regularly the cough seems disproportionate to the physical signs to be found in the chest. There is scarcely any fever or malaise.

Toward the end of this period of invasion the cough begins to assume the typical character which has given its name to the disease. This cough comes in *paroxysms*, during which the child coughs continuously for some seconds, at the same time holding his breath, and at the end of the paroxysm taking a *long stridulous inspiration*, which produces a sound like the word "whoop." During this attack he becomes blue in the face, the eyeballs become prominent and suffused, the veins stand out, and the child presents the appearance of suffocation. This paroxysm is apt to be repeated two or three times, the inspiratory whoop being given at the end of each, until some tenacious mucus is expelled or vomiting is produced.

It may be some hours before another set of paroxysms begins. The child grows to know when the attacks are coming, and will stand for support against some stationary object, and usually with his hands braced on his knees. Epistaxis is often an accompaniment of a severe paroxysm, and after an attack the child is very exhausted and often in a profuse perspiration. These paroxysms are usually more frequent by night than by day, and may be repeated many times in the twenty-four hours. They vary both in intensity and frequency very much. Exercise, shouting, crying, draughts, eating, drinking, or excitement will often develop a paroxysm.

The general health of the patient suffers mainly from the interference with sleep, and from the inability to retain sufficient nourishment on the irritable stomach. Owing to this, many of these children grow very emaciated.

This paroxysmal stage lasts from three to six weeks, but often is continued over a much longer period. In some cases a whooping-habit seems to be developed, which lasts many months. This disease ordinarily disappears very gradually,

the paroxysms becoming less frequent and less severe until they stop.

Whooping-cough—physical signs: In uncomplicated cases auscultation of the chest gives no physical signs. If *bronchitis* is present, as is so frequently the case, the coarse râles and sibilant and sonorous breathing of this disease are found. If *broncho-pneumonia* complicates the disease, we find the characteristic signs of this lesion.

Complications: Bronchitis and broncho-pneumonia are the commonest and most serious complications of pertussis. Hemorrhages from the nose or mouth, into the conjunctiva, or into the meninges, are seen from time to time. The frænum of the tongue is apt to be torn by the persistent coughing. The vomiting after the paroxysms may be looked on as a complication when it is so severe as to interfere with the child's nutrition. Convulsions and various forms of cerebral paralyses may complicate some severe cases in infancy. Hernia and prolapsus ani may result. A latent tuberculosis may be started up by the disease.

Diagnosis: The typical whoop is the diagnostic test of this disease; but in children with a persistent cough without adequate physical signs to account for it, and especially if it appear paroxysmal and accompanied by vomiting and suffusion of the eyes, a fairly probable diagnosis may be made. The presence of pertussis in the neighborhood, or a history of exposure to the disease, aids in making our diagnosis.

Prognosis: Whooping-cough is a disease frequently occurring in very young infants, and in these cases it is an extremely fatal malady, as it is apt to be complicated by broncho-pneumonia, which in itself is necessarily fatal during the first two years of life. In older children, and without complications, it is not much to be dreaded. Infancy and complications are the dangerous sides of this disease.

Whooping-cough—treatment: Children with pertussis should not be allowed to attend school nor to mix with other children. This is a point in which great carelessness exists in every community. In good weather the sick child should be kept out of doors a great deal, if he has no pulmonary complica-

tions, but the habit of taking such children on street-cars and into places where other children are exposed, is to be condemned. If the weather is bad, or if pulmonary complications prevent the child being out of doors, care should be taken to keep the rooms in which he lives well ventilated; but every precaution should be followed to prevent his "catching cold." The child's diet should be carefully attended to, and only the most nourishing and easily digestible food should be given. If vomiting is excessive and interferes with the child's nutrition, the food should be predigested. Often by feeding a little of this food immediately after a paroxysm accompanied by vomiting, it will be retained. In some cases alcoholic stimulants will be useful. If any of the drugs given for the cough seem to irritate the stomach, they should be stopped.

There seems some value in the child's breathing the *vapor* from *creosote* or *carbolic acid*. This may be brought about by wearing a respirator for some time each day, on which a few drops of the medicament are placed, or by having the air of the child's room contain the vapor of the drug. A so-called *vapo-cresoline lamp* is useful for this purpose, but either drug may be vaporized in an ordinary tea-kettle quite as well.

Local applications made directly to the larynx are of little value. In very severe paroxysms with threatening convulsions chloroform may be used.

Internally almost all the drugs of the pharmacopœia have been given. Only a few are of value, and in some cases even these fail. *Belladonna* in increasing doses until the early poisonous symptoms of the drug appear is often very useful. *Antipyrin* seems the next most valuable drug. It often controls the paroxysms like magic. *Bromoform* in two-drop doses four times a day is a newer remedy of some value. In some cases quinine in fairly large doses helps. It is often helpful to produce good sleep by the use of a *hypnotic*, such as bromide, chloral, trional, or sulfonal, which likewise quiet the reflex excitability of the child.

Complications in the lungs should be treated in the usual way.

MUMPS.

This disease, also called **infectious parotitis**, is characterized by constitutional symptoms and inflammation of the salivary glands.

Etiology: The disease is contagious from person to person, but does not seem to attack infants quite as readily as adolescents. No specific germ has as yet been isolated. One attack protects against subsequent ones.

Pathology: Only one, or both, parotids are involved. The submaxillary glands may or may not be coincidently inflamed. The inflammation only goes to the point of congestion, with swelling and obstruction of the ducts. Suppuration is very rare, and as the inflammation subsides the gland returns to normal.

Incubation-period: This varies from two to three weeks.

Mumps—symptoms: The constitutional symptoms begin early and last three to five days. They are fever, headache, irritability, anorexia, nausea, and prostration. The temperature is from 100° to 102° F.; but in severe cases may reach 104° F. Coincidently the glands become painful, swollen, and tender, and the mouth dry. Moving the jaw, as in talking or eating, is very painful, and the presence of any food in the mouth pungent enough to excite the flow of saliva increases the pain. This is the reason acids were used as an old test for mumps. One gland is usually inflamed a day or two before the other, but at times it may be some days before the second gland becomes swollen. The submaxillary glands may be inflamed coincidently or later. After two to three days the swelling of the glands reaches its height, and then gradually disappears. The disease lasts one or two weeks, as the glands become inflamed together, or subsequently.

Complications: In infants these are rare. In adolescents, inflammations of the *testicles*, *breasts*, or *ovaries* are seen in a fair proportion of cases. This inflammation usually subsides without injury to the organ.

Diagnosis: An idiopathic inflammation of the parotid or submaxillary glands is usually mumps. If the parotid is swollen, the lobe of the ear stands out from the head. Be

sure the swelling is not due to some of the cervical lymph-glands.

Prognosis: This is almost invariably good, as recovery is to be expected in a few days to a week.

Mumps—treatment: The child should be kept in the house and fairly quiet. The diet should be liquid and without much flavor, so as not to excite the salivary glands to activity. If fever is high, a little phenacetin should be given. If the glands are painful, hot applications are soothing. Broken doses of calomel are well given at the beginning of the attack.

LA GRIPPE.

Definition: This disease, also called **epidemic influenza**, and **catarrhal fever**, is an infectious malady, characterized by fever, pains, prostration, and catarrhs of the mucous membranes.

Etiology: It is pretty well proven now that grippe is due to a *specific germ*, the *bacillus of Pfeiffer*. The disease occurs epidemically over large areas of territory, and also endemically after the epidemic feature has passed. The germ seems to be disseminated in the atmosphere, and to attack the victims mainly in the winter and spring. Infants and children seem equally prone to the disease with adults. One attack does not confer immunity to subsequent ones.

Pathology: Catarrhal inflammations of the eyes, nose, throat, ears, larynx, bronchi, or intestines are found. Complicating lobar or broncho-pneumonia may be present.

Incubation-period: This is probably short, from a few hours to a few days, but cannot be decided positively.

La grippe—symptoms: The disease usually begins rather suddenly with fever, headache, pains in the back and limbs, and marked evidences of prostration. The fever runs from 100° to 104° F., but very seldom higher. It lasts from two days to a week, and is accompanied by anorexia, nausea, vomiting, and often diarrhœa. In infants convulsions may be present, and restlessness, insomnia, and irritability are quite common. All the symptoms gradually disappear in about a week in the uncomplicated cases.

La grippe—complications: Very few cases run their course without some complication. The commonest of these are in the respiratory system. Few cases occur without some involvement of the nose and pharynx, a severe rhino-pharyngitis being almost always part of the disease. The mucous membrane of the frontal sinuses is likewise inflamed, and thus severe frontal headaches are produced.

A frequent complication is involvement of the *middle ear*, and an acute otitis media, with its symptoms of earache, deafness, and a discharge, follows.

In children especially, the cervical lymph-glands are very often inflamed, and frequently go on to suppuration.

The larynx, trachea, and larger bronchi are likewise usually involved in the catarrhal process, producing an irritating, croupy cough, with substernal pain, but very little expectoration.

All the above inflammations seem to be almost a necessary part of the attack of grippe, and very few cases escape without more or less severe evidences of these.

More unusual, and more serious complications are *broncho-pneumonia* and *lobar pneumonia*. One or the other variety of inflammation of the lung is seen in a fair proportion of the cases of influenza. They both run a rather irregular course, the toxic symptoms of each being very prominent, the duration prolonged, and the mortality high.

In some children the *gastro-enteric* system seems to be particularly vulnerable to the influenza bacillus, and vomiting and diarrhœa are the prominent symptoms of the disease. This complication, however, is seldom very severe, and recovery is the rule.

Diagnosis: During an epidemic the diagnosis is rather easy. In sporadic cases the diagnosis is best made by excluding other conditions by the absence of physical signs. Malaria is differentiated by the enlarged spleen and plasmodia; pneumonia, by the physical signs in the chest; meningitis, by the retracted head and ocular palsies; typhoid fever, by the tympanites, rose spots, and Widal reaction.

Prognosis: When uncomplicated most all of the children recover. Pulmonary complications are apt to be serious,

especially broncho-pneumonia. Latent tuberculosis is often developed by an attack of la grippe.

La grippe—treatment: The child should be put to bed and kept there so long as the fever continues. The diet should be nutritious and easily digestible. The bowels should be moved by fractional doses of calomel. For the aching and fever, nothing is so useful as *phenacetin* and salicylate of sodium, given in doses suitable to the child's age. Quinine may be added in some cases with advantage. Where the depression is extreme, alcoholic stimulants should be used freely. The respiratory complications are best treated by the ammonium salts combined with inhalations. Convalescence should be carefully watched, and general tonic treatment followed until full strength returns.

TYPHOID FEVER.

Definition: This is an infectious disease due to a specific germ, and characterized by a rather typical course of fever with its attendant symptoms, and lesions in the lymphatic glands of the intestines.

Etiology: The direct cause of typhoid is infection by *Eberth's typhoid bacillus*. This is usually taken into the digestive tract along with some form of food or drink, water and milk being the most usual carriers of the germ. It exists and multiplies in the contents of the intestines, in the intestinal lesions, the lymphatic glands, the spleen, liver, kidneys, and the blood. They are discharged from the body with the fæces, and can live outside the body for a considerable time.

The disease is very rare in infants, as they are so universally fed on sterile food. In children it occurs more commonly, but even in them is rarer than in young adults. One attack of typhoid regularly produces immunity for life.

Pathology: There is swelling, followed, in the severer cases, by necrosis and ulceration of the solitary and agminated glands of the ileum and colon. There is also an associated catarrhal enteritis. The lymphatic glands of the mesentery are swollen and inflamed, but rarely go to suppuration. The spleen is enlarged considerably and soft. There is a degener-

ation or the essential cells of the liver and kidneys. The heart-muscle is soft and flabby. Complicating inflammations may be found in the lungs, meninges, peripheral nerves, and veins, but all are rather rare in children.

Incubation-period: This averages from one to two weeks. The first symptoms of the fever are so indefinite that it is hard to set a positive period of incubation.

Typhoid fever—symptoms: The course and natural history of the disease in childhood follow fairly well the type as seen in the adult, except that all the symptoms seem less severe.

The disease *begins* gradually with lassitude, slight headache, anorexia, and often attacks of diarrhœa; or more rarely rather suddenly with a quick rise of temperature and prostration. The fever lasts usually three weeks; during the *first week*, each day showing a little more temperature than the preceding day; during the *second week* the temperature remaining fairly uniform; and during the *third week*, declining day by day to reach normal after the twenty-first day.

The *average temperature* is from 102° to 104° F., but in more marked cases it reaches 105° to 106° F. It often shows marked variations in its course, complications of an inflammatory nature tending to increase it, while hemorrhages and perforation cause it to fall. It may be prolonged for a considerable time beyond the three weeks, remaining at 99½° to 101° F., or even after a period of apyrexia it may rise again and remain up without a distinct relapse.

The *heart's action* grows more rapid as the fever progresses, but in this disease is always somewhat slower than a like amount of fever from another cause would produce. The *pulse* retains its power, but is apt to become dicrotic toward the height of the fever.

The *tongue* is coated down the centre, but the tip and edges remain clean and the tip pointed. The *mouth* becomes dry and glazed, and sordes develop around the lips and teeth. *Anorexia* is a regular symptom.

The *bowels* are more often constipated than loose, but when diarrhœa exists the discharges partake of the "pea-soup" character seen in adults. The abdomen becomes moderately distended and tympanitic at some time during the course of

the fever, due to accumulation of gas in the intestines. Tenderness and gurgling in the right iliac region are unimportant signs.

The *spleen* is regularly enlarged, so as to be felt on palpation extending some inches below the border of the ribs. There seems some connection between enlargement of this organ and absorption from the intestinal lesions.

During the *second week* of the fever the characteristic *eruption* of the disease appears in the shape of isolated, rose-colored, lenticular spots, slightly elevated, and disappearing on pressure. They are usually few in number, appearing on the abdomen and chest, and coming in successive crops which last about three days each.

The *nervous symptoms* are rather marked in children: headache, restlessness, irritability, and later stupor and apathy. Picking at the bed-clothes and subsultus tendinum are not very common. In some cases delirium, hyperæsthesia, stiff-neck, and ocular symptoms may be severe enough to suggest meningitis.

The children usually feel quite sick, and *prostration* is enough to make them glad to stay in bed. As the disease progresses, *emaciation* becomes extreme, and *bedsores* are very liable to develop over bony prominences.

Intestinal hemorrhages are not very common, but do occur toward the end of the second or the beginning of the third week. They may be single or repeated. They add a danger to the disease in showing the presence of rather deep ulcerations, but in themselves are rarely fatal. The blood is usually dark, and mixed with fæcal matter.

Intestinal perforation is very rare. It is accompanied by all the symptoms of intense shock, and a fatal ending is soon to be expected. *Retention of urine* is quite a common symptom at some time in the course of the disease.

Relapses may occur at any time for two weeks after the subsidence of the fever. They are always shorter in duration and milder in their course than the original fever, but usually all the signs and symptoms of the first attack are reproduced.

Typhoid fever—complications: Slight albuminuria is usually present in typhoid, due to a degeneration of the kidney-cells.

More or less bronchitis is rather common, but is usually confined to the larger tubes. Broncho-pneumonia is a much rarer as well as more serious complication of typhoid. Otitis media is fairly common if attention is not paid to cleanliness of the mouth and throat. Phlebitis and venous thrombosis develop in many of the cases, most often in the legs.

Diagnosis: This needs for its confirmation the typical fever, tympanites, enlarged spleen, and the eruption.

In addition, the *Widal reaction* should be found in the blood: if to a culture of typhoid bacilli a drop of blood from a patient suffering from, or lately recovered from, typhoid fever, be added, the bacilli undergo a peculiar agglutination, which does not take place when normal blood, or blood from other diseases, is added. This test, while not absolute, if positive, is confirmatory.

Malaria is differentiated by the presence of *plasmodia* in the blood; *tuberculosis* by local signs of that disease; meningitis by ocular and pupillary signs; and the various forms of enteritis by the more marked intestinal symptoms.

Prognosis: This is better than in adults, as the disease is usually milder in children.

Typhoid fever—treatment: The child should be put to bed and kept there at least two weeks after the fever has reached normal. The diet should be milk, or milk derivatives only, if possible; but if an idiosyncrasy exists against this, broths and eggs may be used. If constipation is present, the bowels should be moved in the beginning by calomel, and afterward, at least every other day, by enema. If diarrhœa is not excessive, it should be left alone. If necessary, it may be checked by bismuth and opium. All discharges from the bowels should be received into vessels containing 1 : 1000 bichloride of mercury solution, and allowed to remain in it at least an hour before being thrown out. All bedding and cloths which are soiled by the fæcal discharges should be soaked in the same solution, or boiled thoroughly by themselves.

The fever had best not be treated by any drugs of any nature given internally. They simply control the symptoms without doing any good. If the temperature is $102\frac{1}{2}^{\circ}$ F. or

over, *cool sponging*, or *cold packs*, or *cold bathing* should be systematically used; being guided by the height of the temperature and the reaction of the child as to the choice of methods. As temperature-reducers, they are valuable inversely in the order given. The temperature of the water should be about 80° to 85° F. in either form.

The mouth and tongue should be carefully cleaned three or four times a day, a mixture of lemon-juice and glycerin being very useful for this purpose.

There seems some value in giving dilute hydrochloric acid in five-to-ten drop doses three times a day, throughout the course of the fever. If the tympanites is severe, use turpentine stupes to the abdomen or pass a rectal tube. If the child is very restless and sleepless, or delirious, bromides may be used as needed. Stimulants in the shape of whiskey or strong wine, are usually needed in the last week of the fever. For intestinal hemorrhage we give opium and apply cold to the abdomen. Intestinal perforation is treated by morphine. Prevent bedsores in the usual way, and be on the watch for retention of urine. Care must be taken after the subsidence of the fever in returning to a normal diet, and this should take place very gradually. Strychnine and iron are valuable during the convalescent stage.

MALARIA.

Definition: This is an infectious disease due to the presence in the blood of a specific organism belonging to the protozoa, and characterized by fever, enlarged spleen, and cachexia.

Etiology: The organism causing the disease was first described by Laveran in 1880, and named the *plasmodium malariæ*. It exists in the blood and destroys the red blood-cells. We do not know in all cases how it enters the blood; nor is its habitat outside the human species exactly located.

There seems, however, strong proof in some cases that certain species of *mosquito* contain the plasmodium and act as the infecting agent.

The *plasmodium* is an amœboid body growing in the red corpuscle, absorbing the pigment from the red cell into itself,

and, as a paroxysm approaches, segmenting into a number of smaller bodies, each of which probably makes its way into another red cell to go through a similar series of changes when its cycle is through.

Different *varieties* of malarial poisoning are due to somewhat different species of plasmodia, the organism of intermittent fever showing some morphological differences from that of remittent or æstivo-autumnal fever.

Malaria attacks persons of all ages—children as well as adults. It exists endemically in certain parts of the country which are usually low-lying marshy places, and is especially prevalent after the country has been flooded or when new ground has been turned up. In moist Southern climates it is especially frequent. It occurs most commonly in the spring and fall, being rare in the winter.

Incubation-period: The poison of the disease may produce symptoms shortly after infection, or remain latent over such considerable periods of time that it is impossible to decide on a definite time of incubation.

Malaria—varieties: There are *two* fairly distinct types of malarial fever seen—the *intermittent* and the *remittent* or *æstivo-autumnal form*.

Intermittent fever begins suddenly with a severe chill, followed by a rapid rise of temperature to often as high as 104° or 106° F., and after a few hours this falls equally as quickly to normal with a profuse drenching perspiration. During the fever there are intense headache, backache, prostration, and often nausea and vomiting. In infants general convulsions quite commonly occur with the rise of temperature. This paroxysm is repeated daily at about the same hour in the quotidian form or on alternate days in the tertian. Between the paroxysms the child feels perfectly well and has no symptoms. In children one or the other stage of the paroxysm is often wanting, the attack seldom being quite as typical as in the adult.

Remittent fever: In this form the disease may begin suddenly or gradually, or may follow one or more paroxysms of the intermittent type. The fever has daily remissions in its course, but never reaches normal. It lasts an indefinite

length of time, depending on treatment and on the patient's remaining in the malarious country.

With the fever the child suffers from headache, backache, marked prostration, and often nausea and vomiting. The tongue is coated white, the pulse is rapid, and the patient frequently passes into the so-called "typhoid state." In children delirium, restlessness, sleeplessness, and often convulsions are present. These cases are often mild, but they frequently, especially in the South, pass into the pernicious form of fever, with an accession of all the symptoms, and often with a fatal ending.

After **either form of malarial infection** has lasted for a sufficient length of time, the so-called **malarial cachexia** develops. In other cases it is seen in children living in a malarious country, even with no signs of preceding fever.

This cachexia is due to the rapid destruction of the red blood-corpuscles by the plasmodia. The children are pale, feeble, languid, and emaciated. They have headaches and digestive disturbances. An examination of the blood will show the presence of a high degree of secondary anæmia, and of plasmodia in the red cells.

Malaria—diagnosis: In *intermittent fever* the paroxysm is so characteristic as to settle the diagnosis easily. In the *remittent form* the diagnosis from typhoid is very difficult. In all varieties we expect to find the spleen enlarged enough to be easily palpated, and in addition a careful searching of the blood for plasmodia will usually find them, and thus settle the diagnosis positively. The therapeutic test with quinine is always helpful. Be careful not to overlook other conditions by carelessly calling them malaria.

Prognosis: This is good if the disease is recognized early and properly treated. Removal from a malarious district adds much to a good prognosis.

Malaria—treatment: *During a chill* hot applications should be made externally and a good dose of hot whiskey be given.

After the chill is over the bowels should be opened by a dose of calomel, and then *quinine* be given in properly sized doses for the severity of the infection and the age of the child.

Children bear quinine well, and should be given relatively large doses. If the malaria is in the form of well-marked *intermittent* fever, a couple of large doses at an hour's interval, and the last one about five hours before the expected paroxysm, is the best way of giving it.

In the *irregular attacks* and in the *remittent* forms it should be given regularly in good-sized dose three times a day. It should be continued in gradually decreasing doses for several days after all evidences of the infection are gone, and the enlargement of the spleen has disappeared.

For children who can swallow capsules, this is the best method of giving quinine. If it must be given in solution, it is best disguised by the syrup of yerba santa. When small doses only are required, the chocolate quinine tablets may be used, each tablet containing one grain of the tannate. If it cannot be given by mouth, it may be given by rectum, either in enema or by suppository, a double dose of the bisulphate being used.

In malarial cachexia *arsenic* should be given with the quinine.

SYPHILIS.

Definition: This is a chronic infectious disease, probably due to some specific germ; but as yet none has been isolated and proven to be the real cause.

Forms: In infancy and childhood the disease may be *acquired* in many ways just as in adult life, but is more often *inherited*, and is then called *hereditary* or *congenital syphilis*.

Acquired syphilis differs in no respect from the disease as seen in adults, and so will receive no separate description.

In *inherited syphilis* the symptoms seem somewhat modified from the regular course and require special consideration, and in this article, when using the term syphilis, the congenital variety will be understood.

Etiology: The disease may originate in the fœtus from the father, the mother, or from both parents. Parents in the *secondary* stage of the disease are almost certain to transmit the taint. If in the *tertiary stage*, or after prolonged and proper treatment, the danger is rather slight. There seems

less danger of transmission from a syphilitic mother than from a syphilitic father. If the mother is infected late in her pregnancy, the child will often escape.

Pathology: The lesions in babies dying of syphilis are not by any means always characteristic. There are usually found in the viscera certain changes of the nature of a new growth of connective tissue, which replaces the proper structure of the organ involved. For instance, there may be fibroid changes found in the spleen, liver, lung, or kidneys, giving these organs the characteristic whitish color and tough consistency which are always found in interstitial hyperplasia. The capsules of these organs are thickened and adherent, and the whole organ is usually enlarged.

In the *bones* the lesions are quite characteristic and more regularly found. There is usually an inflammatory process present at the junction of the shaft of the long bones with the epiphysis. This consists of congestion and greatly increased proliferation of cartilage-cells. This may be found in only one or in many of the bones, and may lead to separation of the epiphysis and diaphysis. These changes are often found in the metatarsal and metacarpal bones and in the phalanges, producing syphilitic dactylitis.

In the late cases there are found *osteophytic growths* on the shafts of the long bones, due to a chronic periostitis, with the formation of new bone-tissue under the periosteum. This produces great enlargement and thickening of the affected bone. These thickenings may be uniform or in nodes.

Syphilis—symptoms: According to the virulence of the infection and the period of incubation in the individual fœtus, depends the condition of the child at birth. We must bear in mind that what corresponds in the acquired disease to the first period of incubation, (until the appearance of the initial lesion;) and to the second period of incubation, (until the appearance of the secondary symptoms), takes place during *intra-uterine life* in the inherited form. In other words, the infant is ordinarily born during the second period of incubation; and, more rarely, after the secondary symptoms have begun to appear. Accordingly, abortion may take place, and frequently does; or a dead, premature, or full-term child may be

born; or a living child with skin lesions and other evident signs of the disease may come into the world; but most commonly a living child is born with no external evidences of disease.

In *infants born with evident syphilis present* the symptoms are a marked degree of malnutrition, with wasted body, wrinkled skin, and senile appearance. In addition, the baby regularly has various kinds of *skin eruption* present. The eruption may be papular or pustular, but the characteristic form is that of *pemphigus*, and this is usually found on the palms and soles. Many of the bullæ dry and form yellow crusts on different parts of the body. These infants usually live but a short time.

In the *ordinary case* the infant appears healthy at birth. During the first month these children show some anæmia and other evidences of malnutrition. By the *second month*, usually the first signs of the disease develop. These are a persistent coryza, called "snuffles," and the eruption. As in all syphilitic eruptions this is multiform, and may be a simple erythema or roseola, or may consist of macules, papules, vesicles, or pustules. It may develop anywhere on the body, but is most apt to appear in regions where irritation is greatest, as around the buttocks. Associated with these skin eruptions are the frequent occurrence at the muco-cutaneous junctions of ulcers, fissures, mucous patches, and condylomata. These form the rhagades about the lips and nostrils, and the ulcers and warts around the anus. During this time more or less inflammation about the nails is also common.

The *epiphysitis* occurring at this time produces pain, tenderness, and often swelling about the *joints*. From this comes a voluntary immobility of the joint—a pseudo-paralysis. Anæmia and marasmus go on increasing during the course of the disease, and from time to time fever may be present. The spleen and liver are regularly enlarged, but not the lymphatic glands, as in the acquired form.

The child frequently dies of *marasmus* or of some intercurrent trouble during the course of the disease; but if the infection is mild or if treatment is given, the evidences of active disease, the *secondaries* as they really are, gradually disappear.

Later, often about the tenth year, signs of late syphilis or what might be called the tertiary stage of the disease, develop in many of these surviving children. These signs are seen most frequently in the *teeth*, *eyes*, *ears*, and *bones*.

The *teeth* belonging to the permanent set take on the characteristics known as *Hutchinson's teeth*. In this condition the upper central incisors are deeply notched by a crescentic depression in their cutting-edge, and the teeth themselves are shaped like a peg or the end of a screw-driver.

In the *eye* the cornea undergoes an interstitial inflammation with the production of opacities.

In the *ear* there is a gradual loss of hearing without signs of inflammatory action.

In the *bones*, the changes due to chronic periostitis are seen, with the production of enlargements and thickenings of the long bones, as the tibiæ, and the growth of nodes on the flat bones—those of the cranium.

Gummata may form anywhere in the skin or mucous membranes. If untreated, they break down and form ulcers when in the skin; but when in the mucous membranes of the nose and hard palate, as they break down they produce destruction of the nasal and palate bones, with perforations and deformities of these parts. Gummata may likewise form in the viscera, but are usually not diagnosed in these situations.

Syphilis—diagnosis: In well-marked cases this is not difficult. If an eruption only is present, it is more difficult; and the *mother's history* must be taken into consideration as to previous abortions, birth of dead children or of children with eruptions. The coryza, fissures about the lips, condylomata about the anus, signs of epiphysitis, eruption, and malnutrition are the points to be looked for.

In the tertiary stage, Hutchinson's teeth, interstitial keratitis, deafness, deformities of the nose and palate, and enlargements of the tibiæ are typical signs.

Prognosis: This is a more dangerous disease in infants than in adults, as the malnutrition so interferes with their growth. The earlier after birth the symptoms develop the worse the prognosis. Much depends on the way treatment is carried out in estimating the prognosis.

Syphilis—treatment: All the ordinary means for the prevention of syphilis should be carried out. If a mother becomes pregnant with what may be a syphilitic child, she should be vigorously treated throughout her pregnancy.

Just as soon as a diagnosis is made the child should be put under *mercurial treatment.* It is best given by *inunction,* using about one scruple of blue ointment daily. The place of rubbing should be changed from day to day to avoid irritating the skin. Internally, gray powder in grain doses three times a day, or bichloride of mercury, gr. $\frac{1}{60}$, three times a day, may be given. Salivation is rare in children, but diarrhœa may be started.

Locally, calomel powder or a calomel ointment is the best application for *fissures, ulcers,* and *condylomata.*

In the tertiary stage large doses of *iodide of potassium* are to be given.

Through all treatment special care should be given to hygiene and food, and the use of iron from time to time is to be recommended.

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